Sport and Physical Activity in the Heat

Maximizing Performance and Safety

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This book is dedicated to two amazing people: First, Tutita Maria Casa, my partner for the past 26 years. With love as the rock-solid foundation we have built a massively rewarding life. You always supported my passion to make a difference, and I will always be grateful. Second, to Kent Scriber, the amazing athletic trainer who began the life-saving care for my exertional heat stroke in 1985. His immediate attention and recognition of the severity set in motion the coordinated care by the EMT's, emergency room physicians, and nurses that allowed me to fully recover and to have the opportunity to live a life in which I can serve others in this realm.

Foreword I

Sport and outdoor activities are at the core of the human spirit. Each of us strives to challenge our individual physical and mental barriers while pushing beyond to meet or exceed our goals. At the same time, health-care providers and most importantly those at the front lines have been tasked to prevent, protect, and manage those individuals who at times exceed their limitations.

Environmental conditions, specifically heat related, have and continue to be a challenge for those in the occupational and sports arenas. The heat poses particular challenges that create hurdles to maximizing performance and increases the likelihood of untoward side effects such as exertional heat stroke. Its effects can be deadly and experienced by the best and least prepared, which highlights our responsibility as leaders to create awareness, education, and guidelines to prevent and manage severe exertional heat conditions in our communities. From youth sports to the college and professional ranks and short and long distance endurance events, heat-related issues are a time-sensitive emergency requiring rapid recognition and management. In tactical, military, and occupational environments, guidelines directing prevention with protocols and processes can place safety as their top priority and preserve our human natural instinct of attaining our goals and success.

Dr. Douglas Casa has been the leader in taking on this difficult task. For years he has researched, guided, and directed health care, sports medicine, and occupational and tactical providers in the prevention and management of exertional heat illness. He has been the voice for those who have been impacted unnecessarily by the deadly effects of exertional heat stroke. This book captures years of research, guidelines, and endless clinical experience which will guide sport performance and policy and assist in implementing safety parameters around this condition. As a practicing clinician while directing policy and establishing best practice protocols, this book will be on the front lines in optimizing exercise in the heat while elevating player safety.

Chicago, IL, USA — George T. Chiampas, DO, CAQSM, FACEP

Foreword II

Adaptation to temperature has been characterizing species since the beginning of life. Whereas ectotherms rely on behavioral thermoregulation, endotherms such as humans use both behavioral and autonomic thermoregulations to maintain their temperature range. Across their evolution, humans learnt to live in cold environments by building shelters, wearing clothes, and using fire to produce heat. On the other side, humans are relatively well adapted to the heat. A biochemical restructuring allowing enzymes and other macromolecules to function at higher than ambient temperature might have represented an evolutionary advantage allowing humans to be active at high intensity for longer periods during strenuous activity to hunt or avoid predators.

Temperature has been recognized as a critical indicator of health from the first medical writings and is still one of the first vital signs monitored. For example, Hippocrates (460–377 BC) was already considering fever as a natural curative response from the body. Experimental researchers on human body temperature regulation developed from the seventeenth century. Santorio Santorio quantified insensible perspiration in 1614 by constructing a sensitive beam balance and weighing himself, his food, and various excretions during both day and night. Antoine Lavoisier demonstrated in 1780 that humans produced heat by combustion processes resulting in the production of carbon dioxide. This concept has thereafter been revisited by Claude Bernard who showed that the blood entering the lungs was warmer than the blood exiting them, whereas venous blood was warmer than arterial blood in several other organs, thus indicating that these tissues were the site of heat production. It was not until the twentieth century that the occupational requirements from both the mining industry and the military lead to a growing research interest in the thermal responses of the active man.

Despite this long history of both adaptation and research, this book explains that heat is still provoking several deaths every year, being the most serious health hazard in active peoples. In addition to the health issues, heat has also a large effect on exercise performance. For example, the terminology “warm-up” is derived from the increase in muscle temperature sought before a high-intensity effort. While an increase in muscle temperature might improve performance during high-intensity

short-duration exercise, metabolic heat production will induce an increase in internal body temperature during prolonged exercise. Depending on both exercise intensity and environmental conditions, this increase in temperature might be compensable or not. The thermoregulatory demands of exercising in hot and/or humid environments represent a cardiovascular and perceptual stress limiting performance and potentially leading to various levels of exertional heat illness.

However, these problems are rarely fatal and the cure is well established. Heat acclimation is the best natural protective measure to adapt for any population exercising in the heat. As detailed in this book, heat acclimation is a relatively rapid process for preserving both the performance and health of the athlete exercising in the heat. This book provides the keys to maximize performance and safety during exercise in the heat in a range of populations including athletes, workers, and the military. The chapters cover a range of key aspects of physical activity in the heat and are written by world-renowned specialists. This book follows a long tradition of books from North American researchers and clinicians on the performance and safety aspect of exercising in the heat.

Doha, Qatar Sebastien Racinais, PhD

Preface

It was Wednesday, January 29, 2014. My colleague Rob Huggins (who is the author of chapter 10) and I were at a swanky New York City steakhouse to meet with the Federação Portuguesa de Futebol (aka the Portuguese Football Federation, FPF) to discuss how we could assist them with their preparation for the 2014 FIFA World Cup to be held in June and July in Brazil. Specifically, they wanted us to help with developing strategies for hydration, heat acclimatization, body cooling, circadian rhythms, and other similar performance issues as their team prepared for the "Group of Death," the term used to describe the anticipated fierce competition in this part of the bracket. All of the key players were present, including the CEO, head coach, manager, COO, and a few others affiliated with FPF. The dynamic Charlie Stillitano and his colleagues Francesco Campagna and Russell Steves from Relevent Sports also were present and were the conduits to bringing us all together. The next few hours would be the culmination of so many years of preparation in my career. As I spent the evening trying to explain the value, process, issues, potential downsides, and investment related to the heat and hydration concepts, I started to realize that while many of the elite soccer programs of the world have the best players, coaches, and leadership, they are often seeking information related to exercise science, sports performance, sports medicine, and sports nutrition. My experience has shown me that elite sports teams can dabble into particular areas of interest as needs arise, but a comprehensive sports science program is often lacking with most of the top organizations around the world.

I was impressed that the FPF recognized the need to address the issues related to heat preparation, and while they were willing to meet and consider all the options, they were not willing to fully commit to the concepts to maximize performance in the heat. Against my strong recommendation, they decided not to properly enact heat acclimatization strategies during their prep in the USA leading up to their trip to Brazil. I had worked out a plan to have them train in Gainesville, FL (with very similar environmental conditions they would face in Brazil) during the 2 weeks immediately before the departure for the World Cup so that proper strategies could be implemented across the board to maximize performance in the heat. The FPF leadership chose an outstanding (besides for the environmental conditions) NYC

area venue (the NY Jets facility in NJ) for training, in part, so that maximal exposure could be bestowed upon the team, including the world-renowned player on their roster. Nevertheless, all of us involved with helping ensure the players were very well prepared still did the best we could to implement appropriate heat and hydration strategies. However, it was challenging to really mimic what they would face in a rainforest and very similar climates without having the team preparing in an environment with genuine heat and humidity.

Unfortunately, it felt as though all of the actual or intended efforts were for naught when a very silly play by a Portuguese player in the first half (of the first game!) against eventual champion Germany left them playing a man short for most of the game. The four points FPF earned for their win, loss, and tie was not enough to guarantee their advancement to the next stage of the tournament, and losing a group tiebreaker to the USA sealed their fate for not making it out of the group round.

The experience with the FPF highlights my passion and those of my colleagues at the Korey Stringer Institute. We work tirelessly to help maximize performance and safety during exercise in the heat. Our exeprience in this realm is captured in the content of this book. The knowledge acquisition related to the topics covered in this book is only the first part of the journey toward making an impact. To make it really work, you need to be able to sell it, practice it, implement, and critique it for the concepts to take shape and make a difference.

I have had the good fortune to collaborate with many other top athletes in the world and the scientists, athletic trainers, physicians, nutritionists, and strength and conditioning coaches who work with them on a daily basis. The quest to enhance performance and maximize safety has been my mission for over 30 years, and it remains to this day. I have been very privileged that my journey to help others maximize performance during exercise in the heat and ensure it is done safely has taken me to seven military bases to work with spanning all the branches of the military, three marathon majors, and the Ironman Triathlon World Championship three times; I also have had the chance to work with the NFL, an elite team in Serie A, as well as other pro soccer teams, professional tennis players, numerous Olympic medal-winning distance runners, elite college football/soccer/track/cross-country college programs, governing bodies of most of the major sports in the world, the NCAA, OSHA, NIOSH, and numerous others. I hope my experiences with these varied entities helped me gain a broad stroke understanding of the issues at hand so that I could compile a book that would be of greatest service to the end user.

The authors I have been able to gather to contribute to this book offer an array of talents, experiences, insights, and perspectives so that you, the reader, will be armed with a wide range of potential skill sets to assist in a variety of circumstances in a variety of sports. A look at the figure on the following page shows the overall considerations when preparing to work or perform in the heat. You need to be equipped with the following information that is provided to you in this book:

(a) An advanced understanding of the human response to exercise in the heat and an appreciation of what makes our capacity unique (Chaps. 1 and 2)
(b) An excellent grasp of the key components that can be manipulated to enhance exercise heat tolerance, including hydration strategies, heat acclimatization plans,

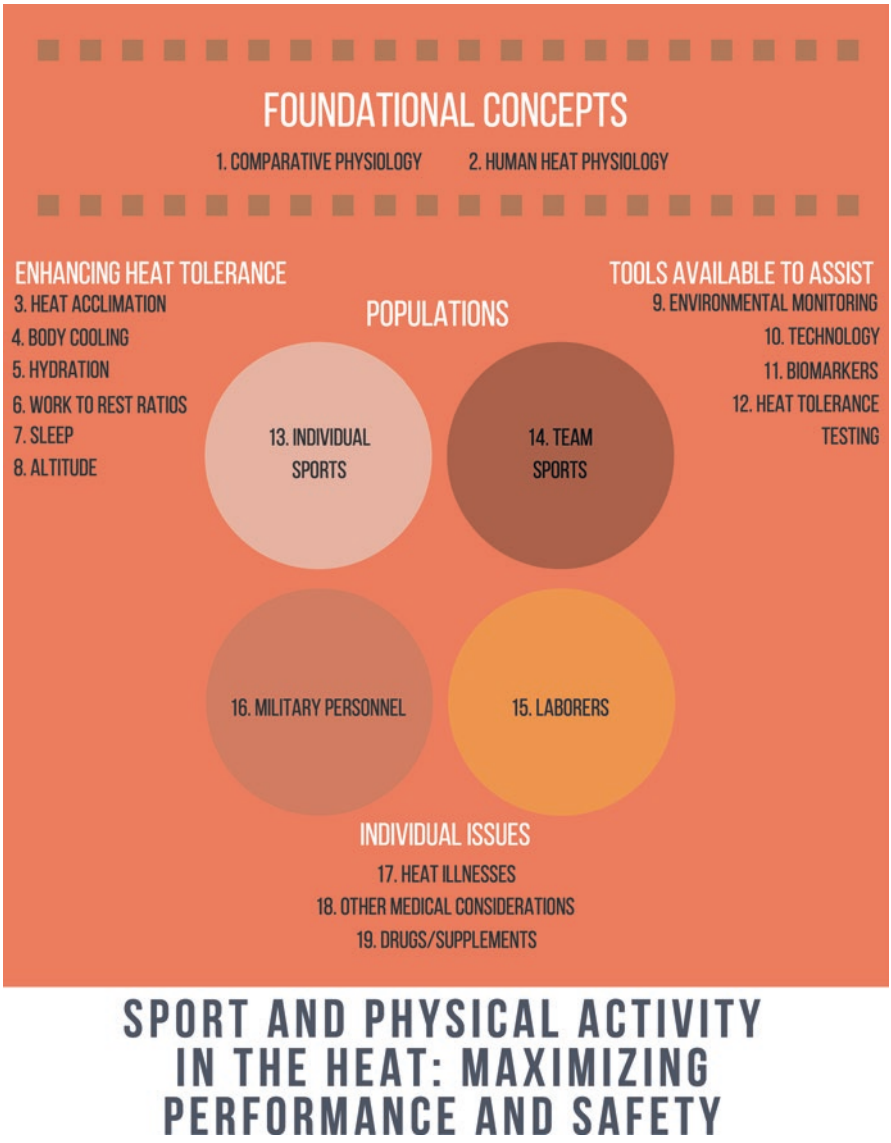

Fig. 1 Overall considerations when preparing to work or perform in the heat (Figure courtesy of Douglas J. Casa and Gabrielle Giersch; Korey Stringer Institute)

body cooling interventions, maximizing quality sleep, modifying work-to-rest ratios based on the intensity/fitness level/environmental conditions, and considering how altitude might influence or contribute to these factors (Chaps. 3–8)

(c) A working knowledge of the available tools that can assist with monitoring physiological strain to evaluate your program, maintain excellence, and constantly seek improvement, including tools, such as utilizing biomarkers, heat

tolerance testing, environmental monitoring, and modern technology to monitor physiological responses that are all part of the process to enhance performance and maximize safety (Chaps. 9–12)

(d) A deep comprehension of the population that you are looking to assist and the particular nuances that laborers, warfighter, or athletes will face within in the construct of their assigned duty or sport (Chaps. 13–16)

(e) An appreciation of the medical/pharmacological issues that can contribute to challenges with exercising in the heat or that can result from the intense effort (Chaps. 17–19)

Ultimately, the process of maximizing performance and safety during exercise in the heat is best done with a comprehensive plan that considers the foundational knowledge, factors that can enhance exercise heat tolerance, and awareness of the best modern tools/knowledge to monitor and modify the individual factors in the populations being served.

Storrs, CT, USA Douglas J. Casa, PhD, ATC, FNAK, FACSM, FNATA

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- Springer—thanks for believing in this book and all the support to make it happen; it certainly is a first of its kind and I am very proud to share it with those who will benefit from the content.

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Part I
Foundational Concepts

Chapter 1
Comparative Physiology of Thermoregulation

Luke N. Belval and Lawrence E. Armstrong

Introduction

From single-cell organisms to the largest animals, the thermal environment challenges both performance and survival. Whether it is the honeybee or the thoroughbred race horse, organisms constantly adapt to the temperatures around them to maintain homeostasis. In athletic, military and occupational settings, thermoregulation is often secondary to the goals of the sport, mission, or job. Similarly, in the animal kingdom thermoregulation is seldom the primary obstacle to survival. Instead, the ability to respond to the thermal environment is controlled via subconscious physiological and behavioral systems. These thermoregulatory systems across species have developed in response to the environments these animals have been exposed to throughout their anagenesis.

The purpose of this chapter is to overview the similarities and differences between a variety of animal species and humans which allow them to adapt and thrive in a given environment. Throughout the mechanisms and systems presented in this chapter, it is important to not dismiss one species' adaptation as superior to another, but rather evaluate the advantages and disadvantages afforded by each system or behavior. We will pay particular attention to the concepts of thermoregulation in animals that apply to human thermoregulatory challenges; as the quest for higher, faster, and stronger performances not only drives our understanding of the

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limitations of physiology but also generates creative solutions to further enhance athletic performance. Finally, we discuss the use of animal models and how multiple approaches can be used to relate this information to exertional heat illnesses.

Comparative Physiology Informs Animal Models of Hyperthermia

Our search of the scientific literature revealed that numerous animal models have been studied to gain insights into human temperature regulation and hyperthermia. Working under the auspices of animal protection committees, which ensure humane treatment of laboratory animals at universities and hospitals, this research has focused on various aspects of thermoregulation including, skin anatomy, organ function, physiological responses, and heat gain/loss/balance. Humans are homeothermic (i.e., constantly attempting to maintain body temperature at a stable set-point), endothermic (i.e., producing considerable internal heat via metabolism) animals that release heat to the environment via dry heat loss (i.e., convection, conduction and radiation) and wet heat loss (i.e., sweat evaporation, respiration).

In addition to these physiologic factors, the design of an animal model of hyperthermia and the resulting clinical complications of heatstroke requires that scientists consider the methods to induce increased body temperature and to measure temperature [1]. Regarding the former, hyperthermia can be induced by viral or bacterial toxins, drugs, exercise, and exposure to a hot environment. Toxins, however, induce unwanted pathologies and increase the risk of investigator infection. Drugs that induce hyperthermia also may induce unwanted side effects and irrelevant compensatory responses. Exercise-induced hyperthermia in an animal model, therefore,

1. must induce natural compensatory responses
2. must mimic human hyperthermia
3. can be generated using commercially available animal treadmills

Therefore, no single animal model is perfect to describe human responses to exercise in the heat and heatstroke. Instead, animal models must be selected to study the responses and adaptations that most mimic human responses for the variables of interest.

Classification of Thermoregulatory Systems

The most basic method of classifying species' thermoregulation considers the methods by which temperature is regulated. While *homo sapiens* and nearly all mammals and birds represent one end of the spectrum as homeothermic endotherms, other

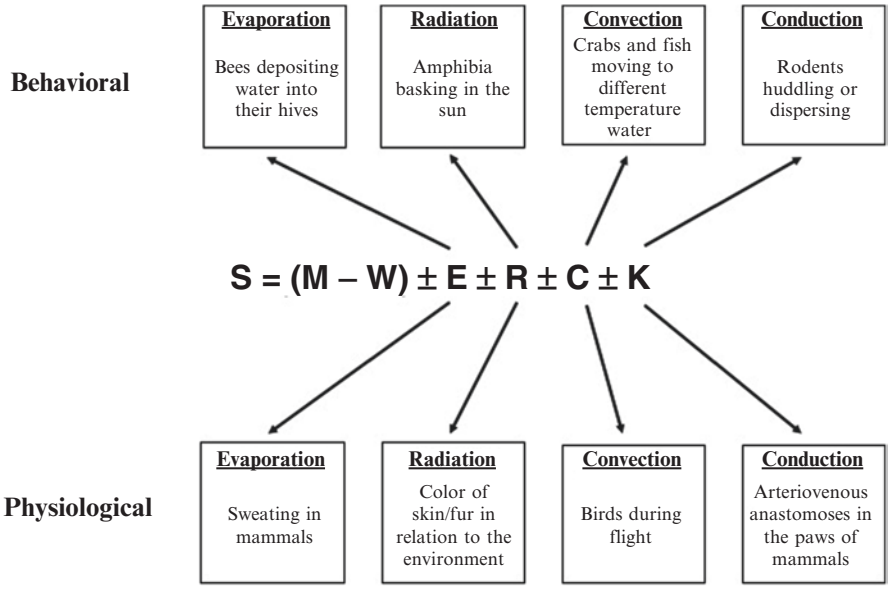

Fig. 1.1 Theoretical representation of the Q_{10} effect. Adapted from [2]

species do not regulate their temperature as closely or utilize environmental sources of heat rather than metabolic heat. Most fish, amphibians, and reptiles can be classified as poikilotherms because their internal temperature is strongly influenced by environmental conditions [2]. Poikilothermy is often coupled with ectothermy, in that heat energy from the environment is necessary for some species to maintain normal body temperature [3]. It is important to note that these categorizations are not exclusive. Endotherms may allow for substantial fluctuation in their body temperature, for example, in the case of hibernation.

While maintaining an internal body temperature that is different from the external environment provides evidence of thermoregulation and classification as a relative homeotherm, evaluation of Q_{10} offers a more nuanced approach. The Q_{10} is defined as the change in metabolic activity of a given organism or system that results from a temperature increase of 10°C [2]. For example, the Q_{10} effect of 2.3 in muscle physiology, following the Law of Aerrhenius [4], is typically the reasoning behind "warming-up" in athletics, with a slight increase in muscle temperature leading to increased athletic performance [2].

In the absence of homeostatic control of internal temperature, a twofold to threefold increase of metabolic activity (i.e., a Q_{10} of 2–3) is observed in response to an increase of environmental temperature [5]. A theoretical uncompensated Q_{10} response (~2.3), such as that in the true poikilothermic ectotherm, is illustrated in Fig. 1.1. Meanwhile, in a perfect homeotherm, a Q_{10} of 1 would be expected because the animal is able to isolate its body temperature responses from changes in the environmental temperature. However, most species are not able to totally isolate

their internal temperature responses, a criterion Q_{10} of < 2 is often used to classify species as homeotherms or poikilotherms [5]. Further, a diurnal rhythm of internal body temperature exists in most mammals [6].

Behavioral Thermoregulation

While homeothermy is defined as the ability to physiologically regulate body temperature in response to environmental changes, nearly all species possess the ability to thermoregulate behaviorally. In poikilotherms, behavioral change is the predominant manner by which they cope with changes in their environments. Metabolically, behavioral thermoregulation is very efficient [3]. This explains why nearly every species has a series of behaviors that alter the effect of the environment, even if they possess highly functioning physiological systems. For some animals, changing posture or moving to a new location is an adequate method of thermoregulation. For reptiles and amphibia, sunning or basking is a typical behavior by which the animal exposes its body to as much solar radiation as possible, leading to internal heat gain [7].

In many animals, activity levels are closely tied to ambient temperature. A Gaussian distribution of activity has been widely observed; as ambient temperatures increase or decrease beyond an animal's thermal preference, it decreases activity to minimize heat gain or loss [8]. For example, cattle decrease rumination as ambient temperature increases [9]. This distribution of varying activity levels corresponding to ambient temperatures is widely conserved across species, suggesting widespread effectiveness.

This idea of a thermal preference has also been studied in fish, which exhibit a distribution of metabolic rates that are proportional to the temperature of water [10]. This thermal preference is not uniform across species. Nocturnal reptiles have been observed to tolerate lower temperatures than similar diurnal species [7]. In fact, even within the same species, thermal preferences can vary greatly. For example, fiddler crabs adapt to different water temperatures, depending on whether they come from temperate or tropical waters [5].

The animal kingdom also utilizes group dynamics to behaviorally cope with a changing environment. Annelids and arthropods in the desert have been observed to burrow underground when ambient temperatures increase and to aggregate in groups when temperatures decrease [8]. Burrowing decreases the radiant load from the sun, while aggregation increases the thermal inertia of a group. The same huddling and dispersion can be seen in rodents [11]. Meanwhile, bees can collectively cool their hive with water from pollinated plants, one drop at a time [8].

In humans, behavioral thermoregulation is considered primarily in terms of thermal comfort [12]. When you sense that you are cold, you may look to add a layer of clothing; if you sense that you are hot, you may remove a layer or you may change the room temperature by adjusting the thermostat [13]. This is based upon integration of information in the pre-optic area of the hypothalamus from cutaneous and

visceral temperature sensors [3]. Instead of a measured physiological response, you are able to use another strategy to better achieve thermal comfort. This same neural pathway exists in vertebrates and demonstrates the importance of the hypothalamus in both physiological and behavioral thermoregulation [3, 14].

In athletics, changing the time of outdoor training to the morning or evening from the middle of the day is another example of behavioral thermoregulation. Just as many animals may seek shade in the middle of the day, smart coaches also alter activities to protect their athletes. Whether this involves moving practice to an air-conditioned facility, increasing rest breaks, or altering the time/location of training, these changes likely will have a greater impact than relying on thermoregulatory responses in an uncompensable environment.

Physiologic Thermoregulation

In homeotherms, physiological mechanisms must compensate for changes in the thermal load placed upon the organism. There must be a constant balance of heat gain and dissipation to maintain the narrow homeostasis of body temperature. Birds and mammals are the typical focus of this phylogeny; however, fish, reptiles, and amphibia also possess some of the same components of temperature regulation.

One of the most basic methods of physiologic thermoregulation involves the countercurrent exchange of heat. In this system, opposing channels of fluid in close proximity allow for rapid exchange of heat between the body's tissues and the external environment. Unsurprisingly, fish utilize this method of thermoregulation very efficiently [10]. In humans, an expanded cavernous sinus, compared to other primates, facilitates countercurrent exchange of heat in the skull [15]. This direct exchange occurs in a continuous manner. However, it relies on the presence of a local heat gradient which can be problematic in animals that have a large tissue mass that does not directly interface with the environment.

Heat Balance Equation

The fundamental methods of heat gain and dissipation in homeotherms can be modeled through the heat balance equation. While other chapters in this book will focus on the specific human aspects of the heat balance equation, it is important to understand that the same principles govern the flow of heat in a variety of organisms.

$$S = \left(M - W\right) \pm E \pm C \pm R \pm K$$

where S is the amount of heat storage in the body, M is the metabolic heat production, W refers to external work (which at rest has a nearly negligible effect on heat

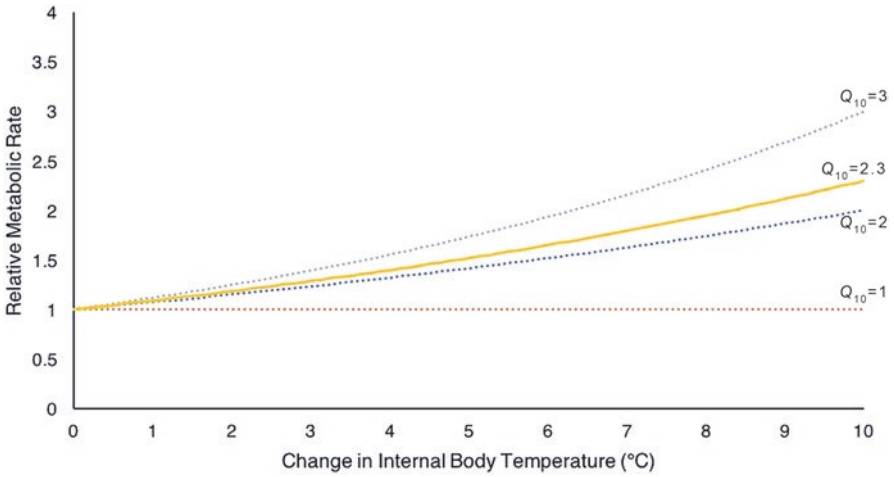

Fig. 1.2 Examples of behavioral and physiological methods of heat balance across species

balance), *E* is evaporation, *C* is convection, *R* is radiation, and *K* is conduction; *E*, *C*, *R*, and *K* usually refer to heat exchange between skin and air [16]. For each component of this heat balance equation, there exists a unique animal adaptation that illustrates the interplay between different avenues of heat flow (Fig. 1.2).

Metabolic Heat Production

Most endotherms maintain a body temperature greater than the ambient environment, which requires a large amount of heat gain [3]. Metabolic heat production is subdivided into either shivering or non-shivering thermogenesis. Shivering thermogenesis occurs primarily as a result of cold exposure and is one of the few thermoregulatory responses observed in snakes [3]. Non-shivering thermogenesis is further classified into four components: thyroid hormone induced increases in metabolic rate, oxidative phosphorylation, muscular work, and brown adipose tissue [3]. Humans are considered relatively efficient with 20–25% of metabolic energy being converted to locomotion during exercise [17]. The remaining energy is released as heat to the tissues. By contrast, the cheetah approaches 10% efficiency when running at 17 km/h [18]. Therefore, due to the excessive heat production, many animals are limited by overheating during exercise [19].

Convection

Convection is the heat exchange between a surface and a fluid, typically air. In free convection, the fluid density determines the rate of heat transfer, whereas forced convection is dependent on the velocity of fluid [12]. In humans, the most common

example of convective cooling is the difference between running indoors on a treadmill and performing the same exercise outdoors. During outdoor exercise, forced convection occurs as a result of both wind and body movements. Meanwhile, indoor exercise at the same intensity is typically considered more difficult from a thermoregulation standpoint because the only convection that occurs is a result of free convection.

For most exercising animals, convective cooling is very similar to humans. An illustrative example of the effects of convection can be found in birds. Despite the fact that most physiologists have identified predominant evaporative cooling methods, the heat balance of a bird while flying could not be achieved without the great amount of convection that occurs during flight [20]. In furred mammals, convection also plays an interesting role in cooling. In the air space between individual hairs, convection loops form as small circular movements of air that gradually bring heat from the skin to the outside environment [6]. While this may not be as efficient as evaporative cooling methods on the skin, these convective loops contribute to some cooling of an otherwise well-insulated animal. However, it is important to note that convection relies on a heat gradient between the body's surface and the environment. When the ambient temperature is greater than the surface, convective cooling is impossible.

Conduction

Conduction is the heat exchange between two solid objects [12]. Physiologically, this occurs within the body as heat transfers between tissues; as a method of heat loss, it appears in limited circumstances because most animals have limited direct contact with a surface that allows heat transfer. Birds and mammals all share fairly similar thermal conductance, which increases in response to increases in blood flow [11, 21]. Meanwhile, poikilothermic lizards possess up to ten times the thermal conductivity of mammals [21].

Thermal conductivity of skin is great in two areas. First, arteriovenous anastomoses provide conductive heat exchange. These pre-capillary blood vessels densely populate the palms and feet of humans and the paws of mammals [22]. Second, similar areas of great cooling potential exist in some animals' tails [23]. Therefore in both humans and animals, the skin surface serves as a target for cooling modalities.

Radiation

Radiation is typically considered a method of heat gain through solar radiation, like the basking of amphibia on a sunny day [21]. While it is not explicitly physiologically controlled by animals, the color of their skin or fur in relation to the environment can impact radiant heat load. Furthermore, the different densities of mammal coats throughout the year can either increase radiant heat load for heat gain during cold weather or minimize radiant heat load during warm weather [24].

When modeling radiative heat and the body, there are six terms to consider regarding net radiation balance: five are the environment's influence on the body and one the body releases to the environment [12]. For example, the color of skin, fur, or feathers can alter the emissivity of a surface, either minimizing heat loss or maximizing heat gain [9].

When compared to other species, humans are notably successful at minimizing solar radiation. Only 7% of the skin surface is exposed to maximal radiation when standing upright [25]. Contrast this to quadrupedal animals that expose a much greater area to the sun, greatly increasing the radiant heat load.

Evaporation

Evaporation as a method of heat dissipation is of particular interest when examining physiologic thermoregulation. For birds and mammals, this is by far the predominant method of heat dissipation. Evaporation relies on the energy required for the phase change of water from liquid to vapor [12] and occurs in a variety of methods across the animal kingdom, as illustrated by the following examples. First, the honeybee regurgitates its stomach contents onto its body [19]; when the fluid from the contents evaporates, the bee cools. Second, rats and kangaroos spread saliva on their skin to dissipate heat via evaporation [3, 23, 26]. Third, storks and vultures defecate on their legs to cool [19]. Most other birds rely predominantly on the evaporation of fluid from mucosal surfaces to cool [20]. This is similar to the respiratory cooling in dogs, typically referred to as panting [27]. Respiration in dogs is linked to stride patterns; thus, the cooling capacity of these animals is limited by the speed of their legs and stride rate [15]. However, in an example of divergent evolution, African hunting dogs have been shown to rely less on evaporative cooling than their domestic counterparts [18].

Sweat glands exist only in the skin of mammals; their density varies greatly, from 20 to 30 glands·cm^{-2} in pigs to >2000 glands·cm^{-2} in some species of cattle [28]. Two types of sweat glands exist, apocrine and eccrine. Apocrine sweat glands are typically found with fur or hair [4], while eccrine glands are predominantly used for thermoregulation [15]. Chimpanzees and gorillas possess approximately two-thirds eccrine sweat glands and one-third apocrine sweat glands, while humans possess nearly 100% eccrine sweat glands [29].

Not all vertebrates have similar sweat glands; for example, cats, dogs, and pigs have eccrine glands only in their foot pads and/or snout; these animals rely more on respiratory panting to release heat to the surrounding air. Bedouin black goats are an interesting counterexample, as they possess a relatively large number of apocrine sweat glands, perhaps to help keep themselves cool despite possessing black fur [15].

Horses, one of the closest thermoregulatory counterparts to humans, possess a very large mass in comparison to their surface area, making cooling difficult. However, horse sweat contains a protein, latherin, that acts as a dispersant for sweat [30].

Their sweat is also slightly hypertonic to blood plasma, which makes electrolyte replacement following exercise a particular concern [28].

One of the downsides to sweating as a means of evaporative cooling is that it requires the loss of body water. In humans, hypohydration becomes a serious concern during prolonged, intense exercise in the heat. Camels demonstrate a unique countermeasure to this phenomenon. Contrary to popular belief, the hump of a camel is fat rather than water. However, during prolonged bouts of sweating, the camel is able to use this fat to yield metabolic water, providing great resistance to hypohydration [19].

What Do Animal Models Reveal About Human Heatstroke?

Physiologists employed a rat model of exertional heat stroke for more than 15 years, at the U.S. Army Research Institute of Environmental Medicine, Natick MA, USA [31]. This model acknowledged that rats spread saliva on fur and skin, across a wide range of ambient temperatures, as an important element of heat dissipation. In fact, evaporation of saliva accounts for approximately 90% of the heat lost by male rats, in a 40 °C/104 °F environment [26]. Similarly, in a hot-dry environment, evaporation of sweat from human skin accounts for >80% of all heat loss during treadmill running. Despite the viscosity and compositional differences between saliva and sweat, many rat physiological responses and compensations are similar to those of humans [1]. For example, the rat model provided the following insights regarding human exertional heatstroke: hepatic ultrastructural changes, acidosis, hyperkalemia, and membrane damage are similar to those observed in humans; individual factors (e.g., age, body weight) influence heat sensitivity, mortality, and survival time; passive and exercise-induced hyperthermia resulted in unique serum enzyme profiles of alanine aminotransferase, aspartate aminotransferase, creatine kinase, and lactate dehydrogenase; low potassium and low sodium diets (versus adequate dietary intake) increased the rate of heat storage and reduced total treadmill work accomplished; gut-derived bacterial endotoxins appeared in plasma after hyperthermia; and endurance training influenced exercise tolerance time, ability to sustain thermal load, and susceptibility to work-induced thermal fatality [31–34].

Researchers in South Africa are widely known for their primate simulations of human classical heatstroke, published during the years 1986–1988. During exposure to a hot environment, the internal body temperature of monkeys and baboons rose to ≥43 °C/109.4 °F, well above the often-cited 40 °C/104 °F threshold of human exertional heat stroke. Due to the taxonomic similarities (i.e., thermoregulatory, cardiovascular) of primates and humans, the primate model of heatstroke resulted in relevant findings regarding cardiovascular responses (e.g., heart rate, mean arterial pressure, peripheral resistance), hyperthermia-induced increases of gut-derived bacterial endotoxins (i.e., lipopolysaccharide, LPS), and

Table 1.1 Factors that influence investigator choice of species to model human hyperthermia and exertional heat stroke

Species	Availability	Cost-effectiveness	Convenience of handling and housing	Similarity to human responses
Baboon	+	+	+	++++
Monkey	+	+	+	++++
Dog	++	++	++	+
Sheep	+++	++	+++	++++
Rabbit	++++	+++	+++	+++
Rat	++++	++++	++++	++++

Adapted from [1]
+, lowest or poorest; ++++, highest or greatest

the relationship between morbidity–mortality and Gram-negative bacterial shock [35, 36]. Because LPS plays an important role in heatstroke symptomatology (e.g., hypotension, shock), South African investigators also studied the protective effects of administering intravenous compounds to primates whose rectal temperature reached 43.5–44.5 °C/110.3–112.1 °F. They observed improved survival times and lower mortality rates following administration of a corticosteroid (i.e., reducing inflammation), an antibiotic (i.e., decreasing bacterial infection), and the antibody to LPS (i.e., counteracting increased plasma LPS concentration) [37–39]. Few or none of the above studies could have involved human subjects, due to the current ethical standards involving human research.

Several other animal models of severe hyperthermia and heatstroke have been developed, with limited success and a smaller number of resultant publications. These models include dogs, chicks, rabbits, sheep, and cows [40]. Sheep are reserved for large-scale experiments in which several variables and functions are studied concurrently [1]; rabbits are seldom utilized in such studies. As shown in Table 1.1, numerous other practical and scientific factors encourage and/or discourage involvement of these animal families.

Summary

The animal kingdom offers unique insights into the complex responses that different species employ to regulate internal body temperature. Whether these involve altering behavior or physiological responses, to better cope with heat stress, humans are not alone in their efforts to thrive in hot environments. It is in light of comparative physiology that we appreciate the thermal load experienced by humans during exercise or occupational labor, and develop strategies, techniques, and products to ameliorate thermal strain and avoid illness or death.

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Chapter 2
Human Heat Physiology

Jonathan E. Wingo, Craig G. Crandall, and Glen P. Kenny

Abbreviations

C	Rate of heat loss via convection
E	Rate of heat loss via evaporation
HR	Heart rate
K	Rate of heat loss via conduction
M	Metabolic rate
$(M - W)$	Metabolic heat production
R	Rate of heat loss via radiation
S	Rate of heat storage
$\dot{V}O_{2max}$	Rate of maximal oxygen uptake
W	External work
WBGT	Wet bulb globe temperature

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Introduction

Heat stress presents physiological challenges that can impair aerobic exercise performance [1, 2] and compromise safety [3]. As such, it is important for athletes, coaches, and practitioners to understand the basic human physiology related to heat exposure. Accordingly, this chapter focuses on human heat balance and the physiological responses associated with sport and physical activity in hot conditions.

Human Heat Balance

Human core temperature homeostasis functions over a fairly narrow range (35–41 °C/95–105.8 °F) [3] with resting temperatures averaging approximately 36.5–37.5 °C/97.7–99.5 °F. Temperature regulation (i.e., thermoregulation) is achieved through a balance between heat production and heat loss. This balance is illustrated by the following equation:

$$S = \overbrace{\left(M - W\right)}^{\text{Heat production}} - \overbrace{\left(R + C + K + E\right)}^{\text{Heat loss}},$$

where S is the rate of heat storage, generally expressed in watts, M is metabolic rate, W is external work, R is rate of heat loss/gain via radiation (heat exchange in the form of infrared waves), C is rate of heat loss/gain via convection (heat exchange between a surface and a fluid such as air or water), K is rate of heat loss/gain via conduction (heat exchange between two surfaces in direct contact), and E is rate of heat loss via evaporation (heat loss in the form of the phase change of water, or sweat, from a liquid to a gas) [4]. If rate of heat production/gain is offset by an equal rate of heat loss, core temperature is maintained and $S = 0$. Conversely, if rate of heat production exceeds rate of heat loss, $S > 0$ and core temperature rises.

The $(M - W)$ portion of the equation represents metabolic heat production which is the difference between the total energy being expended (M) and the portion of this energy that is being used to do external work (W). For example, the efficiency of human skeletal muscle contraction is considered to be about 20% [3, 5]. This means for a task that results in a rate of energy expenditure of 100 W, 20 W are used to perform external work with the remaining ~80 W given off as heat. In order to balance this 80 W of heat gain, a rate of heat loss in the form of $(R + C + K + E)$ is necessary to avoid a net positive heat storage (S). If rate of heat production exceeds rate of heat loss, as is the case during the onset and early phase of exercise, core temperature will rise in proportion to the magnitude of heat storage until heat loss mechanisms (as will be discussed later in this chapter) engage to sufficiently balance heat production and no further heat storage occurs.

The extent to which core temperature rises during exercise is primarily dependent on M in compensable environments (those in which rate of heat production is balanced by rate of heat loss and a steady-state core temperature is sustainable for a given activity) [3]. As shown in Fig. 2.1, within the “prescriptive zone” of compen-

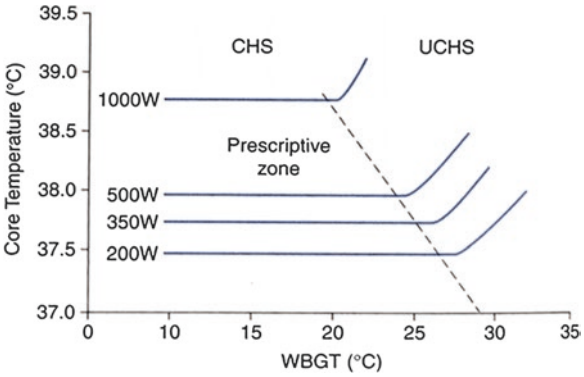

Fig. 2.1 Theoretical core temperatures for someone performing aerobic exercise at different metabolic rates (200 W, 350 W, 500 W, and 1000 W) in different environmental conditions. *WBGT* wet bulb globe temperature, *CHS* compensable heat stress, *UCHS* uncompensable heat stress [7]. Originally adapted from [6]. Reprinted with permission from Sawka MN, Castellani JW, Cheuvront SN, Young AJ. Physiologic systems and their responses to conditions of heat and cold. In: Farrell PA, Joyner MJ, Caiozzo VJ, editors. ACSM's Advanced Exercise Physiology. 2 ed. Baltimore, MD: Wolters Kluwer | Lippincott Williams & Wilkins; 2012, with permission from Wolters Kluwer Health Originally adapted with permission from Journal of Applied Physiology, Lind AR, Vol. 18, © 1963, pages 51–56, with permission from The American Physiological Society

sable heat stress conditions, wet bulb globe temperature (WBGT; an index of environmental heat stress) has little to no effect on the relation between metabolic rate and core temperature [6, 7]. This occurs because despite elevated WBGT conditions, heat loss mechanisms remain capable of offsetting the rate of heat gain during exercise. On the other hand, conditions outside the "prescriptive zone" result in uncompensable heat stress, i.e., WBGTs for which a steady-state core temperature is no longer sustainable because the evaporative requirement for heat balance exceeds the maximal evaporative capacity of the environmental conditions. In such conditions, the relation between core temperature and metabolic rate is no longer independent of the ambient conditions and rate of heat production exceeds rate of heat loss, thereby resulting in an increase in body heat storage and a rise in core temperature [6, 7].

It is important to note that for the avenues of dry heat loss ($R + C + K$) to be effective, a thermal gradient (i.e., difference in temperature) between skin and environment (or in the case of R, between skin and surface temperature of surrounding objects) must exist. The larger the thermal gradient, the greater the magnitude of dry heat loss, assuming environmental temperature is less than skin temperature. Heat exposure challenges human heat loss capability because if ambient temperature exceeds skin temperature, the thermal gradient is reversed and heat can actually be *gained* via one or more avenues of dry heat exchange resulting in R, C, and K being positive instead of negative with respect to S. Furthermore, in such situations the core-to-skin thermal gradient narrows as skin temperature is elevated and thus approaches core temperature. Evaporation is always a negative value in the heat balance equation (only heat loss, no heat gain), and is dependent on the skin-to-air vapor pressure gradient. This means that evaporative heat loss potential in hot-

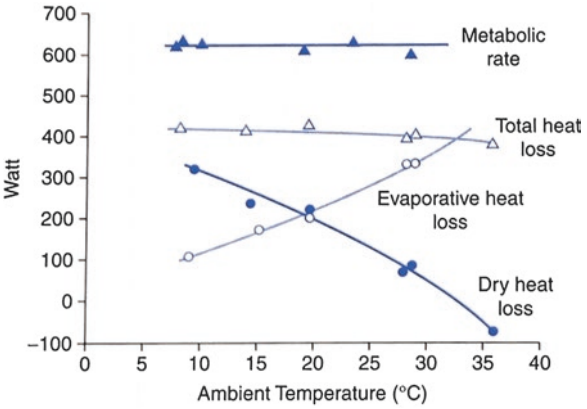

Fig. 2.2 Avenues of heat loss for a subject performing cycle ergometry for 1 h in a variety of environmental temperatures. Dry heat loss refers to dry heat exchange (radiation + convection) and evaporative heat loss refers to evaporation of sweat. Total heat loss is the sum of dry and evaporative heat loss. As ambient air temperature rises, there is a greater reliance on the evaporation of sweat [7]. Originally adapted from [8]. Reprinted with permission from Sawka MN, Castellani JW, Cheuvront SN, Young AJ. Physiologic systems and their responses to conditions of heat and cold. In: Farrell PA, Joyner MJ, Caiozzo VJ, editors. ACSM's Advanced Exercise Physiology. 2 ed. Baltimore, MD: Wolters Kluwer | Lippincott Williams & Wilkins; 2012, with permission from Wolters Kluwer Health. Originally adapted with permission from Nielson M. Die regulation der korpertemperatur bei muskerlarbeit in Skandinavisches Archiv für Physiologie, Vol 79, pages 193–230, © 1938, with permission from De Gruyter (Germany)

humid environments is attenuated relative to evaporative heat loss potential in hot-dry environments. This is because in hot-humid environments more sweat remains unevaporated and thereby drips off the skin, providing no cooling. As a consequence, a greater increase in core temperature would occur under these conditions. In environments with low humidity, the predominant avenue of heat exchange depends on the ambient temperature. Figure 2.2 illustrates data from a person cycling for 1 h under a variety of environmental conditions. Notice that metabolic rate remains constant across the various environments, but as environmental temperature rises evaporative heat loss is relied upon more heavily as the primary avenue of heat loss.

Body Temperature Control System

Human temperature regulation is achieved through a control system involving primarily a negative feedback loop. The control center that integrates peripheral information is generally considered the preoptic anterior hypothalamus [9], with afferent sensory information originating from thermoreceptors located in the body core as well as in the skin [10, 11].

Historically, heat loss thermoregulatory effector responses (i.e., elevations in skin blood flow and sweating) were thought to occur following a positive deviation in core temperature from a "set point" [3]. That said, some reject a "set point" view of thermoregulatory control. Instead, Romanovsky [12] proposes thermoregulatory control is achieved through so-called thermoeffector loops, or essentially independent neural networks with their own efferent and afferent divisions. Under this view, thermoeffector responses are triggered by various combinations of core and peripheral body temperatures, and integration occurs through the common controlled variable (body temperature). Rapid effector responses, such as sweating, under conditions in which core temperature would not be expected to have changed (e.g., forehead sweating within minutes of entering the hot outdoors), support such a view.

The intent of this section is not to argue in favor of one view or the other in terms of thermoregulatory control. Instead, it is important to understand that core temperature must be regulated to avoid catastrophic temperature fluctuations, and, importantly, the effector mechanisms necessary to achieve this regulation can impact performance and safety during sport and physical activity in the heat.

Thermoeffector Responses to Heat Stress

The two primary thermoregulatory heat loss effector responses to heat stress are an increase in skin blood flow (achieved via cutaneous vasodilation) and sweating.

Increased Skin Blood Flow and Accompanying Cardiovascular Responses

The dynamics of skin blood flow responses to heat stress during passive (e.g., resting in hot ambient conditions with solar radiation and/or near hot processes) and active (e.g., associated with the heat gained during exercise) conditions are slightly different. Because the focus of this text is on sport and physical activity in the heat, the coverage of skin blood flow in this section will emphasize skin blood flow responses to a heat stress associated with exercise; especially that performed in hot ambient conditions.

The architecture of the skin vasomotor control includes two neural pathways [13–16]. Glabrous (non-hairy) portions of the skin, such as the lips, palms, soles of the feet, nose, and ears, are primarily innervated by sympathetic adrenergic vasoconstrictor neurons [3]. The remaining non-glabrous (hairy) portions of the skin (e.g., torso, arms, and legs) are innervated by both sympathetic adrenergic vasoconstrictor neurons and sympathetic cholinergic "active" vasodilator neurons [3, 13, 14]. The sympathetic adrenergic vasoconstrictor nerves innervating the skin exhibit tonic

activity, whereas the sympathetic cholinergic nerves do not. The combined effect of withdrawal of the sympathetic adrenergic vasoconstrictor nerve activity and engagement of active cutaneous vasodilation—as would occur during a heat stress—increases cutaneous vascular conductance leading to increases in skin blood flow.

Heat generated in exercising muscles is transferred to cooler blood via convection as the blood circulates through the muscle tissue. The blood then carries this heat to the skin as it traverses the systemic circuit. Increasing skin blood flow thereby raises skin temperature [3]; if ambient temperature is lower than skin temperature, this results in a favorable skin-to-air temperature gradient. Once sweating begins, heat delivered to the skin via increased skin blood flow is lost through sweat evaporation. In this way, sweating and skin blood flow work together to dissipate heat away from the body.

At the onset of aerobic exercise, a generalized vasoconstriction of inactive circulatory beds occurs, which includes the skin circulation [17, 18]. This response has been attributed largely to increased vasoconstrictor tone, not to diminished vasodilator activity [17]. Figure 2.3 [19] shows forearm blood flow responses (primarily cutaneous vascular responses) at the initiation of exercise in normothermic and hyperthermic conditions. Notice the initial declines in forearm blood flow (and thereby, skin blood flow) at the initiation of each exercise bout.

This cutaneous vasoconstriction at the onset of dynamic exercise is relatively short-lived as skin blood flow increases with elevations in core temperature. That said, the onset for cutaneous vasodilation (i.e., increase in cutaneous vascular conductance) is delayed during exercise (compared to resting conditions) [20, 21] (see Fig. 2.4), and the magnitude of the delay is greater with higher intensity exercise [22]. Additionally, the skin blood flow response plateaus at core temperatures of ~38 °C/100.4 °F during upright exercise [23, 24], which is considered well below maximal skin blood flow. This plateau occurs secondary to diminished active vasodilator activity rather than increased vasoconstrictor tone [20, 21, 23].

The cutaneous active vasodilator system is vital for appropriate skin blood flow responses during exercise, as it is estimated to account for as much as 90% of the cutaneous vasodilatory response [13, 14]. This system comprises sympathetic cholinergic nerves but the precise neurotransmitters responsible for active vasodilation are unresolved. Several factors have been implicated, and it is likely the mechanisms are redundant in that if one or more are not operating another can effect the same response. Nitric oxide (NO)—originating from neuronal nitric oxide synthase [25, 26]—contributes to reflex cutaneous vasodilation but the extent of the involvement is variable [14]. Besides NO, other substances implicated in reflex cutaneous vasodilation include vasoactive intestinal peptide [27, 28], histamine [29], substance P or other neurokinin-1 agonists [30], and prostaglandins [31], but a full understanding of these mechanisms has not yet been established [14].

Despite this lack of complete understanding of reflex cutaneous vasodilatory mechanisms, the collective cardiovascular responses to heat stress are clearer. Large increases in skin blood flow during exercise in hot conditions present unique challenges to the cardiovascular system because a competition for available cardiac output arises between the skin and active skeletal muscle. During mild exercise in the

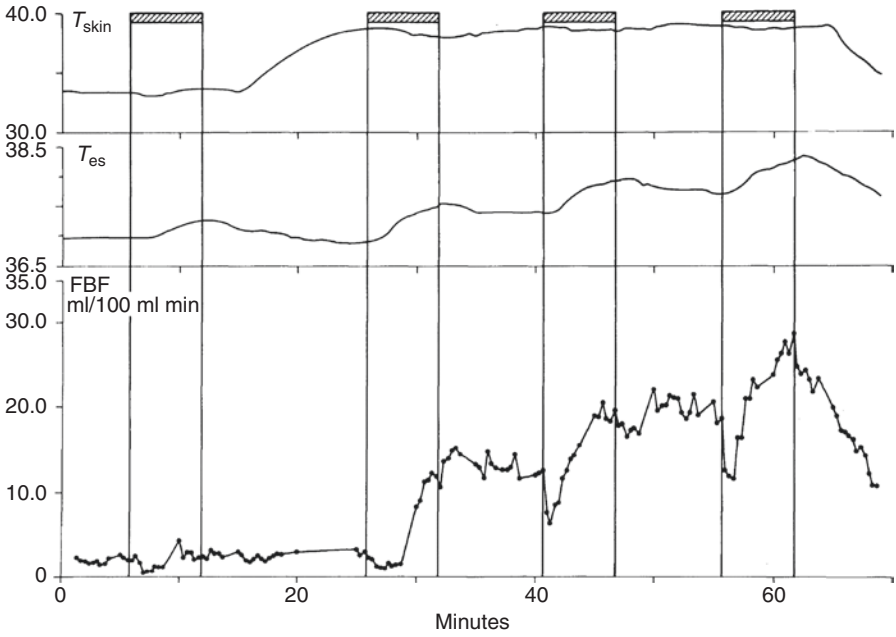

Fig. 2.3 Forearm blood flow (FBF) responses (primarily representative of skin blood flow) during 6-min bouts of supine leg exercise (rectangular regions). The first bout of exercise was performed at normothermic mean skin temperature (T_{skin}); the subsequent 3 bouts were performed at elevated whole-body skin temperature. Regardless of skin temperature, skin blood flow decreased at the onset of each exercise bout, showing cutaneous vasoconstriction even after skin blood flow was already elevated. T_{es} = esophageal temperature. From Johnson and Park [19], with permission. Reprinted with permission from Johnson JM, Park MK. Effect of heat stress on cutaneous vascular responses to the initiation of exercise in Journal of Applied Physiology, The American Physiological Society, Vol 53, No 3, pages 744–749, © 1982, with permission from Journal of Applied Physiology, The American Physiological Society

heat, the increase in cardiac output is augmented to support the added skin demand for blood flow [32]. However, during moderate to heavy exercise in the heat, cardiac output is not increased further by the external heat load [32–34]. Furthermore, for a given exercise intensity and cardiac output, heart rate is higher, which can result in reduced diastolic filling time, and, along with reduced venous pressure and cardiac filling, can reduce stroke volume [33, 35]. Cardiac contractility is increased to account for reduced cardiac filling, and cardiac output is essentially maintained [3].

The preceding discussion raises the question as to whether the competition between muscle and skin results in compromised muscle blood flow during exercise in the heat. The reader is referred to Gonzalez-Alonso, et al. [20] for a thorough review on this topic. In short, available evidence suggests muscle blood flow is not compromised during exercise in the heat when the exercise is performed at light to moderate intensities [20, 36]. Redistribution of blood flow away from other vascular beds (e.g., splanchnic and renal), rather than muscle, liberates more blood for the cutaneous

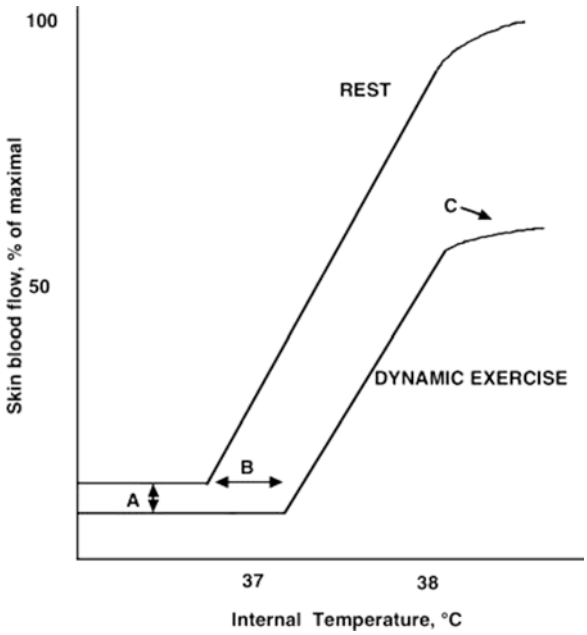

Fig. 2.4 Representative effects of moderate-intensity exercise on thermoregulatory control of skin blood flow. Compared to resting conditions, exercise results in the following: *A* = vasoconstrictor response at the onset of exercise; *B* = increase in the internal temperature threshold for cutaneous vasodilation (and thereby increased skin blood flow); *C* = plateau in skin blood flow above an internal temperature of ~38 °C/100.4 °F, which is well below the plateau in skin blood flow in the resting condition that occurs near maximum. From González-Alonso, et al. [20] © 2008 John Wiley and Sons/Blackwell Publishing, with permission. Reprinted with permission from Gonzalez-Alonso J, Crandall CG, Johnson JM. The cardiovascular challenge of exercising in the heat in Journal of Physiology (London), Vol 586, pages 45–53, © 2008, with permission from John Wiley and Sons

circulation [13, 36] although this may not account for all of the additional blood flow directed to the skin during exercise heat stress [20]. In contrast, prolonged exercise in the heat with concomitant dehydration, because of elevated sweating combined with inadequate fluid replacement, can result in reduced muscle blood flow; interestingly though, muscle oxygen delivery is essentially maintained because of hemoconcentration associated with that dehydration [20]. At high exercise intensities in the heat—such as at maximal oxygen uptake—muscle blood flow to active tissue may be compromised because of reduced blood pressure, not because of reduced muscle vascular conductance [37].

During moderate-intensity exercise, the competition between muscle and skin for blood flow results in lower skin blood flow at any given core temperature compared to resting conditions, but the slope of the skin blood flow-to-core temperature relation appears unaffected (Fig. 2.4). That said, dehydration will increase the core temperature threshold for the onset of cutaneous vasodilation, reduce the slope of the skin blood flow-to-core temperature relation, and lower the maximal skin blood achievable during exercise [38].

Sweating

Sweat secretion during exercise heat stress primarily comes from eccrine sweat glands which are distributed over almost the entire body surface, although the density of glands varies from one body region to another [39]. While the exact neural pathways controlling sweating are incompletely understood, it is known that sweat glands are primarily activated by acetylcholine released from sympathetic cholinergic nerves that binds to muscarinic receptors on the glands [39]. Alpha- and ß-adrenergic agonists can also stimulate sweating [40], but thermoregulatory sweating likely is primarily attributable to stimulation of muscarinic receptors given that atropine (a muscarinic receptor antagonist) eliminates such sweating [41].

As previously mentioned, the primary signal stimulating sweating is an increase in core temperature. Mean and local skin temperature can modify this response by shifting the threshold for the onset of sweating [39, 42] as well as the slope of the sweat rate-to-internal temperature relation [43]. Additionally, periglandular conditions also can modulate the sweating response. For example, skin blood flow and local temperature can both independently modify sweat rate during passive heat stress (i.e., very low skin blood flows and cooled skin each attenuate sweating while warmed skin accentuates sweating) [39, 44]. If shear stress-mediated release of NO occurs in the skin, and low skin blood flow and cooled skin reduce shear stress, then effects on sweating may be related to an NO-mediated mechanism since inhibition of NO attenuates sweating in humans [45, 46].

It is important to note that while the primary signal mediating sweating appears to be an increase in core temperature, sweating in as little as a few seconds following the onset of exercise has been observed prior to any change in core or skin temperatures [47, 48]. The mechanism responsible for the modulation of sweating by exercise, independent of thermal stimuli (i.e., core and skin temperatures), has been attributed to central command [39], metaboreceptors [49], and to a lesser extent, mechanoreceptors [39], but not all studies support this assertion. Discrepant findings may be explained by the thermal state of subjects before exercise given that pre-exercise core and skin temperatures can modulate the effect of exercise on sweating [47, 48].

Regardless of the precise mechanism inducing sweating during exercise heat stress, sweating occurs initially by increasing the number of activated sweat glands. As the heat stress continues, subsequent elevations in sweating occur as a result of increasing the sweat output per gland [39]. Like with regional differences in sweat gland density mentioned above, there are also regional differences in sweat rate. Using an eloquent sweat mapping technique, Smith and Havenith observed highest sweat rates on the central and lower back and forehead, and lowest sweat rates at the extremities [50]. Overall, greater sweat rates occurred with higher exercise intensity, but regional differences persisted. The regional differences did not correspond to regional differences in sweat gland density, so authors attributed the differences to regional variation in sudomotor sensitivity and sweat output per gland [50].

Humans have a tremendous capacity for sweat loss during exercise heat stress, with the highest reported sweat rate equaling >3 L/h [51], although average maximum sweat rates for humans are probably closer to ~1.4 L/h [39, 52]. Sweat rates can vary based on clothing ensemble, environmental conditions, activity duration, and exercise intensity [39, 53]. Table 2.1 highlights the broad range of sweat rates from a variety of sporting activities.

Prolonged sweating can result in severe body fluid deficits if left unattended. Such dehydration results in decreased blood volume (hypovolemia) and plasma hyperosmolality, each of which can diminish sweating sensitivity (slope of sweat rate vs. core temperature) [53], although an effect of hyperosmolality in reducing sweat sensitivity is not a consistent finding [38]. Nevertheless, hyperosmolality appears to have more profound effects on sweating than hypovolemia [66]. Dehydration present

Table 2.1 Sweat rates observed in various sports

Sport	Condition	Sweat rate (L/h)	
		Mean	Range
Swimming [55]	Training (males and females)	0.37	
Waterpolo [55]	Training (males)	0.29	[0.22–0.35]
	Competition (males)	0.79	[0.69–0.88]
Netball [56]	Summer training (females)	0.72	[0.45–0.99]
	Summer competition (females)	0.98	[0.45–1.49]
Ironman triathlon [57]	Temperate competition (males and females)		
	Swim leg		
	Bike leg	0.81	(0.47–1.08)
	Run leg	1.02	(0.4–1.8)
Soccer [58]	Winter training (males)	1.13	(0.71–1.77)
Soccer [59]	Summer training (males)	1.46	[0.99–1.93]
Half marathon running [60]	Winter competition (males)	1.49	[0.75–2.23]
Basketball [56]	Summer training (males)	1.37	[0.9–1.84]
	Summer competition (males)	1.60	[1.23–1.97]
Tennis [61]	Summer competition (males)	1.60	[0.62–2.58]
	Summer competition (females)		[0.56–1.34]
Cross-country running [62]	Summer training (males)	1.77	[0.99–2.55]
Rowing [63]	Summer training (males)	1.98	(0.99–2.92)
	Summer training (females)	1.39	(0.74–2.34)
American Football [62]	Summer training (males)	2.14	[1.1–3.18]
Squash [64]	Competition (males)	2.37	[1.49–3.25]
Tennis [65]	Summer competition (cramp-prone males)	2.60	[1.79–3.41]

Values are mean and (range) or [95% reference range]. Notice generally higher sweat rates during summer training because of adaptations associated with chronic heat exposure. Swim leg, bike leg, and run leg refer to the swim, bike, and run portions, respectively, of an Ironman triathlon. Adapted from Sawka, et al. [54], with permission. Reprinted with permission from Sawka MN, Burke LM, Eichner ER, Maughan RJ, Montain SJ, Stachenfeld NS. American College of Sports Medicine position stand: exercise and fluid replacement, in Medicine and Science in Sports and Exercise, Vol 39/Issue 2, pages 377–390, © 2007, http://journals.lww.com/acsm-msse/fulltext/2007/02000/Exercise_and_Fluid_Replacement.22.aspx, with permission from Wolters Kluwer Health Inc

at the start of exercise also can delay the sweating onset threshold [53]. The culminating consequence of unchecked dehydration is accelerated hyperthermia and an increased risk of a heat-related injury.

Effects of Increased Skin Blood Flow and Sweating on Whole-Body Heat Loss

The net effect of exercise-induced increases in skin blood flow and sweating is an increase in whole-body heat loss. As illustrated in Fig. 2.5, this increase in whole-body heat loss lags the rapid increase in rate of metabolic heat production that occurs in the early minutes of exercise. Furthermore, the delay in reaching a steady-state rate of whole-body heat loss is greater with increasing rates of heat production. Under compensable heat stress conditions (i.e., light, moderate, and high rates of metabolic heat production shown in Fig. 2.5), the differences in rate of heat gain/loss would lead to an increase in core temperature that typically occurs in the early stages of a prolonged exercise bout. Once heat balance is achieved, core temperature would stabilize (typically around 30–45 min irrespective of exercise intensity). This would hold true until a disturbance in the body's ability to maintain heat loss at constant levels occurs, such as progressive dehydration. Furthermore, as the figure shows, there is a point at which a further increase in metabolic heat production will not be matched by a corresponding increase in heat loss (i.e., very high rate of heat production, uncompensable heat stress). This is the point where no further

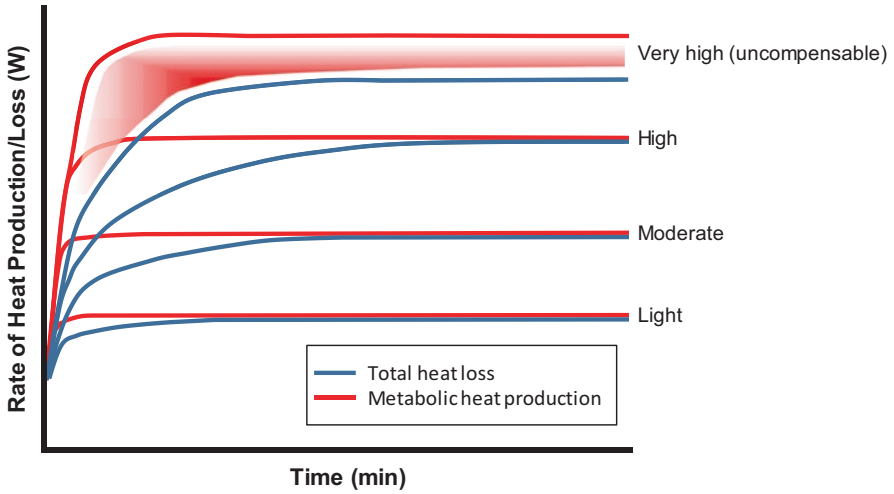

Fig. 2.5 Theoretical whole-body heat loss (dry + evaporative) responses to rapid increases in metabolic heat production associated with exercise at fixed rates of heat production in the heat (30 °C/86 °F, 20% relative humidity)

increases in skin blood flow and sweating are possible because a physiological peak has been attained. At this intensity of exercise, rate of heat storage remains positive and core temperature rises progressively until it reaches a point where the individual stops exercising (volitional); or if the individual continues, and the core temperature is left unchecked, a heat-related injury or death occurs.

Metabolic Consequences of Exercise in the Heat

Numerous studies have demonstrated reductions in maximal oxygen uptake ($\dot{V}O_{2max}$) during prolonged exercise in the heat [33, 35, 37, 67–69]. The mechanism for this impairment is likely an inability to achieve maximal cardiac output relative to a normothermic $\dot{V}O_{2max}$ test. This response is mediated by reduced cardiovascular reserve secondary to elevated core and skin temperatures [1]. It is noteworthy that reduced $\dot{V}O_{2max}$ under conditions of elevated skin blood flow (i.e., prolonged exercise in the heat) results in a higher relative metabolic intensity (%$\dot{V}O_{2max}$) for a given absolute work level, as reflected by elevated heart rate associated with cardiovascular drift [35]. Greater physiological strain associated with higher %$\dot{V}O_{2max}$ during exercise in the heat intensifies perceived exertion and thermal discomfort [3], thereby inducing a slowing of self-paced work [1, 3]. Practitioners using a constant target heart rate to prescribe intensity during exercise in the heat should recognize that doing so reduces the metabolic stimulus of the exercise and so cardiorespiratory training adaptations may be compromised. Allowing heart rate to rise over time while maintaining absolute intensity, however, will result in elevated cardiovascular and thermal strain, which could acutely increase susceptibility to a heat illness. Coaches and practitioners must balance maintenance of a target exercise intensity with ensuring participant well-being [35].

Summary

Humans regulate core temperature within a narrow range by balancing heat production with heat loss. Several avenues of heat exchange may facilitate heat loss but the primary avenue during exercise in the heat is evaporation of sweat. Thermoregulatory mechanisms engaged during exercise—and intensified in hot environments—include elevated skin blood flow and sweating, which are controlled by complex neural networks associated with the preoptic anterior hypothalamus. Elevated skin blood flow is achieved through a combination of cutaneous vasoconstrictor withdrawal and active cutaneous vasodilation. Sweat is secreted as a result of cholinergic stimulation of sweat glands. Both cutaneous vasodilation and sweating may be modulated by local factors, such as NO. The net effect of exercise-induced increases in skin blood flow and sweating is an increase in whole-body heat loss that balances metabolic heat production except under uncompensable heat stress conditions, in which case peak

physiological heat loss capacity has been attained. This would especially be the case during exercise in a hot environment, when the heat gained from the environment adds to the metabolic heat load. Redistribution of blood to the skin, coupled with fluid loss through sweating during exercise in the heat, can present severe cardiovascular challenges to the exercising human. Diminished cardiovascular reserve associated with elevated core and skin temperatures reduces $\dot{V}O_{2max}$ and results in elevated $\%\dot{V}O_{2max}$ for a given absolute work rate. Collectively, the physiological challenges associated with sport and physical activity in the heat require careful consideration on the part of athletes, coaches, and practitioners in order to optimize performance and safety. The ensuing chapters in this book will provide an in-depth examination of the intervening factors that contribute to the thermoregulatory response, the populations that need to consider the implications, and the tools available to enhance our ability to supervise and enhance the human response to exercise in the heat.

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Part II
Enhancing Exercise Heat Tolerance

Chapter 3
Heat Acclimation

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Performance, Cardiovascular, Thermoregulatory, and Fluid Adjustments to Heat Stress: The Rationale for Heat Acclimation

It is nearly impossible to exercise without increasing core body temperature, even in very cold conditions. When humans exercise, a substantial amount of heat is generated as a by-product of metabolism that must be dissipated. In fact, if we were not able to lose metabolic heat to the environment, we would only be able to exercise for a few minutes. When exercise is performed in a hot environment, or when heat loss mechanisms are inhibited as seen with wearing heavy clothing or in very humid environments, the added cardiovascular strain from a rise in core body and skin temperatures can result in decreased performance and possibly dangerously high body temperatures. This cardiovascular strain contributes to impaired aerobic exercise performance, even when subjects are euhydrated [1]. It is estimated that aerobic exercise performance is reduced in the heat by 5–20% in well-trained athletes.

This need for heat dissipation results in a competition for the available cardiac output to provide adequate oxygen and energy sources to muscles while simultaneously

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providing blood flow to the skin for heat dissipation. There are many adjustments to blood flow distribution that occur during exercise to help us meet the competing demands for blood flow. The muscle pump helps to support ventricular filling by preventing blood pooling in the lower limbs. Blood flow is redistributed to the muscles and skin from the splanchnic area (e.g., liver, pancreas, spleen) and the kidneys, two vascular beds that receive approximately 50% of cardiac output under resting conditions. During very intense exercise, blood flow to these regions can be remarkably decreased. Provided venous return is maintained, and therefore ventricular filling and stroke volume, by these adjustments, cardiac output in endurance-trained athletes can increase during exercise by fivefold to eightfold.

During purely passive heat stress, similar adjustments are made to maintain blood pressure in the face of dramatic decreases in peripheral vascular resistance, secondary to high skin blood flow. Whole body skin blood flow in these conditions has been reported to be as high as 7–8 L/min, with an increase of cardiac output to 12–13 L/min in the supine position [2]. However, this high of a skin blood flow cannot be maintained in the upright posture or with exercise. At higher exercise intensities, skin blood flow (and therefore sweat rate, as they are generally controlled in parallel) is reduced due to the competition for a limited cardiac output. During high-intensity exercise, even in very hot ambient conditions, there are clear limits on the ability to increase skin blood flow to the levels required for the thermoregulatory demands [3], resulting in a very rapid increase in mean body temperature. Exercise in these conditions cannot be maintained very long. Studies have demonstrated that even highly trained athletes are not able to continue to exercise when their body core body temperature exceeds over ~40.5 °C/104.9 °F [4, 5] although the actual temperature at which failure occurs likely spans a range of mean body temperatures, as skin temperature has a modifying influence [4, 5].

The effects of body water loss through high sweat rates during exercise in the heat further reduce exercise performance, primarily through a reduction in cardiac output. Although heat stress and exercise can both result in dehydration, environmental heat stress, and dehydration can act independently to limit cardiac output and blood delivery to the active muscles during high-intensity exercise [6]. Additionally, dehydration impairs the body's ability to lose heat, causing a faster rise in core body temperature compared to a euhydrated state. This is because in a dehydrated state, both sweat rate and skin blood flow are lower at the same core body temperature. Even for relatively low-intensity exercise, dehydration increases the incidence of exhaustion from heat strain. Reduced aerobic endurance performance results from an increased core body temperature, elevated heart rate, greater perceived exertion, and increased reliance on carbohydrate as a fuel source.

In summary, physical activity generates heat and this rate of heat gain is magnified by heavy or insulated clothing impeding heat loss, exercise intensity, and hot/humid environmental conditions. This heat gain must be effectively dissipated for continued work in the heat. Shifts in blood flow from the core to the skin and sweat evaporation assist in heat loss, but consequently challenge the cardiovascular system. The blood shunted to the skin vascular beds and sweat-induced plasma volume loss, in turn, decrease cardiac output. The result of these

thermoregulatory alterations is increased heart rate, increased perceived exertion, and reduced aerobic endurance performance. Reductions in total body water occurring either acutely (sweat-induced) or chronically (reduced fluid intake overtime) accelerate heat gain by impairing both sweat evaporation and skin blood flow. Incredibly, heat acclimation serves to enhance cardiovascular stability, thermoregulation efficiency, fluid–electrolyte balance, and attenuate physiological strain benefiting physical activity performance and safety in the heat. For these reasons, heat acclimation is a paramount strategy that should be employed before or during the beginning days of physical activity in the heat. The physiology of these adaptations and how to implement a heat acclimation induction protocol will be explored in this chapter.

The Physiology

Heat and exercise separately represent independent stressors resulting in specific system level and transcriptional responses [7] and alone do not fully produce a heat acclimated phenotype [8, 9]. Rather, it is the combination of exercise plus heat stress that, over repeated consecutive exposures, elicit molecular, central, and peripheral adaptations characteristic of heat acclimation. This driving mechanism of consistent exercise–heat stress must elicit an elevation in both cutaneous and core temperature. In a classic study by Avellini et al. [10], ten untrained non-heat acclimated subjects cycled 1 h at 75% maximal oxygen consumption ($\dot{V}O_{2max}$) for 5 days while immersed to the neck in either 32 °C/89.6 °F or 20 °C/68 °F water. After this period of training, subjects completed a 3-h heat stress test in 49 °C/120.2 °F and 20% relative humidity at 30% $\dot{V}O_{2max}$. Final rectal temperature and heart rate were reduced while sweat rate increased in the 32 °C/89.6 °F water training group but not the 20 °C/68 °F water training group. Such responses to heat stress tests after repeated consecutive exercise–heat stress bouts are hallmarks of a heat acclimated state. The permissive rise in core body and skin temperature via exogenous (and endogenous) heat exposure stimulates the temperature-regulating center in the hypothalamus beginning the cascade of adaptations at effector organs resulting in the heat acclimation phenotype.

The Adaptations and Benefits

Heat acclimation results in numerous adaptations that improve thermoregulation, cardiovascular stability, thermotolerance, fluid–electrolyte balance, and reduce physiological strain leading to enhanced submaximal and maximal aerobic exercise performance in the heat. Heat acclimation is also the best preventative strategy for exertional heat illnesses. This section reviews the remarkable adaptations of heat acclimation and the prophylactic and ergogenic benefits thereof.

Cellular Mechanisms in Improved Thermotolerance and Heat Acclimation

Many of the cellular and physiological mechanisms that underlie adaptations with heat acclimation are not fully understood. This is similar in many ways to our understanding of the physiological adaptations that occur with exercise training. In both cases, acute bouts of stress result in long-term adaptive responses that lead to an improved ability to exercise or tolerate subsequent thermal stresses. The exact signaling mechanisms behind these chronic adaptations likely involve many different molecular signaling cascades and are different depending on the specific tissue. In terms of repetitive heat exposure, every cell of the body is "stressed" to some extent when core body temperature is increased. How this translates into improved function likely involves the actions of heat shock proteins in most tissues.

Heat shock proteins are a class of proteins synthesized by all organisms in response to heat or other stressors. Heat shock proteins were so named because they were observed to increase rapidly at the onset of intense heat. Heat shock protein expression increases during and following exposure to heat stress, and the degree of expression is related to the duration and level of hyperthermia, as well as the rate of rise in core body temperature. Passive heat exposure and exercise each elicit heat shock protein synthesis, with greater expression when these stressors are combined. Importantly, heat shock proteins are also increased in response to other environmental stressors, including cold stress and hypoxia. Thus, they could be considered more generally as "stress proteins" and are implicated in cross-tolerance to novel stressors.

In general, heat shock proteins are thought to protect cells from acute damaging effects of the various stressors. However, they also serve to confer tolerance against subsequent potentially lethal stress. For example, cells that have previously been exposed to non-lethal heat shock can survive a subsequent otherwise lethal heat shock. This is referred to as "thermotolerance," which differs from heat acclimation although the two concepts are complementary. Whereas heat acclimation refers to improved overall function of the athlete following repeated exposures to mild or moderate heat stress, thermotolerance refers to the ability to better withstand an otherwise more severe stress due to previous exposure.

Similar actions of heat shock proteins can be seen in response to other stressors that may occur during exercise, such as hypoxia, ischemia, energy depletion, and acidosis. The improved tolerance stems from their role in the prevention of denaturing structural proteins as well as processing damaged proteins and accelerating repair. This is the foundation for the growing belief that the heat acclimated phenotype may provide greater tolerance and adaptation to many stressors associated with intense exercise. Importantly for the athlete or coach, these factors suggest that the use of heat acclimation may go beyond just improvements in exercise performance in a hot environment. A heat acclimated athlete may be more tolerant to rapid changes in altitude, may be less susceptible to concussive injury, and may have

improved processes to repair damaged muscle [11]. There are numerous labs around the world very interested in these expanded roles for heat acclimation in the management and training of athletes.

Total Body Water and Plasma Volume

Plasma volume typically increases up to 16% over the first 3–5 days of a constant work rate heat acclimation protocol (Table 3.1) and slowly returns to baseline as the exercise–heat stress diminishes and subsequent thermoregulatory and fluid balance adaptations take place. Recent data using the controlled hyperthermia protocol, and thereby maintaining the exercise–heat stress, demonstrate that plasma volume expansion can persist throughout the acclimation protocol [12]. This is a form of physiological habituation [13].

The primary pathways which initially expand total body water include increases in aldosterone and arginine vasopressin secretion during exercise and heat stress. Additionally, the conservation of sodium chloride in the extracellular fluid contributes to the increase in total body water although the mechanisms behind this remain unclear. The expansion of plasma volume is thought to occur through increased production of the intravascular protein albumin, which "pulls" water from the interstitial into the intravascular space. There may also be a reduction in protein loss through the capillaries in the skin, resulting in a net increase in proteins and more water.

The increase in plasma volume provides at least two advantages to the exercising athlete. Most notably, a larger total blood volume will increase cardiac filling pressure (i.e., preload) and help maintain cardiovascular stability during exercise in the heat. This will have numerous effects on cardiovascular and thermoregulatory responses during heat stress, including increasing or maintaining stroke volume, lowering exercise heart rate, and may play a role in lowering the threshold for skin blood flow and sweat rate as described below. Additionally, an expanded plasma volume increases the specific heat of blood, improving the transfer of heat from the exercising muscles to the skin. All these factors can contribute to improved exercise performance in the heat, and possibly in more temperate conditions as well. However, there is some debate whether too much plasma volume expansion can result in a reduction or no improvement in exercise performance if the blood becomes too dilute. In an experimental approach using infusions to acutely expand plasma volume, Coyle and colleagues have shown that plasma volume expansion by 500–600 mL (resulting in an 11% reduction in hemoglobin concentration) can result in a decreased VO_{2max} due to the relative hemodilution [14]. Whether this level of plasma volume expansion in a competitive athlete going through a heat acclimation protocol would be countered by other factors (such as a larger cardiac output) remains a topic of debate.

Table 3.1 Magnitude of performance, physiological, and perceptual changes after heat acclimation

Endurance performance		Magnitude of change
Time trial in the heat	↓	7 ± 7%
Time-to-exhaustion in the heat	↑	23 ± 29%
By protocol duration		
STHA	↑	7 ± 8%
MTHA	↑	21 ± 28%
LTHA	↑	22 ± 29%
Physiological adaptations		
Thermoregulatory		
Resting T_c	↓	0.18 ± 0.14 °C/0.32 ± 0.25 °F
Exercising T_c	↓	0.34 ± 0.24 °C/0.64 ± 0.43 °F
Resting T_{skin}	↓	0.03 ± 0.31 °C/0.05 ± 0.56 °F
Exercising T_{skin}	↓	0.57 ± 0.49 °C/1.03 ± 0.88 °F
Skin blood flow sensitivity	↓	0.25 ± 0.10 °C/0.45 ± 0.18 °F, 42 ± 40%
Max skin blood flow	↑	4%
Sweat sensitivity (onset at T_c)	↓	0.28 ± 0.21 °C/0.50 ± 0.38 °F, 25 ± 10%
Sweat rate	↑	19 ± 21%
Extracellular heat shock protein 72	↑	4% (range: −15 to 32)
Intracellular heat shock protein 72	↑	110–320%
Cardiovascular		
Resting HR	↓	6 ± 5 bpm
Exercising HR	↓	16 ± 6 bpm
Exercising stroke volume	↑	12 ± 16%
Resting plasma volume	↑	4.3 ± 4.7% (range: −1.9 to 16%)
Fluid–electrolyte		
Body mass loss after exercise	↑	38 ± 70%
Sweat sodium concentration	↓	22 ± 16 mmol/L
Aldosterone concentration	↑	25 ± 35%
Arginine vasopressin	↓	5 ± 15%
(see thermoregulatory section for sweat response)		
Metabolic		
Oxygen consumption	↓	2.5 ± 2.0%
Muscle glycogen sparing	↑	35 ± 47%
Blood lactate concentration	↓	1.0 mmol/L (range: 0.8–3.2)
Perceptual		
Ratings of perceived exertion	↓	1–3 units
Thermal sensation	↓	0.8 units (range: 0.3–2.0)

Data are mean ± SD, from Tyler, et al. [20]. *SHTA* short-term heat acclimation (≤7 days), *MTHA* medium-term heat acclimation (8–14 days), *LTHA* long-term heat acclimation (≥15 days), T_c core body temperature, T_{skin} skin temperature, *HR* heart rate

Sweating and Skin Blood Flow

Increases in skin blood flow and sweating are intimately linked, as both are controlled by the same branch of the sympathetic nervous system. Current thinking is that sweat glands are activated and controlled by acetylcholine released from these sympathetic cholinergic nerves, and that a co-transmitter (possibly vasoactive intestinal peptide or substance P, both working partly through nitric oxide-dependent pathways) causes cutaneous vasodilation [15]. In terms of the responses to exercise and heat stress, skin blood flow, and sweating share a similar core body temperature threshold for activation and track together with increased thermoregulatory drive and exercise intensity.

Following heat acclimation, both the initiation of sweating and active vasodilation of the blood vessels in the skin are shifted so that their onset occurs with less of a rise in core temperature (i.e., a lowered "threshold"). This shift in the onset threshold is most likely through central control mechanisms in the hypothalamus. Peripheral changes in the skin contribute to the rate and sensitivity of the sweat glands and blood vessels as well. For example, sweat glands are more resistive to fatigue, so that higher sweat rates can be maintained (particularly in humid conditions). Likewise, sweat glands and blood vessels appear to be more sensitive to acetylcholine and nitric oxide [16]. Collectively, these changes in onset threshold and sensitivity help to begin heat dissipation at a lower core temperature and augment the sweating and vasodilation for a given rise in body temperature, resulting in a lower core body and skin temperature rise during exercise. Although sweating and skin blood flow are actually higher at a given specific body temperature after heat acclimation, the overall earlier rise and better defense of body temperature during exercise in the heat may result in lower demands for skin blood flow. Whether heat acclimation results in greater total body water loss in an athlete during exercise is likely dependent on a multitude of factors. For example, heat acclimated individuals tend to start drinking earlier and drink more fluids during exercise, suggesting there is a shift in the mechanisms driving thirst [17].

Another adaptation that occurs following heat acclimation is that sweat is more dilute, resulting in better conservation of salts (which helps to maintain the drive for thirst). This occurs through an improved reabsorption of sodium at the top of the sweat gland, and serves to help maintain plasma volume. A more dilute sweat also evaporates at a lower water vapor pressure, meaning that the "cooling power" of a given volume of sweat on the skin is improved following heat acclimation.

Heart Rate, Stroke Volume, and Cardiac Output

The "classic" hallmark cardiovascular adaptations to heat acclimation include a lowering of heart rate and a greater stroke volume, both supporting a better maintenance of cardiac output and blood pressure during exercise. The heart is under both

parasympathetic and sympathetic influences from the autonomic nervous system, which provides the appropriate neural outflow to the heart from integrated signals throughout the body. These include feedback from the cardiopulmonary and arterial baroreceptors, muscle metaboreceptors, arterial chemoreceptors, thermoreceptors, osmoreceptors, and central command. As heat acclimation results in many changes causing better cardiac filling pressures, including reduced central body and peripheral temperatures, greater lactate removal, and improved muscle perfusion, the lowered heart rate and better maintained stroke volume is likely the result of a multitude of factors that are improved following heat acclimation. That said, there are also changes within the cardiac muscle itself, mediated through specific heat shock proteins, which results in improved cardiac compliance and myocardial efficiency that may also contribute [18].

Changes in central cardiovascular function with heat acclimation occur quite rapidly and appear to track with the changes in plasma, especially in the case of stroke volume. A lower heart rate for a given exercise–heat stress stimulus is one of the most rapid changes observed with heat acclimation, which then continues throughout the first week of heat acclimation. Cumulatively, these improvements in central cardiovascular function serve to better maintain cardiovascular stability during exercise in the heat.

Metabolic Changes

Heat acclimation has been shown to reduce blood and muscle lactate accumulation during submaximal exercise. As lactate levels in the blood are a function of rates of both production and removal, one or both processes likely underlie these changes. On the production side, oxygen uptake and glycogen utilization at a given submaximal exercise intensity are reduced following heat acclimation. Epinephrine, which stimulates glycolysis, is also lower after heat acclimation. The expanded total body water and plasma volume serve to better maintain cardiac output and lower relative intensity for a given workload, resulting in an increased splanchnic blood flow, enhancing lactate removal.

Heat Acclimation as a Prophylactic Against Exertional Heat Illnesses

At the time of this publication, no experimental study has shown that heat acclimation directly prevents exertional heat illness due to the ethical limitations of purposefully causing potentially fatal exertional heat illnesses. However, virtually every heat acclimation study has reported lower core body temperatures post acclimation due to enhanced heat loss mechanisms. Additionally, the expanded total body water after acclimation likely assists with avoiding an increased core body

temperature rate of rise due to dehydration. Together, these mechanisms explain the substantial drop in exertional heat illness incidence after heat acclimation and substantiate the notion that heat acclimation is one of the best methods to prevent exertional heat illness.

Aerobic Performance in Hot and Temperate Environments

Given the recent and upcoming international competitions in near tropical climates (e.g., Brazil Olympics and World Cup, Tokyo Olympics, and Qatar World Cup), heat acclimation as an ergogenic aid has become a hot topic for athletes, coaches, and researchers alike. Adaptations that result in improved exercise performance and capacity following heat acclimation include an expanded plasma volume, higher sweat rates (of a more dilute sweat), lowered core body and skin temperatures, reduced heart rate, better maintenance of stroke volume, reduced oxygen demand, less reliance on glycolysis eliciting a glycogen sparing effect, improved thermal comfort, and greater power output at lactate threshold [19]. Table 3.1 summarizes data from a meta-analysis that synthesized the magnitude of performance, physiological, and perceptual gains derived from heat acclimation [20]. The wide range of performance and adaptation effects displayed in this table is noteworthy. The magnitude of adaptation is a function of individual fitness level before acclimating and heat acclimation exercise intensity, duration, frequency, number of days, and the environmental conditions (dry vs. humid heat). Notably, time trial and time-to-exhaustion performance in the heat after heat acclimation can be expected to improve by approximately 7% and 23%, respectively [20]. It is logical that time to exhaustion tests after heat acclimation demonstrate greater performance gains as the deleterious effects of heat stress compound and become more pronounced as exercise continues in the heat. If sweat-induced dehydration is addressed and controlled with behavioral strategies, prolonged exercise in the heat can be further improved.

Results from studies examining aerobic performance gains after heat acclimation in cooler temperatures are divided, demonstrating no benefit to as much as 5% faster finish times [20]. The mechanism(s) explaining this improvement in temperate conditions following heat acclimation are not clear. The leading theory explaining faster finisher times in temperate conditions is the expanded plasma volume observed after heat acclimation. A greater volume of plasma leads to increased venous return and subsequent cardiac filling, increasing stroke volume and thus cardiac output at any given submaximal or maximal intensity. Cardiac output increases likely explain the ~5% higher $\dot{V}O_{2max}$ values observed following heat acclimation because oxygen extraction improvements require longer than 1–2 weeks to garner. However, too much plasma volume expansion causes hemodilution reducing hemoglobin's oxygen uptake and release, likely explaining the lack of aerobic performance gains in temperate environments. The optimal increase in plasma volume for greater $\dot{V}O_{2max}$ and aerobic performance gains after heat acclimation is unknown.

There may be additional benefits of heat acclimation such as improved responses to novel environments, such as exercising at higher elevations (hypoxic environments). This concept of "cross-adaptation" refers to the process by which acclimation to one stressor may enhance adaptation to a novel stressor. An increasing number of animal and human studies demonstrate that the heat acclimated phenotype confers some protection to stressors such as hypoxia, ischemia-reperfusion injury, traumatic brain injury, and hyperoxia. How this can impact the athlete and preparation for competitive events is an exciting area of inquiry.

Induction and Decay of Heat Acclimation Adaptations

Heat acclimation or acclimatization develops through frequent exposure to exercise and hot environmental conditions ≥30 °C/86 °F either by artificial (acclimation, e.g., hot room) or natural (acclimatization, i.e., high ambient temperature) means. In general, heat acclimation is a fairly rapid process. Many different strategies that can be employed to induce heat acclimation. The time course, magnitude, and decay of acclimation adaptations will all depend on the specific organ system that is adapting, and the overall process and timing of acclimation strategies. For example, cardiovascular adaptations occur more rapidly requiring as few as 4–5 days in well-trained athletes. Thermoregulatory adaptations such as lower core body temperatures and greater sweat rate generally require longer protocols, up to 14 days.

Although heat acclimation adaptations can be classified into short term (≤7 days), medium term (8–14 days), and long term (≥15 days) [21], the physiological adaptations that impact exercise performance develop relatively quickly with 75–80% of the acclimation process occurring in 4–7 days [22]. The time course and primary adaptations following a period of heat acclimation up to 14 days are displayed in Fig. 3.1, as presented in the excellent review by Périard and colleagues [23]. Importantly, there is a growing consensus that longer heat acclimation protocols, even as much as 4 weeks or longer, may confer further adaptive benefits that could serve to improve exercise capacity. However, this has not been explored in competitive or high-level athletes. Finally, a single exercise–heat stress bout per day is sufficient to induce heat acclimation adaptations as two exercise–heat bouts per day do not appear to speed or enhance heat acclimation adaptations.

Similar to the benefits of exercise training, the benefits of heat acclimation will gradually decline if not maintained. The rate of decline appears to follow similar trends as induction, namely, those adaptive responses that increased first will also be the first to decay [13]. For example, the heart rate improvement is gained and lost more rapidly than the thermoregulatory responses. The actual rate of decline will depend on many factors such as the magnitude of acclimation achieved, the continuation of regular aerobic training, additional heat exposures, and overall fitness. Highly trained athletes tend to have slower heat acclimation decay rates.

As a general rule, it is thought that 1 day of heat acclimation benefit is lost for every 2 days without heat exposure. For example, after employing a 5-day heat

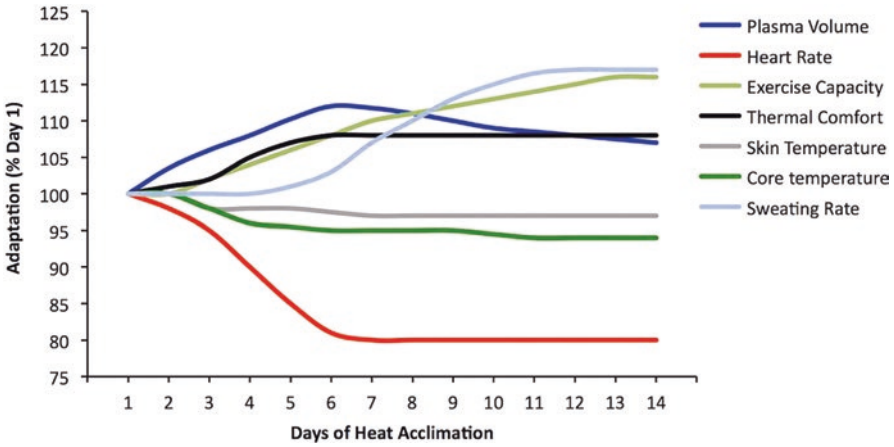


Fig. 3.1 Time course of adaptation to heat acclimation over the course of 14 days. Heart rate decreases and plasma volume expansion occur very rapidly, whereas some of the thermoregulatory adaptations are delayed slightly. Overall exercise performance during submaximal exercise in the heat is improved as a consequence of these adaptations. In terms of exercise performance, 10–14 days appear to be required to gain the full benefits of heat acclimation to a given submaximal exercise and thermoregulatory stimulus [23]. Reprinted with permission from Periard JD, Racinais S, Sawka MN, Adaptations and mechanisms of human heat acclimation: Applications for competitive athletes and sports, in Scandinavian Journal of Medicine and Science in Sports, Vol. 25, Suppl 1, pages 20–38, © 2015, with permission from John Wiley & Sons

acclimation protocol, full decay may be as short as 10 days. However, it has also been reported that 1 day of exercise in the heat is required for every 5 days without heat exposure to maintain benefits [24]. Both these rates of decay are expert opinion and have yet to be empirically tested. It seems that once significant heat acclimation has occurred, only 2–4 days of exercise–heat stress are required to restore most adaptations after about 3–4 weeks without exercise–heat exposure [25]. The term "heat acclimation memory" has been coined to characterize this phenomenon of rapid reinduction. The mechanism(s) underlying this phenomenon are unclear but work from Michal Horowitz in rats suggest epigenetic mechanisms regulating genetic expression and the transcriptional landscape of at least heat shock proteins play a key role [26]. Admittedly, there is a lack of details on heat acclimation decay and reinduction with respect to ideal intensity, frequency, and duration of heat exposures in highly competitive athletes.

In summary, heat acclimation optimizes heat dissipation mechanisms such as sweat evaporation and convection via increased skin blood flow. These beneficial adaptations enhance thermal load mitigation, allowing for better regulation of core body temperature during exercise. Additionally, the expanded plasma volume, improved fluid–electrolyte balance, and corresponding cardiovascular consequences of heat acclimation provide athletes with a competitive edge when performing in the heat, and perhaps in cooler environments. The products of these incredible adaptations also

reduce the risk of exertional heat illnesses. However, the temporary nature of heat acclimation adaptations must be strategically managed by coaches and athletes to coincide optimal heat adaptation with competition.

The Application

Morphological, functional, biochemical, and epigenetic changes serve to reduce the deleterious effects of heat stress resulting in improved submaximal and maximal aerobic performance, thermotolerance, comfort, and safety during physical activity in warm to hot weather following heat acclimation. For these reasons, many athletes strategically integrate heat acclimation into their annual training plan prior to major events or competition(s) in warm to hot environmental conditions.

Understanding the process of heat acclimation requires a basic understanding of the physiology of heat adaptation as previous discussed. This section discusses the "how" of heat acclimation while touching upon on several safety considerations. Safety is a concern when individual and team-based athletes undertake the deliberate act of exercising in the heat to induce heat adaptations, especially if this endeavor is unaccustomed. The induction protocol, training status of the athletes, timing of the induction protocol within the competitive season, food and fluid intake, and environmental considerations must be considered to attenuate exertional heat illness risk and achieve optimal heat adaptation. These parameters are overviewed herein.

Heat Acclimation Induction Protocols

As a general rule, the ideal strategy to obtain "optimal" adaptation is to create a sport specific heat acclimation protocol that closely replicates the work rate and environmental conditions of the competitive setting. However, there are many different factors that need to be considered in designing the ideal heat acclimation protocol. These include, but are not limited to, the specific sport, athlete, competition environment, equipment worn, the length of time available to become heat acclimated, the magnitude of heat acclimation required, the overall stress that can be placed on the athlete during the acclimation process, desired adaptations, and available resources. While many protocols for inducing heat acclimation exist, the adaptive impulse for heat acclimation remains exercise–heat-induced hyperthermia and hypovolemia sufficient to stress thermoregulatory and circulatory effector organs.

With this underlying principle in mind, several laboratory protocols have been developed and are overviewed in Table 3.2. Historically, constant workload protocols employing exercise in the heat for 100 min per day at 40–50% VO_{2max} for 10–14 days was recommended to elicit heat acclimation adaptations in both untrained and trained athletes [22]. Given that the majority of heat acclimation-induced adaptations are actualized within 4–7 days (Fig. 3.1), researchers are

Table 3.2 Various heat acclimation protocols with advantages and drawbacks

Protocol	Duration	Intensity	Length	Pros	Cons
Variable workload					
Controlled hyperthermia or isothermal	$T_c \geq 38.5$ °C/101.3 °F for ≥60 min	Sufficient to achieve and maintain desired T_c	5–14 days	• Sustained thermal strain • Potential optimization of adaptations	• Requires valid T_c assessment • Ideal initial exercise intensity/duration unknown
Self-regulated workload	Varies	Variable	7–14 days	• Practical application in team-based sports	• Less control of exercise variables and adaptive stimuli • Day-to-day improvements not easily measured
Constant workload					
Average fitness	100 min	40–50% VO_{2max}	10–14 days	• Constant exercise–heat stress • Day-to-day improvements easily measured	• Thermal strain/adaptation stimuli reduce as heat acclimation progresses • May ↓ magnitude of adaptation
High fitness	30–45 min	75% VO_{2max} or interval training	5–9 days	• Constant exercise–heat stress • Day-to-day improvements easily measured	• Thermal strain/adaptation stimuli reduce as heat acclimation progresses • May ↓ magnitude of adaptation

Data assume environmental conditions are similar between protocols. Lab studies typically employ isothermal or constant workload protocols as most exercise variables can easily be either controlled or measured. It is more difficult to control exercise variables in self-regulated protocols but this freedom fits well in field studies and team-based sport applications

T_c core temperature, VO_{2max} maximal oxygen consumption

exploring the minimum dose of exercise–heat stress to obtain thermal adaptations and performance gains. High-intensity short duration (75% $\dot{V}O_{2max}$, 30–35 min) exercise–heat stress can achieve comparable adaptations in 9 days [27] while controlled hyperthermia techniques require as few as 5 days in highly trained endurance athletes [28]. Current heat acclimation best practice guidelines suggest at least 60 min training sessions for 7 days in the heat, ideally longer (14 days) to achieve further thermoregulatory and aerobic performance benefits. The exercise intensity and rest breaks should be sport specific while inducing high sweat rates (achieving hypovolemia) and increased core body and skin temperature (hyperthermia).

While each heat acclimation protocol has advantages and disadvantages, the controlled hyperthermia technique has gained popularity among researchers. This protocol is attractive because well-trained endurance athletes achieve cardiovascular and some thermoregulatory adaptations in as little as 5 days [21]. Taylor contends high core body temperature is the primary stimulus for adaptation and sustaining this stimulus throughout the heat acclimation process results in optimal adaptations [13]. With constant workload protocols, the rise in core body temperature gradually reduces as one adapts to the heat reducing this adaptive stimulus. Conversely, with the controlled hyperthermia method, exercise intensity and duration (environmental stress or clothing) is progressively adjusted to achieve a core temperature ≥38.5 °C/101.3 °F for at about 1 h each day. It must be noted, however, that the controlled hyperthermia method is physically demanding and may not be achievable in untrained individuals. This is because exercise intensity must increase each day of the controlled hyperthermia protocol as heat loss mechanisms become more efficient. Untrained individuals may not be able to sustain these higher exercise intensities during the initial ~30 min when the goal is to elevate core temperature roughly 1.5 °C/2.7 °F to at least 38.5 °C/101.3 °F.

Selecting an appropriate exercise intensity during controlled hyperthermia protocols to raise core body temperature ~1.5 °C/2.7 °F may also prove challenging. We recommend consulting a recent publication as a general starting point in selecting an appropriate exercise intensity. Authors report exercise duration and intensity matrices for above-average fitness males to achieve the requisite rise in core body temperature within 30 min in ~40 °C/104 °F and 40% relative humidity environment [29]. Such data are not available for females or lesser fit males. When exercise is of equal relative intensity, trained females perform as well as men in dry heat [30]. However, when selecting exercise intensity measures not relative to individual fitness (e.g., W kg^{-1}, thermal sensation, velocity, ratings of perceived exertion) we suggest to err on the conservative side by selecting a slightly lower exercise intensity for females and lesser fit males until these data become available.

Whether one heat acclimation protocol is superior to another is a source of contention among researchers and practitioners. Very few studies have directly compared heat acclimation induction protocols. One study showed similar adaptations between the constant workload and controlled hyperthermia methods in a small cohort (n = 8) of males with above-average aerobic fitness ($\dot{V}O_{2max}$ range 45–50 mL kg^{-1} min^{-1}) [31]. Comparing protocols across studies is difficult due to differences in sample population (fitness, gender, and n-size), environmental conditions, and exercise characteristics (intensity, duration, mode). As previously stated,

these considerations characterize the magnitude and duration of the adaptation stimuli (hyperthermia and hypovolemia) and thus degree and time until full acclimation. Furthermore, logistical considerations such as the number of athletes to heat acclimation or available equipment may necessitate one protocol over another.

The traditional recommendation during heat acclimation is to sustain hydration and electrolyte balance to defend against rapid and potentially dangerous increases in core body temperature. On the other hand, permissive or volitional dehydration to the extent obtained in 60–120 min of exercise–heat stress (2–3% body mass loss) in combination with hyperthermia controlled induction methods may optimize the two primary stimuli for heat acclimation (hyperthermia and hypovolemia), maximizing adaptations. Indeed, dehydration exacerbates the physiological strain in several systems and can increase thermal strain by attenuating heat loss effectors. A very recent study investigated whether permissive dehydration altered the induction or decay of heat acclimation and performance. Using a balanced cross-over design implementing isothermal strain (i.e., matching core body temperature during training sessions), these investigators reported that permissive dehydration sufficient to induce a mild, transient hypohydration did not affect the acquisition or decay (by 7 days) of heat acclimation or performance variables, including an increased peak power output following heat acclimation [32]. They further reported that, irrespective of hydration status, the trained subjects required more than 5 days to optimize heat acclimation. Collectively, there are not enough well-controlled studies to determine whether adding planned (volitional) dehydration during the heat acclimation protocol results in a greater or lessened adaptive benefit and performance outcome at the time of this publication [33].

Constant workload and hyperthermia controlled protocols are commonly used in laboratory research or single athlete sports such as running or cycling but present many logistical restraints for use in field studies or team-based sports. For team-based sports, logistical limitations include equipment, physiological monitoring, and control over exercise–heat stress. These shortcomings limit the heat acclimation protocol type to the self-regulated workload techniques for team-based sports or for a large number of athletes at one time (e.g., cross-country team).

Over the past 5 years, several field studies using separate self-regulated heat acclimation protocols have demonstrated that 7–14 days of training camp type exercise in hot environments (29–40 °C/84.2–104 °F, 27–50% relative humidity) following a progressive increase in workload and intensity evoked heat acclimation adaptations [34–36]. Not only were most "classic" heat acclimation adaptations realized, but performance improvements in mild conditions (22–23 °C/71.6–73.4 °F) were also observed (yo-yo run test distances increased by 7–44%). It is noteworthy that these studies employed trained endurance athletes and it is unknown whether less fit or untrained athletes would respond similarly. When properly implemented, a well-designed team-based heat acclimation protocol that gradually increases exercise duration and intensity (and equipment if sport mandated) can induce heat acclimation safely, but this is not always the case. In view of the consistently high rate of exertional heat illness incidence during novel exercise–heat stress in team-based sports [37], the National Athletic Trainers' Association (NATA) with ten other organizations published heat acclimation guidelines [38].

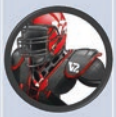
	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
Gear						
Max Practice Time	3 hours	3 hours	3 hours	3 hours	3 hours	5 hours
Contact	None	None	Sleds Dummies	Sleds Dummies	Sleds Dummies	No restrictions

Fig. 3.2 Preseason high school football heat acclimatization guidelines showing a progressive increase in exercise intensity and duration, and equipment [38]. Practice time includes warm-up and cool down. *After 5 days (Friday) through 14 days, two-a-days are allowed but at least 3 h of rest in a cool environment is required between exercise bouts, total practice time cannot exceed 5 h. A two-a-day practice should be followed by a single-practice day or rest day

Figure 3.2 illustrates the current secondary school preseason heat acclimatization guidelines developed from best practices and current evidence [38]. While only 7 days are displayed in the figure, the gradual progression of exercise intensity, duration, and equipment (if applicable) over a 14-day period is recommended to fully induce adaptations. Another model to consider is the preseason heat acclimation guidelines adopted by the Georgia High School Association in 2012. Researchers collected exertional heat illness data over the course of 3 years in interscholastic football in the State of Georgia to develop data-driven guidelines, which resulted in the following guidelines:

Football practice may begin five consecutive weekdays prior to August 1st.

1. In the first 5 days of practice for any student, the practice shall not last longer than 2 h, and the student shall not wear more than shorts, helmet, mouthpiece, and shoes. (NOTE: The time for a session shall be measured from the time the players report to the field until they leave the field.)
2. Beginning August 1st, any student may practice in full pads and may practice two times in single calendar day under the following stipulations:

   (a) A student must have participated in five conditioning practices wearing shorts and helmet before being allowed to practice in full pads.
   (b) If multiple workouts are held in a single day:

   - No single session may last longer than 3 h
   - The total amount of time in the two practices shall not exceed 5 h
   - There must be at least a 3-h time of rest between sessions
   - There may not be consecutive days of two-a-day practices. All double-session days must be followed by a single-session day or a day off [39].

Despite the knowledge of best practices, few high school football programs follow these guidelines. In a cross-sectional study of 1142 high schools in 2011, less than 3% were in compliance with all recommendations [40]. Better adherence and compliance to these guidelines are required to optimize safety during heat acclimation procedures in this population. Several states in the USA have adapted these heat acclimation guidelines into their high school state policy handbooks.

Modification of the heat acclimation protocol may be required and guided by recent illness or injury, or other medical needs. For example, athletes with low fitness levels require a longer acclimation period. In general, athletic teams should follow a 14-day heat acclimation plan to account for the wide range of fitness levels on any given team, thus allowing the least fit athletes to gain all adaptations. Further, equipment laden sports such as American football, lacrosse, and field hockey need additional time to adapt to the uniform and the duration and intensity of practice.

Exposures lost due to injury or illness and designated rest days should not be counted toward the 14-day heat acclimation period. For example, an athlete sitting out preseason practice days 3–6 with an ankle sprain will return to practice on day 7 and resume practice as if they are on day 3 of the heat acclimation protocol. Caution should be exercised here as the exercise intensity and duration and equipment modifications of these returning athletes will differ from their peers. It is critical that strength and conditioning and medical staff acknowledge and understand this difference and implement the proper conditioning program to minimize exertional heat illness risk while inducing heat acclimation.

Endurance Training and Induction Duration

Endurance training evokes a wide array of cardiovascular, pulmonary, and metabolic adaptations that elevate $\dot{V}O_{2max}$. Additionally, during exercise large quantities of endogenous heat is created raising core temperature proportionate to intensity. Consequently, many of the physiological adaptations elicited following high-intensity endurance training are characteristic of the heat acclimated phenotype, suggesting a partial cross-adaptation [13, 26]. Adaptations observed during both exercise in temperate and hot conditions include lower resting and exercising heart rate and core body temperature, expanded blood volume, enhanced sweat rate and sensitivity, and better exercise–heat tolerance [13]. Such parallelism among adaptations has led researchers to suggest that highly trained athletes appear partially, but not fully, heat acclimated [8, 22]. Importantly, it is not the measure of $\dot{V}O_{2max}$ per se that is important when determining the "highly trained status," but recent physical activity which promotes the physiological changes (acquisition of higher cardiorespiratory fitness) and therefore the quasi-acclimated state. Individuals with high $\dot{V}O_{2max}$ and who regularly exercise require fewer exercise–heat exposures to acclimate to the heat. In this population (trained athletes), short-term heat acclimation is generally recommended [21].

Safety Considerations

Exertional heat illness risk is greatest when individuals engage in unaccustomed exercise in warm-to-hot environmental conditions. It is therefore not surprising that the highest incidence of exertional heat illnesses occurs in July and August in the USA during the first several days of fall preseason practice [37, 41]. More specifically, in a 4-year study of NCAA institutions, exertional heat illness incidence rate was greatest during the first 3 days of football practice and gradually declined over the course of the preseason period with a slight spike in the rate at the first two-a-day session on day 7 [37]. It is notable that the risk of exertional heat illness remains elevated until about the time most heat acclimation adaptations are realized (7–14 days).

Several preventative steps should be taken to help minimize exertional heat illness risk before acclimating to the heat. A pre-participation examination is a simple task that can identify predisposing factors that may impede safety when exercising in the heat such as a history of heat illnesses including exertional heat stroke, sickle cell trait, congenital heart diseases, and/or certain medications that affect thermoregulation. By identifying predisposing factors before executing a heat acclimation protocol, the risk of exertional heat illness can be lessened. The NATA recently published a position statement on the pre-participation examination outlining methods for effective, efficient, and systematic implementation based upon scientific evidence and best practice [42].

Factors known to increase exertional heat illness risk while exercising in the heat should also be addressed beforehand (see [43] for review). Briefly, dehydration speeds the rate of heat gain by attenuating sweat rate and skin blood flow heat loss mechanisms. Thus, fluid should be made available before, during, and after heat acclimation exercise bouts. Acclimation progression may be delayed with current illness (especially febrile events) or several days of interrupted sleep or sleep deprivation as these may impair thermoregulatory and exercise efficiency. Finally, and perhaps most importantly, a certified athletic trainer should be onsite during each high-risk practice such as exercising in the heat.

Nutritional Considerations

In non-heat acclimated athletes, sweat contains a relatively high concentration of sodium and other electrolytes. A hallmark of heat acclimation is improved reabsorption of sodium at the superficial section of the sweat gland mediated through aldosterone resulting in more dilute sweat. This adaptation, however, occurs toward the end of the acclimation process. During the first several days of heat acclimation, the sweat produced contains higher amounts of sodium resulting in both dehydration and greater sodium losses. Because sodium is key in sustaining the thirst drive and euhydration, some suggest lightly salting foods during the first 3–6 days of heat acclimation to offset sweat-sodium losses. However, athletes following a Western diet consume approximately 4000 mg of sodium, well above the recommended

1200–1500 mg implicating the mild addition of sodium during the initial days of heat acclimation is not needed. Alternatively, athletes consuming diets low in pre-packaged, highly processed, and preserved foods may benefit from this recommendation.

Environmental Considerations

The principle of specificity states that the human body adapts to specific stressors. Extending this theory to heat acclimation, the type of environmental stress affects the magnitude and maintenance of certain key adaptations. For example, a hot and humid environment during heat acclimation results in a greater sweat rate increase and circulatory adaptations versus hot and dry conditions. Practically, it is highly recommended that the heat acclimation environment emulates the competition environment to enable the athlete to experience the exact nature of the exercise–heat stress and adapt accordingly.

From a safety standpoint, exercising under extreme environmental conditions, even with the intention of acclimating to such conditions, increases the risk of exertional heat illnesses. For example, the risk of an exertional heat illness increased six-fold when the wet-bulb globe temperature (WBGT) reached 82 °F/27.8 °C in a study of intercollegiate football players [33]. Physical activity must therefore be modified during extreme weather days. There are many different WBGT guidelines for activity modification but the American College of Sports Medicine WBGT guidelines are generally accepted for athletic populations. In addition to following these guidelines, other simple methods to reduce heat illness on high heat days are to minimize equipment worn and/or introduce intermittent breaks during practice sessions. This allows for a brief recovery period, reduction in core body temperature, time for hydration and cooling zones through misting, running cold water, cool towels, or similar methods.

Alternative Approaches to Heat Acclimation Induction

Although exercise plus heat exposure induces heat acclimation adaptations, they may not need to be concomitant. Post-exercise hot water immersion has been shown to improve thermoregulation and enhance aerobic performance in the heat. In one study, trained runners ($n = 17$; $\dot{V}O_{2max} = 60.0$ mL/kg/min) completed 6 days of treadmill exercise for 40 min at 65% $\dot{V}O_{2max}$ in 18 °C/64.4 °F followed by either hot (40 °C/104 °F) or thermoneutral (34 °C/93.2 °F) water immersion for 40 min. In the hot water immersion group, not only was post-exercise rectal temperature, skin temperature, and sweat sensitivity lowered after the intervention, but time trial performance in 33 °C/91.4 °F was improved by 5% [44], well above the coefficient of variation for 5-km time trial performance of 2%. The accessibility of this intervention circumvents a number of common limitations to heat acclimation protocols

such as access to environmental chambers, higher prioritized training objectives, and the added stress of exercising in the heat.

The few studies examining the effectiveness of post-exercise sauna bathing to induce thermoregulatory adaptations and aerobic performance gains in the heat have not shown consistent findings. Additional research into post-exercise sauna bathing is required before this modality can be widely recommended in light of the clear thermoregulatory and performance gains with hot water immersion. Theoretically, a greater thermoregulatory stimulus to adapt would be achieved via hot tub versus sauna as greater heat transfer occurs with conduction compared to convection.

When to Induce Heat Acclimation

Determining when to induce heat acclimation adaptions prior to competition in mild to hot conditions is of great importance to coaches and athletes. Recall, heat acclimation adaptations are transient and decay over time (days to weeks) if frequent exercise–heat exposures are not implemented. Of course, the maintenance of these adaptations is logical when practices and competitions both occur in similar environmental conditions. However, when competition occurs in unaccustomed environmental conditions because the athletes practice in cooler environments; the timing of heat acclimation induction must be addressed for utmost application.

Given that cardiovascular adaptations decay within days without exercise–heat exposure and are paramount to the heat acclimation mediated aerobic performance improvements, the current best practice recommendation is to heat acclimate immediately prior to competition. This notion may fit well within the taper phase as exercise volume is generally reduced. When exercising in the heat (35 °C/95 °F, 55% relative humidity), a similar cardiovascular workload can be applied with a 30% reduced mechanical load to the legs versus cool conditions (15 °C/59 °F, 80% relative humidity) [45]. This suggests volume can be reduced and intensity sustained during the heat acclimation-taper phase providing some degree of recovery to the lower extremity prior to competition. Whether the heat acclimation protocol is accomplished prior to traveling to the competition site or at the competition site may be dependent upon travel schedules and available financial and equipment resources at each location. No studies have examined which option would result in optimal performance, but adapting to the specific competition environment is recommended.

Another under-researched strategy in this area is inducing heat acclimation 2–3 weeks prior to competition and incorporating periodic exercise–heat exposures to sustain the adaptations. A recent study demonstrated that post-exercise rectal temperature and heart rate were lower by 0.47 °C/0.85 °F and 28 beats per minutes, respectively, in subjects receiving exercise–heat exposure (120 min, 45% $\dot{V}O_{2max}$) every fifth day 25 days after initial heat acclimation [46]. These results suggest

some of the most important heat acclimation adaptations can be sustained for up to 1 month after initial induction with periodic exercise–heat exposure. Practically, this approach relieves the logistical constraints and physical and mental stress of multiple days of exercise–heat stress to reinduce heat acclimation adaptations immediately prior to competition in the heat.

How Many Days Should I Exercise in the Heat?

It is important for athletes and coaches to consider that the full complement of heat acclimation adaptations may not be realized with short-term heat acclimation protocols (≤7 days). Cardiovascular adaptations are quickly induced but thermoregulatory adaptations such as reduced resting core body temperature and increased sweat rate generally require 8–15 or more days. Practitioners and athletes should consider what outcomes (reduced exertional heat illness risk or improved aerobic performance) and/or specific adaptations are desired when addressing questions of heat acclimation length. So if athletes are acclimating for the purposes of reduced exertional heat illness risk, longer protocols are suggested. For short duration endurance events lasting <90 min, short-term heat acclimation may be sufficient as most heat acclimation adaptations benefiting short duration aerobic performance are actualized within 4–6 days. Long-term protocols would be ideal for endurance events lasting longer than 90 min as larger heat performance decrements are often observed with time-to-exhaustion tests.

Summary

Optimization of heat acclimation adaptations requires personalizing the induction protocol (exercise duration, intensity, number of days) to the athlete's fitness level, competition environment, and adjusting fluid and food intake. The number of athletes, desired adaptations, timing within the training season, and available time and equipment must also be considered. When properly implemented, heat acclimation is a safe prophylactic against exertional heat illnesses and effective ergogenic aid for endurance athletes. The optimal balance between exercise intensity, duration, and frequency in temperate environments to facilitate heat acclimation remains largely unknown.

Case Example *Table 3.3 presents preseason practice sessions for a Central California high school football team to get acclimated in the hot and dry climate (~100 °F/37.8 °C, 20% relative humidity). Both the coaching and the medical staff were in agreement with this practice schedule prior the start of preseason. Additionally, all parties agreed that activity modifications would occur if environ-*

Table 3.3 Practice schedule for preseason high school football following heat acclimatization best practices

	Mon	Tues	Wed	Thur	Fri	Mon	Tues	Wed	Thur	Fri
# of practices per day	1	1	1	1	1	2	1	2	1	2
Total practice time (min)	120	120	180	180	180	240	60	300	180	300
Conditioning specific drills duration (min)	15	20	20	20	25	20	0	20	30	25
Equipment	Helmets only	Helmets only	Helmets only	Shells	Shells	Full gear/ shells	None	Full gear	Full gear	Full gear
Contact	None	None	Sleds/ dummies	Sleds/ dummies	Sleds/ dummies	Full contact	None	Full contact	Full contact	Full contact
Rest breaks per hour	4	4	4	4	4	5	5	5	4	4
Rest break duration	4	4	4	4	4	5	5	4	4	4
WBGT (°F/°C)	81/27.2	82/27.8	79/26.1	80/26.7	83/28.3	89/31.7	92/33.3	86/30	85/29.4	82/27.8

Table 3.4 Ten steps for successful heat acclimation program implementation

1.	Several important precautionary measures should be in place to ensure safe heat acclimation implementation. These include a pre-participation examination, establishing and practicing an emergency action plan, and providing fluid and electrolyte replacement beverages throughout the process
2.	The heat acclimation work rate and environmental (temperature, humidity, and wind) conditions should closely match competition or practice conditions for which the athlete is preparing
3.	Carefully monitoring physiological responses such as heart rate, rectal temperature, and sweat rate throughout the protocol can help determine the time course of adaptation
4.	In team-based sports with a wide range of fitness levels, undergoing a longer heat acclimation protocol is suggested, at least 14 days
5.	The heat acclimation protocol should be determined and agreed upon by the sports medicine team before implementation. A well-planned program includes the progressive increase in exercise intensity, duration, heat exposure, and equipment worn depending on the sport
6.	A certified athletic trainer or other appropriate medical professional trained in the identification and treatment of exertional heat illnesses should be onsite for all competitions and practices. Have emergency equipment onsite for all practices and competitions for the treatment of exertional heat stroke and heat exhaustion
7.	Ensure at least 1 day in the heat for every 4–5 days without heat to minimize heat acclimation adaptation decay
8.	Athletes in team settings who miss heat acclimation sessions due to injury or illness should return to the day of training missed to ensure exercise and environmental stressors match the training state, reducing the risk of exertional heat illnesses
9.	Increase core body temperature to ≥38.5 °C (101.3 °F) for approximately 1 h to sufficiently induce heat adaptations
10.	Introduce rest breaks and intermittent rest during a 2–3 h practice to allow for a reduction in core body temperature, specific periods for rehydration, and use of cold towels or cooling zones to aid in core body temperature regulation

mental conditions worsened. Importantly, the gradual increase in practice duration, conditioning specific drills, equipment, and contact drills over the first 10 days can be observed in Table 3.3. All times are in minutes and no practices occurred over the weekend. Rest and hydration break duration and frequency increased when exercise intensity, duration, or environmental conditions increased.

*Environmental conditions were evaluated before and during practice on the hour with a WBGT device. When indicated (*e.g.*, extreme WBGT values), activity was modified to minimize the risk of exertional heat illness. For example, on the sixth day of practice (second Monday) WBGT was 8 °F/4.4 °C greater, on average, than the previous week equating to a WBGT of 89 °F/31.7 °C. According to the NATA WBGT guidelines, practice time was reduced from a total of 5 h to two separate 2 h bouts; one in the early morning and one in the early evening to avoid the mid-day heat. Due to the lower temperatures, full gear was worn during the morning sessions but only shells were worn in the evening sessions as the WBGT index*

remained near 89 °F/31.7 °C. Due to the oppressive weather on the second Tuesday, practice time was restricted to 1 h with no gear or contact drills and 25 min rest per hour.

The consistently oppressive environmental conditions necessitated employment of several additional precautionary measures to ensure heat acclimation was induced safely and effectively. For every practice, cold water tubs and rectal thermometers were available for the diagnosis and treatment of exertional heat stroke and heat exhaustion. Shade tents were erected on the sidelines for athletes to rehydrate during periods of instruction and rest breaks. When possible, industrial fans circulated air in the tent to aid in sweat evaporation and convective heat loss. The sports medicine staff also instructed athletes to wear light colored clothing. Finally, the head athletic trainer held an educational meeting prior to all practices informing the staff on exertional heat illness recognition, treatment, and prevention.

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Chapter 4
Body Cooling

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Introduction

Body cooling and exercise, with specific foci related to enhancing both performance and improving safety during exercise, particularly in the heat has been well studied within the scientific literature. Investigating the effects of pre-cooling, per-cooling (body cooling during a bout of exercise), and cooling following exercise on performance and safety has resulted in multiple systematic reviews and meta-analyses purporting the benefits of body cooling on exercise performance. This topic is of importance across the vast world of sport and physical activity, particularly during elite competitions taking place in hot environmental conditions.

Events such as the FIFA World Cup and Summer Olympic games, recently occurring during the summer months in cities, such as Athens, Atlanta, Beijing, Rio de Janiero, and upcoming in the cities of Doha and Tokyo, have and will expose

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competitors to extreme heat or situations of warm conditions (i.e., ≅ 30 °C/86 °F or greater) with high relative humidity, which will have the potential to negatively influence the competitor's performance. For example, female runner Paula Radcliffe, one of the front runners competing in the 2004 Olympic Games in Athens, dropped out of the women's marathon where it was cited that the extreme conditions (35 °C/95 °F and 31% relative humidity) may have been the culprit. Conversely, American runner Deena Kastor came from behind to finish third, the first time since 1984 that an American female had medaled in the event. It was noted following the event that Kastor utilized various strategies, including body cooling prior to the event, to assist in optimizing her performance despite brutal conditions.

The goals of this chapter are to discuss the proposed physiological mechanisms related to body cooling on exercise performance and safety risk mitigation related to pre-cooling, per-cooling, and cooling following exercise. Associated with the performance and safety benefits of body cooling in the context of exercise in the heat, we will also focus on the benefits of body cooling on cognitive function based on relationships between cognition and exercise performance.

The Physiology

Maintenance of body temperature at about 37 °C/98.6 °F is tightly controlled by the preoptic anterior hypothalamus within the brain, which sends appropriate efferent signals in response to heat stress related afferent input from the body's thermoreceptors [1]. During exercise, roughly 70–80% of energy that is produced by the working muscles is given off in the form of heat, thus increasing exercising body temperature [2, 3], the magnitude of which is determined by the relative intensity of the physical activity performed. To combat this rise in temperature, the body utilizes the heat loss mechanisms of conduction, convection as well as the evaporation of sweat from the body to dissipate heat. However, it must be acknowledged that these mechanisms can be restricted, eliminated, or reversed depending on the environmental conditions one is exercising in. For example, as ambient temperature exceeds skin temperature (~34–25 °C/93.2–77 °F), there will be a net heat gain within the body, thus requiring the body to rely on the evaporation of sweat from the skin's surface to dissipate body heat. Conversely, in conditions of high relative humidity (i.e., 30 °C/86 °F and 80% relative humidity), heat loss through dry heat exchange will still occur; however, evaporation of sweat from the skin will be restricted due to the high water vapor concentration in the air. In conditions where ambient temperature exceeds skin temperature coupled and relative humidity is high, both dry and evaporative heat losses are restricted, thus reducing the body's ability to dissipate the heat generated during exercise [4].

During exercise, During exercise, it is common for individuals to become hyperthermic (>37 °C/98.6 °F). A body temperature above 38.5 ℃ has demonstrated the potential for premature termination of exercise and can increase the risk and onset of symptoms of exertional heat illness [5–9]. There are two working hypotheses used to explain this response and are termed the critical temperature hypothesis and

anticipatory temperature hypothesis. Both hypotheses suggest that the body employs internal mechanisms to prevent the body from reaching a critical body temperature (40.0–41.0 °C/104.0–105.8 °F) as a protective measure from succumbing to exertional heat illness. With the critical temperature hypothesis, it is suggested that exercise performance will be compromised once body temperature reaches this critical level [10, 11]. The anticipatory temperature hypothesis insinuates that the body detects the rate of heat gain within the body during exercise and adjusts work rate (i.e., exercise intensity) to allow exercise to be completed prior to reaching the critical temperature level [10, 12, 13]. While it is currently unknown what physiological mechanisms are responsible for hyperthermia-related adjustments in exercise intensity, it may be a combination of both scenarios that are involved in protecting the body from exertional heat-related illnesses.

To augment a decline in performance during exercise or competition in the heat and mitigate the risks of exertional heat illness, body cooling methods have been shown to have, in some cases, profound benefits. The premise behind body cooling is that it expands the body's heat storage capacity and/or enhances the extraction of heat from the body, which attenuates the rise in body temperature and allows for an improved period of recovery from exercise heat stress (Table 4.1). The proposed mechanisms related to pre-cooling, per-cooling, and post-exercise cooling are discussed below.

Table 4.1 Definitions and examples of types of body cooling during exercise and competition

Type of body cooling	Definition	Time of cooling	Example sports	Example modalities
Pre-cooling	Body cooling that takes place prior to the start of exercise or competition	A. During warm-up	Running, cycling, football (soccer)	Cooling vests, cold water/ice slurry ingestion
		B. While resting	Tennis, swimming	Cooling vests, cold water/ice slurry ingestion, cold room
Per-cooling	Body cooling taking place during a bout of exercise or during competition	A. During activity	Running, cycling, triathlon	Cooling vests, cold water/ice slurry ingestion
		B. During brief breaks in activity (i.e., halftime or between quarters)	Football (soccer), American football, rugby, lacrosse	Ice-towels, cold water/ice slurry ingestion, misting fans
Post-exercise cooling	Body cooling following the completion of exercise or competition[a]	A. Between bouts of exercise	Tennis, swimming, football (soccer) tournaments	Ice-towels, cold room, cold water/ice slurry ingestion
		B. Following completion of daily exercise	Football (soccer), American football, lacrosse	Cold water immersion

[a]In the case of exertional heat stroke, which is a medical emergency, only cold water immersion (or other modality with known cooling rate >0.15 °C/32.27 °F) should be used to treat the patient

Pre-cooling

Pre-cooling has been extensively studied and has received the greatest attention within the scientific literature where it is purported to lower body temperature prior to exercise in an effort to expand the body's heat storage capacity in order to maintain performance [13–16]. If the method(s), timing, and application of pre-cooling are sufficient, the resulting physiological responses (i.e., reduced thermal and cardiovascular strain) can enhance athletic performance, particularly in the heat [5, 13, 17]. Alternatively, if the pre-cooling strategy is not sufficient to induce a physiological change, there may still be added benefits on exercise performance by altering one's perceived level of exertion, thus indirectly enhancing performance [18]. This may be seen during shorter duration events that are <30–40 min (i.e., 5 km road race, etc.) where the physiological and psychological effects of pre-cooling may be effective.

While it is acknowledged that the decrease in body temperature following pre-cooling increases heat storage capacity and performance, the exact physiological mechanisms remain unclear [10, 19, 20]. Researchers [21–23] have postulated that the inhibition of motor activation within the brain caused by increased levels of hyperthermia is responsible for hyperthermia-related central fatigue. While research investigating the effects of pre-cooling on muscle inhibition and activation are lacking, it is thought that pre-cooling delays the inhibition of motor activation allowing for a greater exercise intensity and improved performance. Furthermore, it is suggested that when implementing pre-cooling strategies prior to exercise, the reduction in body temperature should not exceed 1.5 °C/2.7 °F as this may have detrimental effects on performance due to the decrease in muscle temperature [4]. It must be noted, however, that while pre-cooling may improve exercise performance, it may also have detrimental effects by increasing the risk of exertional heat illness by delaying the activation of the body's physiological heat loss responses to exercise, especially in the heat.

Per-cooling

Similar to pre-cooling, the aim of per-cooling is to reduce thermoregulatory strain during exercise by means of attenuating the rise of body temperature in the heat. Utilizing an effective cooling modality during exercise that mitigates body temperature rise may allow for prolonged exercise prior to reaching one's critical temperature, and thus improve exercise performance in the heat. It is thought that per-cooling may provide benefits during exercise in the heat by providing a source of cooling (i.e., cooling vests, menthol sprays, cold water/ice slurry ingestion) to the body throughout the duration of exercise to facilitate the extraction of body heat, thus enhancing the person's ability to offset increased levels of heat gain resulting from either the environmental conditions or exercise intensity. This may be advantageous

over the utilization of pre-cooling as the benefits of pre-cooling may be short-lived depending on the duration of exercise. For example, during endurance events lasting longer than 60 min, per-cooling strategies may be more effective as the benefits of pre-cooling are negated after 30–40 min of exercise.

Other proposed mechanisms that may be responsible for improved exercise capacity in the heat with the utilization of per-cooling is an increased core-to-skin temperature gradient and cardiovascular/metabolic changes. An increased core-to-skin temperature gradient observed during exercise, especially during uncompensable conditions, preserves the ability of the body to maximize its heat loss potential, thus assisting in keeping body temperature below the critical temperature limit [24]. Conversely, per-cooling may have the potential of having the opposite effects on the thermoregulatory response during exercise. By lowering skin temperature via per-cooling, the physiological mechanisms responsible for heat loss such as the onset of sweating may be delayed, thus unintendedly causing a more rapid rise in body temperature.

In conjunction, body cooling during exercise can assist in mitigating the metabolic [25] and cardiovascular [26] strain that occurs with exercise-induced hyperthermia. The maintenance of central blood volume leading to a decrease in exercising heart rate and increased stroke volume following cooling during exercise in the heat can optimize performance by increasing blood flow and oxygen delivery to the working muscles [10, 24]. Per-cooling may also be effective for improving exercise performance in the heat by attenuating muscle glucose utilization [13, 27] and preventing heat-stress reductions in lactate threshold [28].

Lastly, it must not be discounted that psychophysiological mechanisms may also contribute to exercise performance in the heat and the role of which body cooling may enhance performance. Body cooling, independent of pre- or per-cooling, may reduce perceptual feelings of thermal strain without inducing physiological changes of a reduced body temperature [18]. Recent research [29, 30] investigating the use of a menthol spray has shown that perceptual signals may outweigh physiological signals to increase exercise intensity for improved performance. However, changing thermal perception to improve exercise performance in the heat may increase the risk of exertional heat illness due to an increased rate of metabolic heat production without a perceptual recognition of the change in the body's physiological state and should be approached with caution, especially with inexperienced and/or at risk populations.

Post-exercise Cooling

Body cooling following exercise has been considered a method to enhance recovery between bouts of exercise. While scientific literature on the physiological mechanisms by which post-exercise cooling affects exercise recovery and subsequent performance remain equivocal, it is postulated that post-exercise cooling enhances

exercise recovery by altering the inflammatory, cardiovascular, and thermoregulatory responses to exercise [24].

It is proposed that body cooling following exercise may reduce muscle soreness and increase muscle force generating capacity for subsequent workouts by reducing reactive oxygen species generation and the cellular, lymphatic, and capillary permeability responsible for muscle fiber edema following exercise [24, 31, 32]. Furthermore, post-exercise cooling has been shown to decrease the inflammatory response, which decreases levels of pain and results in a smaller reduction in muscle force generating capacity [33].

Quickly reducing body temperature following exercise, which is often done using cold water immersion (CWI), can have a multitude of effects on the cardiovascular and thermoregulatory systems. With post-exercise cooling such as CWI, the rapid reduction in skin temperature results in faster recovery of central blood volume via peripheral vasoconstriction, which aids in the body's ability to clear metabolic waste and may enhance recovery [24]. Furthermore, the reduction in body temperature following exercise may facilitate the reduction in sensory and motor nerve conduction velocity to decrease perceptions of pain, decrease muscle metabolism to reduce levels of muscle soreness, and return body temperature to baseline at a faster rate. The latter may be more important in situations of repeated bouts of physical activity in the heat as it allows an individual's body temperature to return to baseline levels, especially when repeated bouts of exercise are in close proximity to one another (i.e., 1 h) [34–36].

Cognitive Function

Despite predominant research focusing on physical performance in the heat, many prolonged exercise events require maintenance of cognitive function for performance (i.e., decision making) and/or health purposes (i.e., injury risk). Cognitive functioning is empirically conceived as the potential of remaining lucid during exercise (e.g., adjust to a direct opponent, update an inadequate pacing strategy) but more appropriately refers to (sub)cortical processes that sub-serve the ability to repeatedly and efficiently regulate behavior according to one goal (e.g., working memory, mental set shifting) [37]. While the challenge of optimal self-regulation has been underlined during prolonged exercise in temperate environments, the addition of heat-related strain makes it even more pronounced [37], then suggesting a role for protective cooling maneuvers.

To capture the interactions between the magnitude of body temperature elevation and cognitive performance, an inverted U-shaped curve has recently been proposed [38]. Specifically, hyperthermia initiation (up to ~38.2 °C/100.8 °F) seems to improve a variety of cognitive task performances (simple and choice reaction time, vigilance, psychomotor, and working memory tests). This enhancement in cognitive function, though counterintuitive, could relate to an increased cerebral meta-

bolic ratio of oxygen, thermoregulatory control mechanism-derived arousal changes in regional cerebral blood flow and/or in blood–brain barrier permeability to circulating brain-derived neurotrophic factors. Of interest, this observation emphasizes that all cooling interventions could not be ergogenic for cognitive performance in hot conditions. In particular, procedures aimed at reducing body temperature (e.g., internal cooling, combined methods, CWI prior to and at the beginning of an exercise bout) may be less advisable, since this would inhibit metabolic heat production and therefore place cognitive performance at a non-optimal level. In contrast, a reduction in skin temperature concomitant to a rise of body temperature (e.g., warming-up with an ice vest) may represent a useful strategy to maximize cognitive performance in the heat—especially since an increase in skin temperature per se has the potential to impair cognitive response due to altered cortical activity [39].

When body temperature rises up to ~38.5 ± 0.2 °C/101.3 ± 0.4 °F, the positive effects of hyperthermia on cognition dissipate although changes in brain functioning remain noticed (i.e., increased activity of frontal and temporal brain areas)—a compensating phenomenon thought to counteract heat-related constraints [40]. However, beyond this threshold, both the magnitude of heat strain and cognitive task complexity seem to interact to predict cognitive efficiency; such that the more stressful the combination of hyperthermia and cognitive demands, the larger the cognitive impairment. Correspondingly, as body temperature increases, memory and vigilance performance as measured by dual tasks, tracking, and simple reaction time declines. Explanatory mechanisms for this progressive impairment may rely on the inhibitory influence of subcortical brain regions, which in response to body heat load, act to preserve perceived effort by reducing the command towards the prefrontal cortex. In this perspective, any strategies that would delay or prevent the drift of body temperature toward excessive hyperthermia (i.e., greater than ~39.0 °C/102.2 °F) could preserve optimal cognitive processing alongside physical performance.

The Evidence

Within scientific literature, there are myriad examples supporting the use of body cooling to optimize performance and enhance safety by reducing the risk of heat-related injuries. Examination of the types of cooling modalities, timing of cooling (pre-, per-cooling, and post-exercise), the length of time needed for cooling, and specific sport/physical activity application (i.e., endurance, high-intensity interval exercise, or sprint exercise) have been analyzed to provide clinicians, coaches, and athletes strong evidence supporting the benefits of cooling on performance [10, 13, 19, 20, 24, 41–45]. Similar to the previous section, this section will expand on the evidence of pre-cooling, per-cooling, and post-exercise cooling on exercise performance and reducing the risk of heat-related illness.

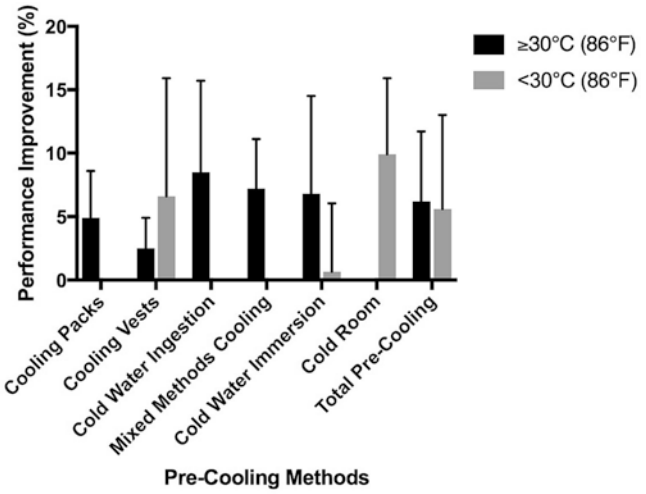

Fig. 4.1 Effects of pre-cooling on exercise performance from different cooling modalities and when exercise is performed in temperate/moderate (< 30ºC) and hot (≥ 30°C) environmental conditions. Total pre-cooling refers to the performance improvement when all pre-cooling modalities are averaged together. Data was adapted from work by Bongers et al. [43] and Wegmann et al. [20]

Pre-cooling

Researchers have examined the influence of various pre-cooling methods on endurance performance, sprint performance, intermittent sprint performance, performance in moderate and hot environmental temperatures [20, 43–45]. Bongers et al. [43] and Wegmann et al. [20] found that pre-cooling enhanced exercise performance in the heat by as much as 5.7% and 4.9%, respectively. Additionally, when mixed methods (multiple cooling methods used at the same time) were employed, the combination of cold water immersion and cold water ingestion was the most effective method to improve performance prior to exercise in the heat (Fig. 4.1). Similarly, pre-cooling has also been shown to be effective during exercise in moderate/temperate conditions; however, this evidence is sparse and the effects on exercise performance (i.e., time to exhaustion, time to completion) is mixed [20].

Duration of exercise, in the context of endurance and intermittent sprint exercise has been shown to exhibit greater benefits of pre-cooling versus short duration sprint exercise [20, 24]. The latter may be due to evidence showing that cooler muscles have decreased power output, increased time-to-peak tension, and reduced rate of anaerobic metabolism [27, 46, 47]. A review by Ranalli et al. [45] found that the performance benefits of pre-cooling on endurance exercise (4.25%) versus anaerobic (sprint) exercise (0.65%), providing further evidence that pre-cooling has greater influences when implemented prior to prolonged exercise in the heat. However, some have speculated that the ergogenic benefits of pre-cooling are limited to 30–40 minutes, and exercise lasting longer than 60 min does not benefit [20].

Along with enhancing performance in the heat, pre-cooling may also be an effective strategy to reduce the risk of exertional heat illness. With the premise of pre-cooling expanding heat storage capacity and/or enhancing heat extraction (cooling modality dependent), a larger window is created from pre-exercise body temperature to potential pathologic levels (>40 °C/104 °F). For example, normal body temperature is ~37 °C/98.6 °F and if a bout of pre-cooling reduces body temperature an additional 0.5 °C/0.9 °F prior to the start of exercise, the exercising individual now has ~3.5 °C/6.3 °F before reaching levels that may put them at risk for exertional heat illness. However, the limitations associated with pre-cooling from a thermoregulatory perspective must not be negated. By increasing heat storage capacity or enhancing heat extraction, the activation of heat loss responses during exercise (i.e., evaporation of sweat from the skin) are delayed, thus minimizing any benefits of pre-cooling during exercise in the heat as the largest heat gains occur during the first 15–20 min of exercise. Nevertheless, evidence supports pre-cooling techniques that provide aggressive whole-body cooling for both safety and performance during exercise in the heat.

Per-cooling

A limitation of using pre-cooling as a strategy to enhance safety and performance during exercise is the ability to cool only prior to the start of exercise. If the bout of cooling is not sufficient to effectively lower body temperature, or if the event is longer than 30–40 min in duration where any effects of pre-cooling are negated, an individual may be at risk of exertional heat illness or be at risk of suffering a degradation of exercise performance. In these scenarios, per-cooling has been considered to be effective.

Recent research investigating the effects of per-cooling on exercise performance has found performance benefits in 15 of 21 studies that were examined in recent meta-analyses [43, 44, 48]. Furthermore, as a whole, per-cooling may afford up to an 8.4–9.9% improvement in exercise performance, which may depend on type of exercise (i.e., time trial or time to exhaustion), the environmental conditions in which the exercise is taking place and type of cooling modality used (Fig. 4.2) [24, 43]. The most effective methods of per-cooling include cold water ingestion, cooling/ice vests, and the utilization of menthol (both orally and topically); however. Due to the limited number of studies examining this and the total number of subjects tested, future research is needed to identify the most effective per-cooling strategy [24]. In general, cooling strategies are beneficial during conditions (i.e., exercising in the heat [~40 °C/104 °F]) that adversely affect the body's physiological capacity to dissipate heat. With an increase in metabolic heat production (increased exercise intensity) during exercise coupled with an environmental heat load that decreases the body's ability to dissipate heat, the added heat load may exceed an athlete's ability to dissipate body heat, thus causing detrimental effects on performance and safety.

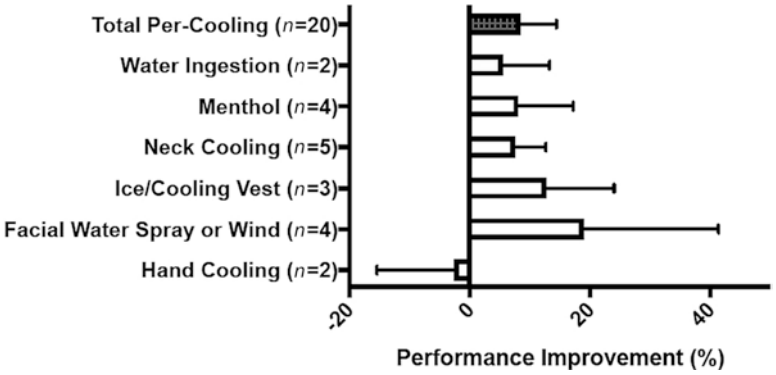

Fig. 4.2 Effects of various methods of per-cooling on exercise performance. The number associated with each cooling modality reflects the number of studies examining that particular method with exercise performance. The data shown is adapted from work by Bongers et al. [24]

Coupled with evidence demonstrating improvements in exercise performance, per-cooling may also provide an advantage above pre-cooling from a health and safety perspective. Cooling during a bout of exercise may attenuate the rise of body temperature, increase heat storage capacity, and improve heat loss potential during exercise; however, it is dependent upon the cooling technique used and the cooling modality's cooling potential. Prior literature [24, 43] has shown that although there were no observable reductions in body temperature with per-cooling during exercise when compared to a non-cooling condition (environmental temperatures ≥30 °C/86 °F), there was no observable improvement in performance, which may be due to the redistribution of blood between tissue compartments. Per-cooling may have been able to increase heat loss potential during exercise and reduce thermal strain, thus allowing individuals to exercise at a higher intensity (improve performance) without an additional rise in body temperature.

Post-exercise Cooling

Body cooling has been used for years to augment post-exercise recovery, with CWI being the most frequently used. A recent meta-analysis [49] and two Cochrane reviews [50, 51] have examined the effects of post-exercise cooling on recovery, finding that post-exercise cooling has the greatest benefits on subjective ratings of recovery. Specifically, symptoms of delayed-onset muscle soreness are reduced 24, 48, and 96 h following post-exercise recovery with CWI being the most effective method of cooling [49]. Post-exercise cooling was also found to reduce ratings of perceived exertion following exercise; however, these benefits were constrained to within just the 24 h following exercise [49].

The evidence on the effects of post-exercise cooling on objective measures of recovery following exercise is equivocal. Post-exercise cooling has been shown to have no effects on recovery outcome measures such as blood lactate, creatine kinase, and c-reactive protein concentrations, nor does post-exercise cooling improve maximal strength or power output following exercise [49, 50]. Furthermore, post-exercise cooling does not appear to influence the inflammatory response to exercise, even up to 96 h following exercise [50]. Contrary to these findings, recent reports show that post-exercise cooling utilizing whole-body cryotherapy ranging from −110 to −195 °C/166 to −319 °F improved maximal strength and decreased the inflammatory response; however, these effects have not been shown to be consistent, thus prompting future research to examine the effects of post-exercise cooling on recovery following exercise [51, 52].

While the effects of post-exercise cooling on exercise recovery and subsequent performance requires further research to determine physiological benefits, body cooling following exercise has profound clinical applications. Exertional heat stroke (EHS), a medical emergency, requires immediate and aggressive whole-body cooling within 30 min of collapse to ensure survival [53–55]. CWI has been shown to be the gold standard method of treatment of EHS due to the large convective cooling capacity of water [56]. Other cooling methods have also been shown to be effective in lowering body temperature below the critical threshold (~40.0–41.0 °C/104.0–105.8 °F) in humans, with tarp-assisted cooling having cooling rates well above the acceptable rates [57, 58]. Independent of specific cooling modality, the cooling rate to optimize treatment of EHS should exceed 0.15 °C min^{-1}/0.27 °F min^{-1} to expedite cooling and reduce the risk of morbidity and mortality [59].

Body Cooling on Cognition

Although limited to date, outcomes in the scientific literature investigating cognitive responses to cooling maneuvers in hot conditions appear varied. For instance, applying head/neck cooling with cold packs or a collar to hyperthermic participants was found effective for negating heat-related impairments on information processing speed, working memory, and executive control (i.e., resolving conflicting situations) [60, 61]. While functional, these local cooling strategies do not appear to offer any reduction in body temperature, which—due to their cooling-related inhibition of autonomic thermoregulatory processes—may only make them relevant as short-term interventions. Alternatively, pre-cooling using CWI (20 °C/68 °F for 60 min) or a cooling tent (~25 °C/77 °F) plus buckets with ice water was also shown to improve performance of hyperthermic participants in most (but not all) indices of cognitive functioning in a battery of psychomotor tasks [62, 63]. Together, the findings from the heat-cognition literature do provide support for cooling strategies to protect cognitive function. However, their enhancing effect does not appear

systematic and seems to depend on three main determinants: the individual's body temperature (and thus timing of cooling implementation), the type of cooling procedure, and the complexity of the cognitive task to perform.

As stated earlier, one of the key mechanisms through which body cooling is ergogenic for endurance performance—and for cognitive functioning—is the increase in the core-to-skin gradient of temperature. Importantly, hydration and heat-acclimation scheduling are well-known strategies to augment this gradient, especially through allowing an increased sweat rate. With this in mind, we believe these interventions should be viewed as complement, effective cooling maneuvers for optimal cognitive performance in the heat [64, 65].

The Application

There are many strategies available to athletes that are purported to improve performance and simultaneously, enhance safety. The feasibility of these may depend on cost, timing, and considerations for drying off (if water immersion is used) after utilization. An ideal cooling modality for performance would help maintain body temperature, reduce skin temperature, be portable, and allow complete function during exercise in the heat. In this section, we discuss various practical applications for use of cooling devices, or strategies, before and during exercise in the heat for the main purpose of performance enhancement.

Pre-cooling

Since the early 1990s, athletes have been using various strategies to reduce body temperature prior to activity, pre-cooling. When considering pre-cooling, athletes must consider evidence and feasibility for their sport. As stated above, the most effective way to reduce body temperature is to utilize CWI. However, in equipment-laden sports, for example, the limitation of utilizing this strategy is that the athlete has to dry off, put on equipment and uniform, and then still participate in pre-event activities. Also, by the time the game begins, the benefits of reducing body temperature prior to the event, may be absent. In this case, consuming an ice slurry pre-event offers some of the benefits of body temperature reductions without the impairment of timing. Furthermore, with the evidence supporting the use of multiple methods of pre-cooling (i.e., ingesting an ice slurry drink and wearing a cooling vest), it is recommended that this may optimize safety and performance during exercise in the heat for events lasting up to 45 min.

Per-cooling

Cooling during activity, or per-cooling, is another tactic aimed at improving safety and performance. Some sports (tennis, American football) offer many breaks where a myriad of strategies can be utilized. Others, however, such as soccer and rugby, present limitations of no intermittent rest breaks where body cooling can be achieved during activity. Therefore, in these sports, the cooling plan would have to include clothing worn by the athlete that does not impair performance in addition to maximizing cooling opportunities during the halftime periods. Other sport settings (i.e., cycling and running) may allow for the use of both pre- and per-cooling strategies and should be considered to optimize performance outcomes. A final consideration surrounding the utilization of per-cooling during exercise is the ability for the source of cooling to provide an effective amount of cooling throughout the duration of exercise as this may be difficult without an energy source, especially during prolonged events in the heat.

Post-exercise Cooling

Athletes have been cooling post-activity to accentuate recovery, reduce delayed-onset muscle soreness, or for perceived benefits of limiting inflammation. Despite limited evidence in support of CWI for many of these perceived benefits, many utilize this tactic following team sport, or intense activities. The benefit of cooling post-activity is that there are no limitations on timing, getting the athlete wet, preparation for activity afterwards, etc. Since the workout has already been completed, CWI has potential to augment performance advantages from the workout in preparation for the next training session or competition.

Complexities Surrounding Body Cooling

Specific recommendations for individual performance characteristics of device feasibility for myriad sports appear in Tables 4.2 and 4.3. The considerations for each sport are separated so what may be feasible for basketball pre-cooling, per-cooling, and post-exercise cooling can be regarded individually. Considerations include athlete wettedness, timing, uniform/equipment potential, and the ability to perform the modality. Athletes and coaches should consider all of these in preparation for an event. In addition, the athlete, coach, strength and conditioning personnel, and sports medicine team should be cognizant of the most feasible cooling modality specific to the sport that is supported by scientific evidence when making decisions on body cooling during exercise in the heat.

Table 4.2 Summary of individual sport characteristics in terms of competition rules and regulations [66]

		Game type	Time duration (min)	Halftime duration	Substitution considerations	Equipment considerations
American Football	Professional	Quarters	15	12	Unlimited	Helmet
	Collegiate	Quarters	15	20	Unlimited	Shoulder pad
	High school	Quarters	12	20	Unlimited	
Soccer	World cup	Halfs	45	15	Max. 3; No re-entry	Shin guard
	Professional	Halfs	45	15	Max. 3; No re-entry	
	Collegiate	Halfs	45	15	No re-entry	
	High school	Halfs	40	10	Unlimited	
Basketball	Professional	Quarters	12	15	Unlimited	None
	Collegiate	Halfs	20	15	Unlimited	
	High school	Quarters	8	10	Unlimited	
Baseball	Professional	Innings	9	Variable	No re-entry	Helmet, chest protector, shin guards (catcher)
	Collegiate	Innings	9	Variable[a]	No re-entry	
	High school	Innings	7	Variable[a]	No re-entry	
Softball	Professional	Innings	7	Variable[a]	No re-entry	Helmet, chest protector, shin guards (catcher)
	Collegiate	Innings	7	Variable[a]	No re-entry	
	High school	Innings	7	Variable[a]	No re-entry	
Decathalon	Olympic	10 events	2 days	N/A	N/A	None
	Collegiate	10 events	2 days	N/A	N/A	
	High school	10 events	2 days	N/A	N/A	

Heptathalon	Olympic	7 events	2 days	N/A	N/A	None
	Collegiate	7 events	2 days	N/A	N/A	
	High school	7 events	2 days	N/A	N/A	
Running	100 K	Race	62.1 miles	N/A	N/A	None
	Marathon	Race	26.2 miles	N/A	N/A	
	Half Marathon	Race	13.1 miles	N/A	N/A	
	10 K	Race	6.2 miles	N/A	N/A	
Cycling	Professional	Race	Dependent on the race	N/A	N/A	None
	Collegiate	Race	Dependent on the race	N/A	N/A	
	Amateur	Race	Dependent on the race	N/A	N/A	
Triathlon	Full ironman	Race	140.6 miles	N/A	N/A	None
	Half ironman	Race	70.3 miles	N/A	N/A	
	Olympic	Race	31.93 miles	N/A	N/A	
	Sprint	Race	16 miles	N/A	N/A	
Tennis	Professional	Game/set/match	Best of 3 sets		N/A	None
	Majors	Game/set/match	Best of 5 sets		N/A	
	Collegiate	Game/set/match	Best of 3 sets		N/A	
	High school	Game/set/match	Best of 3 sets		N/A	
Motor Racing	NASCAR	Race	Race dependent	N/A	N/A	Helmet
	Indy car	Race	Race dependent	N/A	N/A	Flame retardant clothes

(continued)

Table 4.2 (continued)

		Game type	Time duration (min)	Halftime duration	Substitution considerations	Equipment considerations
Lacrosse (Men's)	Professional	Quarters	15	12	Unlimited	Helmet
	Collegiate	Quarters	15	10	Unlimited	Shoulder pad
	High school	Quarters	12	10	Unlimited	Chest protector
Lacrosse (Women's)	Professional	Halfs	30	10	Unlimited	Eye guard
	Collegiate	Halfs	30	10	Unlimited	Chest protector (goalie)
	High school	Halfs	25	10	Unlimited	
Ice Hockey	Professional	3 periods	20	17[b]	Unlimited	Helmet, chest protector, thigh/shin/elbow pads, gloves
	Collegiate	3 periods	20	12 or 15[b]	Unlimited	
	High school	3 periods	15 to 17	15 to 20[b]	Unlimited	
Rowing (eight)	Olympic	Race	2000 m	N/A	N/A	None
	Collegiate	Race	2000 m	N/A	N/A	
	High school	Race	2000 m	N/A	N/A	
Rugby	Professional	H	40	10	Max. 7; no re-entry	Scrum cap
	Collegiate	H	40	10	Max. 7; no re-entry	
	High school	H	30 to 35	10	No re-entry in the first half	
Wrestling	Olympic	3 periods	3	N/A	N/A	None
	Collegiate	3 periods	3–2–2[c]	N/A	N/A	
	High school	3 periods	2	N/A	N/A	

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[a]The variable duration of the halftime in baseball and softball refers to the variation in time that the offensive team has on the bench and their opportunity to cool during this time

[b]Refers to the intermission time between the 1st and 3rd periods in ice hockey

[c]Refers to the breaks in minutes between the 1st, 2nd, and 3rd periods, respectively [66]

Table 4.3 Optimal cooling modalities and times of implementation across sport competition [66]

Sport	Cooling opportunity	Cold water immersion (whole body)	Cold water immersion (partial body)	Water dowsing	Ice towel	Ice bag	Misting fan	Head cooling device	Hand cooling device	Ice/ cooling vest	Cooling garment	Hydration (15–21 °C)	Slushy drink
American Football	Pre-game		▲	∆	△	△	△		△		△	●	▲
	Offensive/ defensive sides			△	△		△		△		△	●	△
	Halftime		△		△	△		△	△		△	●	△
	Post-game	●	●	△	△	△		△	△		△	●	△
Soccer	Pre-game	▲	●	○	△	△		△	△	▲	△	●	▲
	Halftime	●	●	○	△	△		△	△	△	△	●	△
	Post-game	●	●	○	△	△		△	△	△	△	●	△
Basketball	Pre-game	●	●		△	△		△	△	▲	△	●	▲
	Substitutions				△	△					△	●	△
	Halftime		○		△	△		△	△	△	△	●	△
	Post-game	●	●	○	△	△		△	△	△	△	●	△
Baseball/ Softball	Pre-game		▲		△	△	△	△	△	▲	△	●	▲
	Between innings (while batting)		△		△	△	△	△	△		△	●	△
	Post-game	●	●	△	△	△	△	△	△	△	△	●	△
Running (competitive)	Pre-race	▲	●	○	△	△	△	△	△	●	△	●	▲
	During race			△							△	●	△
	Post-race	●	●	○	△	△	△	△	△	△	△	●	△
Cycling (competitive)	Pre-race	▲	●	○	△	△	△	△	△	●	△	●	▲
	During race										△	●	△
	Post-race	●	●	○	△	△	△	△	△	△	△	●	△

(continued)

Table 4.3 (continued)

Sport	Cooling opportunity	Cold water immersion (whole body)	Cold water immersion (partial body)	Water dowsing	Ice towel	Ice bag	Misting fan	Head cooling device	Hand cooling device	Ice/ cooling vest	Cooling garment	Hydration (15–21 °C)	Slushy drink
Triathlon	Pre-race											●	▲
	During bike										△	●	△
	During run			△							△	●	△
	Transitions											●	△
	Post-race	●	●	○	△	△		△	△	△	△	●	△
Tennis	Pre-match	▲	●	○	△	△	△	△	△	▲	△	●	▲
	Between games				△	△					△	●	△
	Between sets				△	△					△	●	△
	Post-match	●	●	○	△	△	△	△	△	△	△	●	△
Motor vehicle racing	Pre-race				△				△		△	●	▲
	During race										△		
	Post-race	●	●	△	△	△		△	△	△	△	●	△
Lacrosse				△	△	△							
	Pre-game		▲	△	△	△	△	△	△	▲	△	●	▲
	Substitutions			△	△		△				△	●	△
	Halftime		△		△	△		△	△		△	●	△
	Post-game	●	●	△	△	△		△	△	△	△	●	△
Ice Hockey	Pre-game				△	△					△	●	▲
	Substitutions										△	●	△
	Intermissions				△	△		△	△		△	●	△
	Post-game	●	●	△	△	△		△	△	△	△	●	△

Rowing	Pre-race	▲	●	○	△	△		△	△	▲	△	●	▲
	During race										△	●	△
	Post-race	●	●	○	△	△		△	△	△	△	●	△
Rugby	Pre-match	▲	●	○	△	△		△	△	▲	△	●	▲
	Halftime	○	○	○	△	△		△	△	△	△	●	△
	Post-match	●	●	○	△	△		△	△	△	△	●	△
Wrestling	Pre-match	▲	●	○	△	△		△	△	▲		●	▲
	Post-match	●	●	○	△	△		△	△	△		●	△

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● is identified as the optimal cooling modality with scientific evidence to support its use. ▲ is identified as a feasible cooling modality to use that is based on scientific evidence. ○ and △ are optimal and feasible modalities to use, respectively; however, its use in improving performance is not scientifically based. Blank cells indicate cooling modality is not feasible for the respective sport and time period [66]

It is also recommended that athletes attempt a similar protocol during training that they plan to utilize on the day of competition in order to acclimatize to the modality and activity following, or with, application of specific strategies. This will allow for ideal preparedness and mindset on game day. For example, if a marathon runner does not respond well to the use of CWI during training for pre-cooling, they should not utilize this for race day, or they should alter the temperature for increased comfort and performance. Another strategy may be that they instead opt to utilize a cooling vest and consume an ice slurry pre-event.

Case Example *An elite marathoner is training for an upcoming race where environmental conditions are expected to exceed 32 °C/89.6 °F. The runner strategizes to use various methods of body cooling before, during, and following the race to optimize performance and recovery.*

- *During training, the runner rehearses and refines the three phases of body cooling (before, during, and following activity) to identify his/her optimal race day strategy.*
- *Prior to the start of the event the runner plans to utilize multiple pre-cooling methods (wearing a cooling/ice vest and consuming ice-cold beverages) during an abbreviated warm-up to limit the rise of body temperature prior to the start of the race.*
- *During the race, the runner plans on consuming ice-cold fluids during the race to minimize fluid losses to optimize hydration status to ensure that sweating, thus evaporative heat losses are not impaired. Also the runner plans to utilize the benefits of the temperature of the beverage in attenuating any rise in body temperature following the period in which the benefits of pre-cooling are negated.*
- *Following finishing the race and following the runners cool down, the runner utilizes CWI to aid in their post-race recovery.*

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Chapter 5
Hydration

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The Physiology

Although the average 70 kg (154 lb) human has 42 L (~11 gal) of total body water, a loss of only ~700 mL (24 fl. oz.) can compromise exercise performance. Maintaining optimal water balance (i.e., euhydration) is essential for everyone but especially important for those performing exercise. Hypohydration and dehydration are often used interchangeably because they have similar physiological consequences, but they have different definitions. Hypohydration is the state of a body water content deficit; dehydration refers to the loss of body water [1]. Rehydration is the process of going from a hypohydrated to a euhydrated (Fig. 5.1).

About 2/3 of total body water resides within our cells; the other 1/3 is extracellular. Extracellular fluid includes fluid that is in the interstitium (i.e., between cells) and plasma. The movement of fluid between the intra- and extracellular compartments is controlled via several mechanisms that are beyond the scope of this chapter [3]. Total body water balance is challenged throughout the day because water losses to the environment occur via transdermal evaporation, feces, respiration,

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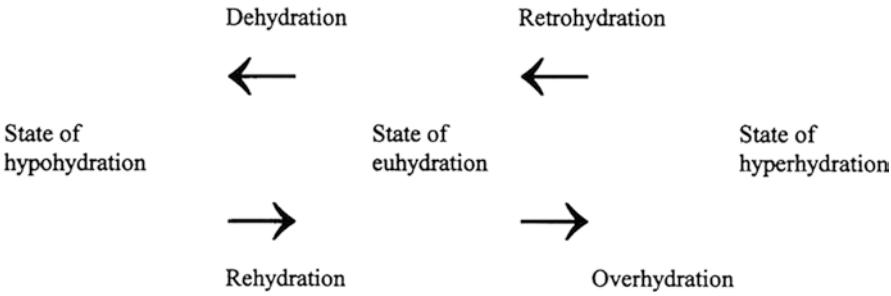

Fig. 5.1 Clarification of terms to describe body water losses and gains during exercise [2]. Euhydration: steady-state condition of normal body water. Hypohydration: steady-state condition of decreased body water. Hyperhydration: steady-state condition of increased body water. Dehydration: water loss leading to hypohydration. Retrohydration: water loss from a state of hyperhydration leading to euhydration. Rehydration: adding water from a state of hypohydration to move toward euhydration. Overhydration: fluid intake that exceeds euhydration, leading to hyperhydration. Reprinted from Casa DJ. Exercise in the Heat. I. Fundamentals of Thermal Physiology, Performance Implications, and Dehydration. J Athl Train. 1999;34(3):246–52

urine, and sweat [4]. The human brain has an amazing ability to detect body water changes and to respond accordingly. Specifically, plasma osmolality (i.e., concentration of the watery part of blood) is closely monitored, and a multitude of physiological consequences occur when it varies more than 1–2% (i.e., ~6 mmol/kg) [5]. When elevations of plasma osmolality occur, as with dehydration, the hypothalamus releases arginine vasopressin (i.e., AVP), also referred to as antidiuretic hormone, from the posterior pituitary. AVP acts on V2 receptors in the kidney, increasing the reabsorption of water such that urine production is reduced and body water is conserved [5, 6]. By reducing water loss, the concentration of urine increases, making it a useful measure of hydration status (discussed below) [7]. This ability to concentrate urine is very effective in defending body water such that individuals can drink a wide range of fluids throughout the day and maintain a relatively stable plasma osmolality [8].

The largest water loss from the body during physical activity is sweating. The evaporation of sweat is an extremely effective mechanism to keep body temperature from reaching dangerously high levels. As skin and internal body temperature increase, afferent nerves communicate directly with the preoptic anterior hypothalamus to initiate a coordinated series of events that release heat to the environment [9]. Sympathetic cholinergic nerves innervate the eccrine glands in the skin, release acetylcholine, and stimulate the release of sweat [9]. As isotonic sweat travels into the secretory and reabsorptive ducts, ions are reabsorbed such that expulsed fluid is hypotonic [10]. This process leads to a reduction of plasma, which in turn reduces total body water (termed “hypovolemia”). Sweat loss also leads to more concentrated blood (termed “hyperosmolality”). Thus, standard exercise in the heat leads to hyperosmotic hypovolemia.

Sodium is the most prominent ion in sweat, but sweat also contains calcium, iron, magnesium, zinc, copper, and potassium [11]. Because high sodium levels in

Table 5.1 Mean ± SD or mean (range) whole-body sweating rates of adults and youth during various sport activities

		Mean ± SD (Range) Absolute whole-body sweating rate (L/h)
Adults	American Football	1.7 ± 0.8
	Baseball	1.0 ± 0.3
	Basketball	1.0 ± 0.4
	Half-marathon running	1.49 (0.75–2.23)
	Rowing	1.98 (0.99–2.92)
	Soccer	0.8 ± 0.4
	Swimming	0.37
	Tennis	1.6 ± 1.1
Youth	American Football	0.78 ± 0.35
	Hockey	1.8 ± 0.1

Data from references [1, 16–18]

sweat have been associated with muscle cramping, it is important to understand what affects these levels [12]. Differences *between* individuals' sweat sodium concentrations can be partially explained by genetics [13]. However, within an individual, several factors can affect sweat sodium concentration. For example, heat acclimatization consistently leads to reduced sweat sodium concentrations [14]. Quantifying sweat sodium concentrations are beyond the scope of this chapter, but the reader is directed to an excellent publication outlining the procedure [15]. Knowing your sweat sodium concentration is important because it may have implications for the choice of hydration beverages (see below).

Given the importance of sweat production for body temperature control, it is no surprise that magnitude of sweat evaporation (i.e., sweat rate) is directly correlated to mean body temperature (i.e., the calculated aggregate of skin and rectal/internal temperature). Internal body temperature will increase dramatically during intense exercise, especially if environmental heat stress is great. Therefore, different sports and sport settings lead to a large variety of body temperature, and thus sweat rates (Table 5.1) [1, 16–18]. See below for instructions on how to calculate sweat rate. Intrinsic factors such as sex, age, training status, and heat acclimatization status can also influence sweat rate and should be considered when developing a hydration plan.

One physiological reason for maintaining euhydration (i.e., optimal water balance) is to support the cardiovascular system. In order to maintain adequate blood flow to the skeletal-muscular and thermoregulatory systems during exercise, the cardiovascular system increases cardiac output. Cardiac output is the rate of blood flow leaving the heart each minute and is mathematically calculated as the product of stroke volume (i.e., amount of blood pumped out of the heart with each beat) times heart rate. Increasing cardiac output is critical to performing exercise in the face of rising internal temperature. With dehydration, cardiac output is affected because there is "less blood to go around," which increases cardiovascular strain. This is evidenced by the physiological phenomenon called "cardiovascular drift,"

defined as the gradual increase in heart rate and decrease in stroke volume that occurs after ~15 min of steady-state aerobic exercise. This occurs even as work output (i.e., running or cycling pace) remains constant. Cardiovascular drift represents strain on the cardiovascular system, is exacerbated with dehydration, and can almost be completely prevented with proper hydration [19]. This is important because cardiovascular drift during exercise can reduce maximal oxygen uptake (i.e., $\dot{V}O_{2max}$) [19] and has implications for exercise training [20]. In periods of severe dehydration, and hence large degrees of cardiovascular drift, the increase of heart rate does not compensate for the large decrease of stroke volume. Thus, cardiac output is not maintained and can lead to decreased blood pressure. Decreased blood pressure may lead to exercise cession and/or syncope. This cascade of events is often referred to as cardiovascular collapse and is the hallmark of heat exhaustion (discussed in Chap. 17).

In the face of dehydration, there is competition for blood flow between the musculoskeletal and thermoregulatory systems. If thermoregulation is prioritized, more blood is directed to the skin and blood flow to contracting muscles may be compromised. This leads to either decreased exercise intensity or cessation of exercise. On the other hand, if blood flow to the skeletal muscle is prioritized, less skin blood flow occurs and thermoregulation is impaired. In reality, neither system is completely prioritized over the other. Dehydration affects both of these systems, so it is no surprise that exercise performance in the heat is compromised when an individual is dehydrated (see below).

The exact effects of dehydration on skeletal muscle are less understood, primarily because of methodological difficulties in assessing blood flow and substrate usage in specific muscle groups. However, we know that dehydration leads to reductions in skeletal muscle blood flow, increased lactate production, and increased muscle glycogen utilization [21]. Also, increased muscle glycogen utilization, as a result of dehydration, can lead to premature fatigue, particularly in long-duration events such as a marathon road race.

Dehydration impairs the body's ability to adequately increase skin blood flow and reduces sweat rate. It has been repeatedly shown that these impairments lead to increased internal temperature [22]. The link between dehydration and body temperature is further emphasized by the fact that *degree of dehydration* directly correlates with the degree of hyperthermia (i.e., elevated internal temperature) that occurs [22]. As discussed below and in Chap. 17, hyperthermia decreases exercise performance and increases one's risk for exertional heat illnesses.

At the other extreme, excess fluid ingestion can also be dangerous and can lead to exertional hyponatremia, which occurs during exercise when fluid is consumed and retained in excessive amounts. It is most prevalent in long-duration exercise when an individual drinks substantially more than he/she loses in sweat, thus gaining body mass [1]. Specifically, the greater the increase in body mass that occurs in an individual during prolonged exercise, the greater the risk of hyponatremia (Table 5.2). Untreated dilutional hyponatremia may lead to cerebral edema, encephalopathy, pulmonary edema, respiratory arrest, and even death [1]. These symptoms may develop during exercise or several hours after exercise is discontinued. Although

Table 5.2 Reducing the risk of exertional hyponatremia

1.	During exercise, attempt to lose no more than 1.5% of the pre-exercise weight. A body weight gain during an endurance event should be treated as fluid excess and as a signal to cease drinking until a minor body weight deficit (−1% to −1.5%) exists [24].
2.	During exercise, fluids containing Na^+ maintain plasma Na^+ slightly better than pure water. However, consuming a large volume of a fluid-electrolyte replacement beverage (5–10 L of a hypotonic Na^+ solution) may induce exertional hyponatremia [25]. Ultraendurance competitors may find it beneficial to take brief rest periods, to consume Na^+-rich beverages and foods (e.g., low-fat soup or stew with crackers), in an attempt to maintain normal plasma Na^+.
3.	Before and after exercise, consuming food and fluids that contain Na^+ slightly offset Na^+ losses in sweat and urine.
4.	Know the signs and symptoms of exertional hyponatremia.
5.	Limit use of nonsteroidal anti-inflammatory drugs because they may impair urine production and cause fluid retention [26, 27].
6.	Exercise in a hot environment (heat acclimatization for at least 8 days, 60–90 min/day) reduces the amount of Na^+ lost in sweat and urine. It also increases sweat rate and an athlete's hourly fluid requirement. This means that heat acclimatization enhances Na^+ retention, decreases the risk of exertional hyponatremia, and defends the whole-body Na^+ level, but slightly increases the risk of dehydration.

Reprinted from [23]. Armstrong LE, Casa DJ, Watson G. Exertional hyponatremia. Current Sports Medicine Reports, Vol. 5/No. 5; pages 221–222, © 2006, http://journals.lww.com/acsm-csmr/Citation/2006/10000/Exertional_Hyponatremia.1.aspx, with permission from Wolters Kluwer Health, Inc

hyponatremia is rather rare (approximate incidence of 1 in 1000 individuals), it could have deadly consequences. It is important to understand the signs, symptoms, and risks associated with this disorder, which are discussed elsewhere [1]. Simple strategies can be implemented to dramatically decrease the likelihood of exertional hyponatremia (Table 5.2) [23].

The Evidence

The Effect of Dehydration on Endurance Exercise

Many times sports scientists are able to verify, via sophisticated experimentation, observations that athletes and coaches have made for years. The effects of dehydration on exercise performance fall within this same category. It was back in 1912 during the Stockholm Olympic games when the Portuguese marathon runner Francisco Lázaro was attempting to prevent dehydration from decreasing his performance in such an important event. He was fast but he had observed that his excessive sweating during exercise had a negative impact on his exercise performance. His plan was to decrease sweating by applying a thick layer of grease on his skin, thus preventing dehydration. His trick worked. He did not get dehydrated, but collapsed from hyperthermia and died from complications related to heat stroke.

Since then, several studies have shown that dehydration has a detrimental impact on endurance-related events like running and cycling. One of the first studies by Armstrong and colleagues investigated the effect of diuretic-induced dehydration when running 1.5, 5, and 10 km on an outdoor track in a mild environment [28]. They reported that running speed declined 3.1–6.7% as a response to dehydration. Montain and Coyle examined the effect of different rates of dehydration as a response to differing amounts of fluid intake, during 2 h of cycling exercise at 62–67% of maximal oxygen consumption in a warm environment [22]. They reported a decline of stroke volume as well as an increase of heart rate and esophageal temperature, proportional to the degree of dehydration. In other words, during exercise in the heat, as dehydration increases, so does exercise induced body temperature and heart rate, coupled with lower stroke volume. In 1997, González-Alonso and his colleagues examined the separate effects of hyperthermia and dehydration that usually appear at the same time during exercise in the heat [29]. They observed that both dehydration and hyperthermia strain the cardiovascular system. However, when dehydration and hyperthermia were superimposed during exercise in the heat, athletes were less able to cope with hyperthermia due to lower cardiac output and blood pressure. Dugas et al. studied six male cyclists who performed six 80-km cycling time trials [30]. Subjects replaced 0%, 33%, 66%, or 100% of fluids based on their weight loss during an ad libitum trial (i.e., they could drink at will). These scientists grouped the 0 and 33% trials as LOW and the 66 and 100% trials as HIGH, fluid intake and observed that low vs. high fluid intake induced hypohydration of −3.75% and −1.5%, respectively. The greater hypohydration was linked to lower power output, slower exercise performance time, but similar internal body temperature. In a running study, Gigou et al. investigated the effect of pre-exercise hyperhydration on performance [31]. The control and experimental trial resulted in dehydration of −3.1% vs. −1.4% of body mass, respectively. Even though no differences were found in 80–90 min running time trial performance, the greater hypohydration induced greater cardiovascular and thermoregulatory strain, as indicated by elevated heart rate and rectal temperature.

It is important to point out that these laboratory findings have been replicated and expanded in realistic field-setting studies [32, 33]. In a series of trail runs, subjects running at a fixed pace while hypohydrated (~3% body mass loss) experienced increased heart rate, rectal temperature and greater perceived exertion [32]. When the same subjects, during a separate trial, ran at maximal exertion, hypohydration led to much slower race times [32]. A follow-up study examined running at a "moderate pace" (i.e., submaximal) while using a heart rate monitor to keep heart rate at the same rate, a practice similar to many running enthusiasts [33]. When subjects began and remained hypohydrated over 12 km, they experienced elevated internal body temperatures and slower completion times. This occurred because, in order to keep the same heart rate between trials, they had to run at a slower pace. Despite the slower pace, deep body temperature was elevated. These studies highlight the cardiovascular and thermoregulatory strain that hypohydration places on the body in a field environment. A review article examining laboratory-based and field-based

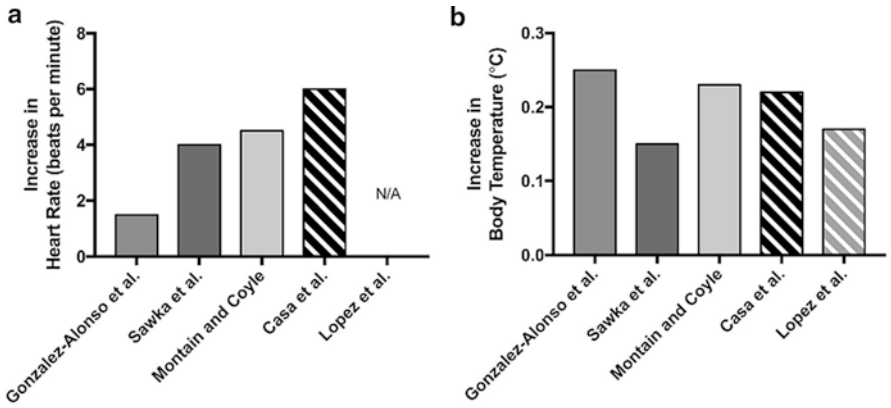

Fig. 5.2 A comparison of laboratory (Gonzalez-Alonso et al. [29], Sawka et al. [35], Montain and Coyle [22]) and field (Casa et al. [32], Lopez et al. [33]) studies examining increases in heart rate (**a**) and body temperature (**b**) for each additional 1% body mass loss. Adapted from Lopez et al. [33]

studies concluded that each 1% body mass loss due to hypohydration led to an increase of 3 bpm in heart rate [34]. Similarly, it has been shown that deep internal temperature is elevated ~0.2 °C for every 1% body mass loss (Fig. 5.2) [33].

Even though we know, from years of research, that high skin temperature can increase sweating independently from internal body temperature [36], the effect of skin temperature on exercise performance was not studied until recently. A study conducted at the U.S. Army Research Institute of Environmental Medicine examined whether dehydration of 4% impaired exercise performance in 10, 20, 30, and 40 °C/50, 68, 86, and 104 °F environments [37]. Indeed, heat stress exacerbated the dehydration-induced decline in performance and, when skin temperature was greater than 29 °C/84.2 °F, performance declined by 1.6% for every 1 °C/1.8 °F increase of skin temperature.

A recent comprehensive literature review examined the effect of dehydration on endurance performance [5]. The authors reported that 68% of the 60 studies showed performance impairment with dehydration >2% of body weight. Also, the position statement of the American College of Sports Medicine (ACSM) regarding exercise and fluid replacement suggests the goal of drinking during exercise is to prevent dehydration greater than 2% of body weight [1]. This statement acknowledges that dehydration influences exercise performance negatively.

Strength and Power Performance

The effect of dehydration on strength is not as well studied as endurance exercise. The literature review by Cheuvront and colleagues examined 276 separate observations stemming from 43 studies [5]. Significant impairment of performance was

observed in 20% (54 out of 276) with dehydration >2% of body weight. These data indicate that the effect of dehydration on strength and power is less clear than in endurance exercise. Judelson et al. critically reviewed all published studies up to 2007 and reported that several methodological differences between studies could explain this variability [38]. They concluded that a 3–4% body weight loss decreased strength by ~2% and power by ~3% after accounting for these methodological factors. In a recent meta-analysis, Savoie and his colleagues summarized the effect of hypohydration on strength and power [39]. They stated that "Hypohydration, or factors associated with dehydration, are likely to be associated with practically important decrements in muscle endurance, strength, and anaerobic power and capacity." Interestingly, these authors also suggested that dehydration of approximately 3% of body weight may improve performance in body-weight dependent tasks such as vertical jumping ability; however, several other studies refute this observation [38].

Mild Dehydration

Even though most data and the ACSM position stand support the notion that dehydration greater than 2% of body weight impairs exercise performance, some data indicate that even mild dehydration of 1–2% body weight loss might also have a detrimental effect on exercise performance. One of the first studies examined the effect of mild dehydration on six male cyclists during a high-intensity performance test [40]. The authors found that time to exhaustion was 30% shorter during high-intensity exercise (90% $\dot{V}O_{2peak}$), even at a low (−1.8% body weight loss) level of dehydration. They suggested that "fluid ingestion during exercise should be designed to fully replace sweat and urine losses." Recently, the effect of mild hypohydration on skeletal muscle metabolism was examined in nine females during a 2 h cycling protocol at 65% of their $\dot{V}O_{2max}$ [41]. The publication reported that progressive dehydration greater than 1% of body weight lead to increased muscle glycogen breakdown as well as increased cardiovascular and thermoregulatory strain, when compared to a euhydrated trial. In a different laboratory study, the effect of 1% hypohydration was examined during a 30-km simulated cycling interval course in the heat [42]. Ten experienced cyclists exercised either euhydrated or hypohydrated (−1% of body mass) while drinking water to maintain initial hydration status. The cyclists generated higher power output during the euhydrated trial with lower body temperature and greater sweating sensitivity. Similarly, the effect of mild hypohydration was examined during a short, maximal effort 5-km outdoor climbing trial in the heat [43]. The cyclists completed the 5-km cycling trial 5.8% slower when hypohydrated by 1%, with greater body temperature and lower sweating rate.

Even though the majority of the data indicate dehydration impairs exercise performance, some scientists argue that dehydration might have a beneficial

effect. In a study during a marathon race in France, Zouhal et al. examined the relationship between athletic performance and dehydration, as expressed by body weight loss during the race in 643 runners [44]. Body weight loss was inversely associated with the finishing time; however, dehydration explained only 4.7% of the finishing time variability. The authors concluded that their data are not in agreement with the laboratory-based findings which suggest that dehydration impairs performance. More recently, some authors have suggested that the dehydration-induced decline in exercise performance results from lack of subject blindness to the experimental design, sensation of thirst, and low air flow especially in cycling studies [31, 37]. For example, ten cyclists completed a 25-km time trial in the heat at various levels of hydration (−3, −2, and 0% of their body weight). In order to blind the participants, hydration state was manipulated by intravenous saline infusion during exercise. No differences were observed in exercise performance despite a higher rectal temperature in the −3% experiment [45]. In another study, Cheung and colleagues examined the effect of dehydration and thirst during a 20-km cycling time trial in the heat [46]. The subjects performed four separate trials in euhydrated and hypohydrated conditions, with or without the presence of thirst. Thirst was manipulated by rinsing the mouth with water during exercise. No differences in exercise performance were observed and they concluded that neither dehydration up to 3% body mass nor perception of thirst, separately or combined, impacted submaximal exercise performance in the heat, for healthy and fit individuals. Interestingly, during the dehydrated but non-thirst trial, subjects' rectal temperature was significantly elevated compared to the euhydrated trial.

Involuntary Dehydration During Exercise

Fluid intake and the sensation of thirst seem to be altered during exercise [47]. Exercising athletes tend to drink less fluid than they lose via sweating, even when they have free access to water or sports drinks. In 1944, Pitts and his colleagues studied drinking behavior during exercise in the heat [48]. They concluded that drinking during exercise rarely exceeded two-thirds of the net water losses due to sweating. Later, this phenomenon was described as involuntary dehydration and defined as "the delay in rehydration by spontaneous drinking after dehydration induced by exercise, fluid restriction, environmental heat and cold" [49]. This phenomenon has been named voluntary dehydration by other authors [4, 50]. In 2007, Passe et al. examined the fluid intake of 18 men and women during an 8 mile run in a 20 °C/68 °F environment [50]. Even though the runners exercised under ideal favorable conditions, with sports drinks available every 2 miles, subjects replaced only 30% of their fluid losses.

The Application

Developing a Personalized Drinking Plan for Exercise

Endurance sports, team sports, high altitude expeditions, military activities, and industrial labor often result in mild-to-severe dehydration. These activities vary greatly in their duration, environmental conditions, fluid availability, terrain and exercise intensity [51]. Also, each person has a unique sweat rate, heat acclimatization status, total fluid intake, and urine production. This variety suggests that a single recommendation for rehydration is not appropriate for all athletes, laborers, and military personnel; this also supports an individualized drinking plan that is specific to each activity [43, 44].

Developing a personalized drinking plan requires 1 h and a bathroom floor scale, to determine sweat rate (liters/hour, L/h). Simply follow these steps [24]:

1. Perform warm-up exercise to the point that you begin to produce sweat.
2. Urinate and void the bowel, if necessary.
3. Weigh yourself naked (kg) on an accurate floor scale (sensitive to 0.1 kg).
4. Exercise for 1 h, simulating your next competition (pace, terrain, ambient conditions, clothing).
5. Measure the amount of fluid (ml) that you consume during exercise, if any (oz × 30 = mL; 1 mL = 1 g).
6. Do not urinate during exercise.
7. Weigh yourself naked, after exercise (kg).

Next, calculate sweat rate by performing a few simple math calculations:

(a) Subtract your final body weight (#7) from your initial body weight (#3) in kilograms (lb/2.2 = kg).
(b) Convert the milliliters of ingested fluid to grams (1 mL = 1 g) and *add* this weight to the body weight difference (calculation a).
(c) If you urinated during exercise (step 6), convert the volume to grams (1 mL = 1 g) and *subtract* this weight from the body weight difference (calculation a).
(d) Corrected sweat rate = a + b − c.

This final value describes the *maximal amount* that you should drink (L/h) during exercise, following the guidelines of the ACSM [1] and the National Athletic Trainers Association [52]. These organizations recommend that (1) fluid intake during exercise should approximate water losses in sweat and urine; (2) a body weight loss of more than 2% should be avoided, to avert compromised endurance performance (see above); and (3) weight gain should be avoided. However, calculated sweat rate has value only if it closely simulates an upcoming event. This exercise should be conducted with air temperature, humidity, clothing, and exercise intensity/mode that are similar to the anticipated competitive environment. Also, sweat rate (calculation d above) may increase after 10–14 days of heat exposure (i.e., heat acclimatization), requiring a greater fluid

intake for a similar bout of exercise. When these factors change, you should reassess your sweat rate and rehydration plan.

Although some authorities recommend drinking only when thirsty, this method is vigorously debated [53]. We recommend "drinking to thirst" only as a secondary strategy, in the absence of an individualized rehydration plan, because thirst is a subjective sensation which varies greatly between individuals, and is influenced by many internal and external factors [54]. For example, our research team [51] reported that thirst was not statistically correlated to total fluid intake, height, body weight, change of body weight, body water balance, or ground speed during a 160-km ultra-endurance cycling event. But, we know that "drinking to thirst" will result in a 1–2% body weight deficit because thirst is not sensed until this level of dehydration exists. We also know that thirst is extinguished by drinking even a small volume of water [1]. Further, no conclusive research evidence explains how different individuals interpret the term "drink to thirst," or how it is accomplished. In our experience [45, 47], the concept is vague and may be easily misinterpreted by athletes (e.g., Should I drink so that thirst is always absent? Should I drink only when my senses indicate that I am thirsty?). Simply stated, "drinking to thirst" means many things to many people.

Daily Water Intake Recommendations

Adequate 24-h fluid intake ensures that water losses in sweat and urine, during athletic and occupational activities, are met and that hydration status is optimal. The Adequate Intake of water (AI) represents a recommended average daily water intake that maintains good health for men and women. Thus, the European Food Safety Authority [55], the World Health Organization [56], and the Institute of Medicine, National Academy of Sciences, USA [57] have published AI in terms of 24-h total fluid intake (plain water + moisture in beverages + moisture in solid foods); the data in column 4 of Table 5.3 [58, 59] also are expressed in terms of 24-h total fluid intake from all sources. The volume of liquids (i.e., not including water in solid foods) that meets these AI are 1.6–2.2 L/day for women, and 2.0–3.0 L/day for men; these volumes can be attained by drinking plain water or beverages. It is important to note that AI do not account for the large sweat losses that are often experienced by endurance athletes, military personnel, and laborers [42, 47, 49], but that self-assessment of hydration status each morning estimates the impact of large sweat losses.

Self-Assessment of Hydration Status During Daily Living

Upon waking in the morning, and during the hours following a bout of prolonged exercise, it is prudent to determine your hydration status, to guide water and beverage intake, throughout the next 24 h, so that you enter your next training session

Table 5.3 Categories of hydration variables for healthy, young women and men

Hydration categories	Percentile range	24-h fluid intake (mL)[a]	24-h urine volume (mL)[b]	Urine specific gravity[b]	Urine color[b]
	Women (59.6 kg)				
	1–10	>3407	>2070	<1.008	<3
Increasing hydration	11–25	2946–3407	1828–2070	1.008–1.011	3
	26–40	2507–2945	1240–1827	1.012–1.015	4
Euhydrated	41–60	2109–2506	951–1239	1.016–1.020	5
	61–75	1745–2108	831–950	1.021–1.024	5
Increasing dehydration	76–90	1507–1744	531–830	1.024–1.026	6
	91–100	<1507	<531	>1.026	>6
	Men (75.1 kg)				
	1–10	>3261	>2250	<1.012	<4
Increasing hydration	11–25	2615–3261	1898–2250	1.012–1.014	4
	26–40	2454–2614	1526–1897	1.015–1.017	5
Euhydrated	41–60	2049–2453	1226–1525	1.018–1.020	5
	61–75	2009–2048	1075–1225	1.021–1.024	6
Increasing dehydration	76–90	1382–2008	875–1074	1.025–1.027	6
	91–100	<1382	<875	>1.027	>6

Adapted from [58, 59]

[a]Moisture in solid foods accounted for approximately 20% of total fluid intake (plain water + moisture in beverages + moisture in solid foods)

[b]Measured in a 24-h urine collection

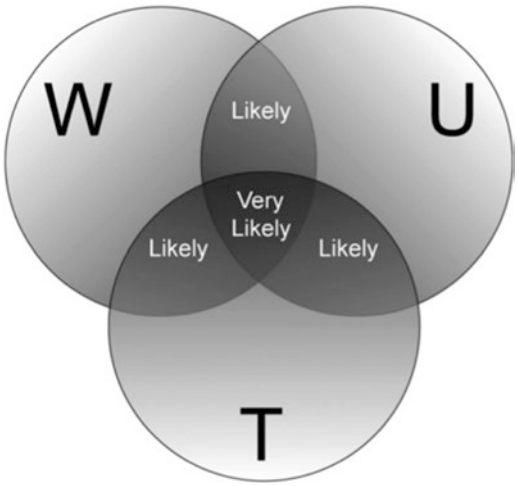

Fig. 5.3 Venn diagram showing the likelihood of dehydration related to certain conditions [61]. Reprinted from Cheuvront S, Sawka M. Hydration Assessment of Athletes. Sport Sci Exch. 2005;18:1–6. *Abbreviations*: *W* body weight, *U* urine color, *T* thirst

optimally hydrated. However, at least 13 hydration assessment methods exist [7, 60] and no single technique is valid in all situations and for all individuals [7]. Some methods require great technical expertise (e.g., stable isotope dilution, neutron activation analysis) and others require laboratory instruments (e.g., plasma volume change, plasma and urine osmolality, bioelectrical impedance) [7]. Further, urine specific gravity requires a handheld light refractometer, and measuring urine volume requires the cumbersome collection of all urine samples throughout a day; thus, most adults will not routinely employ these methods. For everyday use, athletes and laborers want hydration biomarkers that are simple, quick, accurate, safe, and inexpensive [6, 51]. In most field settings and during daily activities, these requirements are met by thirst, body weight change, and urine color.

The fundamental question that underlies hydration assessment is, "Am I drinking adequately (but not excessively) to offset water losses in sweat and urine?". In response to this question, the Venn diagram in Fig. 5.3 [61] allows any healthy adult to evaluate the adequacy of day-to-day water intake and real-time hydration status [62]. This figure represents the principle that hydration assessment should compare information from two or more indices, more than once each day [7]. This self-assessment requires 3–5 min, a bathroom floor scale, and a validated urine color chart; it is most accurate when done in the morning, immediately after you wake. First, consider body weight [62]. If you weigh yourself using an accurate digital floor scale (±0.1 kg) on consecutive days, you can determine your baseline body weight within a few days. If you experience a body weight decrease of 1 lb (0.5 kg) or more, we recommend that you drink additional fluids, above your normal daily intake. Second, urine color is sensitive to dehydration and is responsive to changes of fluid intake [63]. When you are dehydrated, urine appears dark yellow or tan. An 8-category urine color chart is available at hydrationcheck.com. Third, thirst is initially sensed when you are dehydrated by 1–2% of body weight, and becomes more

intense as dehydration increases. The first morning measurement of thirst, in particular, is a strong predictor of dehydration [64]. As illustrated in Fig. 5.3 [61], no single marker provides reliable evidence of adequate fluid intake [7]. When two of these factors are identified, it is **likely** that you are dehydrated. When all three factors exist, it is **very likely** that you are dehydrated [62].

Urine color also allows you to determine when you have consumed *too much* water or beverages [64]. As shown in Table 5.3, well-hydrated women have an average urine color that is pale yellow or yellow, and well-hydrated men exhibit a urine color that is yellow or tan-yellow. However, in 290 urine samples collected from men across 12 days [58], less than 5% had a urine color of 2 (pale yellow), and no samples had a urine color of 1 (clear). Simply stated, very dilute urine samples are rare in men [58] and women [59], comprising only 3–5% of all specimens. Thus, two to four consecutive urine samples that appear pale yellow or clear (i.e., indicating that the body is overhydrated and the kidneys are releasing excess water) indicate overdrinking in most instances.

Replacing Sodium and Other Electrolytes

Human sweat contains over 40 compounds [65]. The most relevant substances for prolonged exercise are the minerals sodium, chloride, potassium, and magnesium, all of which are essential to optimal health and performance. Loss of these electrolytes in sweat is offset by diet, in most athletic and occupational situations. A whole-body electrolyte imbalance most often occurs during prolonged exercise (>4 h), especially when repeated across multiple days [1]. For example, during summer American football twice-daily workout sessions or Ironman triathlon competitions, a loss of 5–10 L of sweat is not uncommon during 5–16 h of exercise. A sweat concentration of 40 mEq sodium/L results in a total loss of 200–400 mEq (4598–9196 mg) of sodium, which exceeds the average daily American intake of 3400 mg of sodium [66] and greatly exceeds the contents of one 12 oz. (360 mL) bottle of sports drink (79–165 mg sodium, depending on the product). Thus, if an athlete's diet does not provide adequate electrolytes and water, the risk of salt-deficiency heat exhaustion and exertional heat cramps increases (see Chap. 17). To regain whole-body salt balance after a large sweat loss (e.g., 5–10 L sweat per day), we recommend that athletes and laborers increase their dietary salt intake, especially if they have a history of exertional heat cramps, or notice white salt deposits on a shirt or cap after prolonged exercise. The following foods contain a high salt content [67]: bacon, center cut, 3 slices (5409 mg sodium); onion soup, dry mix, 1 packet (3132 mg sodium); table salt, 1 teaspoon (2563 mg); tomato sauce, canned, 1 cup (1284 mg); breakfast sausage biscuits, fast food (1080 mg); macaroni and cheese, 1 cup (720 mg); and 2 hot dogs, packaged (713 mg).

Case Example *A unique laboratory-based case report provides some important lessons about hydration during exercise heat-stress [68]. In this case report K.G. was provided a standardized diet with carefully controlled sodium intake for the 7 days leading up to heat exposure (137 mEq Na^+/day). On the day of heat exposure, K.G. alternated 30 min of treadmill exercise (5.6 km/h, 5% grade; mild exercise) and 30 min of rest in 41 °C/105.8 °F and 21% relative humidity for a goal exposure time of 8 h. Participants were instructed to drink pure flavored water ad libitum (i.e., as much and as often as desired).*

During the first 5 h of exposure, K.G.'s body mass substantially increased due to water consumption and retention. At the end of hour 5, he was instructed to curtail drinking. In the 3 h after heat exposure, K.G. complained of nausea and increasing malaise despite no drinking or eating. He was then transferred to a hospital and diagnosed with dilutional hyponatremia due to a serum Na^+ value of 122 mEq/L. Subsequent medical attention resolved the condition such that K.G. was released from the hospital the next day.

Interestingly, the other nine participants in the same research study had no difficulties or complications throughout testing. During this laboratory study, nearly all variables were controlled and/or measured, including diet. The only apparent difference in the actions of K.G. was that he voluntarily consumed twice as much fluid as the other participants. Importantly, his fluid consumption, prior to being told to curtail intake, was 1.9 L/h. His sweat rate was only 0.75 L/h. This 2.5-fold overconsumption of fluid was the likely cause of his hyponatremia.

Hyponatremia can also be related to an imbalance between sodium intake and sodium losses. If sodium loss via sweating occurs at a high rate without adequate sodium intake, hyponatremia may occur. Thus, "salty sweaters" are often advised to increase sodium intake. However in this case example, K.G. had similar sodium intake and losses (via sweat and urine) as the control subjects who did not develop hyponatremia. It should be noted that even if K.G. had supplemental sodium intake during exercise, it would unlikely decrease his chances of developing hyponatremia. Copious fluid intake (i.e., at a rate greater than the sweat rate) can be dangerous. Upon interview, K.G. indicated that his fluid intake actions were driven by the belief that drinking water copiously would "decrease his risk for heat illness."

This case example emphasizes the need for all athletes to measure their sweat rate (described above) and to ensure that the rate of fluid intake during exercise does not exceed sweat rate. It is particularly important that athletes develop *personalized* hydration plans for long-duration events (e.g., >2–3 h). In an effort to "screen for hyponatremia," some ultradistance events require athletes to perform body mass check-ins along the course. Substantial increases in body mass indicate that fluid retention has occurred and that fluid intake should be curtailed. Hyponatremia can be diagnosed and treated by trained medical personnel but, if not recognized, can lead to death; prevention is safer and simpler. Thus, athletes should make fluid and diet preparations part of their training plan.

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Chapter 6
Work-to-Rest Ratio

Riana R. Pryor, David Hostler, Earl R. Cooper, and Andrew J. Grundstein

The Physiology

Core body temperature (T_c) must remain within a relatively tight range (3.3 °C/6 °F) from resting temperature to ensure an individual's health. Exercise, especially in a hot and/or humid environment, is a stressor that can rapidly increase T_c to unsafe levels. The risk of developing an exertional heat illness (EHI) increases as T_c rises, with a high likelihood of exertional heat stroke occurring once body temperature reaches 40.5 °C/105 °F. Work-to-rest ratios (WRRs) can be implemented to mitigate the risk of developing a potentially harmful T_c by decreasing heat gain during exercise and increasing heat loss during rest.

The amount of body heat storage can be calculated using the heat-balance equation by determining heat gain and heat loss [1]:

$$S = M\left(\pm work\right) - E \pm R \pm C \pm K$$

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where S is the amount of stored heat, M represents the metabolic heat produced, E is the amount of evaporative heat lost through sweating, R is the radiant heat that is gained/lost, C is the convective heat gained/lost, and K is the conductive heat gained/lost from the environment. When the body produces a greater amount of heat than it can transfer to the environment or when T_c is lower than ambient temperature, the residual is stored and T_c will rise. Metabolic heat (M) produced through exercise (work) can only contribute to total body heat gain, not heat loss. Exercise can increase one's metabolism 15–20 times the resting rate, greatly contributing to stored heat due to the body losing approximately 80% of its metabolic energy as heat [2]. As exercise intensity increases, so does the heat gain from the skeletal muscle contractions, especially when activity is conducted in a hot environment. Additionally, heat is gained rapidly when the intensity and duration of exercise increases or protective equipment is worn.

Athletes who exercise in a hot environment outdoors during the daytime also gain heat through radiative energy (R) from the sun. Exercising under direct solar radiation greatly increases heat gained compared to exercising in a shaded location or indoors. Even small sheltering measures such as wearing a hat while exercising in the sun can decrease the direct radiative energy gained from the environment.

Strategies to reduce this heat gain during exercise are often centered on a hydration plan and heat acclimatization; however, determining proper WRRs can also be an effective tool to assist with this dilemma. The body will continue to gain heat until exercise intensity is decreased or a rest break is taken. Therefore, when exercise is warranted under hot environmental conditions, strategically increasing the number and duration of rest breaks and taking breaks in a cool or shaded environment, while decreasing the intensity and/or duration of exercise can dramatically mitigate total body heat gain. These strategies can also assist with heat loss.

Heat loss during exercise can occur in a variety of ways, with evaporation (E) being the predominant method. As T_c rises, blood flow to the skin also increases to promote sweating. When sweating commences, heat is transferred from the body to the environment as sweat changes from a liquid form to a gaseous form via evaporation. However, when the environment does not allow for sweat evaporation under humid condition, its ability to cool down the body is limited. Conduction (C) and convection (K) can contribute to either heat gain or heat loss, depending upon the circumstances. Conduction refers to the transfer of heat to or from the body via physical contact with an object such as an athletic surface or chair. Heat gain will occur when a surface is warmer than the body. This is commonly seen in hot environments such as an athlete's hand in contact with a hot athletic playing surface. Conversely, if the object is cooler than the body such as a cold bench in contact with an individual's legs, the body will transfer heat to the object, aiding heat loss. Convection refers to the transfer of heat between the body and fluid, such as liquid and air flow. If someone uses a body cooling modality involving cold water immersion after exercising in a hot environment, body heat will be transferred to the water via convection.

Metabolic heat production can increase significantly during intense physical activity in hot environmental conditions. To best mitigate rising T_c, efforts should be

Table 6.1 Performance and thermoregulatory benefits of rest breaks [3]

Benefits of rest periods during exercise		
	Improve performance	Reduce hyperthermia
Reduce metabolic heat production	↑	↑↑
Dissipate accumulated heat gain	↑↑	↑↑
Clear metabolic by-products	↑↑	↔
Food consumption	↑	↔
Fluid consumption	↑	↑
Cooling modalities	↑	↑↑

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↑ indicates an increase in the benefit; ↑↑ indicates a substantial increase in the benefit; ↔ indicates no appreciable benefit

made to reduce heat/metabolic production during rest periods. While rest periods are often associated with opportunities to rehydrate, they also provide a period of inactivity with decreased metabolic heat production, thereby reducing heat gain. Hydration, rest, and the removal of protective equipment in a location not subject to direct solar heating are strategies that can be employed during rest breaks to improve heat loss. Additionally, rest breaks provide time for enhancing metabolic by-product removal and restoring energy reserves. A description of performance and thermoregulatory benefits of rest breaks is presented in Table 6.1 [3].

The Evidence

Much of the evidence supporting WRRs and repeated exercise bouts in a hot environment comes from occupational worker literature [4–10]. For example, tasks such as fire suppression require multiple rounds of physical activity and passive recovery until the fire is extinguished. Similarly, repetitive lifting is a job requirement in the military and laborer settings and can occur in hot environments. Due to the lack of research regarding WRRs in the athletic population, results from occupational worker settings must be extrapolated to determine best practices for athletes.

Multiple WRRs have been studied and will be explored in this chapter. Maresh et al. investigated cardiovascular and thermoregulatory strain from repeated bouts of exercise using a protocol to simulate a military task with a WRR of 25% work to 75% rest performed in a hot environment [4]. Unacclimated men lifted and carried a 20.5 kg box for cycles of 10 min followed by 30 min of passive rest for a total of 2 h. Ten minutes of box lifting resulted in near maximal heart rate (HR) and a 1 °C/1.8 °F rise in T_c. Despite 30 min of passive rest in the hot environment (38.0 °C/100.4 °F) and maintaining maximal effort, individuals lifted approximately 20 fewer boxes during the second and third bouts of work. During these subsequent

work bouts, HR remained near maximal and T_c continued to rise to approximately 2 °C/3.6 °F above resting temperature. This incomplete recovery may be partially due to the rest break occurring in the same hot environment as the physical activity, as commonly seen in outdoor summer activities such as construction work or soccer games. It is reasonable to think that resting in a cooler environment would aid and possibly lead to a complete recovery. If a cool indoor environment is not available, finding shade outdoors is the next best option to decrease thermoregulatory strain. Another explanation for the poor performance is that the WRR was not appropriate for the maximal exercise intensity. Longer or frequent rest breaks may have been needed for this occupational task.

Typical structural fire suppression involves a 20-min bout of physical activity followed by 30 min of recovery (40% work to 60% rest). During the rest break the firefighter begins a rehabilitation period in which he/she typically rests for 30 min in a cooler environment while rehydrating and applying an external cooling modality such as forearm cold water immersion. At the completion of this rest break, the individual may continue with another task. A 20-min bout of activity results in significant physiological strain. Near maximal HR, a 0.7 °C/1.3 °F increase in T_c, and a nearly 1 kg loss in body mass were observed in healthy individuals of average aerobic fitness [5]. Thirty minutes of passive recovery in a cooler environment returned HR to near resting levels, with most individuals slowing their HR to below 80 beats per minute. Similarly, T_c returned to near resting levels by the end of the rest period. This WRR of 40% work to 60% rest was effective in allowing cardiovascular and thermoregulatory recovery prior to return to physical activity. However, 30 min of continuous rest may not be practical in an athletic setting, such as a fall sport preseason practice, when practice time is limited to 3 h in length to allow athletes to gradually acclimate to a hot environment [1, 6].

To mitigate physiological strain during a 20-min bout of firefighting activities, researchers investigated the effects of 2-min work-rest cycles (50% work to 50% rest). Short, intermittent rest breaks during exercise did not attenuate the elevation of T_c and HR as compared to exercising without short rest breaks [5, 7, 8]. In fact, T_c did not return back to resting levels until 60 min into recovery while HR took over 80 min to return to baseline [7]. These short work-rest cycles were not beneficial in mitigating cardiovascular and thermoregulatory strain during physical activity in heat; however, this study involved uncompensable heat stress created by wearing thermal protective gear and near maximal work. Perhaps short, intermittent rest breaks during exercise could be beneficial during compensable heat stress or during less intense exercise. Furthermore, this WRR of 50% work to 50% rest could be performed in different ways depending upon the demands of the activity. Predominantly aerobic sports such as lacrosse and long distance running may benefit from longer exercise times before resting to mimic the metabolic demands of the sport, while anaerobic sports such as football and sprinting would benefit from shorter exercise times such as 45 s cycles. This comparison of different applications of the same WRR is not well researched.

Multiple bouts of physical activity are common practice in both the athletic and occupational settings. The introduction of a second and third bout of physical

activity is a topic recently studied by Hostler et al. in above average fitness firefighters [9]. The WRR of 60% work to 40% rest for three rounds of fire suppression tasks resulted in a dramatically increased T_c (an additional 1.5 °C/2.7 °F) and increased time needed to complete subsequent mannequin rescue and dexterity tasks compared to individuals who only first completed only two bouts of fire suppression. A fourth work cycle of firefighting activities results in even more drastic changes. Greater peak HR and a quicker rise in T_c occurred in the fourth bout of firefighting activities in heat compared to the first bout of work [10]. A rest duration of 20–40 min between 15- and 30-min work cycles did not allow for complete recovery as illustrated by thermoregulatory strain during subsequent work.

All of the above research illustrates the effects of WRRs on near maximal work intensity resulting in uncompensable heat stress. Both moderate and hard exercise intensities were compared by Sawka et al. to determine differences between continuous and intermittent exercise [11]. Intermittent rest breaks from moderate intensity exercise (a WRR of 75% work to 25% rest) increased exercise time by 20 min compared to continual exercise, with both trials eliciting near maximal HR and high T_c. When exercising at a hard intensity, similar exercise time, T_c, and HR were measured during both continual and intermittent exercise. Despite the addition of intermittent rest breaks, the combination of a hot environment (29.4 °C/84.9 °F) and protective clothing worn created uncompensable heat stress for both intensities. Heat exhaustion occurred in over half of all trials, indicating that the WRR of 75% work to 25% rest is not sufficient to prevent heat illnesses in individuals under uncompensable heat stress. In an athletic setting such as American football preseason sports practices in which athletes wear full body equipment, an uncompensable heat stress environment can be created. In this scenario, the frequency and duration of rest breaks should be increased while the intensity of exercise decreased to enhance safety when environmental conditions are oppressive.

These data show the plausibility of mitigating a decrement in performance and dramatic rise in T_c toward the end of activity by adjusting the WRR during each activity bout by progressively increasing rest time and decreasing exercise time. HR and T_c typically rise with extended continual exercise in heat due to cardiac drift [12]. If WRRs were adjusted to decrease work and increase rest when exercise sessions were prolonged, there would be more opportunities to rehydrate, possibly mitigating cardiac drift while improving physical performance and decreasing fatigue. This strategy may be beneficial in sports games and practices that extend beyond 60 min such as when overtime arises, or during occupational tasks such as military drills that require multiple work-to-rest cycles. For example, during the first hour of exercise, a WRR that emphasizes work could be followed while a WRR that emphasizes rest could be used during the second hour of activity. Again, the effects of varying WRRs on physiological strain in different physical activities are not well researched. Further, perhaps WRRs should be adjusted based on environmental conditions by decreasing work time and increasing rest time as ambient conditions become more oppressive.

The Application

WRR guidelines have been established by many organizations such as the National Athletic Trainers' Association (NATA), American College of Sports Medicine (ACSM), National Institute of Occupational Safety and Health (NIOSH), and the US military [1, 13–15]. Additionally, some organizations have relied on empirical data (e.g., Georgia High School Association rules) to determine specific policy on practice and required WRRs [16]. Other WRR recommendations are consensus derived and have little data to support them, while even well-derived guidelines may have limitations. For example, the NIOSH heat stress guidelines do not specifically address the needs of older workers who may be less able to thermoregulate during exertional heat stress. Despite potential limitations, WRR guidelines should be followed to enhance physical activity safety in hot environmental conditions.

WRR guidelines should adjust the balance of work and rest based largely on environmental conditions and the exercise intensity, although other factors such as clothing and acclimatization are also considered. The environmental thresholds, workloads, and assumptions about how individuals are dressed are used to determine appropriate WRRs but differ by organization or governing body. Environmental exposure can be assessed with a number of different metrics but the wet bulb globe temperature (WBGT), which integrates the influences of air temperature, humidity, ventilation (wind), and radiant heating, is the "gold standard" and most commonly utilized [13–15]. In athletics and the US military, exposure conditions are categorized into levels from low risk to high risk while specific WBGT values referred to as threshold limit values are used in occupational safety.

Physical activity intensity determines metabolic heat production, and the intensity is often classified from light or easy load to heavy or hard load in military or occupational safety settings [14, 15]. In the military, easy work involves weapon maintenance, slow walking with light load, and marksmanship training. Moderate work involves activities such as calisthenics and patrolling, while hard work involves walking with heavy loads and field assaults. Within athletics, the ACSM distinguishes between continuous (e.g., marathon running) and noncontinuous activity (e.g., football, soccer, tennis) but does not specifically designate the degree of intensity [13]. Among the occupational settings, firefighting is considered high intensity short duration work that generates considerable heat stress [9, 17, 18]. On the contrary, hazardous materials mitigation typically involves lower intensity but longer in duration. However, both occupations require protective garments that exacerbate heat accumulation.

Existing WRR guidelines have assumptions made about the amount of equipment or clothing an individual is wearing, which in turn affects heat balance. It is often assumed workers are wearing light summer clothing, soldiers are in battle dress uniform, and athletes are wearing shorts and t-shirts [13–15]. However, this assumption is not always true, such as when athletes wear specialized protective equipment. Therefore, military and occupational safety organizations use clothing adjustment factors if clothing or equipment exceeds expectations. That is, heavy

clothing that impedes cooling results in a lower threshold WBGT value allowed for a given work level. Finally, most policies recognize that unacclimatized individuals are more sensitive to heat and at a greater risk for a heat-related injury by using lower environmental thresholds for determining WRRs [14].

In occupational safety, the American Conference of Governmental Industrial Hygienists provides widely utilized WRRs based on threshold limit values, which are designed to prevent T_c from exceeding 38 °C/100.4 °F [14]. WBGT limits are higher for light work and more rest, and decrease with increasing activity levels and less rest. For instance, a healthy acclimatized individual could safely work at 31 °C/87.8 °F doing light work but only 27.5 °C/81.5 °F for heavy work with 50% work to 50% rest each hour while at 25% work to 75% rest each hour, they could function at 32.5 °C/90.5 °F for light work and 30.5 °C/86.9 °F for heavy work. Similar WBGT-based WRRs are used by other US and international agencies and organizations [14, 19].

The US military employs a similar approach but uses WBGT-based heat categories from 1 (low heat exposure) to 5 (extreme heat exposure) in conjunction with three workload categories to determine appropriate WRRs [15]. In low heat conditions (category 1; 25.6–27.7 °C/78.0–81.9 °F), the work can be continuous for easy and moderate workloads but 20 min of rest per hour is needed for hard work. Under extreme conditions (category 5 or black flag; >32.2 °C/90.0 °F) 10 min of rest per hour is needed for easy work increasing up to 50 min of rest per hour for hard work.

The most widely used activity modification guideline in athletics is produced by the ACSM [13]. Using categories of WBGT provides a set of activity modification for different types of activities (continuous vs. noncontinuous) and sets of individuals (e.g., non-acclimatized, unfit, high-risk vs. acclimatized, fit, low-risk). WRRs are indicated for high heat sensitivity individuals performing noncontinuous activities when WBGTs ≥18.4 °C/65.1 °F and the ratios increase to 50% work to 50% rest for WBGTs ≥27.9 °C/82.1 °F. No WRRs have been created for acclimatized, fit, and/or low-risk individuals performing noncontinuous activity. More specificity in WRRs is provided in Georgia High Schools Association WBGT guidelines (Table 6.2) [16]. Decreased work and increased rest are suggested at greater WBGTs. For moderate conditions ≤27.8 °C/82.0 °F, ≥3 separate rest breaks per hour of at least 3 min each are recommended (85% work to 15% rest), increasing up 20 min of rest breaks per hour when WBGTs range from 32.2–33.3 °C/90–92 °F (67% work to 33% rest). These guidelines also specify that rest time should involve unlimited hydration, no activity, removal of helmets for football players, and the location should be shaded from direct sunlight. For WBGTs over 30.0 °C/86.0 °F, ice towels and spray bottles with ice water should be available to maximize player cooling.

Case Example *When athletes compete in a particularly stressful environment with extreme heat and/or humidity, consideration must be given to developing appropriate parameters regarding the length of physical activity and rest intervals to ensure safety. As discussed in the previous section, selecting an appropriate WRR is essential to mitigate potential EHI due to participation in such climatic conditions.*

Table 6.2 WBGT-based activity and rest break guidelines from the Georgia High School Association [16]

WBGT	Activity guidelines and rest break guidelines
Under 82.0 °F	Normal Activities—Provide at least three separate rest breaks each hour with a minimum duration of 3 min each during the workout
82.0–86.9 °F	Use discretion for intense or prolonged exercise; watch at-risk players carefully. Provide at least three separate rest breaks each hour with a minimum duration of 4 min each
87.0–89.9 °F	Maximum practice time is 2 h. For Football: players are restricted to helmet, shoulder pads, and shorts during practice, and all protective equipment must be removed during conditioning activities. *If the WBGT rises to this level during practice, players may continue to work out wearing football pants without changing to shorts.* For All Sports: Provide at least four separate rest breaks each hour with a minimum duration of 4 min each
90.0–92.0 °F	Maximum practice time is 1 h. For Football: no protective equipment may be worn during practice, and there may be no conditioning activities. For All Sports: There must be 20 min of rest breaks distributed throughout the hour of practice
Over 92.1 °F	No outdoor workouts. Delay practice until a cooler WBGT level is reached

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Determining an appropriate WRR is often difficult if there is no "hard evidence" to depict the length and frequency of activity and rest periods. The NATA, ACSM, and U.S. Department of Defense have all published position statements that set up frameworks around which individuals can model practice policies and more importantly determine the appropriate WRR for each specific scenario. It is the job of the practitioner to select an appropriate WRR for their individual physical activity needs to prevent EHIs [4, 20].

As discussed in Chap. 9, the Georgia High School project aimed to define when the greatest EHI risk rates occurred during high school American football sports practices and how these illnesses might be best mitigated. WBGT thresholds and associated WRRs were identified based on EHI rates that were associated with specific environmental conditions and categorized based on multiple factors to determine safety policies. These factors include practice duration, type of EHI (e.g., *heat syncope, heat exhaustion, and exertional heat stroke), and the WBGT during the acclimatization period (days 1–7 and through the initial 10 days of practice). Practitioners could use a similar strategy to determine which WRR is appropriate, therefore the remainder of this section describes the steps taken in the project to mitigate* T_c *rise and reduce the risk of EHIs.*

Data from the first 3 years of the study were analyzed to determine how to identify WBGT thresholds and how each category would be labeled. All EHIs were defined according to ACSM guidelines [13]. *Results of the initial data indicated that EHI risk significantly increased by approximately 50% when practices lasted longer than 120 min in the first 7 days of practice. This increased risk rate was prolonged to 2.5 h of activity in week 2 and again in week 3 after 3 h, indicating that*

acclimatization was occurring. By validating the reduction in EHI rates over time through the data and the temperatures when EHIs occur, WRRs were more confidently established. Additionally, most EHI cases occurred when the WBGT was below 30.0 °C/86 °F. This, however, was likely due to more practices being cancelled or modified at higher temperatures.

Establishing risk rates at specific WBGT temperatures played a role in determining policy regarding rest breaks, practice intensity, practice duration, and the amount of equipment worn for higher WBGTs [21]. *In a study done on EHI in Marine Corps recruit training, similar WRR recommendations were made to reduce EHI risk* [22]. *After careful consideration of the data from the first 3 years of the Georgia High School study (identifying increased risk when practices lasted more than 2 h or were held at a WBGT greater than 27.8 °C/82 °F), it was determined that a minimum of three rest breaks should be scheduled each hour of activity, lasting a minimum of 3 min each when the environmental conditions were less than 27.8 °C/82 °F WBGT. The number and length of rest breaks for the subsequent WBGT categories were calculated based on previous EHI risk research among active populations and the risk rates from data analysis of the initial data set* [1, 13, 23].

The Georgia High School study analyzed 6 years of data and revised practice policy guidelines were developed after 3 years of initial data collection concluded and risk-rate trends were established. As seen in Table 6.2, the WRR directives were based on WBGT temperature readings and previously established guidelines [22, 24]. *Consideration was given to both environmental and non-environmental risk factor when establishing the revised WRR for the interscholastic football athlete. It is prudent for the practitioner to be mindful of the numerous factors influencing EHI risk during the early phase of heat acclimatization and to determine their own best WRR.*

Table 6.3 Ten steps for successful implementation of work-to-rest ratios

1.	Determine which work-to-rest ratio (WRR) is best for your population based on current best practice guidelines
2.	Determine if there are specific modifications of the WRR based on your geographical location
3.	Plan ahead by having different workout options available depending on the environmental conditions that day and make an appropriate selection
4.	Remain cautious by adding additional rest breaks when prudent
5.	Extend each rest break when exercise intensity or ambient conditions increase
6.	All rest breaks should include adequate opportunities for athletes to fully rehydrate with no required physical activity
7.	Rest in a cool or shaded environment while rehydrating
8.	If the activity requires the wearing of equipment, the equipment should be removed during the break
9.	Have cooling modalities available for use during rest breaks such as ice towels when the wet bub globe temperature is above a reading of 30.0 °C/86.0 °F
10.	Medical staff should talk with the athletes during rest breaks to assess signs and symptoms of exertional heat illnesses

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Chapter 7
Sleep

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Sleep Architecture

The recurring and natural process of sleep involves the regulated and controlled alteration of consciousness without loss of sensory perception or regulation [1]. Sleep is an integral part of one's daily circadian rhythm, representing the cyclical functioning of the hormonal profile that regulates normal physiological function. While the reason for sleep remains debated, the physiological responses that predominate during this time suggest the importance of recuperative and anabolic building processes [1]. Consequently, the relationship between sleep and circadian rhythm functioning is a primal and necessary function for all humans [1, 2].

Sleep onset occurs in a regular fashion at specific intervals during the daily 24-h cycle, often dictated by environmental zeitgebers—an environmental cue, such as the light-dark cycle. During sleep, reductions in conscious control of physical and muscular activity occur, while regulation of physiological functioning is altered to focus on metabolic regeneration of energy stores, hormonal regulation of stress-based hormones, and improvements in neural plasticity [3–5]. Accordingly, the sleep process is fundamental to human behavior and health, as sleep duration and quality seem integral to optimal physical and cognitive functioning; hence,

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Table 7.1 Classifications of the various stages of sleep, as adapted from Colten and Aldevogt [1]

Stage	Duration	Brain activity	Distinguishing characteristics
Stage I (light)	1–7 min	Noise frequency	Transitional phase of sleep, NREM
Stage II (light)	Initially 10–25 min, lengthens in further cycles	Low voltage activity	Needs increased stimuli to produce awakenings, NREM
Stage III (deep)	20–40 min	High voltage, slow wave activity	Slow wave sleep, NREM
Rapid eye movement	Prolonged in late-night cycles	Sawtooth like EEG readings	Dreaming, loss of muscle tone and reflexes

obtaining appropriate sleep seems to be of high importance for athletes to ensure psychological and physiological functioning [2]. The addition of environmental stressors that exacerbate physiological and cognitive responses to exercise, such as heat exposure, emphasizes the need for greater recovery. However, the importance of sleep on exercise performance and its association as a risk factor for exertional heat illness remains contested [5, 6].

Evidence shows that approximately 6–10 h of the 24-h daily cycle is spent in sleep-related activities with a range of 7–9 h of sleep per night being deemed as "normal" [1]. While the term sleep collectively refers to the state of altered consciousness, sleep is further defined as either non-rapid eye movement (NREM) or rapid eye movement (REM) sleep. Within these classifications, it is possible to determine the architecture of the sleep profile, as NREM sleep is further classified into three separate stages (Stages I–III) that are dictated by the frequency and type of brain waves as measured by electroencephalography (EEG), blood pressure, heart rate, breathing frequency, muscle activity, and amount of eye movement [7] (Table 7.1). Each sleep cycle (NREM Stages 1–III and REM sleep) lasts approximately 90 min and depending on individual needs, between four and six cycles are necessary for sleep to be deemed adequate [8]. While there is no explicit definition of "quality sleep," researchers suggest that sleep is most effective when stages are cyclically sequenced throughout a night to then allow appropriate physical and mental conditions on waking [9].

As aforementioned, sleep-wake cycles are distinguished by altered physiological functioning aligned with various stages of consciousness. Included in these changes are various physiological alterations related to the body's thermoregulatory function [1, 2]. Sleep is an important factor in the body's circadian rhythm allowing for daily variation in the thermoregulatory set point [9]. For example, sleep onset is associated with increased melatonin secretion and peripheral vasoconstriction to reduce core temperature, while waking reverses these responses due to the reduction in melatonin concentration after exposure to light or increased muscle activity [9]. Given the relationship between sleep-wake cycle and thermoregulatory function, it stands to reason that exercise in hot conditions may be

further influenced by sleep [10]. Therefore, the purpose of this chapter is to explore the role of sleep and exercise in the heat.

Factors That Control Sleep

To understand the importance of sleep on thermoregulatory control and thus relationship of sleep during exercise in the heat, it is important to understand the physiological regulation and alterations resulting from the sleep-wake cycle. There are many factors that control and assist in the regulation of sleep, and though predominantly dictated by the central nervous system, external zeitgebers also play a role in sleep in onset, duration, and quality of sleep.

Neural

Within the brain, the hypothalamus is an important region that controls one's sleep-wake cycle. Correspondingly, various neurotransmitters are up- and downregulated during sleep that assist in maintaining a non-pathologic period of sleep. γ-aminobutyric acid (GABA)-ergic cells, located in the anterior hypothalamus, are important for regulating the sleep-wake cycle as they inhibit the firing of cholinergic cells in the forebrain as well as inhibit the release of histamine, norepinephrine, and serotonin from the posterior hypothalamus, locus ceruleus of the pons, and raphe nuclei that are responsible for maintaining a state of wakefulness [11]. Furthermore, the inhibition of histamine directly controls one's state of arousal and consciousness, whereas the inhibition of serotonin and norepinephrine leads to the loss of muscle tone, primarily during REM sleep [11]. The neurotransmitter hypocretin has also been found to be directly related to the sleep-wake cycle. While not completely understood, a loss of hypocretin cells has been linked to narcolepsy and cataplexy, which may have adverse effects on normal physiologic function [11].

Hormonal

Melatonin is the hormone that assists in sleep regulation, and is produced and secreted from the pineal gland [1]. Melatonin is secreted largely at night and is inhibited by the presence of bright light and works to decrease the set point of the internal temperature by acting on the hypothalamus, which results in a decrease in internal temperature that prompts the onset of sleep [12, 13]. Cortisol also plays a role in sleep and sleep regulation, and cycles with melatonin in the 24-h circadian rhythm (Fig. 7.1) [1]. Investigations have shown that a presence of bright light before bed inhibits melatonin production resulting in the delayed onset of sleep, and a phase delay in an individual's normal thermoregulatory circadian rhythm, which alters the homeostatic set point for body temperature [12].

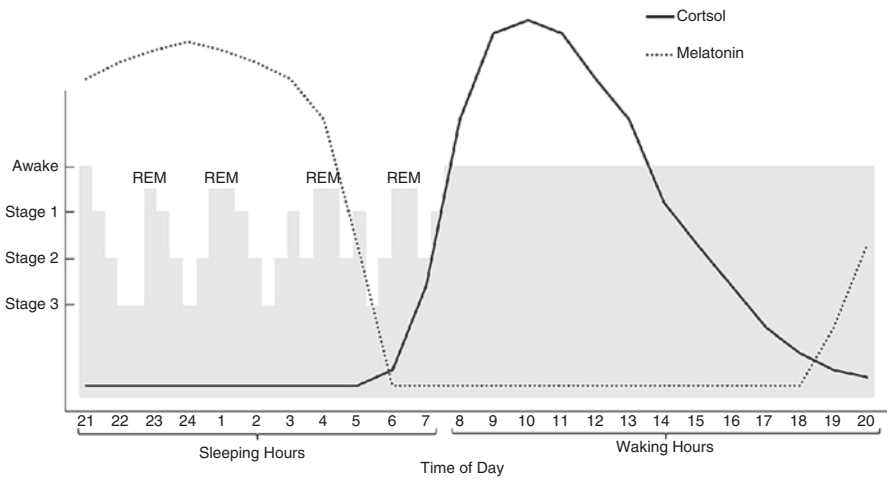

Fig. 7.1 Depiction of the circadian variation of melatonin and cortisol over a 24-h daily period in addition to arousal levels in sleep stages for sleeping and waking hours

External Factors

Zeitgebers such as environmental temperature, light, and alcohol intake may also play a role in sleep onset, duration, and quality. Environmental temperature disrupts sleep by decreasing sleep quality and prolonging sleep onset in colder and warmer environments [12, 14]. Furthermore, bright light of sufficient lux (2500 lux) has been shown to decrease sleep onset due to inhibition of melatonin, and lead to decreased internal body temperature the following day [12]. In combination with the presence of light, the intake of food and social customs surrounding evening activities can also affect both physiological arousal and behavioral patterns related to sleeping. For example, timing of food consumption at night, the use of electronic devices prior to bed time, and other social activities can delay sleep onset time and reduce sleep quality [15]. As a particular example, the consumption of alcohol also reduces the amount of sleep in a given night, disrupting the normal sleep stages, and postpones sleep onset [16].

Sleep and Cardiovascular Function

There is evidence to suggest that sleep loss (>4 h) increases cardiovascular strain during ensuing aerobic exercise bouts (cycling and running of >30 min in duration). Following a night of restricted sleep, exacerbation of cardiovascular strain occurs as evidenced by an increased heart rate at a magnitude of 3.5–4.7% during exercise at submaximal and maximal intensities compared to a night of normal sleep [17, 18]. Such findings highlight that a reduction of cardiovascular efficiency induced by

sleep disruption observed in both maximal and submaximal aerobic performance could be compromised [17].

In addition, indicators of cardiovascular strain (i.e., increased heart rate) were evident during 20 minutes of steady-state exercise following a period of sleep restriction; however, a decrease in heart rate was observed during 60 minutes of treadmill running following 30 hours of sleep deprivation [18]. Such findings contrast with the results observed by Mougin et al. [17], which found sleep restriction to have derogatory effects on performance [17] and no changes in performance [19] and may be a result of differences in the extent of sleep loss and intensity of exercise performed. While further research is warranted, the detrimental impact that sleep deprivation has on cardiovascular function will likely to further exacerbate cardiovascular strain during exercise in the heat. Thus, sleep deprivation and/or restriction would likely further handicap an athlete looking to maximize performance in the heat.

Sleep and Thermoregulatory Function

There is conflicting research in the area in sleep and thermoregulation during exercise. Some research suggests that sleep deprivation induces an increase in internal body temperatures during exercise [20, 21], while other evidence suggests that sleep deprivation actually lowers pre-exercise temperature and has no effect on exercise-induced temperature responses [12, 18, 22–24]. While scientific literature on this topic remains inconclusive, sleep deprivation is postulated to exacerbate thermoregulatory strain during exercise by decreasing the thermal load that the body can tolerate. Following sleep deprivation, a reduction in sweat rate and peripheral vasodilation occurs, which decreases the capacity to dissipate metabolically produced body heat via mechanisms of evaporation and convection [23–25].

Prior laboratory studies examining the effects of total sleep deprivation on thermoregulatory control [24, 26, 27] reported small changes in the threshold and sensitivity of local sweating and cutaneous vasodilation, giving rise to the speculation that sleep loss may be responsible for altered thermoregulatory effector responses [27]. The physiological mechanisms responsible for these changes remain unknown, although suggested causes center on altered central nervous function [24, 26, 27]. Despite finding these local changes of thermoregulatory function during exercise in the heat, there were no reported differences in whole-body temperature responses during exercise in the heat. This may have been due to the methodological design in laboratory research; the length of sleep deprivation in these studies (27–33 h) was not sufficient to elicit any meaningful changes.

Similarly, a recent study examining the effects of partial sleep deprivation on thermoregulatory strain during exercise in the heat found no differences in either rectal or skin temperature following three nights of sleep restriction compared to the control group [22]. Furthermore, when examining the individual variation in temperature responses in this cohort of subjects, only 30% (3 out of 10) of subjects were shown to have any meaningful changes in temperature responses during exercise in

the heat. These responses were in themselves inconsistent, with one subject showing a rise in rectal temperature compared to the control condition, while the other two subjects showed a decline in rectal temperature following exercise [22]. Consequently, despite the intertwined nature of the sleep-wake cycle with thermoregulatory function, the loss of sleep and resulting prolonged wakefulness do not seem to greatly alter the acute exercise-induced thermoregulatory responses.

Despite the above observations, limitations evident in the previous literature to prevent this being a definitive conclusion relate to the shortened period of exercise trial (<1 h) in their experimental design, which may not be long enough to identify any thermoregulatory changes following sleep loss [22, 24, 26, 27]. Tokizawa et al. [21], found that following prolonged exercise in the heat, body temperature was significantly elevated in those exposed to partial sleep restriction versus those receiving a normal amount of sleep. Interestingly, the results from this study found an increased sweat rate in the sleep deprivation trials at the same time points as the increase in body temperature [21], which contrasts the findings of prior literature with evidence of a decreased sweating response [24, 26, 27]. The increase in sweat rate in the sleep deprivation trial may have been responsible for the increase in body temperature based on previous findings [28] but it is unclear as to the specific mechanisms cause the changes in body temperature during the latter bouts of exercise in this study.

Sleep and Cognitive Performance

Sleep deprivation negatively affects cognitive performance (i.e., reaction time, accuracy, vigilance). This is particularly important for soldiers who commonly have to perform under sleep deprived conditions. In addition, sleep deprivation coupled with other stressors such as combat situations and/or environmental heat stress elicits further exacerbation of cognitive deficits and mood disruption in soldiers [29]. Sleep deprivation can lead to decrements in reaction time, mood, and attention [30]. Lieberman et al. investigated the role of sleep deprivation on simulated combat tasks and found that when sleep was restricted (~3 h of sleep per night), combined with heat exposure, there was a significant decline in cognitive performance including visual vigilance, reaction time, match-to-sample tasks, grammatical reasoning, among others [29]. Mood was also detrimentally affected by sleep deprivation and heat exposure [29].

Sleep and Exercise Performance

Anecdotally, sleep deprivation has been recognized as a source of performance deficits during exercise; thus, it has believed that recommending an adequate amount of sleep is essential to achieve optimal performance. While prior research has examined the effects of sleep deprivation on physical performance [17–19, 31–43], the

results are equivocal as to the direct influence of sleep deprivation on physical performance. A collection of research [44, 45] suggests that confounding factors such as length and type of exercise, the extent and type of sleep deprivation, and other individual characteristics (i.e., catecholamine and adrenergic responses) may be responsible for observed performance deficits.

Reviews of the literature [45, 46] have investigated the role of type and length of exercise and their relationship with sleep deprivation and subsequent exercise performance. While some studies show that sleep deprivation has no effect on exercise performance [34, 35, 43, 47], others have found significant performance deficits following sleep loss [18, 33, 38]. Evidence suggests that sleep deprivation, up to 36 h, may not affect anaerobic performance due to the length of time for performing most anaerobic task [34, 41–43]. Evidence is more variable when examining the effect of sleep deprivation on endurance performance. Martin [19], Oliver et al. [18], Pickett and Morris [34], and Chen [37] found mixed results when examining sleep deprivation up to 36 h on endurance performance. The mixed results may be due to methodological differences in the aforementioned studies that included uncontrolled dietary intakes and uncontrolled amounts of sleep in their control conditions. Individual factors and smaller sample sizes may also have been responsible to the lack of conclusive findings as individual variation ranged from 5% improvement to 42% decrement in Martin [19] and from 4% improvement to 9% impairment in Oliver et al. [18].

Naitoh [47] and VanHelder [48] both concluded that performance deficits will not become apparent until the extent of sleep deprivation reaches 46 and 72 h, respectively. Pilcher and Huffcutt [44] also examined short-term, long-term, and partial sleep deprivation on various performance measures and found that long-term sleep deprivation resulted in the largest effect in motor-related and cognitive-related performance deficits. However, it must be noted that partial sleep deprivation (sleep $\geq$4 h <8 h), which is more applicable to athletics competition, military operations, and labor situations (i.e., shift work), also resulted in significant changes in both motor- and cognitive-related performance [44].

The inconclusive findings of the previous literature warrant further investigation to determine how sleep loss may affect performance. Furthermore, examining the effects of sleep deprivation on exercise performance in the heat warrants future research as there is a lack of scientific literature on this topic. Conversely, additional research examining the influence of too much sleep on exercise performance as this area of research has not been investigated in prior research.

Sleep and Health Considerations During Exercise in the Heat

Sleep has been regarded as an essential component of whole-body restoration during times of stress and has been considered the gold standard for post-exercise recovery [15, 49, 50]. While previous literature has examined the effects of sleep on performance, there are only a few that have examined the potential implications that sleep deprivation has on health, particularly during exercise in the heat and the risk

of exertional heat illness. Multiple position statements [51–53], consensus documents [54, 55], and case series [56] have cited that lack of sleep is an important factor related to an increased risk of exertional heat illness, particularly exertional heat stroke. However, these statements are based on clinical observations and previous case series.

While the findings from Moore et al. [22] show that sleep loss alone may not predispose one to increase risk of exertional heat illness, other factors such as dehydration, negative energy balance, and exertional fatigue, which all have been shown to individually increase safety risk may have compounding effects when combined. However, there is a lack of literature examining these factors in combination, requiring future research to determine overall risk.

Despite the rather inconclusive findings on sleep loss and risk of exertional heat illness during exercise in the heat, sleep loss may have compounding effects on other factors associated with increased risk of reduced performance, if not risk of illness in the heat (i.e., reduced muscle glycogen repletion [57], decreased mood state [58]). Previous research has also observed a difference in the self-reported symptoms of heat illness following 24-h sleep deprivation despite no differences in rectal temperature and a reduction of heart rate following a heat stress test [59]. Thus, ensuring adequate sleep along with maintaining normal hydration and metabolic heat balance may attenuate risks of cardiovascular and thermoregulatory strain, ensuring safe participation in sport and physical activity.

Practical Applications for Optimizing Performance and Safety in the Heat Regarding Sleep

In the context of exercise and performance, adequate sleep is important for optimizing performance and safety [43, 44, 60]. Following physical activity, evidence shows that obtaining an appropriate amount of sleep assists in enhancing recovery in preparation for physical activity the following day [61–63]. Current evidence suggests that, on average, sleeping 7–9 h per night along with sleep efficiency ratings >80% can optimize long-term health and reduces morbidity and mortality [60]. Adequate sleep may also aid the whole-body restoration and prevent the cognitive and performance deficits associated with sleep deprivation [43, 44, 50].

Independent of type or mode of physical activity, ensuring that an adequate amount of sleep is attained prior to starting activity will minimize the potential for sleep-related deficits. Identifying when optimal performance is needed ahead of the time, as well as each individual's goals in task or physical activity is vital when implementing a sleep management program [43]. For example, in the context of optimizing performance and safety during military operations, it is recommended that soldiers obtained 7–9 h of sleep in the nights leading up to sustained operations, with important tasks being delayed following the time of sleep inertia (i.e., feelings of lethargy and lassitude in the first 5–30 min following waking) [43, 61]. Furthermore, creating an environment that is most conducive to sleep such as

ensuring the sleep is occurring in a dark room or wearing an eye mask may aid to improve sleep quantity (but not efficiency) following activity or when sleeping environments are not optimal [64].

Exercise in the heat may cause both cardiovascular and thermoregulatory strain and is further exacerbated by individual factors such as dehydration, and acclimatization status [28, 50]. Incorporating an individualized sleep management program in physically active individuals in conjunction with other strategies such as heat acclimatization, individual hydration plans, body cooling, and others can have added benefit for the athlete, soldier, and laborer in optimizing performance and enhancing safety. Athletes are now able to monitor and track their sleep using wearable technology available to the consumer market. Despite inconclusive evidence as to the accuracy and validity of current consumer sleep monitoring devices, there is hope that refinement of this technology will continue to improve to become better objective tools for monitoring sleep and sleep quality. The utilization of an objective measure to improve sleep quality of an athlete during their training regimen may allow for additional improvements in their exercise performance.

Case Example *The Portugal National Soccer Team utilized various tactics to optimize their sleep hygiene in preparation for the 2014 World Cup in Brazil. Portugal resides in the Western European time zone, while Rio de Janeiro, Brazil resides in the Brasilia time zone. This means that the Portugal National Soccer Team would incur a four-hour time difference. Armstrong* [65] *states that for every time zone crossed, it can take up to a full day for an athlete to recover. Thus, the Portugal National Team, in an attempt to help the athletes acclimatize and perform optimally in the Brasilia time zone, traveled to New York City area for their training two weeks before the competition to account for changes to the circadian rhythm and zeitgebers. This travel adjustment resulted in an ideal situation for athletes to train with less influence from the time zone changes and subsequent sleep disruptions. It should be noted that their heat acclimatization could have been further enhanced by traveling and training in a warmer climate within the same time zone as the one they're competing, in order to account for the anticipated heat that they would experience in Rio during the World Cup Games. The Portugal National Team is a good example of what to do in preparation for a competition with regard to sleep and circadian rhythm in order to assist the athletes in optimal performance. However; when taking heat into account, special considerations should be made to allow extra time to further acclimatize the athletes to the warmer climate in the travel destination.*

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Chapter 8
Altitude

Lesley W. Vandermark, Santiago Lorenzo, and Robert F. Chapman

The Physiology

Physiological Response to Altitude Stress

Barometric pressure declines with ascent to altitude, and the physical features and subsequent physiological effects that accompany the decline in pressure can have a profound influence on physical performance. Because the percentages of the individual gasses in air remain constant and are independent of altitude, with a given change in the barometric pressure, the inspired partial pressure of oxygen (PIO_2) will change in a proportional manner. The density of air is also affected by changes in pressure and will decrease with the degree of hypobaria (i.e., low ambient air pressure) of altitude. With regard to the specific issue of altitude and heat-related factors, temperature and humidity are both reduced in a hypobaric environment, relative to what they would otherwise be in a normobaric environment. Ultimately, exercise performance at altitude will be affected primarily by the influence of three factors [1] described in Fig. 8.1.

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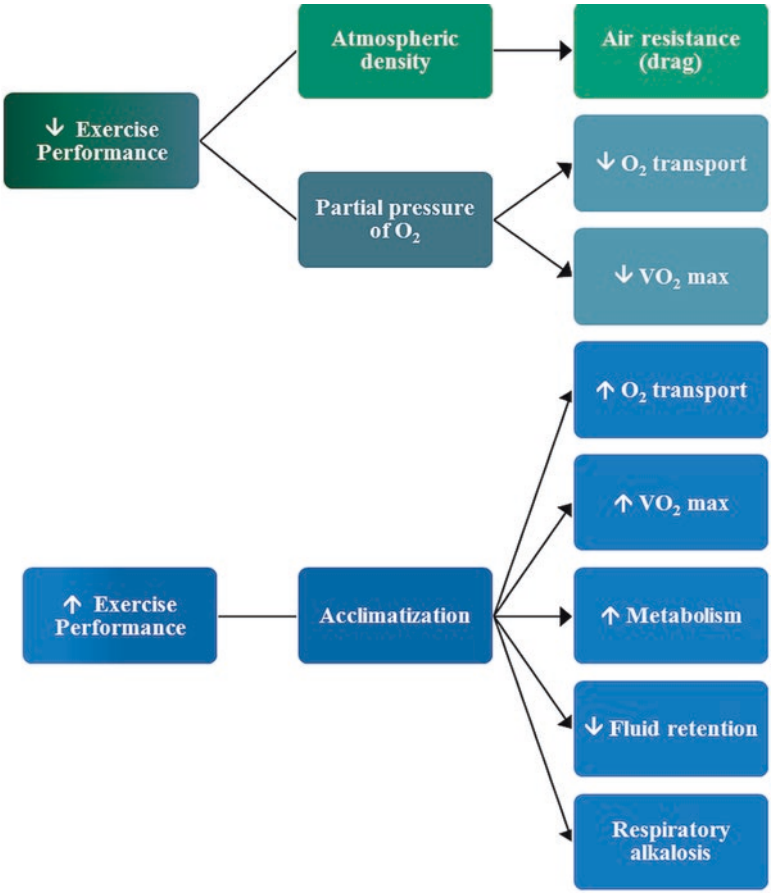

Fig. 8.1 Physiological factors influencing exercise performance at altitude

1. The density of the atmosphere and the resultant effect on air resistance, or drag.
2. The PIO_2 and the resultant effect on oxygen transport and uptake.
3. The process of acclimatization, affecting oxygen transport, metabolism, fluid balance, and acid-base balance.

This chapter will focus on the combined effects of altitude and heat on exercise performance. Therefore, the final two of the above listed factors will be of primary importance.

With acute exposure to altitude, the decline in PIO_2 is translated down each step of the oxygen cascade—that is, altitude exposure results in declines in PO_2 in the alveoli, the arteries, and the capillaries, ultimately impairing oxygen diffusion to the mitochondria in the peripheral tissues. Because of the sigmoidal shape of the oxyhemoglobin dissociation curve, relatively small drops in arterial PO_2 have little effect on arterial oxyhemoglobin saturation (SaO_2) or arterial oxygen content.

Therefore, untrained individuals with normal pulmonary function have the capacity to maintain oxygen delivery and aerobic exercise performance, near to what they would otherwise experience at sea level, at altitudes less than ~1000 m [2]. However, highly trained endurance athletes have been known for some time to be "handicapped to an unusual extent" at altitude [3], and the pulmonary gas exchange limitations common in this population during heavy exercise [4] result in a near-linear fall in maximal oxygen uptake (VO_2max) with ascent from sea level [5]. Contrary to the notion of a "threshold altitude" for aerobic impairment, a statistically significant decline in VO_2max is seen in elite endurance athletes at altitudes as low as 580 m [6]. This is just one of many examples in the literature [7–10] that identifies the ability to maintain delivery oxygen to the periphery as one of paramount importance for endurance exercise capacity at altitude. However, even within the highly trained population, there is substantial individual variability in the response of physiological systems and exercise performance at altitude. For example, distance runners display significant individual variability in the hematological response [11–13], reduction in VO_2max [8], and worsening of exercise performance [9, 13] with acute altitude exposure. With chronic altitude exposure, trained endurance athletes also show substantial variability in the acclimatization response [11], which may be a function of genetic predisposition [14, 15] or the magnitude of the hypoxic stress or living altitude [16, 17]. Ultimately, it is important to keep in mind that the mechanisms by which altitude affects maximal oxygen uptake and exercise performance are *multifactorial* and *variable*.

With arrival at altitude, there are a number of physiological acclimatization responses that take place, all with the goal of trying to maintain tissue oxygenation despite a reduced atmospheric pressure. It is important to note that the severity of these acclimatization responses are often dependent on the nature of specific physiological variable in question and the magnitude of the altitude achieved. To help standardize discussion around altitude nomenclature and the resulting physiological responses, a congress [18] standardized altitude classifications as: near sea level (0–500 m), low altitude (>500–2000 m), moderate altitude (>2000–3000 m), high altitude (>3000–5500 m), and extreme altitude (>5500 m) (Fig. 8.2). Of the multiple physiological systems affected by altitude, the time course of acclimatization responses can range from as short as a few seconds, to time periods covering hours, days, weeks, years, or even generations [1]. For simplicity, and for the purposes of this chapter focusing on altitude and heat, we will confine our review to the physiological responses to terrestrial altitude and the hypobaric hypoxia that exists in that environment.

Perhaps the most immediate response with acute exposure to altitude—one that is typically perceptible by the athlete—is an increase in ventilation, both at rest and during submaximal exercise. Peripheral chemoreceptors, located in the carotid arteries and aortic arch, are sensitive to a drop in the partial pressure of oxygen in the arterial blood. These chemoreceptors respond by stimulating the inspiratory center in the medulla to increase ventilation, and this response helps to defend alveolar PO_2 and SaO_2 with the decline in atmospheric PO_2 at altitude [19]. The sensitivity of the peripheral chemoreceptors is highly individualistic. Interestingly,

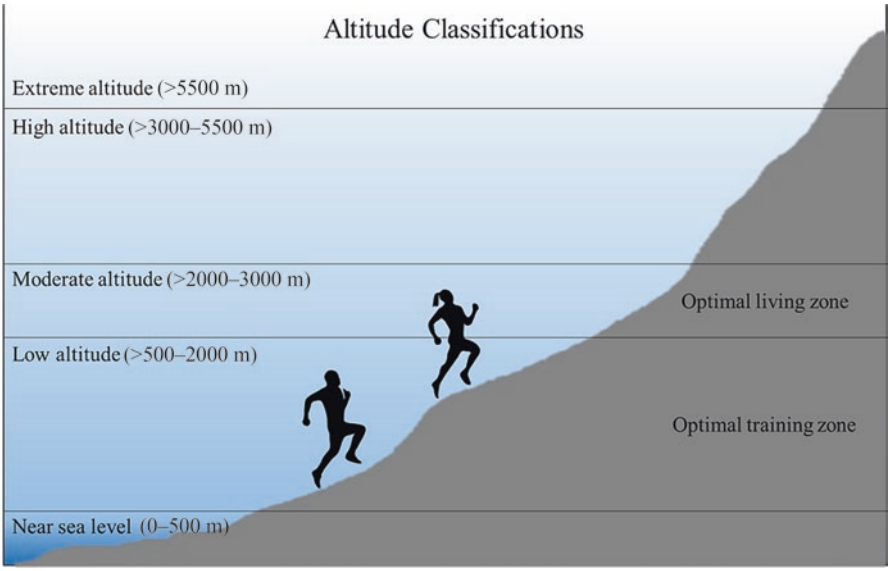

Fig. 8.2 Classifications of altitude exposure as defined in Bärtsch, Dvorak, and Saltin (2008) [18]

endurance trained athletes tend to have a more blunted ventilatory response to hypoxia than lesser trained individuals [20]; however, the sensitivity of the peripheral chemoreceptors to hypoxia appears to be based primarily on hereditary factors [21] and does not appear to be influenced by training [22]. Chronic exposure of 15 days or longer results in an increase in the hypoxic ventilatory response, but this chemoreceptor gain gradually reverts to baseline after return to sea level [23].

The increase in ventilation at altitude, both at rest and during exercise, may help to defend SaO_2 in the face of a declining PIO_2. However, this increase in ventilation is not without consequences. One is the actual cost of the respiratory effort, both in metabolic terms and perceptual terms. As ventilation increases, the work of breathing increases exponentially [24], which requires a larger fraction of the cardiac output to be delivered to the respiratory musculature. At very heavy work rates where respiratory muscle work nears maximal levels, a sympathetically mediated "metaboreflex" causes vasoconstriction in locomotor muscles [25]. While this response helps to ensure adequate respiratory muscle blood flow and oxygenation [26], high levels of respiratory muscle work has been shown to impair exercise performance [27]. In fact, in cohorts of elite distance runners, 27–37% of the increase in VO_2max observed after 4 weeks of altitude training is estimated to go towards simply fueling the increased metabolic needs of the respiratory musculature, rather than the locomotor muscles [28]. Additionally, often an athlete's internal feedback cues regarding sensation of effort during exercise are closely tied to the discomfort of breathing or dyspnea [29]. When athletes complete high intensity exercise at altitude, data suggest that perception of ventilatory effort may play a significant role in workload selection [30], which may ultimately affect the ability to train at altitude.

Fluid Balance

Perhaps one of the most significant consequences of stimulation of the peripheral chemoreceptors with acute altitude exposure—at least where thermoregulatory capacity is concerned—is a decline in plasma volume and total body water. To a small extent, the increase in ventilation (in the typically dry air of altitude) results in a greater insensible respiratory water loss over time than at sea level [31]. At high and severe altitudes, a reduction in fluid intake [32] and anorexia [33] also contribute incrementally to the altitude-mediated loss in total body water. However, by far the largest portion of the decline in total body water and plasma volume at altitude is due to actions taking place at the renal level.

Hypoxia-induced respiratory alkalosis results in a renal excretion of bicarbonate, which is typically complete after 24 h of exposure to mild and moderate altitudes [34]. Alkalosis, independent of hypoxia, results in an ~15% reduction in plasma volume, due to both the compensatory excretion of bicarbonate [35, 36] and an increase in sodium excretion [37]. Alkalosis also causes a shift in fluid to the extracellular space, likely due to increased capillary hydrostatic pressure [36]. Interestingly, hypoxia itself appears to have a direct stimulating effect on diuresis. While the complete mechanism behind a "hypoxic diuretic response" or HDR is still being elucidated, the HDR begins within hours of exposure to altitude and appears to be biphasic [38]. The early phase involves diuresis without natriuresis [39], possibly due to reduced urine concentrating ability [40] or a decrease in circulating anti-diuretic hormone (ADH) [41]. This early phase is followed by a multifactorial response, resulting from downstream effects of peripheral chemoreceptor stimulation (such as increased atrial natriuretic peptide, increased epinephrine, decreased circulating ADH, and decreased kidney sensitivity to ADH), as well as direct effects of kidney hypoxia (such as increased endothelin-I, increased adrenomedulin, and decreased renin and aldosterone) [38]. Each of these factors leads to inhibition of renal sodium reabsorption and a substantial change in blood buffering capacity. However, what happens to buffering capacity in the muscle is less clear, with some studies reporting a decrease in muscle buffering capacity [42] and others reporting an increase in muscle buffering capacity with altitude acclimatization [43, 44].

The diuresis with acute altitude exposure results in an increase in hemoglobin concentration, hematocrit, and blood viscosity, even before erythropoiesis increases red cell mass. While this short-term adaptation does help to defend arterial oxygen content at altitude, most individuals who train chronically at altitude do so for the benefit of increased total hemoglobin mass [4, 45, 46] Within minutes of exposure to hypoxia or arrival at altitude, hypoxia-inducible factor 1-alpha (HIF-1a) is expressed [47, 48]. Among its many effects, one is to upregulate production of erythropoietin (EPO) in interstitial cells in the peritubular capillary bed of the renal cortex [49], which results in survival and proliferation of erythroid precursors from stem cell to reticulocyte [50]. Typically, with chronic altitude exposure, EPO levels peak after approximately 24–48 h, then gradually decline reaching baseline levels normally observed at sea level after ~14–28 days, depending on individual variation

[11] or altitude of residence [17]. Assuming factors such as adequate iron stores are present prior to departure for altitude and dietary iron is supplemented while at altitude [51, 52], total hemoglobin mass increases on average ~ 1% per week over 3–4 weeks at moderate altitudes [52, 53].

The Evidence

There is some evidence suggesting a potential cross-acclimation effect between different environmental conditions. In fact, some studies have shown that heat acclimation can improve performance in cooler environments [54, 55]. Moreover, some research suggests that altitude acclimatization can be used as an ergogenic aid to improve sea-level performance [56, 57]. This means that certain adaptations to a particular environmental stressor can trigger responses that can enhance tolerance in another environmental stressor, as long as they share a common pathway for adaptation.

Although there have been some studies that investigated the use of heat acclimation as a tool to improve tolerance to hypoxia [58, 59], there has not been any research aimed at understanding the effects of altitude acclimatization on performance in hot environmental conditions. There are some benefits, as described previously, to altitude acclimation on exercise in thermoneutral, sea-level environments. On the other hand, some adaptations to altitude acclimatization may be detrimental to exercise in the heat, such as decreased cardiac output and skeletal muscle blood flow, secondary to a decrease in plasma volume.

Thermoregulation and Thermotolerance

Altitude acclimatization can have an effect on thermoregulation, which can certainly play a major role when exercising in the heat. As discussed previously, there is an increased hemoglobin mass that results from altitude acclimatization [52, 53]. Consequently, the increase in arterial oxygen content may allow a greater fraction of cardiac output to be directed to the cutaneous circulation because the oxygen transport requirements for a given level of submaximal exercise would be accomplished with a lower skeletal muscle perfusion [60]. Thermoregulatory responses via enhanced sweating may also be influenced by the increase in hemoglobin mass, which is observed after altitude acclimatization. One study suggested that increasing hematocrit (by 400 ml infusion of whole blood) resulted in an enhanced thermoregulatory response, which was due to enhanced sweating that decreased core temperature and reduced the skin blood flow response [61]. Finally, the study illustrated that exposures to a hypobaric hypoxic and warm environment (i.e., ten sessions over a 2-week period) enhanced the thermoregulatory response,

indicated by an increase in the forearm vascular conductance (FVC) in response to changes in esophageal temperature [62]. However, this FVC response could be an adaptation from the atmospheric pressure or temperature during the intervention exposures. Finally, it's important to keep in mind that the loss in plasma volume that accompanies altitude exposure [63, 64] may also impair the ability to thermoregulate by attenuating skin blood flow responses, particularly when exercising in hot environments.

Heat Shock Proteins

Heat shock proteins (HSPs), particularly HSP-72 and HSP-90, play key regulatory roles in protein transport across cell membranes, refolding denatured proteins, and preventing cell death in response to stressors [65]. The increased in HSP expression have been associated with enhanced cytoprotection, such that cells survive longer when exposed to heat stress [66]. There is a potential for altitude acclimatization and cross-tolerance to hot environments via enhanced HSP expression from studies done in humans and animal models [67, 68]. Taylor et al. [68] reported that 10 days of intermittent hypoxic exposure (75 min at ~3000 m of altitude) increased basal levels of HSP-72 in cells of eight healthy males. Likewise, a study done with animals (yak and rabbits) indicated that an enhanced HSP response to hypoxia may occur in altitude-acclimatized mammal [67].

The enhanced HSP-90 expression has been linked, at least in part, to the sodium-reabsorbing actions of aldosterone in the kidneys and sweat glands [69] that may lead to plasma volume expansion and reduced sodium content in sweat. HSP-90 regulates steroid hormone receptors and could therefore affect mineralcorticoid receptors in the sweat glands and kidneys to reduce sodium losses, leading to more dilute sweat and plasma volume expansion [65]. While there are no studies that firmly establish the relationship between HSP expression and systemic altitude acclimatization adaptations, these thermoregulatory adaptations via increased HSP-mediated pathways can potentially enhance exercise performance in hot environments.

Hypoxia-Inducible Factors

Altitude acclimatization increases hypoxia-inducible factors HIF-1α and HIF-1β. The enhanced HIF-1 pathways in various tissues in response to altitude exposures may initiate several of the altitude acclimatization responses such as an increased ventilatory response [70], increased erythrocyte production [48], enhanced angiogenesis via vascular endothelial growth factor (VEGF) [71], increased glycolytic enzyme expression [72], and enhanced vasodilatory mechanisms via inducible nitric oxide synthase (iNOS) to improve tissue perfusion [73]. Although the

relationship between the HIF-1 pathway and systemic responses in humans during exercise heat stress has not been explored, there is a potential for HIF-1 mediated changes to modulate some of the heat acclimation responses. HIF-1 induces angiogenesis (via VEGF) [71] and vasodilation (via iNOS) [73], which could increase maximal skin blood flow and sweating. These enhanced thermoregulatory responses can improve performance in the heat. Likewise, the HIF-1 mediated increased capillarization, glycolytic enzyme activity, and altered skeletal muscle metabolic rate could potentially be linked to the increased exercise performance and lactate threshold seen after heat acclimation [55].

HSPs and HIF-1 Cross-Regulation Pathways

There is evidence suggesting that HIF-1 can directly upregulate heat shock factor during hypoxia exposures, which leads to a further up-regulation of HSPs [74]. The cross-regulation between the HIF-1 and HSP pathways is perhaps the strongest piece of evidence that may suggest a potential for some physiological and cellular responses to chronic hypoxia that may carry over to improve tolerance and performance in hot environmental conditions. The cross-tolerance between hypoxic and heat acclimation via HIF-1 and HSP interactions have resulted in cytoprotective adaptations that can potentially lead to enhanced performance in hot environmental conditions [65].

Fluid Balance

Perhaps, one of the biggest challenges of the use of altitude acclimatization as a cross-tolerance model to enhance performance in the heat is the loss of plasma volume and total body water. This loss of plasma volume can have negative effects on overall cardiac function (leading to reduced muscle blood flow), as well as impaired thermoregulation (potential lower skin blood flow) during exercise heat stress. The fluid volume dynamics may depend on the altitude and duration of the acclimatization [63]. This loss in plasma volume takes place within 24 h of the hypoxic stimulus [64]. Although hemoglobin concentration and hematocrit increase, the total number or circulating red blood cells does not increase. In fact, a real increase in circulating number of red blood cells does not occur until 3–4 days. The loss of plasma volume can have a significant impact on cardiovascular function during exercise in hot environments. With a loss of plasma volume from altitude acclimatization, exercise in hot environmental conditions can further challenge the cardiovascular system because of the enhanced thermoregulatory demands for skin blood flow coupled with hyperthermia and dehydration, which may impair the adequate delivery of oxygen to working muscles [75].

Cardiovascular Function

It is clear that exercise in the heat imposes a greater cardiovascular and thermoregulatory strain compared with the same absolute exercise intensity in temperate environments. As previously mentioned, a decrease in plasma volume from altitude acclimatization could further increase this strain in hot environmental conditions. The cardiovascular adaptations from altitude acclimatization may vary with the degree and duration of the altitude exposure. Although a study reported no differences in submaximal and maximal cardiac output following 28 days of altitude acclimatization at moderate altitudes (2500 m) [56, 57], other studies have shown that following acclimatization at high altitude (3000–4500 m) there is a decline in cardiac output at rest and during submaximal and maximal exercise, secondary to a reduction in stroke volume [76–78]. Although these responses are multifactorial and variable, the reduction in stroke volume could be largely attributed to the reduction in plasma volume, although a study also suggested a diminished myocardial contractility [79]. The time course for recovery of stroke volume and cardiac output upon the return to sea level is not well understood although it seems that the restoration is not immediate [77]. The decreased cardiac output, stroke volume, and muscle blood flow can be accompanied by a higher arteriovenous oxygen difference (a-vDO_2), thus maintaining VO_2 despite the lower oxygen delivery [57]. Although these cardiovascular adaptations could be beneficial to exercise at altitude (reduced work of the heart, given the increase in blood viscosity due to elevated red blood cells and reduced plasma volume), it is not clear that they would transfer to exercise in hot environments.

Oxygen Transport and Delivery

As previously described, within 24 h of altitude exposure there is a decrease in plasma volume, which results in an increase in hemoglobin concentration and hematocrit [64]. The increase in total number of circulating red blood cells occurs within 3–4 days of altitude exposure. Hemoconcentration due to altitude exposure leads to an increase in arterial oxygen carrying capacity, so more oxygen can be delivered to the tissues at a given level of cardiac output. Although hypothetical, this adaptation may allow a greater fraction of cardiac output to be directed to the skin for thermoregulatory purposes during exercise in the heat.

Within a few hours of altitude exposure there is an increase in the concentration of 2,3-diphosphoglycerate (2,3-DPG), secondary to a rise in intracellular pH. The concomitant rightward shift in the oxygen-hemoglobin dissociation curve enhances oxygen release at the tissue level. This adaptation is observed after 3 days of living in a simulated altitude environment and training near sea level. However, 2,3-DPG returned to baseline values 1 day after elimination of the hypoxic stimulus [80]. Therefore, any potential for enhanced exercise performance in the heat is unclear.

Muscle Function

There are skeletal muscle adaptations following hypoxia that may affect muscle function during exercise heat stress. These include an increase in myoglobin content, a reduced diffusion distance between skeletal muscle fibers and capillaries (due to possible loss in muscle mass), decreased lactate production and enhanced lactate clearance, improved mechanical efficiency, and enhanced skeletal muscle buffering capacity [81]. The altitude acclimatization effects of skeletal muscle and its adaptations on exercise heat stress remains to be elucidated.

Performance

There have not been any studies that investigated the use of altitude acclimatization as a cross-tolerance model to improve exercise performance in the heat. Given the wide variety of adaptations to altitude acclimatization, some may be beneficial when exercising in the heat, whereas others may not. Potential benefits of altitude acclimatization include increases in blood hemoglobin concentration, increased hematocrit, elevated buffering capacity, and improvements in the structural and biochemical properties in skeletal muscle. Some other adaptations that may negatively impact exercise in the heat include decreased cardiac output and skeletal muscle blood flow, secondary to a decrease in plasma volume. Clearly, studies are warranted to elucidate the potential use of altitude acclimation as an ergogenic tool to improve exercise in hot environmental conditions.

The Application

The response of an athlete to altitude training is variable in both how acclimatization is achieved, as well as the impact on athletic performance. The extent of performance change and altitude adaptation is ultimately individualistic for each athlete; thus, training decisions may vary even within a team. To gain these adaptations as a mechanism to maximize performance and safety, altitude training models have been developed and researched with a wide variety of athletes (i.e., runners, cyclists, team-sport athletes.)

Other considerations for altitude training, such as time required at specific altitude, hydration planning, and timing of return for event performance must be considered to gain maximum benefit. The following discussion of these aspects will provide some of the many considerations for an athlete aiming to enhance both performance and safety while exercising in the heat. The information provided in Table 8.1 may be useful for maximizing performance and safety while exercising in the heat via altitude training.

Table 8.1 Tips for successful implementation of altitude training for heat safety

1. Utilize the live high, train-low model
2. Perform easy-moderate intensity exercise at moderate altitude
3. Perform moderate-high intensity exercise at low altitude
4. Heat acclimatize during training, either in a naturally warm environment or a simulated warm environment
5. If heat acclimatization is not possible during training, leave altitude with enough time to heat acclimatize closer to sea level
6. Hydrate appropriately before, during, and after exercise
7. Time the return to sea level either in the first few days, or more than 10 days prior to competition

Training Models

The three most common paradigms for altitude training include altitude exposure in daily living, exercise, or in combination. The live high, train-low (LHTL) model is the most commonly adopted altitude training model worldwide. A review of the LHTL model by Levine and Stray-Gundersen in 2006 describes two essential features for success with this model. A large number of these studies suffer from experimental design issues, most of which are in the area of adequate pre-altitude/hypoxia controls, such as group training effects, training camp effects, training levels prior to the altitude camp, and iron storage levels and supplementation [82]. Without these controls, it becomes difficult to partition out the variance in outcomes that is truly due to the effect of altitude training or hypoxic exposure, and what is due to some other factor.

One central issue to the concept of altitude training for sea-level performance is the ultimate effect of chronic training in hypoxia. For years, coaches, athletes, and many sport scientists have operated under the assumption that training in a hypoxic environment will augment the training response, particularly in muscle structure, vascular development, or metabolic pathways. Part of this belief stemmed from early reports (i.e., 1962) on miners who were permanent residents at high altitude had significantly greater cytochrome c reductase activity and myoglobin content compared to lowland residents [83]. Indeed, in animal models, hypoxic training has been shown to increase Type IIa fibers, as well as increase citrate synthase, hexokinase, and fatty acid β oxidation enzymatic activity [84]. In humans, a number of studies have shown similar improvements in Krebs cycle enzymatic activity with hypoxic training [85], and mitochondrial and capillary densities have been reported to be significantly increased after chronic training in hypoxia [86]. However, in most human studies that have shown significant skeletal muscle changes with hypoxic exercise, most have utilized unique protocols using a smaller muscle mass or training one leg in hypoxia and the other leg in normoxia, and often the same absolute workloads are used in hypoxia [1, 87]. During whole body dynamic exercise, like endurance running, training at altitude has been shown to limit exercise intensity and oxygen flux, which may actually promote deconditioning over time

[88–91]. Current evidence suggests that HIF-1a signaling in skeletal muscle displays only a modest response to hypoxia [92], and structural changes within skeletal muscle—over and above those that occur with training in normoxia—are less likely to occur when hypoxia is added to chronic exercise [87]. Therefore, athletes gain hematological benefits from chronic altitude exposure, but minimizing negative effects from hypoxic training is questionable.

The concept of LHTL altitude training, as proposed by Levine and Stray-Gundersen [57] theorizes that by chronic living at a moderate altitude, a significant increase in total hemoglobin mass will significantly increase maximal oxygen uptake. Moreover, by completing training at a lower altitude, training speeds and muscle capillary oxygen flux will be maintained near what they are at sea level, preserving the positive training response in the periphery to exercise [91].

In an elegant, well-controlled study, athletes who lived high and trained low daily for 4 weeks showed an improvement in VO_2max, maximal steady state, and 5000 m run time at sea level, where groups who lived and trained at moderate altitude (and a sea-level control), showed no change in 5000 m run time [57]. Subsequent iterations to the LHTL model have shown that similar performance improvements can be seen with going to low altitude only for high intensity workouts (i.e., workouts that are performed at a workload faster than the maximal steady state) [91, 93]. This finding makes completing the LHTL model of altitude training much more logistically convenient, as travel to low altitude only needs to be undertaken two to three times per week. Still, there are only a handful of locations where it is convenient to live above 2000 m, but be able to travel to near sea level in a short period of time. Since these initial studies, a number of laboratories from multiple countries have independently shown the advantage of the LHTL altitude training model, compared to traditional "live high—train high" altitude training (LHTH) [94–99]. A recent meta-analysis using terrestrial altitude studies has shown increases in maximal power of 4.0 ± 3.7% in elite athletes and 4.2 ± 2.9% in sub-elite athletes completing LHTL protocols, but a maximal power improvement only 1.6 ± 2.7% in elites and 0.9 ± 2.9% in sub-elites completing LHTH protocols [100].

Evidence on the live low, train high (LLTH) model describes minimal benefit to athletic performance. The LLTH model uses intermittent exposure to hypoxic environments, typically using artificial hypoxia, and rarely daily ascent. Either all or some of the athlete's training takes place at moderate or high altitude. A common assumption is that increased physiological stress from training at altitude would elicit a greater adaptation, thereby enhancing athletic performance. However, the literature on the LLTH model does not support its use for improving endurance performance [101]. Theories to describe this include: (1) artificially induced hypoxia is likely not as potent as a field environment; (2) brief exposure time does not meet the threshold for adaptation; (3) overall exercise intensity is lower at altitude, thus creating less of a training stimulus. The artificial intermittent exposure may be a primary factor in the limited exercise enhancement that comes from these modalities. As described previously in this chapter, the increased physiological and perceptual strain results in the inability to sustain high intensity exercise. Thus, if exercise intensity cannot be maintained, some detraining may occur, although the body may

overcome the short-term environmental stress without long-term adaptation in the LLTH model. In addition, LLTH models are typically carried out in laboratory settings, which may be impractical for athletes not near an environmental laboratory. Either all or some of the athlete's training may occur at altitude, but studies of both full and partial training at altitude have shown inconsistent performance benefit [101, 102].

Timing

Once a training model is selected, several other considerations for exercise training utilizing altitude must be made. Timing of the program, including time living at altitude, as well as return to sea level prior to competition may impact performance in the heat. The long-duration exposure of the LHTL model can be as brief as 4 weeks, training at low altitude only two to three times per week, can be used to gain performance benefit [57]. Timing of return to sea level should be planned to benefit from both improved performance and enhanced safety while exercising in the heat. Anecdotal evidence suggests that some period of maximum benefit for performance exists upon return from training or living at altitude for an event occurring at sea level. A review by Chapman et al. [103] used anecdotal evidence from elite endurance coaches that use altitude training regularly to describe this period of time. Generally, three periods of time were described to impact performance: first 48 h, 3–14 days, and 14 or more days. Athletes were permitted to perform either in the first 48 h, or 14 or more days following return from moderate altitude [103]. These periods of time were viewed as permissible due to the perceived performance benefits of the altitude training, but providing additional time to adapt to the sea-level environment. Also, some hematologic adaptations may remain elevated for 2–4 weeks post-return [4]. This may be of benefit to athletes aiming to improve performance in warm environments because heat acclimatization and proper hydration may be established in this time at sea level.

Fluid Balance

Fluid balance is commonly compromised during training at altitude due to the mechanisms described previously in this chapter. Maintenance of adequate hydration should be considered during training at moderate and low altitude. Hypoxia and hypohydration have been shown to independently cause reduction in aerobic time-trial performance, with an even greater reduction in performance in combination [104]. Thus, hypohydration may further limit altitude exercise performance described earlier in this chapter [88]. Rudimentary hydration indicators may be used over time to maximize safety and performance both at altitude and sea level. This may include estimation of sweat rate at a given work intensity, analysis of urine

specific gravity, volume, and frequency, and body mass change over time. Exercise at altitude, whether in a warm or cold environment, results in a similar sweat rate as the same exercise intensity, with additional respiratory water loss [104, 105]. Additional information about hydration assessment and fluid replacement can be found in Chap. 5.

To our knowledge, only one study has been done to specifically elucidate the impact of altitude acclimatization and training on exercise performance in a hot (presumably sea level) environment. A comparison of aerobic training in either hot or hot and hypoxic environments revealed that a performance benefit was only evident from training in a hot environment, without changes in hemoglobin mass, or perceptual measures of intensity [106]. Although theoretical links in physiological crosstalk theory are compelling, recommendations for maximal training benefit should be taken as such, theoretical. Based on expert opinion and physiological rationale, living or training at altitude may provide some benefit to thermoregulation and exercise performance, if fluid balance is well controlled.

Case Example *Perhaps one of the most well-known and commonly cited examples of altitude training for performance in the heat is from Deena Kastor (D.K.) in the 2004 Olympic marathon in Athens, Greece. Joe Virgil, a coach for D.K., described her training leading up to the 2004 marathon, including the use of altitude training to enhance physiological function in the hot, humid environment in Athens* [107].

Training for the event included typical race preparation training, and incorporation of a LHTH model, with only a small portion of training occurring at low or sea-level altitude. D.K. moved to Mammoth Lakes, CA (2400–3100 m) to set up training camp, and to focus the training program on altitude adaptation. Over a period of years, D.K. performed the majority of training at moderate altitude, and incorporated high altitude (~3000 m) training at frequent intervals. To practice the race course, D.K. and coaches utilized a course in Mammoth Lakes that was nearly identical in terrain to the Athens course, but at moderate altitude (2100–2400 m) instead.

Mammoth Lakes, CA is not a warm or humid environment like Athens, thus D.K. used other means to prepare for the heat in addition to her aerobic training. They anticipated that D.K. would need to prepare by acclimating to the heat, and hydrating appropriately. To provide some heat stimulus without a naturally warm environment, she created artificial warmth by wearing extra clothing during training to prevent some heat loss to the environment. To prepare for hydration maintenance in the warm environment, they utilized gut training and trained D.K. to take in fluid every 15 min to make hydration more regular and comfortable. Lastly, they anticipated that heat acclimatization would be necessary. They went to Crete, the largest Greek island, 2 weeks prior to competition to prepare. Crete provided two very necessary environmental components: altitude among several mountains and a hot-humid environment. These 2 weeks were likely enough to provide D.K. with appropriate heat acclimatization prior to the race without sacrificing her altitude adaptations.

The temperature on race day was 102 °F/38.9 °C, and 54% relative humidity at the start of the race; within the expected range. The race course had one 8-mile incline section in the second half; the peak at approximately 70 m, considered near sea level altitude. Overall, D.K. finished the race with a Bronze medal, and only 1:16 min away from her target time. The intense altitude training D.K. did leading up to the event was likely a major component to her Olympic success in 2004 [107]. *This case exemplifies the potential impact and benefit of utilizing altitude training to enhance performance in warm environments. When utilized with other elements, such as hydration and exercise heat exposure, altitude training can benefit performance in warm environments.*

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Part III
Tools Available to Assist

Chapter 9
Environmental Condition and Monitoring

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The Physiology

Human Heat Balance Modeling

A complex balance sets up between the human body and the ambient environment during physical activity in extreme heat, with the physiological responses and impacts to heat well documented for adults populations [1, 2]. When compared to sedentary, indoor conditions that are relatively static, modeling the human heat balance under changing activity levels and varying ambient environments (i.e., weather conditions) becomes increasingly complex. These factors increase the difficulty in accurately modeling the human heat balance as additional avenues of heat gain, such as metabolic and radiant heat loads, must be considered.

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Human energy balance models are commonly used to establish the effects of the fluxes of energy, as listed in Eq. (9.1), that either add heat or remove heat from the body, effectively balancing to give a surplus (+S), deficit (−S), or a balance ($S = 0$) of energy storage.

$$0 = \Delta S + M + K + R \pm C - E \tag{9.1}$$

ΔS is the energy sum (or storage) per time, changing constantly with environmental and metabolic conditions; hence, we use the change in heat storage over a set time period. Under moderate-weather outdoor conditions, the heat gains include the metabolic heat flux (M), conductive heat flux (K), and net radiation (R) experienced by a human, while the losses include convective heat loss (C) and evaporative heat loss (E) [3, 4]. Under extremely high temperatures, the air temperature will rise above the skin temperature, thus convection will add heat to the budget. All energy fluxes are expressed in watts per meter squared of a human's surface area (W m^{-2}) (the average human's surface area is 1.8 m^2; [5]). From Eq. (9.1), however, it follows that air temperature alone is seldom the reason for heat stress. There are six key factors that combine to determine heat stress or strain to a given individual, which include environmental parameters (air temperature, radiation, humidity, and wind velocity) and behavioral parameters (metabolic rate, clothing) [6].

Under extreme heat conditions in conjunction with high metabolic activity, the environment experienced by the athlete is far from the "normal", static conditions under which many energy balance models were originally tested. For example, metabolic heat production for a sitting person is 58.15 W · m^{-2} (1 MET), yet during exercise, M can rise above 14 METS (or 814 W · m^{-2}), thus becoming the largest energy gain that drives heat stress during physical activity [6]. This heat production by the body is directly linked to an increase in core body temperature (T_c), with the rise in T_c accelerated under hot conditions during which the body is either limited or prevented from dissipating heat to the environment, and thus the body can no longer remain at an equilibrium T_c of ~37 °C/98.6 °F. As shown by Eq. (9.1), the energy balance is controlled by simultaneous transfers of heat to and from the body, which are simplified using empirical, direct, or rational indices.

Common Heat Indices

Heat stress indices are used to predict the physiological strain from stressful environmental conditions in combination with higher activity levels and often provide a single value to represent the interactions between humans and basic weather parameters. Three types of indices exist:

1. Empirical: developed from human studies based on physiological response of exposure to various factors of metabolic and environmental loads; tested under varied conditions.
2. Direct: employs measurements of weather conditions in order to infer the thermal environment that a human experiences with appropriate, simple, and

practical guidelines accounting for intensity and clothing. Direct indices are often used in epidemiological studies and do not assess physiological responses.
3. Rational: based on a complex and integrated combination of environmental and physiological variables, employing the heat balance equation in a more complex manner than direct or empirical indices.

Here, the most common indices used in sports and physical activity settings in North America are presented. Classic reviews of the history, limitations, and nature of over 50 indices are available elsewhere [4, 7–10].

Wet Bulb Globe Temperature Index

Created 60 years ago by Yaglou and Minard [11], the outdoor wet bulb globe temperature index (WBGT) is the most widely used direct heat index today, applied in athletics, occupational safety, and the military. The WBGT was originally developed to control outbreaks of heat illness in training camps in the US Army and Marine Corps [8, 11]. It was adopted by the American College of Sport Medicine (ACSM) in the 1980s and is largely used across sporting entities to define extreme heat limits, with recent developments of variable thresholds for acclimatization and clothing [4, 12, 13]. The index integrates the influences of air temperature, humidity, mean radiant temperature, and wind speed and applies a weighted average between the natural wet bulb temperature (T_w), dry bulb temperature (T_a), and globe temperature (T_g) as follows:

$$\text{WBGT} = 0.7T_w + 0.2T_g + 0.1T_a \tag{9.2}$$

Although it is the most widely used heat stress index, historical limitations of the WBGT have been presented that many users are unaware of [8], with literature pointing to a combination of the limitations (listed in Table 9.1) potentially causing an increase in exertional heat stroke (EHS) from organizational and treatment failings [14], the misuse (or non-use) of the WBGT, and the recommendation that improvements are needed in the weightings of the variables under certain climatic conditions [8].

Heat Index and Humidex

The heat index (HI) and the humidex (HX) use inputs of air temperature and humidity to convey a "feels like" temperature to the public. The HI is a simple hot weather version of the apparent temperature—an index based on human heat balance model developed in the United States by Steadman [15]. The HI is widely used by the U.S. National Weather Service for heat warnings and advisories. The HI has been used at times for heat stress prediction in athletes (e.g., Falmouth Road Race [16]; New York State Public High School Association 2016 [17]; Oregon School Activities

Table 9.1 Comparison table of the common heat indices presented in this chapter used to predict heat stress during sports and physical activity [6, 11, 15, 19, 22, 23, 25–28]

Index and citation	Equation, type, inputs	Benefits	Drawbacks
Wet bulb globe thermometer (WBGT) Yaglou and Minard (1957) [11]	Equation (9.2) Direct index T_a, T_w, T_g	– Most widely used – Basis for International Standards and organizations, defined for reference environmental and personal conditions – Permissible heat exposure thresholds limits are published widely, used in practice, and adjusted for clothing, work rate, and acclimatization	– Often calculated without T_g due to measurement difficulty – Only uses environmental conditions before adjustments – Low sensitivity to cooling effect of moving air – Conservative and thus can be often ignored – Effects of metabolism and clothing not included in model – Non-standardized instrumentation often used
Heat index (HI) Steadman (1979) [15] Humidex (HX) Mastterton and Richardson (1979) [19]	HI: Simplified rationale index from the apparent temperature HX: Direct index T_a, RH/dew point	– Widely used for public messaging of extreme heat – Works well for general population	– Not created or tested for athletes or workers functioning at high metabolic rates – Underestimates heat stress in sun and at high metabolic heat loads – Variations in equations exist
Environmental stress index (ESI) Moran et al. (2001) [22]	Equation (9.3) Empirical index RH, T_a, SR	– Correlated with the WBGT – Tested in hot/dry and hot/humid conditions – Fast response to conditions with pyranometer	– Not correlated with any physiological variable that reflects strain – Often difficult and/or expensive to measure onsite solar radiation – Effects of metabolism and clothing not included – Correlation well with HR, T_{re}, and PSI (Moran et al. 2001) [22] – Only uses environmental conditions
Heat stress index (HSI) Belding and Hatch (1955) [23]	Equation (9.4) Rational index T_a, RH, R, M, windspeed	– Considers all environmental factors and work rates, including radiative exchanges – A good indicator of sweat rate – Led to development of PSI	– Based on constant skin temperature of 35 °C/95 °F – Difficult to use in practice (collect all data for) – At 100% HSI, no convective heat loss assumed – Rational analysis often indicates safe continuation of sports even with HSI > 100% (Brotherhood 2008) [6]

(continued)

Table 9.1 (continued)

Index and citation	Equation, type, inputs	Benefits	Drawbacks
Physiological strain index (PSI) Moran et al. (1998) [25]	Equation (9.5) Empirical T_{re}, HR	– Represents the combined strains of the thermoregulatory and cardiovascular systems – Easily calculated – Discriminates among effects of hot, hot-humid, and normal conditions – Tracks heat strain across work-rest cycles	– Difficult to measure T_{re} – Does not incorporate environmental parameters – Conditions exist where the PSI is significantly under- or over-estimated (Belding and Hatch, 1955) [23]
Thermal work limit Brake and Bates (2002) [26]	Derivatives of Eq. (9.1) T_a, RH, T_g (or R), wind speed	– Less complicated than other indices – Does not require a special interpretation or knowledge	– Not widely validated – Assumes a simple metabolic working rate can be maintained – Mainly tested for occupational purposes
UTCI Bröde et al. (2012) [27] Fiala et al. (2013) [28]	Multivariate mathematical model Rationale index T_a, v_w, RH, radiant temperature	– Validated by many studies across climatic conditions and for relevant combinations of weather and climate parameters – Very sensitive to changes in ambient environment – Automatically alters clothing based on T_a	– Incorporates one metabolic heat load (2.3 METS at 4 km · h^{-1} activity speed), and thus the output provided is only sensitive to the environmental changes – Not applicable to high metabolic heat loads – Clothing model is based on seasonal European clothing

Association 2016 [18]). In a similar sense, the HX was also developed for warnings and advisories in Canada [19], and is used in some locations within Europe. Neither index was developed for use at high metabolic output, and thus makes a number of assumptions that may lead to underestimation of heat stress for athletes and other active individuals, including that the person is shaded, has a low activity level, and is not heavily dressed [20]. For example, the HI may underestimate heat exposure by 8.3 °C/15 °F in full sun, as the solar radiation is not an input value [21].

Environmental Stress Index

The environmental stress index (ESI) is an empirically derived index, purposed to be the first index directly measuring solar radiation (SR) at a high response time, and also a valid alternative to the WBGT. Introduced in 2001 by Moran et al. [22], it was validated using large databases of physiological tests in the heat showing high linear correlation with both the WBGT and the Physiological Strain Index (PSI), as

well as with heart rate (HR), rectal temperature (T_{re}), and SR [22]. The ESI is based on measurements of T_a, relative humidity (RH), and SR as follows:

$$\mathrm{ESI} = 0.63T_a - 0.03\mathrm{RH} + 0.002\mathrm{SR} + 0.0054\left(T_a \cdot \mathrm{RH}\right) - 0.073\left(0.1 + \mathrm{SR}\right)^{-1} \quad (9.3)$$

Heat Stress Index

The Heat Stress Index (HSI) is a rational index devised in 1955 by Belding and Hatch [23] and represents the ratio of evaporation required to maintain heat balance (E_{req}) to the evaporative capacity of the environment (E_{max}). Because the E_{req} is dependent upon the human energy balance, it follows that all energy fluxes in Eq. (9.1) much be known. Hence, the $E_{req} = M + R + C$, indicating the amount of evaporative heat loss required to achieve thermal energy balance. Through knowledge of both E_{req} and E_{max} (based largely on the vapor pressure of air and windspeed; [24]), the HSI can be calculated as follows:

$$\mathrm{HSI}(\%) = \frac{E_{req}}{E_{max}} \cdot 100 \quad (9.4)$$

The resultant value is between 0 and 100, with an HSI of 100 representing the most oppressively humid conditions due to the required evaporation reaching the maximum possible evaporation (i.e., $E_{max} = E_{req}$), and thus heat loss cannot occur via sweating. Again, a similar limitation to that of the ESI exists, where the input variables for E_{req} and E_{max} are difficult to measure and/or calculate, and thus the HSI is difficult to use in practical real-world conditions.

Physiological Strain Index

The physiological strain index (PSI) is an empirical index developed by Moran et al. [25], based on rectal temperature (T_{re}) and heart rate (HR). Using Eq. (9.5), the index rates the physiological strain from 0 to 10, and is based on the assumption that a T_{re} rise of 3 °C (36.5–39.5 °C) (5.4 °F [97.7–103.1 °F]) and HR rise of 120 bpm (60–180 bpm) results in maximal heat stress.

$$\mathrm{PSI} = 5\left(T_{re_t} - T_{re_0}\right) \cdot \left(39.5 - T_{re_0}\right)^{-1} + 5\left(\mathrm{HR}_t - \mathrm{HR}_0\right) \cdot \left(180 - \mathrm{HR}_0\right)^{-1} \quad (9.5)$$

where T_{re_t} and HR_t are measurements taken at any time, t, throughout a measurement period, and T_{re_0} and HR_0 are the starting measurements taken at $t = 0$. Although PSI is a convenient index for clinical use, the assumption of HR range in the formula may warrant clinicians to interpret the output differently depending on the

exercising individual's age since cardiovascular capacity can be influenced considerably by age.

Thermal Work Limit

A further rationale heat stress prediction tool is the thermal work limit (TWL), defined as the "limiting (or maximum) sustainable metabolic rate that euhydrated, acclimatized individuals can maintain in a specific thermal environment, within a safe deep body core temperature (<38.2 °C/100.8 °F) and sweat rate (<1.2 kg · h^{-1}) [26]." The model itself derives from the use of Eq. (9.1) by setting the sum to equal the metabolic heat load based on the established T_c and SR limits provided above (thus the M = TWL at specified limits). Largely used in occupational settings, it is purposed for the protection of workers under high metabolic loads in hot environments, and allows both thermal strain and productivity decrement due to heat (or a reduced work rate) to be quantitatively assessed [26]. It is often used in conjunction with the WBGT index, and is an output given on the Kestrel WBGT heat stress meter (Nielsen-Kellerman, Boothwyn, PA, USA). Due to the model complexity, it is often calculated with a sensor using environmentally measured inputs with the T_c and SR limits set.

The Universal Thermal Comfort Index

Created in Europe by the COST Action 730, the universal thermal comfort index (UTCI) is a dynamic multivariate rationale model—which refers to the time dependency of the physiological responses prior to reaching a steady state [27]—providing an output value in °C that compares a response environment to a reference environment across a range of T_a (−50 to +50 °C/−58 to +122 °F). The reference environment is based on a constant metabolic heat load (2.3 METS or 135 W · m^{-2}), wind speed (0.3 m · s^{-1} at 1.1 m), walking speed (4 km · h^{-1}), radiant temperature equal to T_a, and a RH of 20% with vapor pressure capped at 20 hPa for T_a > 29 °C/84.2 °F). The final output of the model is determined by comparing the actual model human response to the reference conditions across the T_a range, and determining the offset from the reference line (thus the UTCI final output value is the T_a plus offset value, in °C). The response function is based on a linear combination of 7-indicators of thermal strain (e.g., sweat rate, core temperature), and the simple output values will range from extreme heat stress to extreme cold stress [27, 28]. In the original UTCI model, the metabolic heat and activity speed are held constant (2.3 METS at 4 km · h^{-1}, respectively), thus the model is highly sensitive to the environmental changes, and in a practical sense would only be applicable to people walking at the given MET and speed (e.g., coach, spectator), and thus not applicable to heat stress in athletes.However, recently Brode et al. have expanded the UTCI model to consider varying metabolic intensity, clothing, and exposure times. These updates may expand the potential use of this model in athletics [29].

The Evidence/Epidemiology

The complex interplay of multiple risk factors (e.g., health condition, physical fitness, type and intensity of activity, availability of rest breaks) in developing heat injuries are critical factors to consider; however, limitations in the prediction of heat illness arise when these are not considered and one assumes accurate prediction of heat injury risk solely from environmental conditions (i.e., weather parameters). However, environmental monitoring has its advantages in predicting the potential risk ahead of the time based on weather forecasts, allowing organizations and individuals to modify the planned activity or behaviors in an effort to mitigate such risk. Several well-documented epidemiological and case studies in athletics and military have suggested the associations between environmental conditions and heat injury occurrences [16, 30–33]. These data provide the evidence to support the use of environmental guidelines to control for organizational extreme heat risk, especially in the context of mass participation events, organized sport practices and games, and military training.

U.S. Marine Corps was the first organization to strategically monitor WBGT to assess the heat injury risk during training. They conducted an epidemiological study documenting the exertional heat illness (EHI) incidents during 1982–1991 at Parris Island, South Carolina, revealing that the number of EHI incidents rose substantially at WBGT index values (Eq. 9.2) greater than 18.3 °C/65 °F [30]. Furthermore, the EHI incidents increased 26-fold when compared to the incident rates reported under WBGT 18.3 °C/65 °F when the WBGT value was between 23.9–26.7 °C and 75–80 °F, which is the border line threshold to implement activity modification in the military environmental flag system (i.e., green flag) [34]. Case series by Ravacha et al. [31], which documented six Israeli Defense Force EHS fatalities reported that five of the six cases occurred at a WBGT ≥27 °C/80.6 °F, or a heat load corresponding to the "green flag" threshold or above. A similar environmental threshold was observed in the EHS fatalities documented in American football athletes across the contiguous United States, where the median value of the WBGT from the incidents that took place in the morning and afternoon practice sessions were 27.7 °C/81.9 °F and 30.0 °C/86 °F, respectively [32]. Modifications to work to rest ratio and clothing, as suggested by the WBGT activity modification by the American College of Sports Medicine (ACSM; see section "The Application") may have played a key role in preventing the above-mentioned fatalities, if adhered [13]. Cooper et al. [33] studied the association between EHI—including heat cramps, heat exhaustion, and heat syncope—and the recorded WBGT among Division I university football programs across the United States. In this cohort, the rate of heat illness per 1000 athlete-exposure (AE) increased by sixfold (1.44/1000 AE) when the WBGT was 27.8–28.9 °C/82–84 °F (recommended threshold to limit intense exercise by the ACSM guideline) compared to WBGT 24.5–25.6 °C/76–78 °F (recommended threshold to decrease total duration of activity by the ACSM guideline).

A study on runners at the Falmouth Road Race, an 11.3 km summer road race in Falmouth, Massachusetts, presents the largest dataset on EHI and EHS incidents related to ambient weather conditions, using the T_a and the RH to further calculate the HI [16]. The Falmouth Road Race medical tent admission data from a total of 18 years were analyzed against the weather conditions in the respective year. T_a explained 65% and 75% of EHS and EHI incidents per 1000 finishers, respectively, and RH explained 14% and 22% of EHS and EHI incidents per 1000 finishers, respectively. The HI, which combines the influences of temperature and relative humidity, had the strongest correlation, explaining 74% of EHS and 75% of EHS and EHI incidents per 1000 finishers, respectively.

Most large-scale epidemiological studies on EHS and EHI focus on the number of incidents encountered in a population during a given time period, but fail to report confounders (external variables correlating with the outcome) beyond the basic demographics of the patient (e.g., age, sex, body size). Further epidemiological studies that report heat casualties or illness with environmental conditions as well as personal and temporal factors are warranted to further define the most accurate environmental threshold that places individuals to heat injury risk.

The Application

Heat safety guidelines for activity modification utilize both environmental and non-environmental information (e.g., type and intensity of activity; individual factors such as health, fitness, and acclimatization; and amount of clothing or equipment) that can influence risk factors for heat illnesses [35] (see Table 9.1). In athletics, the WBGT is the most commonly used measure of heat exposure. The ACSM has developed a widely used set of WBGT heat safety guidelines that accounts for the non-environmental factors by creating separate recommendations for continuous activity (e.g., road race) and non-continuous activity (e.g., football, tennis, soccer), which is in turn subdivided into categories for heat sensitive individuals (i.e., non-acclimatized, unfit, and/or high risk) and those who are less heat sensitive (i.e., acclimatized, fit, and low-risk). Thresholds for activity modification are lower for continuous activity and for heat sensitive individuals. For example, the "cancel level" for continuous activity and competition is a WBGT of 27.9 °C/82.1 °F, 30.1 °C/86.1 °F for non-continuous activity involving non-acclimatized, unfit, or high-risk individuals, and >32.3 °C/90.1 °F for non-continuous activity involving acclimatized, fit, or low-risk individuals. For less oppressive conditions beginning at a WBGT of 22.3 °C/72.1 °F, the ACSM recommends adjusting rest-to-work ratios and total duration/intensity of activities for more heat sensitive individuals and beginning at 27.9 °C/82.1 °F for acclimatized, fit, and low-risk athletes. In establishing these policies, the ACSM assumes athletes are wearing shorts, T-shirt, socks and sneakers, and that acclimatized individuals have trained in heat for at least 3 weeks.

Particular athletic governing bodies employ a variety of thresholds for canceling or adjusting activities depending on the nature of the sport as well as the gender and age of the athlete [36]. The International Tennis Federation for junior tennis players, and the World Tennis Association for female tennis players employ the ACSM recommended participation cutoff of 32.3 °C/90.1 °F WBGT, and require increased rest breaks between sets when the WBGT reaches 30.1 °C/86.1 °F. The Federation Internationale de Football Association for soccer players requires additional cooling breaks at 30 and 75 min at WBGTs ≥32.3 °C/90.1 °F. Finally, a set of WBGT activity modification guidelines have been developed with specific recommendations for American football players who wear protective equipment (Table 9.2) [37]. At WBGTs of 30.6–32.2 °C/87.1–90.0 °F players are restricted to helmet, shoulder

Table 9.2 Regional heat safety activity guidelines based on the Georgia High School Association policy

Cat 3	Cat 2	Cat 1	Activity guidelines
<27.8 °C/82.0 °F	<26.5 °C/79.7 °F	<24.5 °C/76.1 °F	Normal activities—Provide at least three separate rest breaks each hour with a minimum duration of 3 min each during the workout
27.9–30.5 °C/82.2–86.9 °F	26.6–29.2 °C/79.9–84.6 °F	24.6–27.2 °C/76.3–81.0 °F	Use discretion for intense or prolonged exercise; watch at-risk players carefully. Provide at least three separate rest breaks each hour with a minimum duration of 4 min each
30.6–32.2 °C/87.1–90.0 °F	29.3–30.9 °C/84.7–87.6 °F	27.3–28.9 °C/81.1–84.0 °F	Maximum practice time is 2 h. For football: Players are restricted to helmet, shoulder pads, and shorts during practice. If the WBGT rises to this level during practice, players may continue to work out wearing football pants without changing to shorts. For all sports: Provide at least four separate rest breaks each hour with a minimum duration of 4 min each
32.3–33.3 °C/90.1–91.9 °F	31.0–32.0 °C/87.8–89.6 °F	29.0–30.0 °C/84.2–86.0 °F	Maximum practice time is 1 h. For football: No protective equipment may be worn during practice, and there may be no conditioning activities. For all sports: There must be 20 min of rest breaks distributed throughout the hour of practice
≥33.4 °C/92.1 °F	≥32.1 °C/89.8 °F	≥30.1 °C/86.2 °F	No outdoor workouts. Delay practice until a cooler WBGT level is reached

Values are wet bulb globe temperatures (°C/°F) [41]. Conversions were made from °C to °F and rounded to the nearest whole number. Reprinted from Applied Geography, Vol. 56, Grundstein A, Williams C, Phan M, Cooper E, Regional heat safety thresholds for athletics in the contiguous United States pages 55–60, © 2015, with permission from Elsevier

pads and shorts during practice but must remove all protective equipment for WBGTs ≥32.3 °C/90.1 °F.

Within occupation safety settings, WBGT threshold limit values (TLVs) are employed based on categorically defined workloads, including "resting", "light", "moderate", "heavy", and "very heavy" [38]. As metabolic heat production increases with activity, the allowed exposure to a WBGT value decreases. The limits for acclimatized workers are similar among different agencies and organizations, and range from 32.0–33.0 °C/89.6–91.4 °F for resting to 23.0–25.0 °C/73.4–77.0 °F for very heavy work [38]. The above TLVs may be adjusted to account for clothing ensembles that may inhibit cooling relative to the assumed lightweight summer working clothing. For instance, the TLV for someone working in double layer cloth clothing would decrease by 3 °C/5.4 °F relative to someone in the baseline work clothing ensemble [38].

The US military employs a similar procedure in their WBGT-based recommendations for activity. They use five WBGT heat categories and three workloads defined as "easy work" (e.g., marksmanship training), "moderate work" (e.g. calisthenics, patrolling), and "hard work" (e.g. field assaults; walking with heavy loads) [39]. The allowed work load is varied depending on the heat category. For instance, for WBGTs between 27.8–29.4 °C and 82.0–84.9 °F; ("green flag") there is no activity limit for easy work, 10 min per hour of rest required for moderate work, and 30 min of rest per hour for hard work. The guidelines are established for acclimatized soldiers wearing battle dress uniforms but correction factors can be applied to WBGTs for soldiers wearing body armor or other protective clothing to account for increased heat strain posed by reduced cooling.

As demonstrated, the WBGT is used in various regions around the world, yet due to variations in climates regimes, a universal set of WBGT activity guidelines may not be appropriate for all locations, as individuals acclimatize to the environments where they work or train. An athlete from Minnesota experiencing a typical August day in Georgia, for instance, would not be acclimatized to such heat, and thus extra precautions and time to acclimatize are needed. Therefore, WBGT activity modification thresholds should be adjusted based on local climate conditions. As an example, the ACSM recommends canceling continuous activities for WBGTs ≥27.9 °C/82.2 °F but some literature indicates that this may be too high for marathons in regions with cooler climates where athletes may not be acclimatized to hot conditions. Empirical research suggests that in areas poleward of 40° latitude, marathons should be canceled with lower starting WBGTs ≥21 °C/69.8 °F [40]. For non-continuous activity, Grundstein et al. [41] established three activity modification categories for areas in the contiguous United States with hot, moderate, and mild climate-based regional extreme WBGTs (Tables 9.2 and 9.3). The hot climate areas would continue to employ the ACSM [13] thresholds, the moderate climate areas would shift thresholds down 1.3 °C/2.3 °F and the cancel conditions from 32.3 °C/90.1 °F to 31 °C/87.8 °F and the mild climates would shift thresholds down by 3.3 °C (5.9 °F) and cancel conditions to 29 °C/84.2 °F. Similar adjustments are made to the football-specific WBGT heat safety (Table 9.2) [37].

Table 9.3 Regional heat safety guidelines for low-risk acclimatized individuals based on American College of Sports Medicine guidelines

Cat 3 (ACSM 2007)	Cat 2	Cat 1	Activity guidelines
≤10.0 °C/50 °F	≤8.7 °C/47.7 °F	≤6.7 °C/44.1 °F	Normal activity
10.1–18.3 °C/50.2–64.9 °F	8.8–17.0 °C/47.8–62.6 °F	6.8–15 °C/44.2–59.0 °F	Normal activity
18.4–22.2 °C/65.1–72.0 °F	17.1–20.9 °C/62.8–69.6 °F	15.1–18.9 °C/59.2–66.0 °F	Normal activity
22.3–25.6 °C/72.1–78.1 °F	21.0–24.3 °C/69.8–75.7 °F	19.0–22.3 °C/66.2–72.1 °F	Normal activity, monitor fluids
25.7–27.8 °C/78.3–82.0 °F	24.4–26.5 °C/75.9–79.7 °F	22.4–24.5 °C/72.3–76.1 °F	Normal activity, monitor fluids
27.9–30.0 °C/82.2–86.0 °F	26.6–28.7 °C/79.9–83.7 °F	24.6–26.7 °C/76.3–80.1 °F	Plan intense or prolonged exercise with discretion
30.1–32.2 °C/86.2–90.0 °F	28.8–30.9 °C/83.8–87.6 °F	26.8–28.9 °C/80.2–84.0 °F	Limit intense exercise and total daily exposure to heat and humidity
>32.3 °C/90.1 °F	>31.0 °C/87.8 °F	>29.0 °C/84.2 °F	Cancel exercise

Values are wet bulb globe temperatures (°C/°F) [41]. Conversions were made from °C to °F and rounded to the nearest whole number. Reprinted from Applied Geography, Vol. 56, Grundstein A, Williams C, Phan M, Cooper E, Regional heat safety thresholds for athletics in the contiguous United States pages 55–60, © 2015, with permission from Elsevier

Case Example *The Georgia High School Association (GHSA) provides an excellent case study of how a governing body developed an empirically based set of heat safety guidelines. The GHSA was prompted to action after a series of tragic and well-publicized deaths of high school football players and as being identified as among states with the greatest number of football hyperthermia deaths* [32].

Historically the GHSA had minimal restrictions regarding the nature of practice sessions (i.e., type, timing, length of practice, clothing). The original policy regarding practice for fall football included a specific starting date for practice and guidelines for the type of equipment worn during the first 5 days of practice. However, there were no stipulations on the length of practice and number of practice sessions held during a single day, nor stipulations regarding hydration/rest breaks or a means to evaluate the environmental conditions. Given the vagueness of the practice policy within the GHSA practice policy guidelines, it was not uncommon for high schools to hold multiple sessions per day (in some cases as many six sessions per day) and schedule practice sessions lasting in excess of 3 h.

As a result of this situation, the GHSA approached a group of researchers to help them address this problem of heat-related deaths. After a discussion that included administrators from the GHSA, coaches in the field, and the members of the rules committee, it was agreed that the following questions needed to be addressed:

- *Should the current rule be modified?*
- *What is a reasonable amount of time before players begin wearing equipment (pads) in practices?*
- *What should be the time interval between multiple practice/session days?*

- *What should be the duration of a practice session related to EHI risk?*
- *Who are the vulnerable student-athletes when compared to EHI risk?*
- *What is the best environmental variable to use when evaluating heat stress—WBGT or HI?*

A consensus was formed by all parties who had a vested interest in this project to share information and knowledge in order to make the best decision, to learn from each other's experiences, and to have open discussions/debates to confirm any decisions/policy creation that are made. Experts were contacted from other state high school organizations (Texas and Arkansas) as well as prominent researchers who had expertise in EHI prevention, bringing everyone together for an EHI summit to consider the best approach for addressing these issues and develop a comprehensive plan that would mitigate EHI risk.

Methods

An epidemiological study was conducted for three consecutive football seasons (2009–2011) utilizing high schools who represented different regions in the State of Georgia. The state was divided into five geographical regions (North, Central, Southeast, Southwest, and Metro Atlanta) with five schools representing each region. To ensure that participation numbers were consistent, each region was represented by schools that had equivalent enrollment numbers. The National Athletic Trainers' Association (NATA) Position Statement on EHI was used to define EHIs [35]. A reportable EHI was any heat illness event that resulted in a loss of participation or required an evaluation by a member of the athletic medical staff. A reportable athlete-exposure (AE) was based on the National Collegiate Athletic Association criteria and defined as an individual participating in one-team session (practice or game) [42]. A full-time athletic trainer was identified for each participating institution, and they were responsible for documenting all EHIs and exposures using a standardized computerized form.

It was decided that the WBGT would be the required method to assess environmental measures rather than the HI. This decision was based on organizations such as the NATA, ACSM, American Pediatric Association, and the United States Department of Defense whom all use the WBGT and it is widely accepted as the gold standard for environment measure for EHI/EHS prediction. Each participating school was given a WBGT weather monitor (QuestTemp-34, Quest Technologies, Oconomowoc, WI, USA), which recorded weather data throughout the duration of practice. WBGT values were recorded at the specific time an EHI occurrence was documented. Data collection periods ran from August 1 through September 30, which represented the reporting period. In addition to the environmental data, player characteristics (e.g., age, grade level, body mass index), equipment worn, EHI types, and type of practice (e.g., conditioning, practice, scrimmage) were documented for analysis. EHI rates were calculated for practice days, duration of practice, and WBGTs. At the conclusion of the third season, the researchers convened with the leadership of the GHSA to review the data.

Outcome

Results from the epidemiological study showed that the greatest risk for EHI was during the first 5 days of practice and when practice sessions lasted longer than 2 h during the same 5 days. Notable increases in EHI risk were observed when there were multiple practice sessions in a given day as the environmental conditions became more stressful (i.e., higher WBGT reading), which suggested the implementation of the work to rest ratio recommendations. As a result, the GHSA developed stringent practice policies for hot weather focusing on heat acclimatization, practice length, and numbers of sessions per day, including work to rest ratios and a WBGT index specifying practice modifications for respective observed environmental conditions. The GHSA utilized the data collected from the first 3 years of the project. Through collaborative discussions, they implemented all of the suggested practice policy changes to create a new and comprehensive pre-season practice policy that address a variety of player-safety issues [37]. Furthermore, GHSA observed a dramatic reduction in the EHI incident rates during the 3 year post the implementation of the WBGT guideline. The findings from this project provide clear evidence that policy change can be implemented when empirical evidence is produced to support such changes. Hard epidemiological data connected to weather conditions can tell the story to a skeptical audience (coaches and administration) by using facts to support policy change. Utilization of this type of approach eliminates the need for "expert opinions" and eliminates any bias by special interest groups. In addition, the researchers of the Georgia Project saw the need to be flexible in designing new policy and rules—seeing the bigger picture for sport safety. Through the involvement from all parties involved, there was an equal sharing of governance and ownership in the policy-making process. Change and revision is based on a partnership that is built on trust and through regular communications a positive outcome is achievable (Table 9.4).

Table 9.4 Ten steps for successful implementation of heat guidelines

1.	Create a working partnership with all interested parties (coaches, administration, medical)
2.	Explore what steps others have taken to mitigate risk
3.	Review the literature—determine the best parameters to explore
4.	Share individual knowledge to make the best decision regarding player safety
5.	Determine what areas are in need of revision and how they may have a positive impact
6.	Create methodology to collect data to support any new policy revisions
7.	Utilize certified athletic trainers to help with data collection and determine best application of possible policy changes
8.	Make recommendations regarding policy change based on collected data (3 year minimum to establish any trends)
9.	Once new guidelines are established, follow-up with results analysis after 3 years of data collection
10.	Communication … Communication … Communication

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Chapter 10
Technology

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Introduction

Recent advances in technology and algorithms related to the continuous monitoring of physiological variables (e.g., hydration status, heart rate, internal body temperature, and skin temperature) and variables associated with training load (e.g., Global Positioning System [GPS] and accelerometry devices) has enabled the coach, clinician, and researcher to generate a great deal of useful data that can be used to monitor and optimize performance during exercise in the heat and prevent injury or illness. The use of technology is rapidly advancing in the field of exercise science, sports medicine, and military, with the sole intent of being more informed so that data-driven decisions can be made to optimize performance and safety on the field, battlefield, or in the laboratory. Major areas where technology is currently being used to improve performance during exercise in the heat are in the areas of hydration and sleep, thermal strain, or internal body temperature (direct and indirect

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methods), heart rate monitoring, and training load monitoring. When using performance monitoring technology, it is critical for the end-user to understand the variables, metrics, and outputs and well as the limitations of these systems so that useful information can be garnered. Although much of the information presented in this chapter reference team sport and military athletes, the technological advances presented in this chapter can be used and applied to almost any setting where heat and hydration monitoring may be necessary.

Measuring Hydration Using Wearables: Technology and Challenges

Hydration and fluid homeostasis is a complex and dynamic physiologic process that depends on various factors such as circadian rhythm, fluid availability, exercise/physical activity, and heat stress. Developing individualized hydration strategies based on one's needs and other situational factors (i.e., exercise in the heat) allows for the mitigation of physiologic strain to optimize performance and enhance safety. Coupled with individualized hydration strategies is the understanding of one's current hydration status, which can assist in dictating the need for fluid.

Hydration status fluctuates throughout the day within a narrow (<1% body mass change) range when activity is controlled and the individual has unlimited access to fluids [1]. During environmental heat stress and/or physical activity, the rate of fluid loss often exceeds the rate of fluid gain or (i.e., voluntary dehydration) [2]. As the level of dehydration increases, one's ability to sweat may be affected with increased threshold for the onset of sweating, which delays one's ability to dissipate body heat effectively [3, 4].

The ability to assess hydration status has been a long sought after area of discussion and contention. To date, the clinical diagnosis of dehydration using a single reference standard remains unreliable as numerous investigations have failed to establish an accurate and valid method for measuring hydration status in all situations [5, 6]. For clinical, laboratory and field settings, urinary, hematologic, and body mass change measures are often utilized to assess changes in hydration status; however, the application of these measures is situational dependent (i.e., acute or chronic changes in hydration status) warranting careful consideration on which measure to use to have the highest diagnostic accuracy at a given time point [5, 6].

While situational dependent measures of hydration status exist, the application of these measures in real-world settings may not be feasible. For example, for an athlete on the practice field or a soldier in forward deployment, it is nearly impossible to obtain urinary, hematologic, or measure changes in nude body mass during physical activity to gather an individual assessment of hydration status. Furthermore, the equipment needed to analyze hydration measures cannot be employed in many settings. With these acknowledged challenges and complications, recommendations exist whereas the utilization of body weight changes before and after physical activity, thirst sensation and urine color may be able to identify the likelihood of being dehydrated from both a time and cost-efficient way [1].

These challenges bring rise to the need of current technology that can offer a noninvasive method of real-time assessment of hydration assessment. However, the use of technology is wrought with challenges that the end-user must consider before deciding to use such modalities to supplement their training to optimize their performance and safety. To date, noninvasive methods to assess hydration status such as bioimpedance have been unable to distinguish hydration status. Furthermore, any technology that is being used to assess hydration status must be able to accurately and reliably track changes in hydration status within a range of 0.5% body mass change to optimize performance and safety in a meaningful manner. An "ideal technology" would capture thirst, urine specific gravity, and body mass, given that fluid regulation is dynamic and that the literature suggests we need two or more of the three indices to distinguish hypohydration from euhydration with confidence [1].

The Evidence

Bioelectrical impedance is one of the common technologies that has been previously studied to identify levels of total body water; the premise of utilizing bioelectrical impedance is to utilize electrical conductive capacity of water to determine total body water. Evidence suggests that bioelectrical impedance may not provide an accurate [7, 8] and valid assessment of hydration status due to the inability of the technology to track fluid shifts between the extracellular and intracellular compartments [9]. This becomes particularly problematic during exercise as body water is in constant movement between these body compartments. A review by O'Brien et al. [9] found conflicting results in prior literature examining the ability of bioelectrical impedance is measuring total body water independent of the type of bioelectrical impedance used (single or multi-frequency). In addition, other techniques such as bioimpedance vector analysis, using regression analyses to plot vectors associated with total body water change, are limited based on the fact that such analyses are formulated from normally hydrated persons which negate its applicability in other scenarios [5].

Wearable sensors (i.e., impedance, photoplethysmography [PPG], spectroscopy, and others) and the utilization of smart water bottles (e.g., Hidrate Spark 2.0 bottle) linked to a smart device to track fluid intake are being proposed as new methods to track one's hydration status; however, there is an apparent gap in the scientific literature examining the validity and accuracy of such devices compared to clinical standards. Developing highly controlled laboratory studies comparing these wearable sensors to clinical standards of hydration assessment during exercise is needed to examine the validity and reliability of such measures to assess true hydration status. Also, use of smart water bottles that can track fluid intake lack the evidence to support the efficacy of using such technology to enhance hydration practices; however, there is potential promise that such available feedback could be used to change behavior surrounding hydration needs on a daily basis.

The Application

While there currently is no evidence supporting the validity or reliability of any noninvasive technology in assessing hydration status during exercise, there is hope that future-independent, highly controlled, laboratory studies will identify the efficacy of such noninvasive measures. Furthermore, once the efficacy of such technology is verified, the application spans athletics, military, and labor settings. With the utilization of smart devices in conjunction with the hydration assessment technology, individuals would be able to accurately track their hydration status throughout the day and during physical activity. Coupled with appropriate feedback, individuals would be able to meet their daily hydration needs, which would have a direct impact on enhancing both their performance and safety during exercise in the heat.

Measuring Sleep: Technology and Challenges

As previously described in Chap. 7, sleep is paramount for leading a healthy and productive life; however in today's society, sleep is often the aspect of daily life that is sacrificed for life's other demands. Sleep has been considered the "gold standard" for post-exercise recovery due to the recuperative and restorative effects that sleep has on the body. Furthermore, the effects of sleep deprivation or restriction on physiologic function and the effects of exercise on sleep physiology have been extensively studied [10, 11] and regular bouts of physical activity have demonstrated improved sleep quality [12, 13]. Moderate intensity exercise taking place in the evenings was shown to have positive effects on sleep [14, 15]; however, high-intensity exercise has been implicated in disrupting sleep by increasing wakefulness, thus requiring athletes, coaches, and clinicians to establish a training program with an aim at maximizing the potential for optimizing sleep to prevent detrimental outcomes on performance [13]. Additionally, evidence has demonstrated that sleep disturbances are evident during intense periods of a training program [16] and during busy times of an athletics season [17, 18] among elite athletes.

While evidence has shown that optimizing one's sleep assists in attenuating any declines in exercise performance, other evidence does not provide the same conclusive findings [19, 20]. Methodological differences related to the length of sleep deprivation or restriction, the timing of exercise, the length of exercise, and the type of exercise confounds the results making it difficult to distinguish the true relationship of sleep on exercise performance. Furthermore, most research examining the effects of sleep on exercise and exercise performance has been performed in "good sleepers" which may not be indicative and representative of a larger population of athletes, especially those with some underlying sleep pathology [13].

Another challenge associated with investigating the relationships between sleep and exercise performance is the method in which sleep is quantified. The "gold standard" diagnostic tool to quantify sleep is with the use of polysomnography (PSG), which combines measures of electroencephalography, electrooculography,

electromyography, electrocardiography, pulse oximetry, and respiration to determine an overall assessment of one's sleep. While PSG has been found to be an accurate and valid diagnostic tool to determine pathological conditions, it lacks the ecological validity to track one's sleep over the course of time due to cost constraints and the requirement of the individual to report to a sleep laboratory to be assessed. In the case of an athlete training and preparing for competition removes them from their normal routine, which may cause negative outcomes related to exercise performance.

Alternative less expensive means of quantifying sleep in individuals has been previously investigated and validated. The use of validated scales such as the Pittsburgh Sleep Quality Index (PSQI) [21] and the Karolinksa Sleep Diary [22] has been previously used to gauge one's subjective assessment of their sleep. While getting a subjective assessment of one's sleep can be an effective means of investigating the relationships of one's sleep to exercise performance, it is limited as it does not provide an additional physiological rationale for any adverse effects of sleep on exercise performance.

Various software programs have creatively integrated the questions from these validated scales into "pre-practice or session" questionnaires that each player can complete either daily upon exiting the locker room or training facility or at specific previously identified intervals over the course of the season using a tablet connected to the cloud. The sleep responses can then be funneled and alerts can be set for those athletes who are "outside of their norm." For many collegiate sports that begin training in the summer months of the northern hemisphere, the monitoring of sleep during this time of routinely increased physical stress speaks to the importance of measuring/monitoring the amount and quality of sleep to ensure that recovery is optimized. This method of sleep data acquisition is not without challenges as well. Compliance is routinely an issue and thus achieving the buy-in from all members of the team players, medical staff, coaches, etc. is critical to the data that is obtained.

The Evidence

As aforementioned, the use of PSG is the gold standard method of measuring both the structure and depth of sleep [23]. Although PSG is the clinical standard for assessing the quality of one's sleep, the widespread utilization of PSG is limited due to cost, invasiveness, lack of ecological validity and impractical nature of assessing sleep in multiple persons over a period of consecutive nights. With the noted limitations of using PSG, other methods have been developed to assess sleep in more real-world scenarios. The use of actigraphy, small wrist-worn devices that monitor movements during sleep over an extended period of time, has grown exponentially in scientific literature and has been identified as a reliable and valid method of sleep monitoring by the American Sleep Disorders Association [24, 25]. With the use of actigraphy, the raw sleep data is translated to one's sleep-wake score based on computerized algorithms that have been developed by numerous commercial sleep analytics companies.

Although the use of actigraphy has immensely grown and has been identified as a valid and reliable measure of sleep and sleep quality [26], there are inherent limitations surrounding the reliability and validity of such devices. While the comparison of actigraphy to PSG has shown high sensitivity (>80–90%), the resulting specificity of detecting wakefulness during sleep is poor (<60%), which calls into question the true accuracy of actigraphy devices in quantifying sleep [27–29]. This challenge may be due to a combination of the device used, the scoring algorithms, or the individuals that are using actigraphy.

The use of actigraphy and the validation of scoring algorithms has been primarily tested in populations of "good sleepers," thus calling into question the validity and reliability in special populations such as infants, children, and adults with sleep disorders or other medical disorders that may affect the resulting actigraphy measure during sleep [26]. As an example, prior literature has shown large discrepancies between the scoring of sleep using actigraphy compared to PSG in children with intellectual deficits or motor-related handicaps [30].

On the other hand, the use of actigraphy has been found to be a good method for assessing sleep disorders such as insomnia or as a supplementary tool to use in the assessment of sleep-disordered breathing, narcolepsy, and sleep-schedule disorders [26]. Furthermore, actigraphy has also been shown to be sensitive to changes in sleep derived from either pharmacologic or non-pharmacologic interventions meant to manipulate sleep or the schedule of sleep [26]. In recent years, the use of actigraphy outside of the clinical population but in a real-world environment, has grown [31, 32]. When assessed over a multi-night period or longer (up to a year), the use of actigraphy has shown very good inter-individual validity, which may be useful in other areas of research assessing the effects of sleep deprivation and/or sleep restriction on cognitive and physical performance tasks [26].

The Application

With the continued widespread use of using actigraphy to measure sleep and the advancement in technology in accurately depicting sleep and sleep quality using such devices, such a tool could be invaluable to various settings such as athletics, military, and labor settings. Being able to measure and quantify sleep over the course of an athletics season, during military training or operations and during various labor settings could assist those supervising these individuals in making appropriate clinical decisions and modifications. The knowledge of the derogatory effects of poor sleep quality, sleep deprivation and/or sleep restriction, coupled with the ability to accurately monitor and track one's sleep will allow for more directed interventions to ensure that individuals needing to perform, both cognitively and/or physically, can do so at an optimal level.

Measuring Thermal-Work Strain: Technology and Challenges

Individuals engaged in activities or occupations involving heavy workloads, hot environments, or thermally insulating protective clothing and equipment are often at greater risk of heat illness. Examples include athletes competing at very high work rates in extremes of temperature; miners and steelworkers working in foundries or near blast furnaces; and firefighters or military personnel who often wear personal protective equipment (PPE) designed to mitigate the threat of fire, chemical, biological, nuclear, or explosive agents or devices. Ineffective management of heat strain under these and other conditions can lead to heat exhaustion, collapse, or even death from heat stroke [33]. The psychological pressures associated with team or squad settings can also increase the risk of heat strain as individuals may ignore and attempt to "work through" the initial symptoms or sensations of heat illness in order to avoid what they perceive to be letting their team-members down [34].

Personal physiological monitoring has long been suggested as a means to assess thermal-work strain and prevent injury [35, 36]. The physiological strain induced by heat is often assessed in combination with the strain of performing work as thermal-work strain as it is the combination of the two that most commonly leads to exertional heat illness. Assessing thermal-work strain generally involves measuring some combination of internal body temperature, skin temperature, heart rate, and sweat rate or water loss. Robinson et al. [37] proposed an index that was an equally weighted combination of all four of these parameters. While Hall and Polte [38] suggested an index that used heart rate, change in sweat rate, and change in internal body temperature. Pandolf and Goldman [39] used the convergence of skin temperature and internal body temperature to predict time to exhaustion. While the technique showed promise, it operated best in hot humid environments. Frank et al. [40] also introduced a two-parameter heat strain measure in the form of their cumulative heat strain index; however, it was Moran et al. [41] who proposed a real-time index that could be computed as a weighted combination of heart rate and internal body temperature. This Physiological Strain Index (PSI) calculates an index (from one to ten) that can represent the physiological strain during multiple bouts of work and rest periods by combining how hot an individual is getting (think of a car's temperature gauge or a human's internal body temperature) with how much work is being done (e.g., a car's tachometer or heart rate). The common element among these methods of assessing thermal-work strain is that a measurement of internal body temperature is needed in real time. Outside of a lab environment, the requirement to measure or infer internal body temperature has been a limiting factor in real-time assessment of an individual's thermal-work strain state. Several different methods can be employed to measure or estimate internal body temperature in an ambulatory environment, including: direct measurement, indirect measurement (temperature correlates, zero heat flux approach), and estimation from regression equations.

Table 10.1 Summary of ingestible thermometer pills

Name	Accuracy (°C/°F)	FDA 510 K #	Comments
HQInc	±0.10 °C/0.18 °F	K880639	Analog, individual calibration
Jonah™, VitalSense™	±0.05 °C/0.09 °F	K033534	Digital
BodyCAP e-Celsius™	±0.20 °C/0.36 °F	Unknown	Digital, logging

The Evidence

Direct Measurement

Since the late 1980s ingestible thermometer pills have been used to track internal body temperature in ambulatory settings. These pills measure intestinal tract temperature as they pass through the body. While temperatures vary slightly with location in the intestinal tract [42], they have been used successfully in both laboratory and field settings [43, 44]. Validation studies have found that the pills compare favorably to either rectal or esophageal probe methods [45, 46] and fall within acceptable limits of agreement (±0.4 °C/0.72 °F) and bias (<0.1 °C/0.18 °F) when compared to esophageal temperatures [47]. When the pills are higher in the intestinal tract, they respond in a similar fashion to esophageal probes with faster reactions to body temperature changes. As the pills get lower in the intestinal tract (usually 6–8 h), they exhibit temperature responses similar to rectal probes with slower reactions to temperature changes. In general, the pills will measure slightly higher temperature than when taken rectally [48, 49]. Three common ingestible pills systems are in use today: HQInc, Philips/Respironics, and BodyCAP e-Celicius™ [52]. Table 10.1 provides the specifications for each of the pill systems and the FDA 510K pre-market certification numbers.

The HQInc (Palmetto, FL) system uses a silicone-coated epoxy potted pill (Fig. 10.1). The pill requires a calibration and provides a temperature reading by varying the frequency of a simple radio transmission around 262 kHz. Temperature data are recorded externally with a data logging device that receives and decodes the pills' radio frequency signal. The logging device requires a calibration code to be entered for the swallowed pill to provide accurate temperatures. As these pills are analog based, the internal body temperature reading can get noisier the further away the monitor is from the pill. Additionally, the data logger can mistakenly receive other pills from other participants in close proximity, so multiple readings are often necessary to confirm measurement in the intended participant.

The Jonah™ ingestible pills (Philips/Respironics, Bend OR) were originally developed to try and address some of the issues with HQInc pills. The pills (see Fig. 10.2) have a food grade polycarbonate shell, are digital in nature, and are pre-calibrated at the factory. They transmit actual internal body temperatures in a pseudo-random scheme four times per minute at approximately 40 MHz. Pill readings can either be received and displayed on a VitalSense™ logger unit or via the Equivital™ EQ-02 system (Equivital, Cambridge UK). Additionally, the VitalSense™ system can also use a skin temperature form of the pill.

Fig. 10.1 HQInc ingestible thermometer pill and data logger [50]. Copyright © HQ, Inc. Reprinted by permission

A relatively new (2016) ingestible thermometer the BodyCAP (Caen, France) system has a smaller form factor pill that can log up to 15 h of data internally. This logging capability can allow for long periods where the pill can be separated from the data logger.

In controlled studies, ingestible pills can provide very robust data; however, they have some critical limitations. First, the pills are contraindicated for anyone with gastrointestinal problems such as Crohn's disease or diverticulitis. While thousands of ingestible pills have been used throughout the world, caution should be exercised even when used in a healthy population. A relatively small number of medical device adverse events reported concerning ingestible thermometer pills illustrate the need for caution in their use. A 2015 Medical Device Adverse Event Report [53] details how a healthy participant in a research study had to have surgery for removal of a thermometer pill stuck in the small intestines. The report noted that while the patient was healthy there was a history of Crohn's disease in their family. A further report details how a research participant had to have a pill removed from their esophagus. The patients believed they were healthy, but during the pill removal procedure it was noted that they suffered from a congenital narrowing of the esophagus.

The thermometer pills can also suffer from inaccuracies from ingested fluids when they are in the upper portion of the gastrointestinal tract. Wilkinson et al. [54] show this effect for up to 10 h. While this problem can be overcome by administering pills in advance, participants may pass the pill within 12 h.

Fig. 10.2 Philips Respironics Jonah™ Pill and VitalSense® skin temperature patch, and the VitalSense® data logging system [51]. Reprinted with permission from Philips Respironics

An alternative means of avoiding aberrant data due to fluid ingestion is to use the pills as a rectal suppository, but this can discourage participant use/recruitment. Finally, continuous monitoring over many days can be prohibitively expensive as a single pill can cost roughly $50. These limitations in directly measuring internal body temperature in ambulatory settings have motivated the search for alternative and noninvasive techniques.

Indirect Measurement

Temperature correlates are body sites where either surface temperature or subsurface temperature can be used to approximate internal body temperature. However, finding suitable sites and developing algorithms capable of approximating internal body temperature from them is challenging. Skin surface temperatures can vary quite differently from internal body temperature based upon the environment, clothing, and sweat rate. Generally, humans thermoregulate by increasing and decreasing blood flow to the skin and sweat rate. Thus, in many circumstances there is no clear correlation of skin temperature to internal body temperature.

During periods of intermittent work, internal body temperature and tympanic "aural canal" temperature diverge to suggest other means of assessing internal body

temperature during exercise [55–57]. From a safety and clinical standpoint, aural canal temperature should not be used to assess temperature in exercising individuals especially when monitoring for safety or in the diagnosis of a heat illness. Overall, Lim et al. [58] report in their review article that using external measurements such as axillary or tympanic temperatures have proven unreliable and should never be used when diagnosing or monitoring for safety in individuals exercising in the heat.

Another noninvasive approach is the zero heat flux (ZHF) method [59] where an insulated area of the skin is heated until there is no heat flow. The temperature of the skin is then assumed to be equivalent to deep body temperature. Most of the work on this approach has been in laboratory and clinical settings [60] with recent work focusing on improving measurement of dynamic temperature changes [61], and decreasing the technique's response time [62]. In clinical settings, these devices have demonstrated good agreement with esophageal measures, while custom sensors developed for ambulatory environments have had varying degrees of success depending on environmental conditions [63]. Work by Xu et al. [64] reports the difficulty of the heat flux approach by demonstrating how the relationship between internal body temperature and heat flux on the skin surface can be affected by the environment, sensor placement, clothing, and sweating.

Regression and multi-parameter approaches or those that utilize noninvasive physiological measures to accurately estimate code body temperature are becoming more well known. Kaufman and Coleman [65] have patented a 12 parameter regression model derived from data from 60 volunteers but, do not present any validation data. Niedermann et al. [66] report an internal body temperature estimation equation based on determining two independent factors derived from principal components analysis. The method relies on three skin temperature measurements, heart rate, and two skin heat flux measurements. While Niedermann's results were reasonable (see Table 10.2), they were collected from relatively few subjects. Additionally, many heat flux and skin temperature instrumentation sites are not practical for field use outside of the laboratory. Richmond et al. [67] presented a technique needing only three sensors (heart rate, insulated skin temperature, and clothing micro-climate). This algorithm achieved reasonable results (see Table 10.2) but utilizes a custom array of sensors that require careful placement for accurate results. Finally, Seng et al. [68] expanded the idea of a Kalman filter approach first suggested by Buller et al. [69] by adding, in addition to heart rate, skin temperature, and separate algorithms for periods of activity and inactivity.

The estimated internal body temperature (ECTemp™) model uses sequential observations of heart rate to track internal body temperature over time. The simple, yet effective approach was proposed by Buller et al. [69] and updated with a more general model in 2013 [70]. The ECTemp™ assumes heart rate can be used as a "noisy" observation of internal body temperature. Physiologically, heart rate reflects both the metabolic rate (blood flow to the muscles) and the rate of blood flow to the skin. Thus, heart rate contains information about both heat production and heat loss from the body. The ECTemp™ relies on a Kalman filter approach often used in engineering tracking problems, where a variable of interest is tracked using (1) information about how that variable changes over time (transition model) and (2) a

Table 10.2 Performance of internal body temperature estimation algorithms

	Development data				Validation data					
Model	*n*	Bias (°C)	LoA (°C)	RMSE (°C)	Study	*n*	Description/clothing	Bias (°C)	LoA (°C)	RMSE (°C)
Buller 2013 [70]	17	–	–	0.27	Buller 2013 [70]	89		−0.03 ± 0.13	±0.63	0.30 ± 0.13
					Buller 2015 [72]	27	Chem. Bio	−0.02 ± 0.25	±0.49	0.21 ± 0.11
						16	Young college athletes	−0.01 ± 0.19	±0.37	0.28 ± 0.15
					Showers 2015 [71]	33	Hot/wet environment	−0.01 ± 0.19	–	0.29 ± 0.15
					Buller 2016 [73]	43	35 °C Chem. Bio PPE	−0.15 ± 0.19	±0.37	0.22 ± 0.10
						95	40 °C short + T-shirt	−0.07 ± 0.26	±0.51	0.22 ± 0.14
Niedermann 2014 [66]	5	–	–	0.28	Niedermann 2014 [66]	5	Hot	–	–	0.28 ± 0.15
						5	Hot	–	–	0.34 ± 0.15
						5	Cold	–	–	0.33 ± 0.15
Richmond 2016 [67]	5	–	–	0.21[a]	–	–	–	–	–	–
Seng 2016 [68]	34	0.03	±0.45	0.23	Seng 2016 [68]	5	–	−0.11 ± 0.19	±0.53	0.29 ± 0.15

N number of study participants, *LoA* 95% limits of agreement, *RMSE* root mean square error, – not included or reported, *Chem. Bio* chemical biological clothing, *PPE* personal protective equipment

[a]Standard error of estimate (SEE), a measure of precision comparable to RMSE in this context

series of "noisy" observations. The model was developed from 17 US Army Soldiers engaged in 24 h of field training (ambient temperature 24–36 °C/75.2–96.8 °F and a relative humidity range of 42–97%). Soldier activities ranged from periods of inactivity including sleep to high work-rate activities such as fox-hole digging. The data used to train the model included internal body temperatures from 36 to 40 °C/96.8 to 104 °F and the ECTemp™ algorithm has been validated across a large number of laboratory, field, and operational settings (Table 10.2) including: cool (11 °C/51.8 °F) to very hot (42 °C/107.6 °F) environmental conditions, various clothing ensembles (shorts and T-shirts, combat uniforms, body armor, encapsulating PPE), work rates from sleep to heavy exercise, hydrated versus under-hydrated conditions and heat acclimated versus non-heat acclimated conditions.

Overall the ECTemp™ algorithm has been validated in over 14 studies with more than 300 subjects and generally performs reliably from study to study, with similar RMSEs (<0.30 °C/0.54 °F), biases, and limits of agreement (LoA; generally less than ±0.63 °C/1.13 °F. The LoAs for ECTemp™ found in these studies are similar to comparisons of rectal and esophageal probe measures which on average have LoAs of ±0.58 °C/1.04 °F. The ECTemp™ algorithm has been used as a surrogate for internal body temperature while assessing thermal-work strain via the physiological strain index (PSI) with R^2 values above 0.90 PSI, RMSE values around 0.6 PSI, and limits of agreement around ±1.1 PSI units [71].

The Application

During the latter part of the twentieth century, individual sensors were light and wearable enough to be incorporated into systems that monitored several physiological parameters. Pioneering work circa 1955 from the U.S. Army's Quartermasters Research and Development Center, Natick MA produced a telemetry system to measure the physiological impact of clothing (see Fig. 10.3) [74].

While individual and prototype systems were built as early as the 1950s, it wasn't until the mid-1990s to early 2000s that true real-time physiological monitoring systems were developed. The evolution of a small size data logger for the HQInc ingestible core thermometer pill (sponsored by National Aeronautics and Space Administration [NASA] and the US Army) allowed for large scale field testing of an integrated sensor system that measured motion, heart rate, internal body temperature, pedometry, and geo-location during military training events [44]. This system was utilized by researchers from Yale University and NASA to track the physiology of mountain climbers during an ascent of Mt. Everest [75]. Similarly, Massachusetts Institute of Technology (MIT) researchers were able to web cast Boston Marathon runners' internal body temperature, skin temperature, and heart rate during the marathon as part of a project named "Marathon Man" [76]. Fig. 10.4 shows a real-time monitoring system used to show the thermal-work strain index of a squad of warfighters using internal body temperature pills and heart rate monitors [77].

Fig. 10.3 Thermal physiological strain monitoring system, Quartermaster Research and Development Center, Natick MA (Circa 1955) [74]. Reprinted from Friedl K, Buller M, Tharion W, Potter A, Manglapus G, Hoyt R. Real time physiological status monitoring (RT-PSM): Accomplishments, Requirements, and Research Roadmap. Technical Report. Natick, MA: United States Army Institute of Environmental Medicine; Report No.: TN16–02 (ADA 630142)

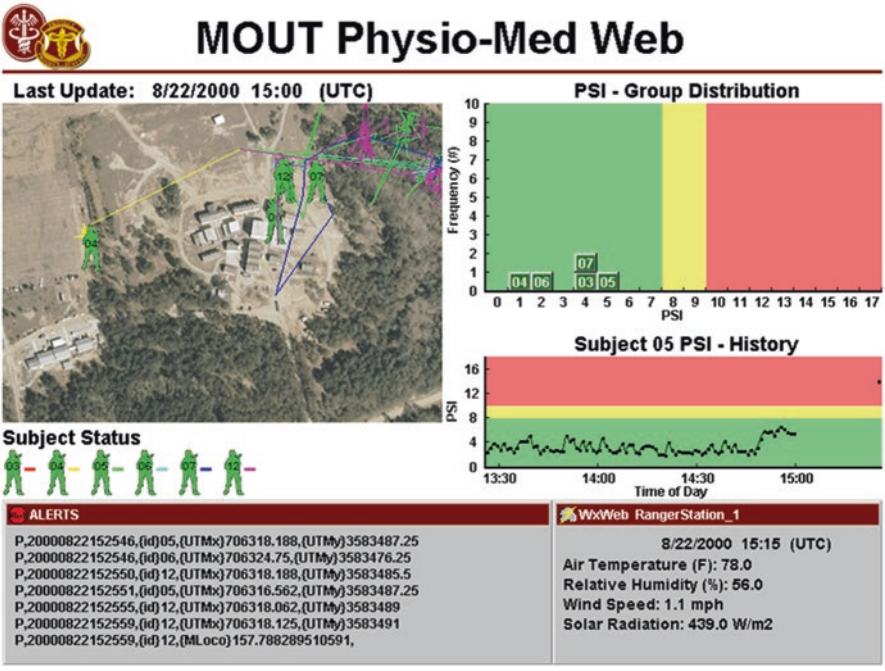

Fig. 10.4 Physiological monitoring system that measured the thermal-work strain index in real time

Other real-time physiological status monitoring work tried to combine both thermoregulatory modeling with real-time sensors. An integrated system developed by the US Army attempted to use the SCENARIO thermoregulatory model and multiple sensor inputs including real-time environmental information in a systems level approach. A combination of physiological and environmental sensors, and physiological and physics-based heat transfer models were used to attempt to estimate life signs and multiple health states (e.g., thermal, hydration, and cognitive) [78]. While the system was able to provide real-time physiological monitoring, the technological limitations of short distance radio communications and the core thermometer pill hampered acceptance of these systems.

The advent of low-cost integrated Bluetooth Low Energy (BTLE) wearable sensor systems and algorithms such as the ECTemp™ has overcome many of the early limitations to thermal-work strain monitoring systems. Cost-effective heart rate bands can be used in place of more expensive physiological monitoring systems that receive ingestible core thermistor pill data and low cost sensors can readily be linked to smart phones/tablets via BTLE. Simple thermal-work strain monitoring apps can use this information to manage training events and personnel and mitigate the risk of thermal injuries (e.g., see Fig. 10.5a). The increased processing power and connectivity of smart phones can be used to "telemeter" individual data back to medical personnel or a thermal-work strain management team (e.g., see Fig 10.5b).

Whether using ingestible thermometer pills or an internal body temperature estimation algorithm, the current state of technology allows for a simple assessment of internal body temperature to be integrated into a monitoring system. Regardless of which combination of commercial sensors, algorithms, and software one chooses; internal body temperature monitoring systems can be assembled, tested, and deployed more easily than ever.

Measuring Training Load: Technology and Challenges

Quantifying exercise performed during training and competition is critical to ensure that athletes are not pushed beyond their physical limits or reach a state of overtraining [80]. It is relatively easy to quantify closed-skill exercises, such as resistance training and distance running, which are exercises that generally follow a predetermined sequence, are performed in a relatively controlled conditions, and involve minimal interaction between athletes [81]. For a resistance training session, a strength and conditioning coach can plan and record all the exercises performed, resistances lifted, number of sets and repetitions, and rest intervals. This is not the case when tracking many open-skill exercises, such as soccer or lacrosse, since they are exercises that are highly unpredictable, performed in relatively uncontrolled conditions, and involve frequent interaction between athletes [82]. For instance, a soccer coach can record each athlete's playing time during a match but the distance covered and velocities reached during that time will vary considerably by position, game conditions, and level of competition [83, 84].

Fig. 10.5 (**a**) Smart phone thermal-work strain app showing a physiological strain index score computed from monitored heart rate and ECTemp™ algorithm. Courtesy of MIT Lincoln Laboratory (**b**) Equivital Black Ghost human monitoring systems (http://www.equivital.co.uk/)-Custom Equivital Black Ghost Screen showing an Estimated PSI computed from heart rate and ECTemp™ and predicted physical strain index (PSI) 15 min into the future along with other physiological parameters (Tharion et al. [79], Buller et al. [72])

Many athletics programs have started to use GPS devices to address the challenges of tracking movements of groups of athletes during open-skill exercises. GPS devices provide timing and locational data from transmissions received from a network of 32 satellites orbiting the Earth. A GPS device calculates the relative distance from one of the satellites based upon the length of time between signals received from that satellite. When a GPS device receives signals from three or more satellites, the global coordinates of the GPS device can be determined. Changes in the global position of the GPS device between measurements can be used to measure the distance traveled by the athlete. In addition, movement velocity can be determined by changes in the frequency of the GPS satellite signals or by dividing the change in position between measurements by the change in time.

Many different distance and velocity-based statistics can be used to summarize the exercise performed during practices and games. Total distance traveled was one of the first GPS metrics investigated and one of the most frequently used to quantify exercise performance [85]. Other commonly reported metrics are the average and maximum velocity of movement. However, these metrics may not adequately describe bouts of high velocity, intermittent exercise. For example, only 3% and 1% of total distance covered during soccer matches are at high velocity (>18 km h^{-1}) and sprint velocity (>21 km h^{-1}), respectively [86]. Consequently, the average velocity will be more representative of standing, walking, and jogging movements than brief high-velocity movements.

A common approach to generating useful metrics from GPS data is to define different ranges of velocity, known as velocity zones, and recording the amount of time or distance each athlete travels within each zone. Suppose a team decides to evaluate athletes on how much distance they cover within three different zones: low (0–3 m s^{-1}), moderate (3–6 m s^{-1}), and high velocity (>6 m s^{-1}) [87]. Using these velocity zones, a coach can determine which players covered the most high-velocity distance versus those that covered large distances at lower velocities. This information can be extremely vital to ensure that athletes are completing the distance and velocities necessary to improve physical fitness without compromising health and wellness. With these metrics, "individualized optimal ranges" and "individualized norms" can be tracked and utilized to alert the athlete when they venture outside their own ranges. For example, these data can be used to alert an athlete in real time who is not used to exercising in the heat that they are spending too much time in high-velocity zones during play and should be more efficient or subbed out of play.

Challenges common to the collection of GPS data, regardless of exercise in the heat, are that in order to make informed decisions regarding training load, data must be collected on one individual athlete for a number of training sessions to allow for the appropriate amount of normative data to be generated. Depending on the sport, this may take up to a year to understand the full range of training load experienced by the athlete in different parts of the season. Another challenge specific to GPS monitoring is that the technology, the algorithms, placement, and supporting software is constantly being improved and or modified. This can present challenges when attempting to gather normative data for an individual over a season or succes-

sive seasons. Last, often the triangulation of satellites (as previously described) used to monitoring have been shown to be variable with increasing cloud cover, tall buildings, and when transitioning from indoor to outdoor activities. Early GPS monitoring technology often would lose signal and data from a training session when moving from indoors to outdoors (e.g., football or soccer halftime from locker room to the field) which would result in missing data and inaccurate speeds, distances, and time spent in various zones related to these metrics. More recent GPS technology has improved allowing for quicker acquisition of satellites, but this still remains an issue unless specific measures are taken to confirm that satellite signals are locked.

The Evidence

The precision of GPS devices in measuring timing, locational, and movement-related data has been a frequent research topic in exercise science. Whether a GPS device is suitable for measuring a given physical activity is dependent on its sampling rate; commonly reported in hertz (Hz) or the number of measurements per second. A GPS device with a sampling rate of 1-Hz is sufficient for general navigational purposes since movements are generally linear, and the margin of error is negligible in comparison to the total distance traveled.

However, 1-Hz GPS devices are inadequate for measuring movements occurring in less than a second [88], such as the rapid changes of direction that occur during many team sports. Even with sampling rates of 5-Hz, GPS devices have been shown to have poor precision when measuring high accelerations and decelerations [89]. As such, it has been recommended that GPS devices require sampling rates of 10-Hz or greater to accurately measure the movements of team sport athletes [85].

Investigations of team sport training and matches performed in hot environments frequently incorporate GPS technologies to measure on-field performance. A study by Aughey et al. [90] investigated internal body temperature, hydration, and running performance in professional Australian football players during 16 matches, including eight played in hot environments. Internal body temperature increased over the course of the matches played under hot environmental conditions with corresponding decreases in total distance. Interestingly, high-intensity running (>4.17 m s^{-1}) and maximal accelerations (>2.78 m s^{-1}) remained consistent throughout the matches. Another study by Duffield et al. [91] examined thermoregulatory responses during Australian rules football matches played in warm conditions (e.g., wet-bulb globe temperature= > 29 °C/84.2 °F). The greatest increase in internal body temperature was observed during the first quarter of the match (2.1 ± 0.7 °C/3.8 ± 1.3 °F), which is consistent with the normal initial rise in internal temperature observed during exercise in the heat. Average internal body temperature peaked during the final quarter (39.3 ± 0.7 °C/102.7 ± 1.3 °F). Internal body temperature and rise in internal body temperature demonstrated strong positive correlations with moderate-intensity and high-intensity activity velocity. Given that exercise intensity is the

largest contributor to heat production within the body, these findings highlight the value of GPS technology as a means of quantifying player workloads and intensity during outdoor exercise. When aligned with other factors well known to alter heat gain and heat loss during exercise in the heat, information related to intensity of exercise garnered from GPS technology can potentially be used to explain these alterations in internal body temperature and potentially mitigate heat illness or injury in subsequent athletic contests.

The Application

While GPS devices have enormous potential for monitoring athlete performance and risk of injury, organizations must be aware of the substantial commitments necessary to successfully integrate this technology into practical use. First and foremost, the GPS systems used to track competitive athletes require a substantial financial investment, typically between $500 and 5000 per unit. In addition, personnel must be available for all data collections and not only proficient in handling and troubleshooting the GPS hardware but also capable of organizing, cleaning, analyzing, and reporting GPS data. The following section provides a general overview of the steps required in order to utilize GPS data.

At least 10 min prior to data collection, all GPS devices should be turned outdoors to allow for proper synchronization with satellite signals. As measurements can vary between devices, each athlete should be assigned the same GPS device at each data collection. Users should also record important details of the data collection such as attendance, device issues, and the sequence of exercises performed. Following data collection, all GPS devices should be gathered so that data can be extracted and uploaded to a computer or data storage system promptly.

After data has been uploaded, the next challenge is to reduce the massive amounts of data that has been collected into more reasonable quantities. Tracking 11 players over the course of a single 90-min match with 10-Hz GPS devices will result in the collection of approximately 594,000 data points. Over 100 million data points may be collected if a season's worth of training sessions and matches are recorded from an entire team.

Users should ensure and evaluate the quality of GPS data before data analysis begins. This includes examining the raw data for any irregularities (e.g., rapid spikes in the velocity trace) and assessing signal quality metrics such as the number of satellites interacting with each device. Although no gold standard criteria have been established, a recent review recommended that devices should be interacting with at least six satellites to ensure quality data [92]. All poor-quality data should be excluded and replaced to ensure that analysis and interpretation are not distorted.

In-depth analysis can be conducted on the GPS data collected during a single match or training session from individual athletes. To illustrate, Fig. 10.6 displays the movement velocities of two different soccer players (Player A and Player B) over the first half of a soccer match. Notably, Player A has a much lower max

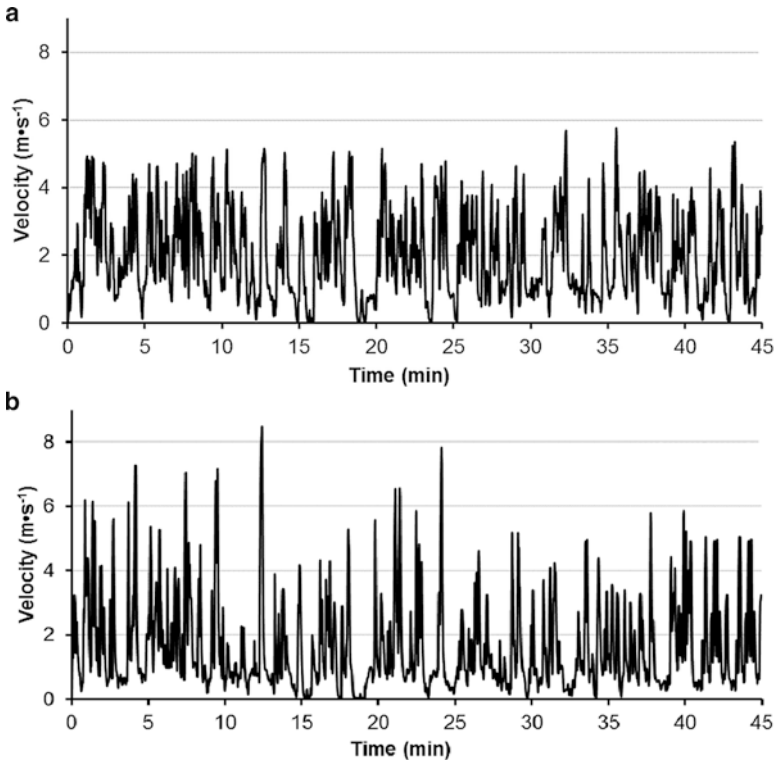

Fig. 10.6 Movement velocity of two different soccer players (Player A and Player B) over the first half of a soccer match

velocity than Player B (5.8 $m \cdot s^{-1}$ vs. 8.5 $m \cdot s^{-1}$). Interestingly, Player A had a higher average velocity over the half than Player B (2.0 $m \cdot s^{-1}$ vs. 1.6 $m \cdot s^{-1}$) and covered a greater overall distance (5394 m vs. 4343 m). Greater insight can be obtained by assessing the time spent in three velocity zones (Low, 0–3 $m \cdot s^{-1}$; Moderate, 3–6 $m \cdot s^{-1}$; High, 6+ $m \cdot s^{-1}$). For instance, it can be determined that Player B spent more time than Player A at both low velocities (38 min vs. 34 min) and high velocities (1 min vs. 0 min). In contrast, Player A spent a much greater time at moderate velocity than Player B (11 min vs. 6 min). Consequently, these data show that Player A spent most of the half jogging at a consistent velocity while Player B's half was much more of an intermittent nature, alternating between walking and sprinting. This indicates that Player A may be at greater risk of heat illness based on the findings of Aughey et al. [90] discussed in "The Evidence" section.

Longitudinal analysis of group trends may also provide valuable information that can be used to better ensure player safety. Fig. 10.7 displays the average distance traveled within the low, moderate, and high-velocity zones by soccer players during the first 6 weeks of the season. Note that the average total distance was considerably higher in the first 2 weeks (33,255 m and 32,815 m, respectively) during preseason

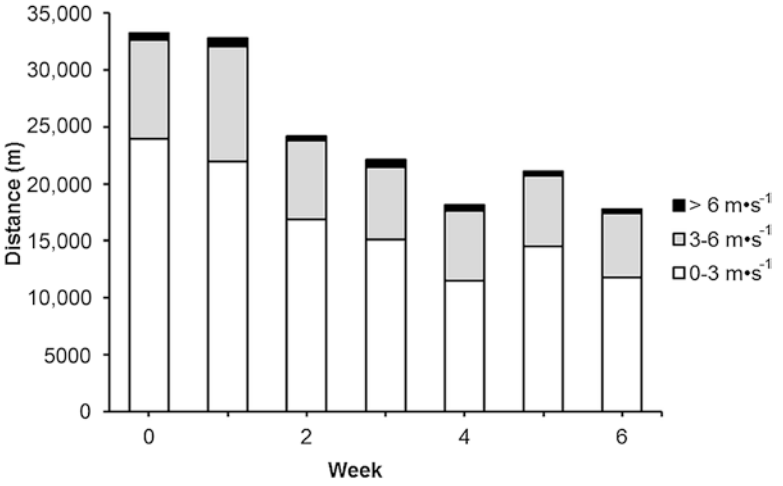

Fig. 10.7 Average distance traveled within three velocity zones (0–3, 3–6, and >6 m·s^{-1}) by college men's soccer players over the first 6 weeks of the season

training camp (Fig. 10.7). Monitoring player workloads during this part of the season is essential due to the hot and humid environmental conditions. Over the course of the season, high-velocity distance remained consistent with changes in total distance resulting primarily from low and moderate velocity exercise. A soccer coach could use GPS data such as this to quantify the level of physical demands placed on the players during preseason camp and focus on limiting unnecessary low and moderate-intensity exercise for better player safety.

Case Example *You are an exercise scientist at a prominent university in North America and the coaches of the men's national soccer team comes to you based on your expertise in monitoring players during competition and training. The coaches are preparing for the next 2022 FIFA World Cup that will be held in Qatar, where despite the move to the winter months for the first time in the history of the Word Cup, ambient temperatures will still be between 78–86 °F and 26.0–30.0 °C and have the potential to reach 90 °F/32.3 °C even in the evening. They are concerned about the team's performance in the heat and are planning to undergo a 21-day heat acclimatization (HeatAcc) protocol leading up to their departure. In addition to the HeatAcc, they would like to monitor the players during training and friendly matches leading up to the World Cup.*

As someone with extensive knowledge in the area of sports performance monitoring and thermal physiology, you have come up with the following plan. First, you suggest a comprehensive approach that will measure as many aspects of their training, lifestyle, physiological changes, and performance. First, from a hydration perspective, you suggest to keep record of daily first morning measures of body mass, urine color, and level of thirst to help insure that hydration requirements are being met. To make things easy, you suggest using a mobile compatible

software system that syncs to the athlete's individual body mass scale that you will place in their homes; furthermore, you implement a system where the athlete can log their first morning urine color to a standard urine color chart, and input their first morning thirst scale. The system would be programmed to send alerts when athletes meet two out of three criteria for euhydration (e.g. change in body mass of >1%, dark urine color > 4, thirsty (yes or no?)). This would allow them to intervene well before training and arrive to training in a euhydrated state. Furthermore, to ensure that athletes are hydrating adequately, you provide them with smart water bottles that log the amount of fluid that they consume. You also suggest that pre- and post-practice body mass are obtained each day to determine sweat rate and fluid needs to replace before the next training session. Second, you suggest that the men's national team wear a wrist-worn actigraphy device that tracks sleep, heart rate-variability, and sleep quality. Even though you know that compared to PSG these devices lack specificity, right now it is the most convenient and easy-to-use way to gather this information from the athletes. Third, you suggest using a 10 or 15 Hz GPS device equipped with software that will generate reports, alert individual player loads outside of each athlete's norm, collect heart rate data, and can be worn during training, ideally under the uniform on the chest or upper back. The coaches inform you that they have been tracking using GPS and heart rate for over 1 year now. You smile because you know that you have enough data to generate normative values for each player, which allow you to make more informed decisions about the players training loads, speed, and heart rate zones. Lastly, you suggest that the players ingest temperature pills on a daily basis or at least prior to intense training sessions in the heat during the HeatAcc process to ensure that the players are reaching internal body temperatures in excess of > 102.5 °F/39.2 °C for a minimum of 1 h during training to ensure that physiological adaptations are optimized. To confirm that adaptations have occurred, you choose to compare average and peak internal body temperature, heart rate-based load measurements, distance covered and time spent in various speed zones, and sweat rates during a match from earlier in the season to a friendly match just prior to traveling to Qatar. As expected, although average and internal body temperatures were similar before and after HeatAcc, it took players longer to reach the peak internal body temperature after HeatAcc. Furthermore, they were at lower heart rate zones in the later stages of the match yet, intensity and distance and time in higher speed zones was increased indicating increased performance. Additionally, because of your suggestion to utilize temperature pills, you were able to notify the medical staff about one athlete's internal body temperature at half time that reached 106.2 °F/41.2 °C; this allowed the medical staff to immediately immersed the athlete in a life-saving ice water bath until his temperature was reduced to 102 °F/38.9 °C and protected the athlete from experiencing prolonged exercise-induced hyperthermia. Come to find out, this athlete was not feeling well and had digestive issues and an illness that were not reported from the day before. So not only did your suggestions assist with monitoring for HeatAcc status, you very well saved this athletes life from exertional heat stroke!

Summary

Now that you have a thorough understanding of the various existing technologies that are useful for the maximization of performance and safety, it will enable you as the exercise scientist, the coach, the drill instructor, or the fitness professional to make more informed decisions before, during, and after exercise in the heat. When current technology related to hydration, sleep, internal body temperature, and training load are routinely monitored in high-performance athletes, warfighters, and the like, injuries related to heat illness or overtraining can potentially be avoided and performance can be optimized. Understanding the challenges, evidence, and proper application of these technologies is critical for successful implementation and when implemented correctly, can become a valued member of the team or unit.

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Chapter 11
Biomarkers

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The Physiology/The Challenges

Exertional heat illness (EHI) pathophysiology is complex, variable, and difficult to assess in real time, directly, and categorically. Assessment of increased susceptibility to, onset and severity of, and recovery from EHI can potentially be measured by biological markers (biomarkers) that are consistently present in EHI development and during recovery from EHI. Protein or molecular biomarkers are measurable indicators that may be most often measured in urine or blood samples while genetic

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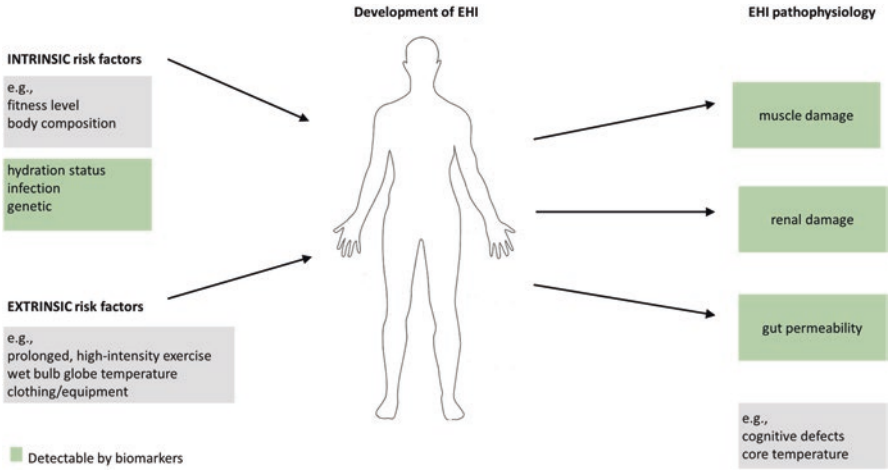

Fig. 11.1 Blood biomarkers may detect intrinsic risk factors or specific aspects of EHI pathophysiology including hydration status, tissue damage, and ensuing inflammation

biomarkers can be assessed in any tissue from which we can extract genomic DNA (e.g., whole blood, saliva, hair, skin).

Currently, there are no biomarkers that can be used to confidently predict susceptibility to, severity of, and recovery from EHI. Much of the training/research is based on classical rather than exertional heat stroke (EHS). However, markers of hydration state, muscle damage, and immune function have all been explored as possible indicators of risk for or recovery from EHI. Genetic susceptibility for EHI may intersect with heritable risk for diseases/conditions such as rhabdomyolysis or malignant hyperthermia; markers of increased risk for these diseases have also been explored as possible indicators of risk for EHI and particularly EHS.

EHS biomarkers that have been investigated thus far result from key aspects of pathophysiology associated with EHS. Theoretically, biomarkers can be assessed for EHS risk as well as for EHS severity (Fig. 11.1). Although there are several extrinsic and intrinsic risk factors for EHS, biomarkers can be assessed for select intrinsic risk factors such as hydration status and infection. Genetic susceptibility to rhabdomyolysis or malignant hyperthermia may also be an intrinsic risk factor for EHS that can be assessed by genetic biomarkers.

Biomarkers of Hydration State

Dehydration contributes to EHIs via different mechanisms. Reduced blood volume due to dehydration causes cardiac insufficiency whereby cardiovascular output inadequately provides blood flow to exercising muscles and other tissues. In this instance, dehydration is a major contributing factor to *heat exhaustion*. Via a distinct mechanism, dehydration can also be a major risk for *exertional heat stroke*. Reduced

blood volume during dehydration contributes to an inability to reduce core temperature via convection and evaporation (i.e., sweating). Monitoring hydration state identifies risk for EHI and performance effects of exercise in hot and humid environments.

Daily body water turnover includes losses via respiratory, insensible, urine, feces, and sweat losses amounting to up to ~10% of total body water. Dehydration results in a hypohydrated state that can be detected most simply via changes in body mass. Acute changes in body mass can be attributed primarily to body water loss with 1 g of body mass loss equaling 1 mL water, assuming specific gravity of water to be 1.0. Using body mass changes to estimate hydration status, however, requires an established hydrated body mass. This can be best defined per individual by weighing a person who is well hydrated, on multiple mornings upon waking, before food consumption. Euhydrated body masses over 2–3 mornings will provide the average weight of a person when he/she is hydrated so that any mass losses from that value can be assumed to be loss in water over a short time. Over hours or just a few days, accounting for food consumption and bowel movements will also increase accuracy of using body mass changes to estimate level of dehydration. Using body mass change to assess hydration state is not effective over many days or weeks because long-term changes in body mass may be due to fluctuations in body composition or actual tissue loss or gain. Over long periods of time, using this measure of hydration state requires occasional reestablishment of a euhydrated baseline weight.

Biomarkers of hydration state in urine and blood also provide relatively accurate, but contextualized assessment of level of dehydration (Fig. 11.2). Urine color,

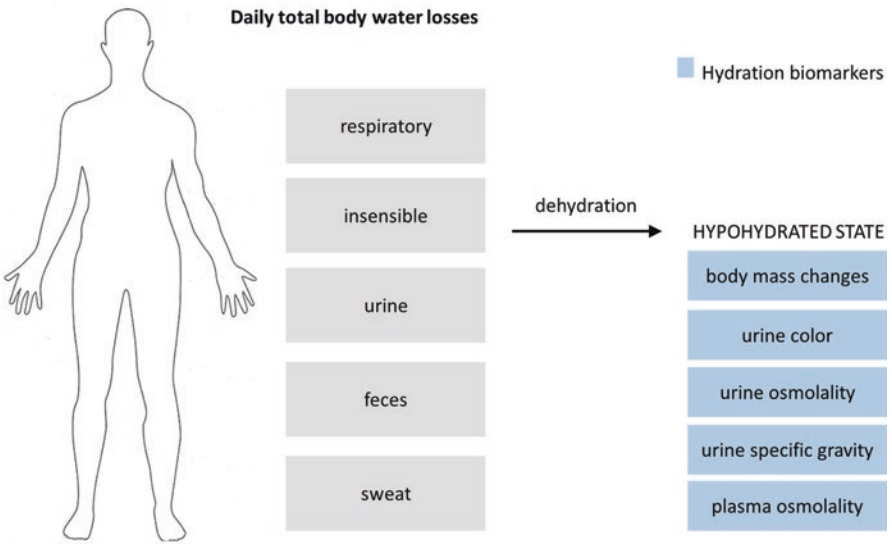

Fig. 11.2 Daily body water losses via respiratory, insensible, urine, fecal, and sweat losses can be detected via acute shifts in body mass, blood plasma osmolality, and urinary markers such as urine color, osmolality, and specific gravity

specific gravity, and osmolality, plasma sodium and osmolality, and hematocrit are well-researched biomarkers of hydration. During dehydration, water moves from the interstitial fluid space rather than from intracellular water to the extracellular fluid space to preserve plasma osmolality [1]. In the event that dehydration continues, intracellular fluid is protected and plasma osmolality increases as relative water volume in this compartment decreases; water is lost from plasma up to five times that lost from other body water compartments during dehydration [2]. As water volume in circulation decreases and solute concentration increases, plasma osmolality increases from average euhydrated values ~290 to >300 mOsm kg^{-1} and plasma/serum sodium increases from ~145 to >150 mmol L^{-1}.

Resultant shifts in plasma osmolality (increased with dehydration) are detected at neurons in the supraoptic and paraventricular nuclei in the hypothalamus. These neurons terminate at the posterior pituitary gland at which secretory nerve terminals release arginine vasopressin (AVP) which easily enters the bloodstream at this site. AVP ultimately impacts the utility of urine as a biomarker. At the kidney, urine is formed via filtration, reabsorption, and concentration through kidney tubules. Whether water is reabsorbed at water-permeable sections of the tubular network is determined by the extracellular fluid osmolality in the medulla surrounding the relevant ducts and by hormonal regulation of fluid regulating hormone, arginine vasopressin (AVP). Water is reabsorbed to conserve body water and the resulting urine output has higher specific gravity, osmolality, and is darker in color. Urine biomarkers appear to be more sensitive to low or moderate levels of hypohydration than blood biomarkers [3]. Other biomarkers including salivary osmolality and potential cellular markers of dehydration have been studied, but these have yet to be established as accurate.

Biomarkers of Muscle Damage

Muscle “damage” incurred through overload forces adaptation and drives many positive responses for musculoskeletal development and performance enhancement. However, there are certain types of exercise (high-load eccentric contraction) [4] that induce profound damage that results in significant inflammation, tenderness, and temporary strength and power degradation as part of the repair process. During exercise in hot and humid environments, muscle or tissue damage may occur not only from the mechanical effects of exercise, but also from the damage associated with prolonged hyperthermia. In extreme cases, potentially fatal muscle tissue breakdown and necrosis resulting from exercise, hyperthermia, injury, or other unrelated causes is termed rhabdomyolysis. Assessment of the degree of damage can be used to quantify the magnitude of the training stress as well as to attempt to determine the recovery from high-intensity training to infer readiness and training status. Markers of muscle damage may also indicate the severity of tissue damage during exercise-heat stress and the recovery process from exertional heat illness and stress-induced tissue damage.

Most commonly employed measures of muscle damage are indirect in nature, with the most typical methods including blood-based biomarkers, maximal force development, or subjective assessment of muscle soreness [4]. Though direct assessments do exist in the form of biopsy or magnetic resonance imaging (MRI), these techniques are invasive or costly. Additionally, they are not without their own limitations. For example, the process of biopsy itself can cause muscle damage [4, 5] and induce responses that may mistakenly be inferred to have been caused by the exercise bout. Additionally, MRI signal intensity appears to peak *after* muscle soreness starts to dissipate and strength is reestablished [6], thus raising questions about whether the method is capturing a clear picture of the time-course of damage per se.

Typical laboratory methods used to induce muscle damage include downhill running and eccentric-emphasis contractions, such as maximal eccentric bicep curls. While the downhill running protocols have been found to induce force losses of as much as 10–30% for up to 24 h post exercise [7], high-force eccentric contractions have been found to result in 50–60% reductions and effects can persist for as much as 2 weeks [8]. There may even be a bimodal effect for strength loss with an immediate (and large) decrease after exercise, followed by some recovery, and then a second (smaller) decrease a few hours later that is more persistent [4]. Consistent with the magnitude of the force reductions, the delayed onset muscle soreness (DOMS) associated with these protocols also differs slightly, with greater DOMS associated with the maximal eccentric contractions and exposure to heat stress [9]. The levels of soreness appear to be associated with the swelling that occurs with muscle damage [10], though there is some disagreement about whether the time-course coincides [11]. The subjective nature makes assessment of DOMS problematic, particularly if that is the only measure of damage or recovery employed. Likewise, force production is somewhat dependent on motivation, which could be less than maximal, especially if still sore. For the assessment of recovery from tissue damage associated with exertional heat illness or heat stroke, laboratory-based testing or DOMS assessment are not contextually useful tests.

A more objective means of muscle damage or recovery assessment post exercise-heat related injury may therefore be the use of biomarkers. Blood proteins have been used to provide indirect evidence of damage, including lactate dehydrogenase, myoglobin, troponin, aspartate aminotransferase, aldolase, and creatine kinase (CK), among others [12, 13]. Of these, CK has received the most attention, probably because of the relative magnitude of its response as well as the low cost of the assay [4].

Much like the differences in force production and DOMS seen for downhill running vs. high-force eccentric contractions, CK also appears to differ in terms of magnitude (100–600 IU for downhill running vs. 2000–10,000 IU for eccentric exercise) and time of peak appearance (12–24 h following downhill running vs. 4–6 days following eccentric exercise) [14–16]. Theoretically, CK serves as a surrogate damage marker as it released into the blood through disrupted muscle membranes. However, its appearance is a function of both production in the muscle and clearance from the blood. As such, values can change depending on subsequent activity either because of further “massaging” of CK from the damaged muscle [17], enhanced clearance [18, 19], or even due to changes in lymph flow [20].

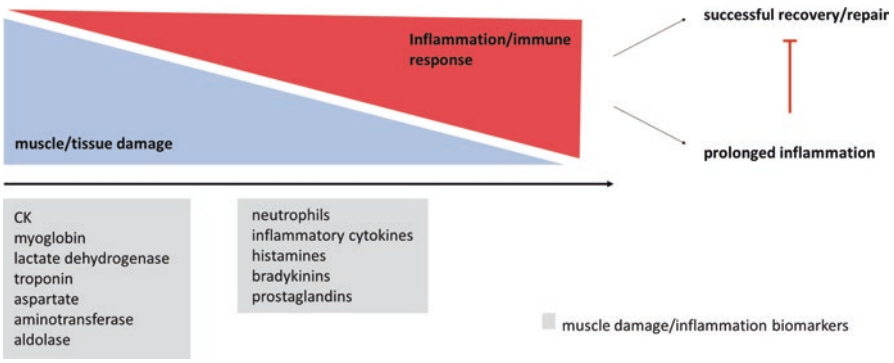

Fig. 11.3 Muscle damage biomarkers may detect tissue damage by-products that leak into circulation while inflammation/immune biomarkers will detect tissue damage-induced inflammation and repair processes

One criticism of the use of CK (or the other blood proteins) as a damage biomarker is the high individual variability of the response [4]. However, although the relationship is not perfect, individuals with the greatest damage as assessed by MRI also tend to have the greatest CK response [11]. Though the variability may be problematic for acute studies, particularly if employing small numbers of subjects, it may become far less of an issue when examining individual changes over time and when used in an applied setting to gauge damage and recovery. Additionally, other biomarkers have been explored that also correspond to muscle damage and, if used in conjunction with CK, may provide a more comprehensive picture of muscular trauma or insult. Recent evidence indicates that aldolase may have less variability than CK while also being related to DOMS [12].

During recovery from muscle damage, inflammation occurs and thus markers of muscle damage include markers of inflammation (Fig. 11.3). Neutrophil accumulation at the site of damage to eliminate necrotic tissue causes the release of proteolytic enzymes as well as the production of reactive oxygen species that further increase membrane permeability and damage, thus enhancing the CK and myoglobin (Mb) cascade [21, 22], as well as inflammation [4]. The inflammatory response is supported by the release of cytokines, including interleukin (IL)-1, IL-6, IL-10, IL-13, and tumor necrosis factor (TNF)-α. Not surprisingly, plasma increases in neutrophils precede their appearance at the site of damage and may provide an earlier indication of degree of trauma. Much like CK, their appearance also appears to be intensity and exercise dependent [23, 24]. The inflammatory cytokines IL-6, IL-8, and TNF-α (in circulation) seem to be particularly responsive to increasing demands of exercise [25], with IL-6 providing a unique contribution to the "damage" model as it appears to be responsive even to repeated bouts of activity and may be indicative of continued inflammation even in the absence of high levels of damage [26]. Currently, additional work remains to be done to elucidate the time-course of the inflammatory cascade as related to muscle damage and recovery and how hyperthermia can alter this response.

Other markers associated with recovery and neuronal responses to muscle damage have been studied as potential indicators of the degree of muscle damage and recovery from tissue injury. Histamines, bradykinins, and prostaglandins have all been associated with DOMS and this may be due to their interaction with afferent neurons relaying pain reception from the injured muscle to the central nervous system [27]. Circulating levels of these markers may indicate muscle damage post exercise, but the challenge in assessing these biomarkers is that these markers have not been as well characterized as a marker like CK.

Biomarkers of Immune Function

In athletes, functions of both the innate and acquired arms of the immune system are often observed to decrease after heavy exertion, typically 15–25% with recovery of immunity within 24 h [28, 29]; prolonged heavy training sessions in particular have been shown to decrease immunity [30] (for an overview of the immune system you are referred to Chap. 2 in the textbook, Exercise Immunology [31]). Athlete immune health is affected in unique ways during exercise in hot and humid environments and biomarkers of immune function respond differently in such cases.

During regular training and competition, many athletes experience exertional hyperthermia (internal body temperature >39.5 °C/103.1 °F) and dehydration and a few athletes also experience more extreme thermal stress such as EHI casualties (internal body temperatures can be >41 °C/105.8 °F). It is important to distinguish between the increase in body temperature that accompanies a fever (internal body temperature maintained at >37.2 °C/99.0 °F) and the increase in body temperature that accompanies passive heat exposure (e.g., when taking a hot bath or sauna) or vigorous physical activity. During passive heat exposure or vigorous physical activity, the hypothalamic temperature set point remains the same, but problems with heat dissipation cause body temperature to rise. During a fever, an increase in cytokines which act as endogenous pyrogens such as IL-1, IL-6, and TNF-α raise body temperature via an increase in the hypothalamic temperature set point. Pyrogenic, pro-inflammatory cytokines may contribute to heat storage during a fever in response to infection, inflammation, and perhaps muscle/tissue damage.

One area of continued research interest and the subject of a recent review [32] focus on the putative involvement of immune dysregulation and inflammation in altered thermal tolerance, EHI and EHS. Recent research showed that the inflammatory response to muscle damaging exercise (increase in circulating IL-6, a known pyrogen) correlated positively ($r = 0.67$) with the subsequent rise in body temperature during exercise-heat stress 30 min later [33]. Although an augmented inflammatory response may increase heat strain and the risk of EHI during acute exercise-heat-stress, research points to an important role for inflammation in acquired cellular thermal tolerance (e.g., cytoprotective role for heat shock protein 72) and the more chronic phenotypic adaptations associated with heat acclimation

[34]. Markers of inflammation and cellular thermal tolerance include cytokines, C-reactive protein (CRP), and heat shock protein 72 (HSP72).

Stress hormones respond to exercise and heat stress alter immune cell (leukocyte) function and leukocyte trafficking whereby exercise-induced increases in circulating catecholamines, epinephrine and norepinephrine, and cortisol [35] are involved in the immediate and delayed increase in circulating leukocytes (leukocytosis) and decrease (lymphopenia) after exercise. In the late 1990s, one of the pioneers of exercise immunology, Dr. Roy Shephard, hypothesized that exercise in adverse environments, with stereotyped stress hormone responses over and above that seen during exercise in favorable conditions, may cause greater disruption to immune function and host defense [36]. Exercising in the heat where internal body temperature increases by >1 °C/1.8 °F compared with thermoneutral conditions (where internal body temperature increase is <1 °C/1.8 °F) increases circulating stress hormones including catecholamines and cytokines, with associated elevations in circulating leukocyte counts [37]. Thermal clamp studies, where individuals exercised immersed to mid-chest in cold water (thermal clamp condition) or warm water, demonstrated a significant contribution for the rise in internal body temperature in the leukocytosis and cytokinemia of exercise [38, 39].

However, most of the available evidence does not support the contention that exercising in the heat poses a greater threat to immune function compared with exercising in thermoneutral conditions [40]. A number of studies have examined whether exercising with additional heat stress is harmful to the important functional properties of immune cells. With the exception of one study that showed a subtle reduction in stimulated lymphocyte responses after exercise with additional heat stress [41], tightly controlled laboratory studies show a rather limited effect of exercise with additional heat-stress on innate immune cell functions (e.g., neutrophil function, monocyte function, and natural killer [NK] cell activity) and mucosal immunity (e.g., saliva immunoglobulin-A (IgA)) [37, 40]. It is noteworthy that individuals exercising in the heat tend to fatigue sooner or reduce their work rate so their exposure to exercise stress in the heat tends to be self-limiting.

Continued research efforts are required to investigate the influence of various heat acclimation strategies on immunity and infection, including conventional exercise heat-stress approaches and novel post-exercise heat-stress approaches (e.g., taking a post-exercise hot bath [42] or post-exercise sauna [43]). The effects of not only heat stress, but blatant EHI, including EHS are likely much more dramatic on immune function than effects of simply exercising in the heat and thus markers of infection may indicate greater risk for EHI and markers of immune function may be informative about recovery from EHI. For detailed discussion of earlier studies investigating the effects of heat stress on immunity, please read the International Society of Exercise and Immunology position statement and earlier reviews [36, 37, 40].

The Evidence

The main concern with circulating biomarkers is that they often result in false-positive (low sensitivity) and false-negative (low specificity) diagnoses. This can have serious consequences for the patient due to misguided treatments that are not effective at preventing long-term organ injury. The search for new biomarkers is not a new endeavor for the field of EHS or other medical problems, as it has been going on for 15 years or more with little or no success. However, it continues to garner significant interest from the medical and research communities because there is so much variability in the use and diagnostic accuracy of available biomarkers. Perhaps one of the most important aspects to consider is reliance on a panel of biomarkers and their pattern of changes over time, rather than focusing on specific values for one or two biomarkers alone. This point was introduced several decades ago by Hubbard et al. [44] with respect to changes observed in a rat heat stroke model and also represents the approach that clinicians take in diagnosing the condition. Table 11.1 summarizes biomarkers related to EHI that are associated with different aspects of EHI risk or pathophysiology. Of course, each clinician and/or researcher will place emphasis on a different subset of biomarkers that they regard as having greater potential for prediction of severity, which continues to be a matter of considerable debate and inconsistency.

Biomarkers of Immune Dysfunction with Exertional Heat Stroke

EHS is often associated with immune disturbances consisting of a redistribution of circulating lymphocyte populations. The clinical blood test used to assay relative numbers/percentages of different types of cells in circulation is the CBC/differential (Complete Blood Count with differential analysis) and is an automated counting and biochemical analysis using flow cytometry and hematology equipment, as well as manual counting in some cases. CBC/differential provides information about different types and abundance of white blood cells (leukocytes), red blood cells, and platelets in a whole blood sample. Patients may present with elevated suppressor, NK cell and total lymphocyte counts and an attenuated helper-to-suppressor ratio. T lymphocytes showed attenuated mitogen activation through 24 h of recovery compared to the response to exercise alone. Unfortunately, it can be difficult to dissociate the effects of exercise vs. hyperthermia on cell immune disturbances and in some cases, these may have existed prior to exercise-heat stress in those patients with pre-existing illness. It was demonstrated in a baboon model of classic heat stroke that increased lymphocyte and T suppressor-cytotoxic cells were directly correlated with the severity of hyperthermia. That is, moderate heat stroke was associated with leukocytosis at 3–12 h of recovery whereas leukopenia was induced by severe heat stroke, which correlated with increased IL-6 production. The mechanisms proposed to account for changes in cell distribution include alterations in

Table 11.1 Summary of researched blood biomarkers associated with specific aspects of EHI/EHS pathophysiology

Pathophysiology associated with EHI/EHS	Biomarkers related to EHI/EHS pathophysiology
Immune dysfunction	CBC/diff Endotoxin/LPS Inflammatory cytokines (e.g., IL-1α, IL-1β, IL1-ra, IL-6, TNF-α, sIL-6r, sTNFr, IL-10)
Coagulation	Inflammatory cytokines CBC/diff (platelets, fibrin) TF (tissue factor) PT aPTT Fibrinogen D-dimer
Systemic inflammatory response syndrome (SIRS)	Inflammatory cytokines IFN-γ IL-2r IL-8 Acute phase reactants (e.g., CRP)
Organ damage	CK AST ALT UA Mb LDH Cr BUN Blood glucose
Miscellaneous tissue/cellular damage	HSP72 ANP BNP cTnI Cardiac fibronectin CKMB FABP (H-FABP, I-FABP) HMGB1 NO Serum nitrite/nitrate

regional blood flow, stress hormone release (cortisol, catecholamines), exercise, cytokines or endotoxin stimulation.

During heat exposure, there is a reflexive increase of blood flow to the skin surface to facilitate heat loss to the environment and reduce the rate of total body heat storage. This increase in blood flow is facilitated by a reduction in splanchnic blood flow to sustain adequate blood pressure. Prolonged reductions in gut blood flow cause ischemic stress to the epithelial membrane of the gut causing them to become "leaky" due to a breakdown of tight junctions. Endotoxin that is normally confined to the gut lumen is then able to leak into the circulation. The innate and adaptive immune systems work in concert to sense the presence of a pathogen and initiate the

appropriate systemic inflammatory response. The innate immune system, consisting of monocytes, macrophages, and neutrophils, recognizes pattern-associated molecular patterns (PAMPs) on the cell surface of pathogens to recognize their presence.

Toll-like receptor (TLR) 4 is a class of pattern recognition receptors that detects lipopolysaccharide (LPS; the lipid moiety on the cell surface of endotoxin) and elicits the appropriate immune response through stimulation of various gene transcription factors (e.g., NF-kB). Endotoxin is typically only detectable in the most serious cases of heat stroke that are associated with extreme body temperatures or when active cooling was not rapidly or aggressively initiated. It is noteworthy that splanchnic blood flow is not decreased until internal body temperature of 40 °C/104 °F is reached. Primates showed increased circulating endotoxin at rectal temperatures in excess of 41.5 °C/106.7 °F with a precipitous rise as temperatures reached 43 °C/109.4 °F. In classic heat stroke patients, endotoxin was detected at ~42.1 °C/107.8 °F and remained elevated despite cooling. Endotoxin was undetectable in patients exposed to heat stress that induced a mild hyperthermia (internal body temperature >39.5 °C/103.1 °F), suggesting that this was not robust enough a stress to induce gut leakage. In a young football player, high circulating endotoxin levels were associated with hemorrhagic necrosis of the liver on the second day of practice despite a body temperature as low as 40.6 °C/105.1 °F. However, multiple exercise-heat stress exposures are a risk factor for EHS, particularly early in the spring/summer months when acclimatization has not yet been achieved.

Importantly, neutralization studies support the hypothesis that endotoxin leakage is an initiating event for stimulation of a systemic inflammatory response syndrome (SIRS) that leads to multi-organ damage and/or death. This was shown in endotoxin-tolerant rats that were protected from heat stroke mortality as well as in dogs that were treated with antibiotics to reduce gut flora levels and showed improved 18-h survival rates (more than threefold compared to controls). It appears as though extreme heat can overcome the protective effects of endotoxin neutralization; primates treated with anti-LPS serum showed a reversal in heat stroke mortality until body temperatures were elevated to 43.8 °C/110.8 °F, at which point the protection disappeared due to irreversible organ damage. Paradoxically, C3H/HeJ mice (mice that lack the TLR4 gene due to a spontaneous mutation) have a diminished response to bacterial infection, but increased SIRS mortality due to the inability to induce the full complement of immune responses**.** The apparent paradoxical function(s) of the immune system at different stages of the SIRS is not a new phenomenon and has complicated interpretation of the action of several immune modulators (e.g., cytokines, discussed below).

Coagulation

Disseminated intravascular coagulation (DIC) may manifest as microvascular thrombosis or consumptive coagulation, which are very different clinical manifestations. Microvascular thrombosis is characterized by excessive fibrin deposition and/or platelet aggregation that occludes arterioles and capillaries to predispose to

multi-organ system dysfunction. Cytokines stimulate microvascular thrombosis through the activation of neutrophil adhesion and the release of reactive oxygen species that stimulated endothelial activation and injury. Tissue factor (TF) is a cell surface receptor that initiates coagulation following exposure to trauma, burns, and inflammation. TF expression is regulated by endotoxin and several cytokines including TNF-α and several interleukins that are stimulatory or inhibitory influences. This form of DIC is common with sepsis or trauma. On the other hand, consumptive coagulation is characterized by excessive blood loss due to platelets and coagulation proteins being consumed at a faster rate than they are produced resulting in hemorrhagic complications. This may include bleeding from venipuncture sites or the gums, which has been associated with fatal outcome in classic heat stroke patients.

Prothrombin time (PT), activated partial thromboplastin time (aPTT), fibrinogen, and D-dimer represent clinical biomarkers of coagulation. The aPTT in combination with PT is used to assess clotting time in response to exogenous TF, thus serving as a sensitive measure of the responsiveness of the coagulation pathway. PT is often significantly prolonged in heat stroke patients. Fibrinogen is an acute phase protein released by the liver that is indicative of liver damage at low levels or clinical signs of systemic inflammation when elevated. DIC may be difficult to diagnose, but low fibrinogen levels in combination with prolonged PT or aPTT are strong clinical indicators of disease severity in critically ill patients.

Biomarkers of the Systemic Inflammatory Response Syndrome

Hyperthermia can cause widespread tissue/cellular damage that may positively feedback to cause SIRS during which inflammation is dramatic, prolonged, and can cause multi-organ failure and death (Fig. 11.4). Please refer to *Pathophysiology of Heatstroke* [45] for comprehensive information about various mechanisms of heatstroke pathophysiology, including SIRS. Inflammatory cytokines are released by several cell types (e.g., macrophages, T and B cells, endothelial cells) and elicit pleiotropic, overlapping actions in a variety of disease states, including heat stroke. For many decades, consensus of the research community was that cytokines were adverse mediators of heat stroke that induced hyperthermia, coagulation, and multi-organ injury leading to death. Within the past decade or more, the dual actions of cytokines have come to light and it is now realized that the pro- vs. anti-inflammatory effects of cytokines depend on the milieu in which they function.

Despite evidence showing that cytokine injection induces symptoms similar to heat stroke, these studies do little to shed light on the actions of these proteins since they stimulate supra-physiological levels that are not relevant to the natural clinical condition. Several cytokines, including IL-1α and β, the IL-1 receptor antagonist (IL-1ra), IL-6, soluble IL-6 receptor (sIL-6r), IL-8, IL-10, IL-12, interferon (IFN)-γ, TNF-α, and sTNFrs are increased in the circulation of heat stroke patients with different timing and patterns of release in classic heat stroke vs. EHS patients. In a mouse EHS model, a very rapid increase in circulating IL-6 was observed at col-

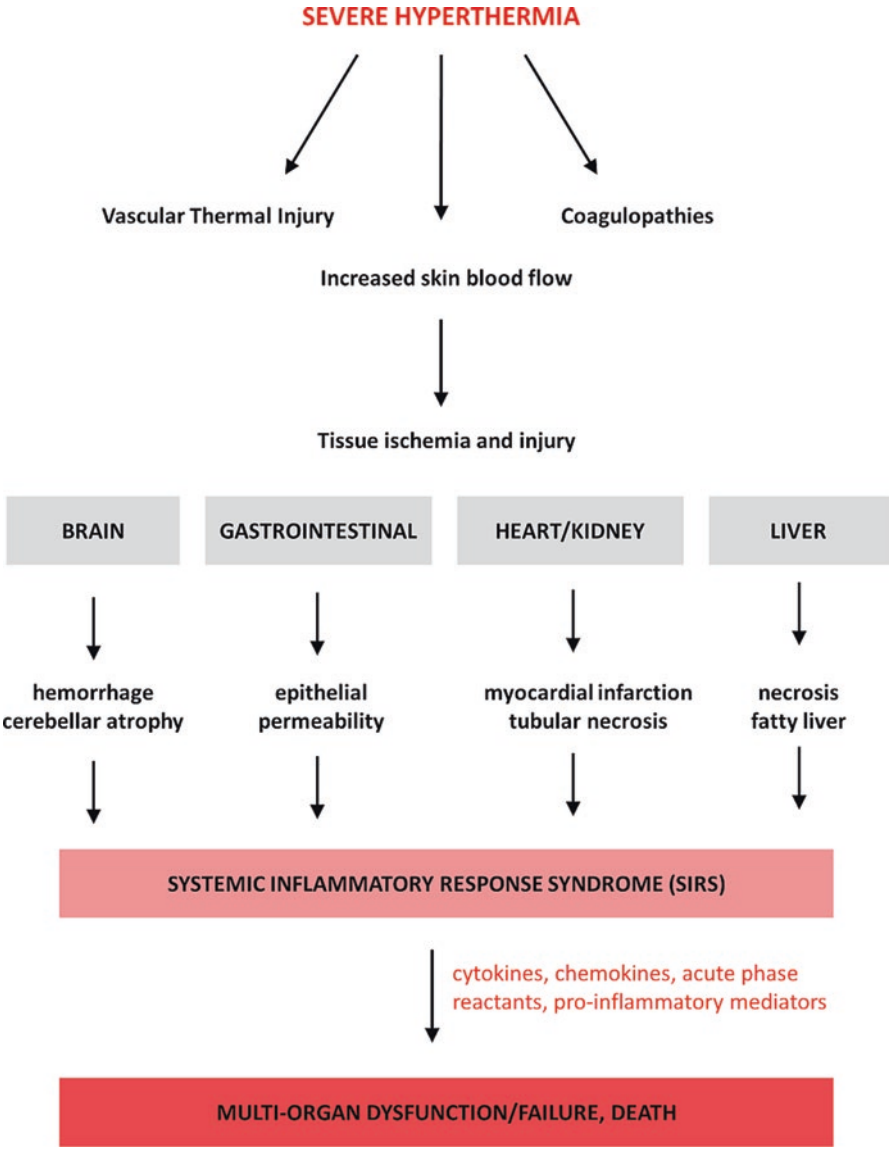

Fig. 11.4 EHI pathophysiology may include widespread tissue ischemia and injury that contributes to a systemic inflammatory response syndrome (SIRS) characterized by a marked and prolonged inflammation which can be detected in blood via cytokines, chemokines, and acute phase reactants. Figure modified from Leon LR (2015). Pathophysiology of Heat Stroke. Colloquium Series on Integrated Systems Physiology: From Molecule to Function. San Rafael, CA: Morgan & Claypool Life Sciences. DOI:https://doi.org/10.4199/C00128ED1V01Y201503ISP060 [45]

lapse, peaked at 30 min of recovery, and disappeared by 3 h, whereas IL-10 was not elevated at any time. This contrasts with classic heat stroke where both IL-6 and IL-10 peak at 3 h of recovery in the mouse. Moderate exercise superimposed on heat exposure would be expected to alter the pattern of circulating cytokine and chemokine production in EHS. It has been suggested that this response may be reflective of an endocrine reflex to exercise in heat that initiates survival pathways and early-onset tissue repair mechanisms. IL-6 is the one cytokine that has been most strongly and consistently considered an adverse mediator of heat stroke although the anti-inflammatory properties of this cytokine are starting to be realized. The view that IL-6 has only pro-inflammatory actions in heat stroke is based on two observations: (1) IL-6 is often elevated in 100% of the patient cohort and (2) sustained elevations in IL-6 in the recovery phase are correlated with mortality, which has been demonstrated in animal models and classic patients [46]. IFN-γ has also been observed in the circulation of classic patients more consistently than other cytokines (>50%), but appears to have little correlation with morbidity or mortality. However, EHI patients showed high IFN-inducible gene expression and IFN-γ levels that were elevated concomitantly with several other cytokines (IL-1β, IL-6, TNF-α, IL-2r, and IL-8). This appeared to be a consequence of pre-existing viral infections in these patients, which suggests that IFN-γ may be a sensitive biomarker to detect susceptibility in some patients prior to exercise-heat stress. It is difficult to provide a precise description of the time-course and pattern of cytokine release with heat stroke since there is so much interindividual variability that depends on the severity of heat stroke, activity at the time of collapse, immunological state of the patient, cooling methodology, and other intrinsic factors. More importantly, few neutralization studies have been performed, which are critical to understand the actions of these proteins. To-date, gene knockout studies indicate that most cytokines have protective effects at low concentrations, presumably due to their role as stimulants of the acute phase response. However, once high concentrations are detected in the circulation, this is likely a reflection of cellular spillover indicating that production is overwhelming metabolic/clearance pathways and the SIRS is venturing out of control and will likely lead to organ damage that will require more aggressive treatment. Furthermore, cytokines are involved in so many endogenous processes during homeostasis as well as cellular disturbances that it is virtually impossible to identify their specific function in the SIRS, which comprises so many simultaneous ongoing pathophysiological responses. Given that cytokines are not biomarkers routinely measured in the clinical setting, they are of interest from a research point of view, but provide little or no diagnostic value for physicians that have the challenge of treating EHS patients.

Biomarkers of Organ Damage

Traditional clinical biomarkers used to assess organ and/or tissue damage include CK, aspartate and alanine aminotransferase (AST and ALT, respectively), uric acid (UA), and Mb for skeletal muscle; AST, ALT, and lactate dehydrogenase (LDH)

for liver; and creatinine (Cr), blood urea nitrogen (BUN), and blood glucose for kidney. Unfortunately, an aspect of exercise-heat stress that is often ignored or misunderstood with respect to biomarker interpretation is that many biomarkers are altered by heat as well as exhaustive exercise, and released by multiple organs and tissues. Both of these factors can significantly limit the diagnostic value of biomarkers for accurate clinical assessment of organ damage. For example, CK is released from skeletal muscle, but high circulating levels may be indicative of rhabdomyolysis, myocardial infarction, or acute renal failure. Similarly, high AST and ALT values may be interpreted as an early clinical sign of liver damage in EHS patients, when it is more accurate to interpret this response as a consequence of intense endurance exercise. High BUN levels may be completely unrelated to EHS as they are also observed with other conditions, such as fever. Clearly, the interpretation of biomarker patterns must be made in the context of the environment, activity, immunological health, and other factors that the patient experienced prior to collapse. For example, many heat stroke victims have pre-existing conditions that induce thermoregulatory (e.g., fever) or inflammatory biomarker (e.g., IL-6) responses that were present prior to the exercise-heat stress. It is also important to distinguish between classic and EHS responses as the timing of peak responses and recovery differs between these conditions. For example, high enzyme levels are observed early in classic heat stroke patients, but may not be elevated until 24–36 h after collapse in EHS patients. Organ biopsy is the only accurate method to determine the degree, type, and dynamics of cell injury since blood enzymes and other biomarkers have been shown to normalize despite residual histological abnormalities [47]. A runner presented with normal liver enzyme and bilirubin levels within 60 days of EHS despite persistent liver damage through 12 months of recovery. Similar findings for BUN, AST, and ALT were observed in a rat classic heat stroke model that showed residual histological kidney and liver abnormalities despite normalization of those circulating biomarkers. Obviously, organ biopsies are not feasible in EHS patients such that reliance on circulating biomarkers becomes a more important aspect of clinical management to determine when an individual is ready to return-to-duty/play or normal activity without incurring a subsequent organ injury event.

Rhabdomyolysis is a form of skeletal muscle injury caused by the leakage of muscle cell contents into the circulation or extracellular fluid. Damaged muscle cells release Mb that is subsequently filtered by the kidneys; if the filtration threshold for the liver is exceeded (e.g., with severe muscle damage), Mb will appear in the urine as a reddish-brown color. High levels of Mb can be toxic to the kidney nephrons resulting in the overproduction of UA, progression to acute renal failure, coagulopathy, and even death if not rapidly detected and treated. CK, aldolase, LDH, ALT, and AST are all biomarkers of skeletal muscle injury and rhabdomyolysis that are influenced by several factors including type, intensity, and duration of physical exertion, as well as sex, environmental temperature, and altitude.

Hyper- and hypoglycemia are common in exertional and classic heat stroke, respectively. Hypoglycemia may occur with EHS, but this is rarely seen unless the

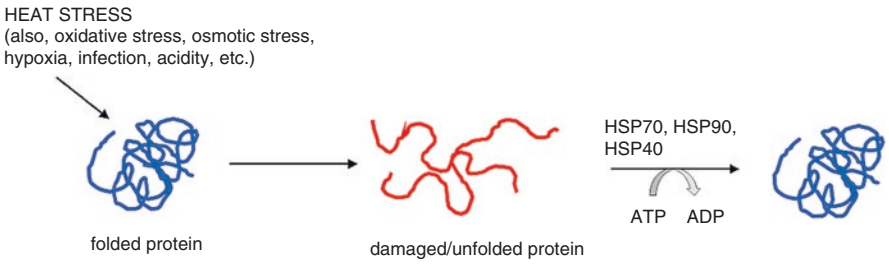

Fig. 11.5 Damaged/unfolded proteins during heat stress are repaired and refolded by complexes of stress-inducible and constitutive HSPs including HSP70 family members, HSP90, and HSP40

victim experienced recent prolonged caloric restriction with strenuous physical exertion. It has been suggested that disturbances in glucose homeostasis may be indicative of heat injury to the liver due to alterations of the phosphoenolpyruvate carboxykinase (PEPCK) pathway that regulates gluconeogenesis. However, heat-induced alterations of the PEPCK have not been experimentally validated as a mechanism for hypoglycemia.

Biomarkers of Cellular Heat Stress Response

Hyperthermia that leads to EHI and EHS induces tissue/cellular damage at the protein level, causing proteins to unfold and aggregate. A fundamental response to unfolded cellular protein is the chaperone response, which involves the expression of various chaperone and heat shock proteins (HSPs) that cooperate to repair or discard damaged intracellular protein. A core component of the chaperone response is heat shock protein 72 (HSP72, HSPA1A), a member of the 70kD heat shock protein family that is specifically induced during heat stress (and other stressors) and works with other HSPs to refold denatured protein (Fig. 11.5). HSPs have been proposed as potential biomarkers of EHS or EHI severity, but the challenge is that HSPs respond to diverse stimuli and have fundamental and diverse cellular functions. Furthermore, circulating levels of HSP72 increase post exercise-heat stress, but quickly return to baseline levels during recovery [48]. It is unclear whether HSP72 remains elevated and for how long after EHI or EHS. Cellularly and in circulation, it appears that HSP72 does not remain chronically elevated. It is oversimplified to consider HSP72 as a promising biomarker of EHI simply because it is a protein that responds to cellular heat stress. Further research needs to characterize its kinetics during EHI and EHS to accurately define it as a biomarker. Similarly, further research is required to define other components of the chaperone response as potential blood biomarkers of EHI risk, severity, or recovery.

Novel Biomarkers

Atrial natriuretic peptide (ANP), brain natriuretic peptide (BNP), and cardiac troponin I (cTnI) are biomarkers of cardiovascular dysfunction that have been shown to increase in response to heat strain. ANP type A and B mRNA expression peaked shortly after heat exposure of rats that reached a body temperature as high as 42 °C/107.6 °F. Cardiac fibronectin is a biomarker of myocardial damage that peaked from 4 to 8 h after heat exposure suggesting a direct effect of heat toxicity or ischemia.

Cardiac troponin I (cTnI) is present exclusively in cardiac muscle with higher sensitivity than CK-MB or troponin T for the detection of cardiomyocyte damage. The increased sensitivity of cTnI with EHS is likely due to its release from cardiac tissue alone since CK-MB is released from skeletal muscle of endurance-trained athletes after exercise. EHS was associated with cTnI levels in a dog that experienced systolic hypotension, multiform PVCs, and irregular myocardial echogenicity with poor left ventricular systolic function. The prognostic value of cTnI for assessment of cardiac damage with heat stroke has only recently been investigated in more depth. This investigation was driven by the observation that heart rate and blood pressure responses were more predictive of heat stroke severity than the severity of hyperthermia [49]. This suggested that a biomarker of cardiac dysfunction or damage, such as cTnI would be more sensitive to predict impending collapse and/or severity than reliance on a specific internal body temperature value [49]. cTnI was found in a rat classic heat stroke model to be significantly elevated at the time of collapse and correlate with heat stroke severity during 24 h of recovery to be significantly elevated at the time of collapse with slow resolution to baseline values through 24 h of recovery. Fingerstick testing of cTnI for point-of-care (POC) assessment of acute coronary syndrome has been proposed and was tested for its feasibility in the rat heat stroke model. The cTnI POC test correlated with multiple biomarkers of organ damage (kidney, liver), which demonstrated its predictive value for diagnosis and potentially early treatment of visceral organ damage during recovery. CK-MB (cardiac form of creatine kinase) and Mb may detect myocardial infarction in the absence of heat, but cTnI may be a more sensitive and specific biomarker of cardiac damage with classic heat stroke. It is interesting to point out that CKMB (cardiac form of creatine kinase) and Mb, despite their use to detect myocardial infarction in the absence of heat. These results suggest that cTnI is a more sensitive and specific biomarker of cardiac damage with classic heat stroke. Whether these findings will extend to the EHS condition remains unknown and may be complicated by the expectation that strenuous physical exertion may elicit cTnI release despite the absence of cardiac damage under these conditions. cTnI is not currently included in traditional clinical heat panels, which routinely consist of a complete blood count, metabolic profile, and markers of kidney and liver damage/dysfunction.

Fatty acid-binding proteins (FABPs) are intracellular lipid chaperones that have been strongly linked to metabolic and inflammatory pathways. There are nine types of FABPs that are named after the tissue where they were first identified, although most tissues express several isoforms of the protein. Heart-FABP (H-FABP) is released from injured myocardium and has release characteristics similar to Mb, although the cardiac tissue content of H-FABP is much higher than Mb with a higher plasma peak and more rapid release than either Mb or cTnI. This combination of biomarkers may be optimal for assessment of cardiac injury. Intestinal-FABP (I-FABP) is elevated in response to rat intestinal injury and in patients with intestinal ischemia and a SIRS. A progressive increase of plasma I-FABP levels was observed in response to a 60-min cycling test at 70% Wmax as measured by urinary lactulose/rhamnose ratio. FABP2 is a protein found in enterocytes of the small intestinal epithelium. Peak FABP2 concentration at 30 min of recovery was significantly higher in a mouse EHS model compared to controls and remained elevated at 3 and 24 h of recovery. FABP2 was also significantly elevated in a classic mouse model of heat stroke suggesting this is specific to heat-induced ischemia of the gut wall in the absence of exercise.

High mobility group box 1 (HMGB1) is a pleiotropic cytokine released in response to inflammation and tissue damage, including sepsis. Necrosis is the premature death of tissue or organ cells due to external factors, such as pathogens. HMGB1 is secreted by necrotic cells as well as oxidative stress-challenged macrophages and functions as an alarmin, which are endogenous PAMPs that initiate the restoration of homeostasis following infection or inflammatory insults. HMGB1 has been shown to interact with TLR4 (and TRL2) to enhance cytokine secretion in response to LPS. EHS patients show peak plasma HMGB1 levels within 6–13 h of clinical presentation that was positively correlated (r = 0.80) with the Acute Physiology and Chronic Health Evaluation II (APACHE II) score. Plasma and liver HMGB1 levels in rats correlated with increased plasma levels of AST and ALT at 77 min of heat exposure that induced hyperthermia to 43 °C/109.4 °F. HMGB1 monoclonal antibody pretreatment attenuated plasma ALT, AST, IL-1β, IL-6, and TNF-α and alleviated swelling of liver sinusoid endothelial cells. Elevated HMGB1 levels are often observed late in the recovery period and are associated with poor outcome suggesting it may be a sensitive biomarker of disease severity.

Nitric oxide (NO; otherwise known as endothelium-derived relaxing factor) regulates thermoregulatory vasodilatory responses during heat exposure and is produced in response to bacterial translocation, IL-1β and TNF-α production. Hyperthermia to 41.5 °C/106.7 °F was associated with increased intestinal expression of inducible NO synthase (iNOS). Increased NO concentration was detectable in peripheral splanchnic beds of hyperthermic animals and associated with tight junction permeability. NO is thought to mediate the hypotensive response to heat stroke due to high nitrite/nitrate levels in patients and increased NO levels observed in the circulation and portal venous (but not arterial) blood of hyperthermic rats. Serum nitrite/nitrate levels were positively correlated with the APACHE II score (a measure of heat stroke severity). Despite compelling evidence in favor of a role of

NO in the mesenteric vascular response to heat stress, in vivo and in vitro investigations have provided conflicting results. Sensitivity to acetylcholine, which causes arterial vasodilation through NO release from the endothelium, was decreased in vascular segments of the mesenteric arteries heated to 41 °C/105.8 °F. However, rat mesenteric arteries precontracted with norepinephrine did not show an attenuated response to acetylcholine when heat stressed to 42 °C/107.6 °F. The nonselective NOS inhibitor L-NAME reduced splanchnic blood flow and heme-NO production but had no effect on total heating time or thermal load in a rat heat stroke model. However, the protective effects of NOS inhibition appear to be dose-dependent as higher doses lowered heat tolerance and increased reactive oxygen species generation. It appears as though intact NOS activity is required for normal heat tolerance whereas overproduction of NO may cause a loss of splanchnic resistance that leads to heat stroke collapse.

NO affects multiple physiological processes in the kidney including salt and fluid reabsorption, renal hemodynamics, and renin secretion and has been implicated in acute renal failure in response to heat stroke. Effective renal plasma flow was negatively correlated with nitrite/nitrate levels in exertional heat stroke patients suggesting the downstream cytotoxic effects of NO metabolite production mediate acute renal failure. Pretreatment with the selective iNOS inhibitor L-N6-(l-iminoethyl) lysine (L-NIL) immediately prior to heat exposure was effective in attenuating renal iNOS and peroxynitrite immunoreactivity and protected against renal ischemia and damage in a rat heat stroke model.

The Application

Further research is required to validate the use of the many biomarkers that have been explored in EHI susceptibility, severity, and recovery assessment. Research should focus on assay of biological samples from EHI and EHS patients. Although exercise-heat stress may induce cellular and tissue responses that may be assayed in circulating blood, pathophysiology of actual EHI/EHS may reveal precise and accurate biomarkers; testing this requires large subject sample sizes.

Additionally, because of the widespread nature of EHI/EHS pathophysiology, and multiple roles of many of the studied biomarkers, it is recommended that biomarker analysis of EHI/EHS include simultaneous assay of multiple biomarkers. Taxonomizing responses of multiple validated biomarkers will provide more accurate information regarding EHI specifically. For example, because HSP72 is a candidate biomarker that may respond to a variety of stresses, to truly assess the risk for EHI in an individual based on HSP72, it is recommended that other biomarkers be assayed simultaneously to increase accuracy of interpretation. As the field advances, information about genetic susceptibility and efficient, noninvasive, inexpensive assays will help identify individuals with high risk of developing EHI and inform clinicians about progress in recovery from EHI.

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Chapter 12
Heat Tolerance Testing

Rebecca L. Stearns, Patricia A. Deuster, Josh Ben Kazman, Yuval Heled, and Francis G. O'Connor

The Physiology

Exertional Heat Stroke (EHS) is a life-threatening illness that affects both athletic and military populations [1–5]. It continues to be among one of the top three leading causes of death in these cohorts and remains largely preventable [1–4]. Although clear guidelines have been established for the successful treatment of EHS, the process of returning individuals who have survived EHS is much less clear. This is largely due to an inability to systematically research this topic in a human population coupled with the relatively low number of occurrences when compared to other major causes of death in the general population.

The initial purpose of the Heat Tolerance Test (HTT), originally developed by the Israeli Defense Force (IDF), was to determine a soldier's ability to return to duty following an EHS [6, 7]. Generally, EHS victims—treated aggressively and appropriately—have uncomplicated recoveries, assuming they are otherwise

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healthy [1, 8–13]. When optimal care is not applied or available, the recovery process can become varied and complicated [3, 4, 10–14]. Currently no validated, objective measure or procedure has been identified that can evaluate the body's ability to handle exercise heat stress (generally termed heat tolerance) following an EHS. This is analogous to testing for return to duty/play after a concussion, where consensus for an objective test to determine severity or prognosis is not yet available. Current concussion management incorporates clinical tools, which may include neurocognitive testing, functional rehabilitation assessment, and clinical judgment. We believe a HTT should be considered a realistic tool to incorporate into a larger decision-making process, comparable to concussion return-to-play, a tool that considers other factors impacting an individual's ability to return to a pre-EHS lifestyle.

Although the HTT has been determined to be a functional test of heat tolerance, the definition of heat tolerance is very loose and may depend on factors within and outside the individual's control. Currently insufficient data are available to support any HTT as a diagnostic test, but it does serve as a functional test to assess a person's responses within that moment in time for the conditions in which they are tested. This does not account for modifiable factors such as aerobic capacity, training, acclimatization status, or body fat, which have all been demonstrated as significant predictors of HTT outcomes. Additionally, it is impossible to identify whether risk factors present after an EHS were a result of, or potentially present prior to, the EHS, and therefore represent a non-modifiable risk factor.

At this point it is not certain which particular biological markers can be used to identify recovery or future risk for EHS, and the same holds true with regard to the diagnostic feasibility of the HTT. Importantly, the need for accurate assessment of an individual's ability to return to duty/sport is very different for military and athletic populations. This chapter will address the military model by focusing on how it was introduced to the military setting and adapted within athletic population. In addition, we will consider special circumstances as to when and how HTT can and should be used within sport and various models of HTT that have been used.

Heat Tolerance Physiology

Prior to discussing how a HTT is executed, it is important to review the thermoregulatory process the HTT is expected to challenge. Ultimately, heat tolerance depends on an individual's ability to mitigate and/or avoid extreme body temperature elevations when performing work in a thermally stressful environment. Although the factors and the body's coordinated efforts to tolerate various thermal conditions are dynamic and complex, the majority of the body's thermoregulatory capabilities in hot environments rely upon blood flow distribution, body composition/size, sweat evaporation, fitness/intensity of exercise, and acclimatization status. The heat balance equation provides a general summary of how the body compensates when exercise and heat exposure/production are combined:

$$S = M\left(\pm W\right)\text{-}E \pm R \pm C \pm K$$

S represents overall heat storage, *M* represents metabolic heat production, *W* represents mechanical work, *E* represents evaporation of sweat, *R* represents radiation (e.g., the sun), *C* represents convection (heat transfer through either air or water), and *K* represents conduction (heat transfer through direct contact, e.g., through a persons' clothes). Heat gain is indicated by a positive value, whereas heat loss is indicated by a negative value. Options for heat loss include the *E*, *R*, *C*, and *K* pathways, whereas options for heat gain include the *M*, *R*, *C*, and *K* pathways. Note that for an exercising individual, *M* will always be positive and contribute to heat gain while E will always contribute to heat loss [15].

The extent of heat loss depends on the temperature difference between the surfaces receiving and producing the heat, coupled with the heat transfer method (e.g., *M*, *E*, *R*, *C*, *K*) [16]. Conduction makes up a small amount of the total heat exchange (approximately 1%) and does not contribute greatly to cooling of the body [16].

During exercise in high ambient temperatures, when air temperature rises above skin temperature, cooling by radiation and convection is lost and sweat evaporation is the main method the body uses for cooling. However, sweat evaporation is only effective when humidity is low. Unfortunately, the rate at which the body can cool via evaporation decreases as humidity rises because sweat that cannot evaporate will drip off the body. Without evaporation, sweating is unable to contribute to cooling the body and thus only leads to further dehydration via fluid loss [16].

Whereas these factors will largely determine the heat loss potential, it is important to note that the factors contributing to the body's heat production and heat tolerance can be greatly impacted by the individual's body size (waist circumference, body fat), exercise intensity, hydration status, clothing/equipment/load, fitness status, dietary supplements, drugs, sleep deprivation, fatigue, sex, and age [17–23]. These will all be important when determining the cause and prognosis of an individual who has had an EHS.

Physiologic Markers of Heat Tolerance

Some biological markers have been associated with heat stress response and recovery. The majority of these markers are only detectable within the acute recovery window (and have not been associated with long-term recovery or as a predictor for reoccurrence), but they do represent a potential for future research to determine if such a marker could be useful.

Traditional markers to monitor post-EHS recovery include those related to liver function (aspartate aminotransferase, AST; alanine aminotransferase, ALT), kidney function (blood urea nitrogen, BUN) and muscle damage, most notably creatine kinase (CK). However, these markers can be altered by a variety of perturbations, and therefore are not necessarily specific to EHS cases [24]. Whereas traditional

recovery for EHS calls for these biomarkers to return to normal values, followed by a progressive return to activity, examples can be found for cases where despite a normal value, the individual has still not fully recovered from the EHS episode due to the inability to fully return to their pre-EHS activity level [25]. Therefore, current biomarkers remain as supporting values to aid in determining progression back to activity.

The Evidence

As mentioned previously, the initial purpose of the HTT developed by the IDF was to determine a soldier's ability to return to duty following an EHS. This protocol has also been adopted by several other national military organizations to assist in gauging if a military member can be returned to service. The HTT currently has limited utilization in the US military, typically being utilized only in complicated cases or those with evidence of sequela. The development and current uses of the HTT within both the Israeli and US models are outlined here.

The Israeli Model

The common practice in the IDF Medical Corps during the last few decades has been that all exertional heat related injuries patients undergo a standard exercise HTT about 6 weeks following the injury as part of the "return to duty" process [6, 7]. It should be noted that the test is not performed before complete clinical recovery. The IDF heat tolerance/intolerance criteria are mostly based on the changes in rectal temperature and heart rate during the test. The HTT protocol is performed during the early hours of the morning and for practical reasons, it does not control for time-of-year and patient acclimatization. Before the test, the subjects go through general medical exam and their baseline rectal temperature should be lower than 37.5 °C/99.5 °F in order to be cleared for the test. The HTT is performed in a special controlled environmental chamber under 40 °C/104 °F and 40% relative humidity. During the test, the subject is wearing light clothing (shorts and no shirt for men). The HTT consists of walking on a treadmill for 120 min at 5 km/h (3.1 mph) with a 2% incline [26, 27]. The tested subjects are instructed to avoid tobacco and caffeine prior the test, not to perform any exercise and not to drink alcohol for at least 24 h prior to the test, to sleep at least 7 h during the night and to drink 0.5 L of water during the hour prior to the test [26–28].

During the test, rectal body temperature and heart rate are continuously monitored and recorded, and sweat rate is computed from body weight prior to and after the test, corrected for fluid intake and urine output (if required). Heat intolerance is determined when rectal body temperature elevates above 38.5 °C/101.3 °F, heart rate elevates above 150 bpm, or when either value does not tend to reach a plateau

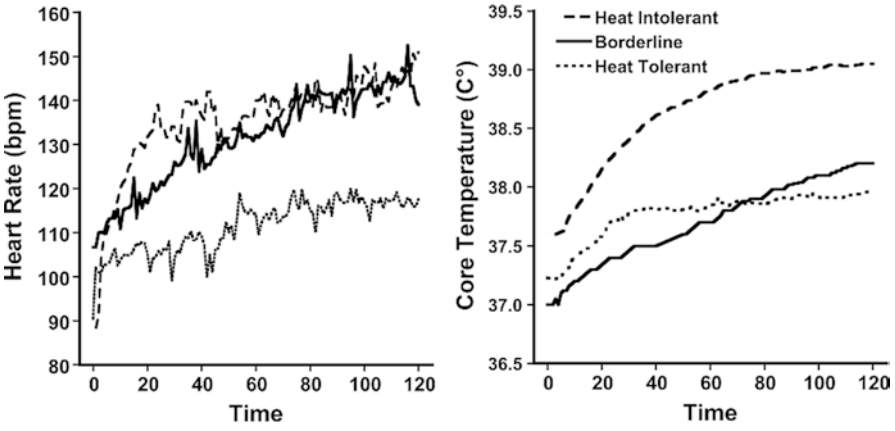

Fig. 12.1 Results of IDF heat tolerance tests for three participants, based on heart rate (left panel) and rectal temperature (right panel) over 120 min for (a) one who was clearly heat tolerant (dotted line), (b) one who was definitely heat intolerant (dashed line), and (c) one where the results are ambiguous (solid line)

[7]. Rectal temperature dynamics in heat tolerant and heat intolerant individual are presented in Fig. 12.1. Sweat rate is expected to be 0.5–1 L/h [7]. According to the IDF protocol, if the thermoregulatory response is abnormal (~5–10% of post-EHS cases), the soldier is scheduled for a second test that can be performed between 1 and 3 months later, depending on the test results and the severity of the prior heat injury. If the second HTT is again abnormal according to the standards, the subject is defined as heat intolerant and cannot continue his service in a combat military unit.

According to the IDF's decades of experience and hundreds of cases, this process has been proven to be successful with almost no indications of heat intolerance symptoms after returning to duty. Nevertheless, not all HTT results are straightforward. Although the final rectal body temperature and heart rate are significant criteria to the heat tolerance state, the dynamics of these measures, even if below the intolerance thresholds, may be important. For example, the "tendency to plateau" in borderline cases warrants clinician's subjective interpretation of the HT result. Therefore, supportive measures have been suggested by the IDF Institute of Military Physiology [18, 26] that quantify the dynamics of the physiological changes. The first supportive measure is the Thermal-Circulatory Ratio (TCR) index [26] which is calculated by the ratio between rectal body temperature and heart rate (TCR = rectal temperature/heart rate), where a higher value is associated with heat tolerance. A maximum TCR value of 0.28 °C/bpm / 0.50 °F/bpm or less at the end of the 120-min test has been suggested to be an effective measure to distinguish between heat tolerant and heat intolerant with specificity and sensitivity of 89% and 100%, respectively [26] (Fig. 12.2). In addition, a TCR value of 0.32 °C/bpm / 0.58 °F/bpm or higher, calculated after 60 min was found as a significant measure to determine heat tolerance with 100% sensitivity and 69% specificity. The latter threshold may

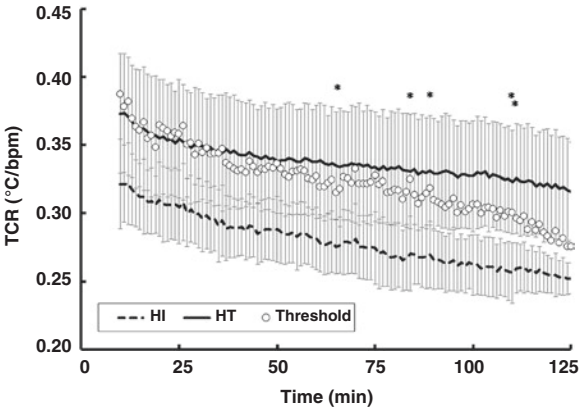

Fig. 12.2 Changes in Thermal-Circulatory Ratio (TCR) values (mean ± SD) during heat tolerance testing. *HT* heat tolerant; *HI* heat intolerant; *O* suggested threshold differentiated line between HT to HI [26]. Reprinted with permission from Ketko I, Eliyahu U, Epstein Y, Heled Y. The thermal-circulatory ratio (TCR): An index to evaluate the tolerance to heat. Temperature. 2014; 1(2):101–6

even assist in significantly shortening the HTT for those individuals whose TCR value matches this criterion. This index, although only a supportive measure, can surely contribute to prevent a misdiagnosis of heat intolerance (false negatives), with an adequate specificity to account for "false positives."

A second supportive measure that aimed to refine the distinction between heat tolerant and intolerant individuals during HTT, mostly in borderline cases, is the magnitude of increase in rectal body temperature during the HTT [18]. It has been showed that an increase of more than 0.45 °C/0.81 °F in rectal body temperature during the second hour of the test can be a good supportive measure to distinguish between a positive and a negative test. A value higher than this may be referred to as lack of tendency of the rectal body temperature graph to plateau during the HTT, which may be helpful in identifying heat intolerant individual.

It should be emphasized, however, that the HTT criteria have been set for relatively young and fit individuals (soldiers). Therefore, old, unfit, ill, and/or obese individuals may not meet the heat tolerance criteria due to conditions, which are not necessarily associated directly with their thermoregulatory dysfunction. Moreover, although HTT results may point to certain thermoregulatory related pathologies, it is not necessary that every heat intolerant state should be defined as clinical condition or disease. In many cases, heat intolerance results from low or inappropriate functional or physiological capabilities to cope with certain conditions (exercise and/or heat) which may be improved with proper rehabilitation and training.

Heat Tolerance Testing Within the US Military

In the US military, HTT is being used on an "as needed" basis for service members whose next duty assignment or training venue demands quantitative data as to whether the service member can be returned to duty after an EHS event. The results of the HTT are provided to the referring clinician to assist in making a prudent return to duty decision. This complex decision is made as a result of integrating the clinical picture, the HTT results, and the operational needs of individual and unit. It should be remembered that this decision is made to begin an appropriate acclimatization strategy unique to the environmental and operational stresses, and not to immediately return to full duty.

Although most HTT follow the IDF model—treadmill walking at 3.1 mph on a 2% incline for 120 min in an environmental chamber set at 40 °C/104 °F, 40% relative humidity, a slightly more demanding exercise protocol has been used by the Naval Health Research Center for Special Operations Forces. The person is asked to walk at 3.3 mph on a 4% grade. These two test protocols require an oxygen uptake of approximately 14.8 mL kg min^{-1} for the low and 18.7 mL kg min^{-1} [1] for the higher intensity exercise. Both would be considered moderate activity, but the relative workload would vary depending on the maximal aerobic capacity (VO_2max) of the person being tested. For example, if the person's VO_2max was 30.0 mL kg min^{-1}, then the relative workload would be about 50% of maximum. If the person being tested had a VO_2max of 60.0 mL kg min^{-1}, then the relative workload would be only 25% of maximum. Thus one would expect heart rate and rectal temperature to be higher in the less fit person as they would be working harder. This will be discussed further when the role of aerobic capacity is explored.

For the low intensity protocol, a person is considered heat intolerant if he/she meets any of the following criteria: a heart rate ≥150 bpm, a rectal temperature ≥38.5 °C/101.3 °F, or failing to achieve a plateau in rectal temperature between 1 and 2 h (change in rectal temperature ≥0.45 °C/0.81 °F). Given the low intensity nature of the exercise, these cut points are reasonable. Currently, the Consortium for Health and Military Performance has conducted over 120 HTT in the past 10 years. The participants have ranged from those with no prior history of any exertional heat illness event to those who have had severe episodes of EHS. Many of the participants that completed the low intensity protocol also completed a comparable exercise test but in a thermoneutral environment. Additionally, body mass index, percent body fat, VO_2max, sweat rate, urine specific gravity, and a variety of other measures were obtained.

One important detail to be considered is how to evaluate test results. The results of IDF HTT are shown in Fig. 12.1 for three participants, based on heart rate (left panel) and rectal temperature (right panel) over 120 min: (a) one who was clearly heat tolerant (dotted line), (b) one who was definitely heat intolerant (dashed line), and (c) one where the results are ambiguous (solid line). The last one would make the return to duty decision more complicated.

The heat tolerant participant's rectal body temperature, heart rate (Fig. 12.1), and change in rectal body temperature between hours one and two were all well within the tolerant criteria. The maximal heart rate achieved was less than 130 bpm and the maximal rectal body temperature was maintained at or less than 38.0 °C/100.4 °F. Likewise, the change in rectal body temperature was clearly less than 0.45 °C/0.81 °F. This test is easy to interpret, as all three criteria were met. Contrast that test with the HTT represented by the dashed line, where the participant's heart rate, rectal body temperature, and change in rectal body temperature were all consistent with criteria for being heat intolerant. Again, that test is clear cut. Importantly, the person's aerobic capacity was likely low as noted by the high heart rate at a relatively low workload, and the thermal responses indicated difficulty performing light exercise under this environmental condition. Now refer to the HTT represented by a solid line, where we have an ambiguous test and the person administering the test must make a clinical decision. First, the heart rate hovers right below 150 bpm and the rectal body temperature is below 38.5 °C/101.3 °F—around 38.0 °C/100.4 °F. However, when we review the change in rectal body temperature between hours one and two, the increase is greater than 0.45 °C/0.81 °F. These results are challenging because again, the exercise intensity is low. One question to ask is how would this person do when wearing body armor and carrying a heavy load, which would increase the absolute workload. Ricciardi et al. [22] showed that simply wearing body armor (10 kg) increased O_2 uptake by ~12% at slow (~2.4 mph at a 5% grade) and 17% at moderate (~3.8 mph at 10% grade) paces in a thermoneutral environment. In addition to body armor, service members often carry more than 40 kg, which could increase metabolic load by more than 50% [21]. Thus, in this ambiguous case we would want to determine the patient's VO_2max and if low, prescribe an exercise program and consider a heat acclimation protocol prior to returning the service member to duty. Importantly, the occupational demands of the service member would be taken into consideration.

When interpreting HTTs, it is important to consider the roles of fitness and fatness, transient deconditioning (due to EHS), and long-term pathology (likely due to EHS, or other factors). The two factors most strongly related to increases in rectal body temperature (Trectal) and heart rate during the HTT are percent body fat (Trectal, $r = 0.20$; heart rate, $r = 0.38$) and VO_2max (Trectal, $r = -0.13$; heart rate, $r = -0.45$). In the military, heat intolerant and ambiguous cases typically undergo a second HTT after 3 months. It is common that more time to recover and recondition results in improved HTT results. Individuals who fail a single HTT may simply require additional time to get back into shape. In contrast, individuals who fail multiple HTTs may require closer monitoring, or have an unidentified pathology, especially if they are otherwise aerobically fit and lean.

The importance of aerobic capacity cannot be understated—it matters and those who are physically fit aerobically will do better as noted above. Likewise, those carrying less body fat will fare better both during the HTT and in warm environments. Again, physical training and acclimation may be key. Although women have been found to be 3.7 times more likely to be heat intolerant than men ($p < 0.01$), the

increased risk is largely explained by differences in fitness (i.e., VO_2max) and percent body fat [17, 20]. These findings just reinforce the importance of fitness and fatness.

Finally, questions regarding whether different tests are needed for more fit persons and whether there should be a separate test for women have been posed. However, if a male or female service member cannot easily pass the current HTT, then they need to find a way to improve their readiness—a different test would not rectify the situation. In contrast, if an individual from an elite unit has had an EHS and needs to be evaluated for return to duty, a more strenuous test may be in order—especially given the need to wear body armor and other loads they carry. Data regarding the optimal workload for the missions could be used to develop a more operationally relevant HTT.

The Application

Using Heat Tolerance Testing for the Athlete

Generally, when EHS victims are treated aggressively and appropriately, they have uncomplicated recoveries, assuming they are otherwise healthy [8, 9]. In the event that optimal care is not applied or available, the recovery process can become varied and complicated. This is when a HTT for an athlete may be helpful. Most guidelines suggest the following for athletes recovering from EHS [1–3]:

- Monitor athlete until normal blood work is obtained.
- Wait at least 7–21 days before any activity is performed.
- Obtain physician clearance before any activity or progression for return to activity starts in conjunction with the first two criteria.
- Initiate activity with low intensity exercise in a cool environment to progressively increase to higher intensity exercise in a cool environment.
- Begin exercise progression within a warm environment once exercise in a cool environment is successfully achieved.
- Begin a period of heat acclimatization prior to full return to activity.
- Have a medical professional monitor for signs and symptoms of exertional heat illness.

Body temperature (either via rectal or gastrointestinal temperature) should be monitored during these early training stages and during the initial 1–2 weeks after returning to sport to ensure safe participation. Likewise, HTT could be considered when this progression is stalled due to reoccurrence of symptoms and/or an inability to recover from a previous day's training session. Importantly, if the individual had not received the standard of care for EHS (i.e., if the individual is not completely cooled to a rectal temperature of 102 °F/38.9 °C or lower within 30 min of the onset of EHS via cold water immersion), an HTT may be in order. Additionally, a HTT

might be considered when the person has had a previous episode of EHS as this could suggest other underlying pathology (e.g., malignant hyperthermia) [27, 29] or incomplete recovery from the previous EHS.

Variations of the Heat Tolerance Test for the Athlete

Use of the standard HTT protocol has been documented within the athletic population; however, it has received criticism for not being sport specific or reflective of the demands of sport [30–32]. Although the traditional HTT has been recognized as a good initial screening tool for advanced heat testing within the athlete population, it does not provide any further feedback regarding the individual's ability to handle exercise in the heat at higher intensities, much like those likely encountered in sport. Some preliminary steps have been taken to create a sport specific heat tolerance test for athletes [30–32], but no standardized or validated protocol has been established. Nonetheless, a monitored exercise test at a higher intensity in the heat could certainly provide additional information and greater assurance for the athlete's ability to handle similar loads during training and competition. A standard HTT should always be conducted first, and only with successful completion of this test should further progression or testing at increased intensities be considered. Certainly, if the athlete fails the standard HTT, they should not be allowed to continue to progress back to activity and consideration should be given to modifiable factors to focus on and thereby improve future testing results. The athlete would need to have the time to modify these factors (e.g., fitness, body fat, heat acclimatization status) before re-testing in 2–3 months. Inter-athlete variability and recovery time variations should also be considered as these can differ between individuals and cases. Table 12.1 provides a summary of reported methods that have been used to modify the traditional HTT to either a more intense test or a more sport specific test.

The protocol of Roberts et al. [32] is reflective of an attempt to recreate conditions that culminated in the individual's heat stroke and is specific to that person. Similarly, the protocol put forward by Johnson et al. [30] was used for an individual

Table 12.1 Various modified heat tolerance test protocols [30–32]

	Johnson 2013 [30]	Mee 2015 [31]	Roberts 2016 [32]
Exercise mode	Cycling	Running	Running
Intensity	70% VO_2max	9 km/h^{-1}	10.5–12.9 km/h^{-1}
Duration	90 min	30 min	70 min
Incline (%)	N/A	2%	0%
Air temperature	36 °C/96.8 °F	40 °C/104 °F	25 °C/77.0 °F
Humidity	50%	40%	60%
T_{rec} passing criteria	39.5 °C/103.1 °F	39.7 °C/103.5 °F	39.5 °C/103.1 °F
HR passing criteria	None	None	None

Note: T_{rec} rectal temperature, *HR* heart rate

who struggled while cycling in the heat. Finally, Mee et al. [31] were the first to create a modified HTT and examine its repeatability, which did demonstrate good agreement and low variability within participant's individual results. However, similar to the standard HTT, Mee's modified version does not identify an individual's susceptibility to a future heat illness, but limited to assessing physiological responses in the heat at a higher intensity.

Interpreting Success Within the Athlete Model

Returning an individual from EHS in a military setting may have greater consequences compared to an athlete. Failing a HTT could impact their career, but passing a service member and returning them to duty too early could place others at risk. This consequence is serious given that one individual who is not able to perform can compromise military success.

The options to monitor and ensure that the athlete is progressing, performing safely, and within expected ranges are far greater compared to the military model. For example, an athlete who has successfully completed a HTT and been approved to begin a heat acclimatization protocol should be monitored by medical professionals throughout the duration of this period. The medical professional can then monitor body temperature (either rectal or gastrointestinal) during exercise, and implement modifications or breaks as needed. Such options are rarely present in military scenarios. As noted above, athletes should be monitored during the initial weeks of returning to activity and demonstrate a successful full return to normal practices before tapering active medical monitoring.

Athletes may also benefit from having greater flexibility in their recovery time. Although uncomplicated cases may return to play within 2–3 months from their EHS, those with complicated cases may need 6–12 months. In these cases, a series of HTT may be beneficial to monitor the athlete's thermoregulatory responses, especially with a failure on the initial standard HTT, or if a sport specific HTT is performed. Once moderate to intense activity in the heat has been successfully documented and verified by body temperature monitoring, the medical team for the athlete can consider full return to participation. As with any return from a catastrophic injury, careful evaluation and monitoring is strongly encouraged, especially within the first 1–2 months of full return to activity.

Case Example *In February of 2014, 6'3" 300 pound lineman, Hunter Knighton, was a redshirt freshman starting spring football training at University of Miami. He reported to training despite feeling sick and worked through an intense practice. About 40 min into practice he collapsed with EHS. He was immediately transferred to the local hospital, but unfortunately proper policies were not in place to aggressively cool Hunter on-site. The lack of adequate on-site cooling (*e.g.*, cold water immersion, rotating cold towels, dousing with cold water) sent Hunter down the path of fighting for his life and long-term complications. His rectal temperature*

when admitted to the local emergency department was 109 °F (42.8 °C). Cooling blankets were applied, and Hunter was hospitalized for a total of 18 days, where he had experienced kidney and liver failure and had multiple seizures. In March 2014, he was finally released from the hospital.

In October of 2014 after 7 months of recovery, he was referred to the Korey Stringer Institute (KSI), University of Connecticut, for follow-up testing and an assessment of heat tolerance. He failed his first HTT in just over an hour, but this test served as a baseline for future assessment and a clear indication that he should not be exercising in the heat at this point. His fitness assessment and demonstration of being heat intolerance revealed two modifiable factors that could be influenced; aerobic fitness and heat acclimatization. First, he was instructed to slowly work on improving his fitness over the course of the next few months. In February of 2015, his VO2max demonstrated an approximate 35% improvement but he still failed the HTT. However, he was able to extend his time before failure by 32%. These remarkable improvements in fitness and duration for the HTT proved to be promising, and Hunter was encouraged to take another few months to recover and regain fitness. He returned in May 2015 for his third HTT, which he passed, a total of 450 days after his initial EHS.

After Hunter successfully passed the HTT, he was provided recommendations consistent with the EHS recovery literature, which included:

1. *Continue to perform conditioning workouts with the team outside to maintain heat acclimatization status. When pre-season starts for the fall, the university will naturally follow the mandated NCAA heat acclimatization guidelines to slowly introduce and progress to the next and last progression step for return to play: exercise in the heat with protective equipment. Intensity is a primary factor in body temperature increases, therefore this factor must be considered when a graded progression is made during the training period.*
2. *Hunter needs to report if he is not feeling well before practice; if he does, modifications to cut the intensity of practice or remove him from practice should be made.*
3. *Body temperature monitoring is strongly suggested when performing new/unique exercise or conditioning sessions, especially in warm to hot environments. This may represent a total of 3–4 weeks of the year (*i.e.*, the first 1–2 weeks of spring football and pre-season in August) since these are generally the riskiest times of the year for heat illness. Monitoring will help to ensure safe participation and confirm no need for exercise modification. For intense exercise in the heat, it is not unusual for body temperature to raise above 101 °F/38.3 °C. However, additional breaks and more frequent monitoring is called for if body temperature rises over 103 °F/39.4 °C and activity should be ceased at 103.5 °F/39.7 °C (until it returns back down to 103 °F/39.4 °C).*
4. *Fluid needs should be monitored, as demands increase with warm weather exposure and increase in activity. Matching fluid losses to fluid intake can help regulate body temperature during exercise.*

5. *All exercise progression should be conducted with the discretion and direct observation of a medical professional. Always monitor athletes for signs and symptoms of illness, and have emergency treatment protocols and equipment ready. Team practices may need to be modified based on extreme weather conditions.*

Because heat acclimatization has a significant impact on thermoregulatory responses, the medical staff recommended a heat acclimatization protocol for Hunter prior to the start of the fall pre-season. This gave him extra time to work during a period where he could be monitored more closely than during formal practices, which had not yet started. Hunter continued to practice with the team and on September 3, 2015 (18 months after his EHS) Hunter took his first teps back onto the game field, returning fully back to football.

Table 12.2 Ten steps for successful implementation when working with a complicated exertional heat stroke (EHS) case

1.	Evaluate modifiable and non-modifiable risk factors present for the EHS
2.	Wait until lab values return to normal before any activity takes place
3.	Once lab values return to normal and athlete has received physician clearance, evaluate opportunity to improve modifiable risk factors (e.g., fitness), if present, begin slow progression to improve those risk factors
4.	Allow return to activity progression to occur over the course of 2–3 months
5.	Schedule a heat tolerance test (HTT) 2–3 months after initial return to activity. If HTT is passed, then progress to sport specific exercise acclimatization and return to activity
6.	If initial heat tolerance test is failed, evaluate if further progression can be made with modifiable risk factors. If so, continue to address these over the course of the next 2–3 months and schedule another HTT
7.	If second HTT is failed repeat step 6, especially if HTT demonstrates significant improvement in performance and outcomes. If no improvement is seen and no further modifiable risk factors can be addressed, consider genetic testing for evaluation of non-modifiable risk factors. If HTT is passed, progress to sport specific exercise acclimatization and return to activity
8.	If first two HTTs are failed, after another 2–3 months, schedule a third HTT. If no improvement is seen and no further modifiable risk factors can be addressed, consider genetic testing for evaluation of non-modifiable risk factors. If HTT is passed, progress to sport specific exercise acclimatization and return to activity
9.	If at any stage a HTT is passed, be sure to educate the athlete regarding individual risk factors and strategies to reduce risk for future events prior to return to play. Begin gradual progression back to sport/exercise to eventually return to full participation with team/training
10.	Once athlete has been progressed and cleared for return to activity, recommend body temperature monitoring (via ingestible thermistor or rectal thermometer) during initial 2–3 weeks of training to ensure body temperature is responding normally and within a safe range (or any other period where heat illness risk may be high). Consider monitoring once environmental conditions start to get warm and pose a potential heat illness risk

Note: Complicated exertional heat stroke (EHS) = not treated with cold water immersion (CWI) and cooled to 102 °F/38.9 °C within 30 min, also resulting in extended hospital stay (>1 day) or blood labs that are elevated past 21 days from release of hospital or failure of a standard HTT 3 months after the EHS

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Part IV
Populations

Chapter 13
Individual Sports

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Introduction

Strain on the human system is inherent in all sporting disciplines, the magnitude of which is contingent on a multitude of physical, psychological, and environmental stresses. The strain individual sport athletes accumulate during activity will fluctuate based on factors such as intensity (maximal vs. submaximal), mode (simple vs. complex movement), duration (short vs. long), and environmental heat and cold transfer (evaporation, convection, radiation). Several other factors both intrinsic (e.g., heat acclimatization, hydration state, sleep deprivation, fitness level, motivation) and extrinsic (e.g., exercise intensity and duration, clothing and equipment, education and awareness of exertional heat illness among coaches, athletes, and medical staff) also add to the dynamics of thermal and physiological strain experienced by athletes [1]. Understanding sport and exercise-specific physiological demands placed on athletes will better equip coaches, athletes, and medical practitioners to anticipate and effectively mitigate potentially dangerous strain.

For individual sport athletes and sport federations alike, optimizing performance and maximizing health and safety is of considerable importance. Sport governing bodies play a large part in mitigating heat-illness risk through provisions of policies,

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rules, and guidelines that ensure minimal safety and health standards are employed [2]. However, due to a multitude of physiological and environmental factors contributing to heat-illness risk (Table 13.1), it is not possible for precautions across sport disciplines to be universal. Athletes, practitioners, and other support staff looking to maximize performance in the heat should implement sound tactics focused on reducing thermal strain before, during, and after training and competition. Therefore, it is imperative that comprehensive intervention recommendations be established for each sport based on its own unique demands.

The purpose of this chapter is to detail the challenges individual sport athletes face, describe physiological- and performance-related evidence behind these challenges, and provide recommendations and practical strategies that can be used to mitigate them. Specifically, we review the environmental and physiological stressors inherent in a variety of individual sports, and discuss how performance is influenced during heat-stress conditions. Although mental and emotional stresses are key influences on strain, the focus of this chapter will center on physiological and environmental contributors to heat storage and their effect on performance. Further, we demarcate individual sports by intensity domain, allowing for discussion of sports based on groupings of (1) high-intensity/anaerobic, (2) intermittent, and (3) endurance/aerobic. Endurance/aerobic sports have been subdivided into groups of running, cycling, swimming, triathlon, and auto racing to allow for a more in-depth discussion of differential challenges between multiple modes of activity.

High-Intensity/Anaerobic Sports

The Challenges

Individual sports that are high intensity in nature (e.g., 100–400 m sprinting or field events in athletics) are unique with regard to performance in the heat. Unlike activities at low or intermittent intensities, high-intensity performance may be improved in warm conditions [3, 4]. Dissimilar to longer duration event, heat storage time is minimal and sufficient time between bouts is available for heat dissipation. Sprinting, sprint cycling, sprint-swimming, throwing, and jumping performance is significantly influenced by peak power output or peak running/cycling/swimming velocity. When exercise duration is short, thermal and cardiovascular stress is normally not a significant factor unless the athlete is either exposed to high thermal stresses or hypohydrated prior to training or competition. However, sprint and power athletes should still be cautious when training involves repeated efforts over a longer duration in thermally challenging conditions.

Table 13.1 Individual sports and their risk factors

Risk factor	High environmental temperatures	High exercise intensity	Limited recovery between events	Water/food restriction	Limited access to fluids	Radiative heat exposure	Prolonged, continuous activity	High body fat %	Limited access to medical facilities	Large sweat losses	Protective clothing as a barrier to dissipate heat
Throwing	X										
Jumping	X			X							
Squash/ badminton		X									
Tennis	X	X	X			X				X	
Combat sports				X							X
Water sports						X					
Auto racing	X					X					X
Sprinting	X	X				X					
Middle distance	X	X				X					
Long distance	X	X				X				X	
Ultra-distance	X				X	X			X	X	
Open-water swimming	X				X	X	X	X			
Triathlon swimming	X	X				X	X				X
Road cycling	X	X	X			X	X			X	

(continued)

Table 13.1 (continued)

Risk factor	High environmental temperatures	High exercise intensity	Limited recovery between events	Water/food restriction	Limited access to fluids	Radiative heat exposure	Prolonged, continuous activity	High body fat %	Limited access to medical facilities	Large sweat losses	Protective clothing as a barrier to dissipate heat
Mountain biking	X	X	X		X	X	X		X	X	
Triathlon	X	X				X	X				X

The Evidence

Most of the knowledge gathered on the influence of heat stress on speed and power comes from laboratory rather than field studies. There have been retrospective studies analyzing track performance during varying environmental conditions. Guy et al. examined elite level track performance (e.g., 100 and 200 m during 1999–2011 *International Association of Athletics Federation* World Championships) and noticed a marked increase in sprint performance (~2% reduction in finish time) during hot (>25 °C/77 °F) vs. cool (<25 °C/77 °F) weather conditions [4]. The top annual performances in sprint and middle-distance events (100–1500 m) were also seen in host cities with traditionally warm conditions [5].

As ambient temperatures fluctuate, local muscle temperatures are affected passively through heat transfer between the environment, skin, and muscle. Muscle temperature has been shown to subsequently affect performance in a dose-response manner [3]. That is, as muscle temperature increases or decreases there is a positive or negative effect on performance, respectively. Although not fully supported, short-duration exercise has been observed to increase 2–5% for every 1 °C/1.8 °F increase in muscle temperature [3]. Improvements in muscle function and power are evident when a muscle is warmed up prior to exercise [6] or when exposed to warm conditions [7]. This appears to be linked to enhanced muscle contractility and neural drive [3]. However, improvements in power output diminish when sprinting bouts are repeated [7, 8] or when central body temperatures elevate to detrimental levels (~39 °C/102.2 °F) and performance suffers from a reduction in voluntary muscle activation [9].

The time of day of exercise can affect short-duration sprint and power performance [10]. This phenomenon is linked with the body's natural circadian rhythm as internal body temperature fluctuates throughout the day, with the morning hours showing lower body temperatures than in the afternoon. Afternoon performance testing has shown peak performance measures such as vertical jump height, sprint cycling [10], and outdoor sprint velocity [11], when compared with morning testing. When body temperature is low, high ambient temperature can increase local muscle temperature to a greater degree, subsequently improving muscle contractility, force, and short-duration performance [3]. This is an important consideration for sprint and power athletes while training and competing in the morning.

The Application

Compared to intermittent and aerobic-based sports, high-intensity/anaerobic sport athletes are less affected in hot environmental conditions. However, there are several practices these athletes can employ to maximize performance and minimize risk of exertional heat illness during training and competition (Fig. 13.1). Sprint and

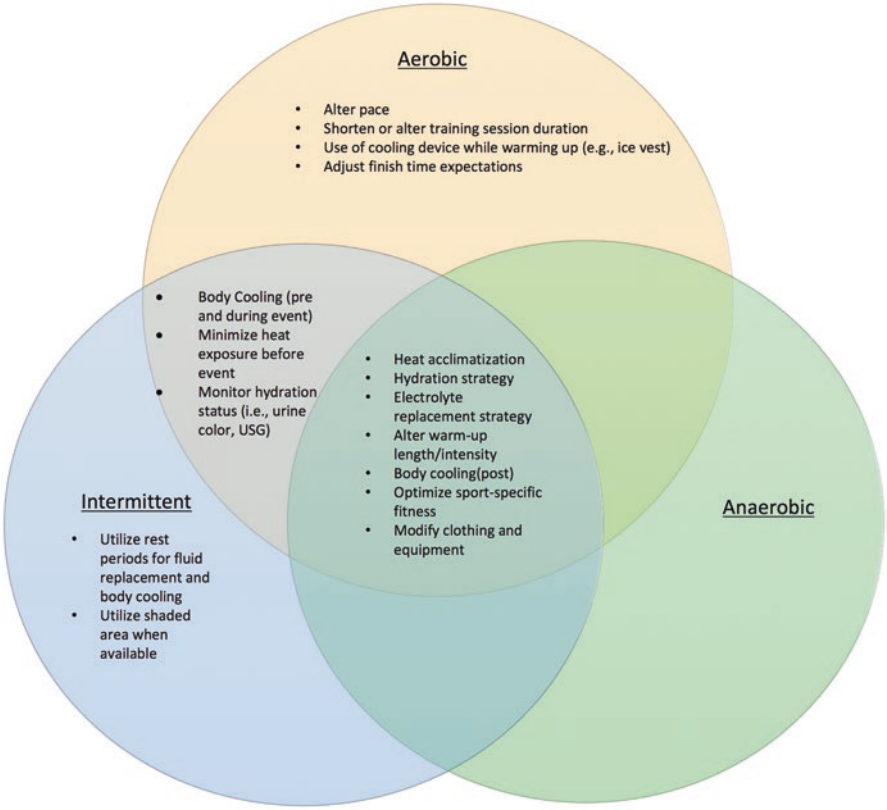


Fig. 13.1 Strategies for maximizing performance and safety in the heat

power athletes should warm up effectively to increase muscle temperature during both hot and cool conditions, especially in the morning when body temperatures may be low. Both passive and active heating may be used to increase muscle temperatures; however, athletes should avoid prolonged and intense warm-up session in the heat which may raise internal body temperature too high. While there is no optimal internal temperature that can be generalized for all athletes, raising body temperature to levels that could induce heat illness should be avoided. Although progressive dehydration is not a primary concern for sprint and power athletes, attention should be paid to maintaining adequate hydration and electrolyte balance. Maintaining adequate hydration and electrolyte balance is of particular importance when intensely training in hot and humid conditions.

Intermittent

The Challenges

Intermittent-intensity sports vary greatly in activity type and load influencing variables such as intensity, duration, and length of rest period. Intermittent-intensity racquet sports such as tennis, badminton, and squash require short and explosive changes of direction, complex neuromuscular actions (e.g., forehand, backhand, serve), and brief recovery periods between points (e.g., normally 15–20 s between points). In contrast to many other sports, competition duration is variable, with some matches lasting less than an hour (e.g., 30–40 min during a normal squash match) and others lasting for several hours (e.g., 3–5 h during tennis matches extending to five sets). Combat sports such as wrestling, martial arts, and boxing have a different intermittent activity profile as rounds are fixed around 2–5 min with ~60–90 s breaks between rounds.

Intermittent-activity sports are played in a variety of environmental conditions ranging from mild (i.e., indoor events or temperate outdoor conditions) to extreme (i.e., high outdoor ambient temperatures, humidity, solar radiation, and reflective solar energy from court surfaces). The risk of exertional heat illness from environmental factors during indoor individual sports is relatively low; however, the absence of air conditioning or limited airflow while indoors could make this environment challenging. Intense and repetitive movement can produce high levels of metabolic heat; however, mild conditions make heat loss characteristics favorable. As such, indoor intermittent-intensity sport athletes are more often limited by intrinsic factors such as fitness, hydration, and fatigue state. However, concerns associated with combat sports are different than other indoor intermittent sports. Combat athletes are classified by body mass, which is used to match participants of relatively similar body characteristics in efforts to reduce injuries and mass-associated advantages (i.e., facing a smaller and/or weaker opponent). The primary concern surrounding these athletes is rapid weight loss behavior prior to weigh-in for the purposes of competitive advantage.

Outdoor individual sport athletes, as in tennis, will incur similar activity stresses as indoor intermittent sport athletes however will experience added thermal stress from varying environmental conditions and longer overall durations of exercise and heat exposure. In addition to challenging physical demands and environmental fluctuations, tennis requires multiple-day match exposure over the course of a tournament (i.e., Grand Slam finalists play seven matches in 14 days) and can entail same-day matches (i.e., singles and doubles match on the same day). In many regular tournaments, individuals will need to play five singles matches in less than 7 days to win a tournament.

The Evidence

Combat athletes can lose 2–10% of body mass prior to a weigh-in and will typically begin the process of rapid weight loss 2–3 days prior to competition [12]. Extreme hypohydration and vigorous exercise in heat-stress conditions can cause rapid increases in body temperature. Weight loss techniques such as fluid and food restriction, saunas, vigorous exercise in non-breathable clothing, and bulimia have been historically used to rapidly "cut" weight [13]. Although the effectiveness of rapid weight loss on performance is controversial, with some evidence pointing to clear augmentation and others showing impairment [12], it is clear that starvation and fluid restriction can be fatal and should be avoided [14].

In heat-stress conditions, outdoor intermittent sport athletes can experience significant sweat and electrolyte losses. Young adolescent tennis players have shown sweat rates of 1 L h^{-1}, while older adult players can exceed 2.5 L h^{-1} during intense practice or competition in the heat [15]. Often, tennis players will not fully replace fluid deficits from previous same-day matches, particularly junior tennis players [15], which can have negative impacts on physiological strain and performance during subsequent play. This "carry-over" effect is frequent in tennis with multiple same-day matches and sometimes relatively short recovery periods between play [15]. Slowly accumulating dehydration can lead to excessive thermal and cardiovascular strain and can have a marked effect on racquet sport performance. Davey et al. showed tennis serve accuracy can decrease around 30% and ground stroke accuracy around 69% while dehydrated [16].

Evidence suggests the intermittent nature of tennis in healthy athletes allows for adequate thermoregulatory responses under a variety of environmental conditions. Bergeron and colleagues have shown internal body temperature in elite-junior tennis players reach 38.7 °C/101.7 °F, with few players exceeding 39 °C/102.2 °F [17]. Similarly, Tippet et al. showed professional female tennis player's average internal body temperatures of 38.6 °C/101.5 °F and peak temperatures over 39 °C/102.2 °F [18]. It's been proposed that when thermal and perceptual strain increases, point duration and effective playing time decrease to modulate external workload and internal heat load [19]. However, during intense and closely contested matches athletes may maintain a high external workload, which would be expected to increase thermal load even further if the competition is held in oppressive environmental conditions [20].

The Application

Intermittent sport athletes should look to maximize sport-specific endurance capacity. Optimal sport-specific aerobic capacity is critical for recovery between short bursts of activity or demanding training sessions. Athletes can train sport-specific endurance capacity by utilizing work to rest ratios and activity patterns inherent to

their sport. Maintaining heat acclimatization year-round and modifying training loads in extreme environmental conditions can help to minimize thermal strain leading up to demanding events. Because sweat rates can vary based on a multitude of factors, the need for individualized hydration strategies to account for fluid and electrolyte losses during competition should be emphasized. Identifying sweat rates can be useful in determining fluid intake during rest periods (i.e., changeovers, set changes, or between rounds). Measuring sweat-electrolyte concentrations can aid in determining appropriate fluid-electrolyte beverage composition. Effective and long-term weight control programs and modifications are an important aspect of maximizing performance through physical preparedness. Athletes should be educated on the adverse effects and risks of rapid weight loss. A licensed sports dietician should be consulted for reasonable weight loss goals and strategies. In efforts to protect the health and safety of their athletes, combat sport governing body should consider rule provisions or monitoring tactics to reduce the incidence of rapid weight loss tactics.

Endurance/Aerobic Running

The Challenges

Running presents significant challenges on the cardiovascular and thermoregulatory system, particularly when intensity and distance increase during heat-stress conditions. The risks of reduced performance and exertional heat illness while running will typically vary by event distance and duration of run time. Middle-distance events (800–5000 m) are much higher in intensity than longer events, leading to higher rates of metabolic heat production. However, run time is relatively short in these events (e.g., <2 min and <14 min for elite 800 m and 5000 m runners, respectively) and the risks of exertional heat illness are therefore relatively low. Performance at these distances will be influenced less by environmental conditions when compared to longer events. However, performance can be impacted by factors such as dehydration, fatigue, and illness. Longer events (10 km–26.2 miles) are characterized by lower intensity and thus a lower rate of metabolic heat storage compared to middle-distance. However, heat storage time is increased. Factors such as physical fitness, heat acclimatization, and fluid imbalances can have a bigger impact on performance during longer duration events. Progressive dehydration is common during prolonged running in the heat and presents distinct challenges as both the skin and working skeletal muscle compete for blood flow. Conversely, if fluid consumption is excessive and body water accumulates, runners are at increased risk for hyponatremia (i.e., low sodium concentration in the blood). At ultra-distances, such as those covered in an ultramarathon (>26.2 miles with some races extending beyond 100 miles), the intensity is considerably lower than in a traditional marathon. Ultramarathon runners are normally very well trained, heat acclimatized, and

careful with hydration and fuel replacement practices. However, ultramarathon runner incurs distinct challenges related to muscle damage, fluctuating environmental conditions, and sleep deprivation as races can extend beyond 24 h.

Both duration and intensity are important factors every runner must navigate safely as inappropriate pacing in hot environments can be detrimental. During typical training a runner is likely to alter or stop activity when significant heat stress is perceived; however, highly motivated and competitive athletes will often times disregard perceptions of stress during a competition. Middle-, long-, and ultra-distances present unique challenges for the body's regulatory system and even more so when running in conditions are unfavorable for heat dissipation (e.g., high ambient temperature, humidity, and solar radiation).

The Evidence

High ambient temperatures can have a significant effect on running performance, specifically longer duration running. Guy et al. found that at World Championships athletes in aerobic endurance sports performed worse (~3% reduction in finishing time) in hot conditions (>25 °C/77 °F) than in mild conditions (<25 °C/77 °F) [4]. However, middle-distance events were only somewhat affected and greatly varied in performance improvements and impairments. As discussed in the high-intensity/anaerobic section, high ambient temperatures can actually augment running performance during sprinting and shorter-duration runs.

One of the greatest decrements to endurance aerobic performance is the development of hyperthermia (i.e., internal temperature >38.5 °C/101.3 °F) [21]. Competition for blood flow between skin for heat loss and active skeletal muscle for metabolism of oxygen for adenosine triphosphate generation can create a challenge for the cardiovascular system to overcome. In heat-stress conditions, thermoregulatory burden is high and sweat rate increases, and the activity may be sustained for several hours resulting in considerable fluid and electrolyte imbalances [22]. Excessive fluid losses (i.e., reduced blood plasma volume) can greatly impact cardiovascular capacity to compensate to heat stress and provide oxygen to working muscles, which can impair performance [21]. Dehydration can additionally lessen thermoregulatory benefits gained when acclimatizing to the heat. The positive physiological adaptations that occur during heat acclimatization (e.g., decreased internal body temperature and increased heat dissipation mechanisms) have shown to diminish when the individual is hypohydrated. Pinchan et al. found similar work capacity decrements in both heat acclimated and unacclimated individuals in a hypohydrated state [23]. The effect of dehydration on run performance also appears to be dependent on run distance. Armstrong et al. found that fluid loss equivalent to just 2% of body mass can impair running performance at 1500, 5000, and 10,000 m distances [24]. However, fluid losses have a greater effect on longer runs as performance decreased by approximately 5% at 5000 and 10,000 m and by just 3% at 1500 m.

The Application

All runners should use precaution when training or competing in heat-stress conditions, particularly in conditions of elevated ambient temperatures, humidity, and solar radiation. Prior to competing in thermally challenging conditions, several preparation strategies should be used to maximize performance and decrease the risk of heat-related illness. Runners can reduce heat strain through tactics such as behavioral modification and heat acclimatization. Gradual increases in distance and intensity during training will help to maximize physiological heat loss mechanisms through acclimatization (i.e., increased sweat rate and decreased sweat-sodium concentration). A sensible progression in exercise duration and intensity will allow for adequate acclimatization in 10–14 days. Although running in the heat does improve heat tolerance, caution should be used when running long distances or at high intensities in elevated heat-stress conditions. Runners should consider rescheduling hard training sessions when conditions are dangerous. In addition, runs should be avoided when runners are experiencing or recovering from illness.

When exercising in the heat, runners can reduce cardiovascular and thermal strain by adequately and regularly hydrating prior to and during a run. Efforts should be made to minimize body fluid losses to less than 2%. Fluid consumption and concentration should be appropriate based on sweat rate and electrolyte losses. Additionally, excessive fluid intake should be avoided to reduce the risk of hyponatremia, especially during longer duration and lower-intensity runs (e.g., marathon and ultramarathon). Runners should minimize heat exposure in the days immediately leading up to competition to reduce the cumulative effects of thermal load. Race athletes can additionally employ cooling strategies (i.e., cooling towels, ice slurry, ice cubes down the back) to reduce body temperature and perceived thermal strain before or during exercise. **In addition, race organizers should do everything in their power to limit casualties due to heat. An exemplary case is described in** Case 1.

Case 1: Vermont City Marathon 2016—An Example of Proper Race Management in Unseasonably Hot Conditions *The Vermont City Marathon takes place in the Northern Vermont city of Burlington at the end of May. The average temperature for this time of year is 67 °F/~19 °C which does not typically impose excessive risk of exertional heat illness. However, in 2016 in the weeks leading up to the race the forecast was for unseasonably hot temperatures into the mid-80s. At this time, it was too late for athletes to become heat acclimatized as they would for a race which typically occurs in a hot climate. Therefore, the race management took all necessary steps to minimize risk/limit damage of exertional heat illness at the race. This includes three tiers:*

1. *Alert runners to what they can do to limit the risk of exertional heat illness*
   - *Hydrate prior to the race*
   - *Monitor urine color to ensure proper hydration*

- *Adjust race finish time expectations*
- *Learn signs and symptoms of exertional heat illness*

2. *Increase ability to minimize overheating*

- *Nine additional misting stations*
- *Five additional ice station*
- *Four roving ice patrols*
- *Doubling the ice on order*
- *Increase water supplies*
- *Extend maximal finish time from 6 to 6.5 h*
- *Messaging to runners to slow down, hydrate, and keep an eye on one another*
- *Alert community to place hoses in their front yards to help cool athletes*

3. *Improve monitoring*

- *Added shuttle busses to pick up athletes that drop out*
- *Close race course if wet bulb globe temperature >82 °F/27.8 °C*
- *Increased EMS presence*
- *More volunteers staffed at points towards the end of the race*

Unfortunately, this race had to be stopped prior to all capable athletes crossing the finish line. However, with proper preparation and dissemination of information to participants the risk of severe exertional heat illness was mitigated. The athletes that did not get to finish were upset but understanding. As athletes it is important to remember when event temperatures are higher than expected to first think of steps to improve safety, and second think of steps to maintain performance. In many cases, these steps run in parallel.

Swimming

The Challenges

Swimming is a unique sport in that it takes place with the body mostly submerged in water which limits risk of exertional heat illness because there is a large gradient for heat movement from the skin's surface into the surrounding environment. Also, water has a specific heat that is higher than that of air, meaning that as a swimmer occupies a position in the water, the water next to the skin can absorb more heat before it warms. Finally, as swimmers constantly move, the water next to the skin is continuously being replaced and a microclimate of warmer water next to the skin's surface cannot form. The temperature gradient, specific heat, and convective cooling force related to water's effective removal of heat from a human body is exactly the reason that cold water immersion, with water circulation, is the gold standard for the treatment of exercise-associated hyperthermia. However, there are several factors related (specifically to open-water swimming) that can increase the risk [25].

This section will not discuss in-pool swimming events because they are generally shorter in length (i.e., <16 min) and the temperature of competition pools is mandated to be between 25–28 °C/77–82.4 °F which allows for appropriate loss of excess metabolic heat.

The risk of exertional heat illness during open-water swimming is related to uncontrolled water temperatures, long duration, radiative heat gain, and body composition. Many open-water swimming competitions occur in warmer bodies of water such as in the Middle East or the South China Sea where water temperatures can reach 32 °C (89.6 °F). As water temperatures approach that of body temperature, the gradient for the removal of metabolic heat decreases, and heat storage increases. Open-water events are typically longer (5–25 km for official competitions, with distances as far as 46 km being completed by those that circumnavigate the island of Manhattan). As distance increases, so does the storage time for metabolic heat. However, the highest risk for exertional heat illness would be in the 5–10 km distance which typically spans 1–2 h of higher intensity swimming. Open-water swimmers are typically exposed to sunlight which can increase radiative heat gain. Lastly, competitive open-water swimmers have been documented to have a higher percentage of body fat when compared to in-pool swimmers [26]. This aids in buoyancy over long distances and in thermoregulation because water temperature is more frequently cold. However, when water temperatures are high the excess body fat can inhibit transmission of metabolic heat from the core to the skin for dissipation into the environment.

Triathletes introduce an additional risk factor as they frequently wear neoprene wetsuits during the swim leg due to the cold water temperatures, and the buoyancy advantage that the suits offer. However, the suit can become a risk factor when water temperatures increase. Tight-fitting, full-body suits create a microclimate of water trapped within the suit which does not allow for the same levels of convective heat loss to the environment as described above. Reduced heat loss due to wetsuits must be carefully considered when choosing to wear a wetsuit in races.

Overall Risk in Open-Water Swimming

The Evidence

Swimming in warm water (32 °C/89.6 °F) increases sweat rate, and body mass loss, and the gain in body temperature over time [25]. This does not appear to increase body temperature above normal thermoregulatory thresholds (i.e., >39 °C/102.2 °F), and it is not clear if the increased body temperature negatively effects performance [27]. However, based on the tragic death of Fran Crippen at a 2010 open-water race held in Abu Dhabi where water temperature was reported to be near 31 °C/87.8 °F, the Federation Internationale De Natation implemented a maximum water temperature of 31 °C/87.8 °F in 2013.

Heat acclimatization is a consistent recommendation when athletes plan to exercise in warm terrestrial environments. However, because internal body temperature when swimming in warm water does not tend to rise more than a degree, there is little stimulus for water-based heat acclimatization. Although there is anecdotal evidence related to the psychological benefits of water temperature acclimatization, there is no thermoregulatory or performance benefit to acclimatizing in warm water swimming [28].

Wetsuits have been shown to decrease oxygen uptake, minute ventilation, heart rate, and rate of perceived exhaustion when swimming at a constant velocity [29], which can be linked to a 14% decrease in drag provided by the wetsuit [30]. Additionally, wearing of a wetsuit was shown to improve performance by an average of 188 m over 30 min (or about 6 min saved time over the course of a 3.84 km swim) [31]. They appear to be safe when used within stated water temperature guidelines. Regulations regarding the usage of wetsuits are complicated and depend on the governing body of the event, as well as the level of competition (Table 13.2).

The Application

All water temperatures should be measured by race officials within 2 h of the event start time at a depth of 40 cm to ensure safe conditions. Following water temperature measurement, race organizers and athletes should abide by the rules outlined by governing bodies regarding cancellation or shortening of events. In open-water swimming, athletes need to be sure to begin events well hydrated and take breaks to consume cool fluids as necessary. In triathlon, athletes do not wear a full-body wetsuit in their swims if the water temperature is above 26 °C/78.8 °F. Athletes should be prepared for swimming in open-water conditions both with and without a wetsuit. It is important that triathletes do not feel dependent upon the wetsuit for the added buoyancy in the face of elevated water temperatures.

Cycling

The Challenges

The incidence of exertional heat illness in cycling is lower than that in running. This is due to higher velocity and thus increased evaporative and convective cooling provided by airflow as athletes cycle. Risk is also reduced in comparison to running because the amount of muscle mass that is activated during maximal cycling is a smaller percentage of total muscle mass, which in turn creates a lower rate of metabolic heat production. However, there are a number of unique factors related to cycling that may increase the risk of exertional heat illness, especially the heat-related or fluid balance-related muscular cramps.

Table 13.2 Water temperature cutoffs in swimming events

Water temperature			Governing body	Athlete group	Regulation
°C	°F				
13.0	55.4	<	ITU	All	Swim leg cancelled[a]
16.0	60.8	<	ITU	All	Swim leg of triathlon shortened
16.0	60.8	<	FINA	All	Minimum water temperature
20.0	68.0	>	USAT	Elite	No wetsuits allowed for elite athletes and swims less than 3k
20.0	68.0	≥	ITU	Elite	No wetsuit for swims up to 1.5k
22.0	71.6	≥	ITU	Elite	No wetsuit for swims greater than 1.5k
22.0	71.6	≥	ITU	Age-group	No wetsuit for swims up to 1.5k
22.0	71.6	>	USAT	Elite	No wetsuits allowed for elite athletes and swims greater than 3k
24.5	76.1	≥	ITU	Age-group	No wetsuit for swims greater than 1.5k
25.6	78.0	>	USAT	Age-group	No wetsuits allowed for athletes aiming for age-group or overall victories
25.6–28.9	78.0–84.0	Between	USAT	Age-group	Wetsuits allowed but athletes do not qualify for age-group or overall victories
28.0	82.4	≥	ITU	All	No wetsuits
28.9	84.0	≥	USAT	Age-group	No wetsuits allowed
31.0	87.8	≥	USAT	All	Swim leg of triathlon shortened
31.0	87.8	≥	FINA	All	Cancellation of swim event
32.0	89.6	≥	ITU	All	Cancellation of swim leg

FINA Fédéracion Internationale De Natation, *ITU* International Triathlon Union, *USAT* United States of America Triathlon

[a]The cancellation water temperature may be as high as 16 °C/60.8 °F if air temperature is below 14 °C/57.2 °F

In road cycling, the risk factors for exertional heat illness include long duration, high exercise intensity, high environmental temperatures, exposure to radiative heat from the sun, and lack of recovery time between consecutive stages. Road races typically occur over distances between 100 and 250 km, which take the athletes 3–7 h to complete. The race stages can occur over mountainous roads that increase muscular demand and thus increase metabolic heat production. As road cycling is classically competed during summer months, race temperatures routinely increase above 30 °C/86 °F. The temperature the riders are exposed to can be higher than the measured air temperature due to radiative heat gain. High heat exposure results in cyclists losing large volumes of fluid over the course of a stage through sweat production. For example, an athlete with a pedestrian sweat rate of 2 L h^{-1} over a 7-h stage could potentially lose 14 L of fluid. Because gastric emptying rates seem to peak between 1.2 and 1.5 L h^{-1} [32], theoretically by the end of this stage, our hypothetical athlete could be at a fluid deficit of 3.5–5.6 L. This becomes a particular

concern when the race, such as the Tour de France, is held as stage a race in which the athletes participate in races for consecutive days. As demonstrated in the calculation above, it is imperative when races occur on back-to-back days that adequate fluid consumption occurs following completion of the stage.

In mountain biking, two additional risk factors must be considered. Access to fluids can be a challenge in mountain biking races. In road cycling, nearly unlimited access to fluids is accomplished either by "team cars" that closely follow the athlete, or the designation of "feed zones" in which all athletes slow in order to receive provisions from team directors. Fluid availability in mountain biking can be improved by race courses taking place on a looped course with aid stations. However, this is not always the case. The largest exertional heat illness risk associated with mountain biking is due to race sites commonly being located in rural areas farther from medical treatment. Mountain bike races generally are held in off-road trails in state or national parks. At times these locations can be difficult to access by car which can make the transport of appropriate cooling equipment (e.g., whole-body cooling tubs) to the medical tent difficult [33].

The Evidence

Exercise intensity in road cycling during professional level stage racing occurs mostly at a moderate intensity (i.e., below 70% of VO_2max). However, because the stage lengths average >250 min in duration, individuals can still spend 90 min above 70% and 17 min above 90% of their VO_2max [34]. This level of exercise intensity produces a large amount of metabolic heat. Of particular concern are long uphill sections during road riding, or mountain bike races. In both of these situations, the riders are exercising at a high intensity while their velocity is typically lower. This causes an increase in heat production and a decrease in evaporative and convective cooling capacity [35].

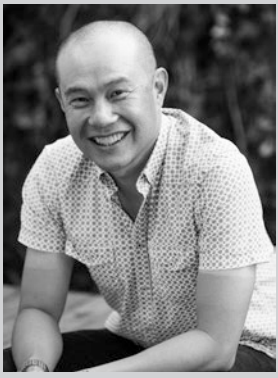
Heat acclimation within a controlled laboratory setting or acclimatization achieved by traveling to a warm climate for training is beneficial for cyclists planning to compete in hot environments, not only because it decreases the risk of exertional heat illness but also because it can improve performance [36]. Additionally, the plasma volume expansion associated with heat acclimatization can improve performance in moderate temperatures as well [37].

Pre-cooling can have a benefit when cycling events taking place in hot environments are short (i.e., time trial, or short mountain bike race) [38]. This cooling should be performed with a cooling "ice-vest" or cold drinks such as an ice slurry drink, as opposed to exposure mild cool environment in order to lower starting body temperature, although care must be taken because cooling to the point of shivering can be counterproductive [39]. Pre-cooling should not be used as a substitute for proper acclimatization. Also, pre-cooling will not necessarily improve performance when heat acclimatization has already occurred. However, there are perceptual

advantages to pre-cooling even in heat acclimatized athletes which may be beneficial for some athletes [40].

Fluid loss incurred during a single day of a multi-day cycling race should be replaced in the rest time prior to the next stage. The beverage should amount to ~150% of the body mass deficit assuming that 1 L of fluid loss is equal to 1 kg of body mass loss [41]. Additionally, beverages containing glucose and sodium have shown improved absorption and retention following exercise-heat dehydration [42] (Box 13.1).

Box 13.1: Maximizing Performance in Elite Cycling
Pioneer of Sports Physiology—Allen Lim, PhD



Dr. Lim is an accomplished physiologist who thinks outside of normal convention while applying physiological principles to sport. He worked as the team physiologist for professional cycling teams participating in Grand Tours between the years 2005–2011. Currently, he offers consultation for cyclists wishing to maximize their performance. He was kind enough to share his time and insights with us as we prepared our chapter on maximizing performance in the heat during cycling.

Question—What tactics would you utilize to prepare an athlete for racing in a grand tour?

A.L.—We made sure that we acclimated our athletes and optimized their fluid intake during training rides. For acclimation athletes were chronically and passively exposed to heat in a sauna following training sessions so that core temperature would remain elevated for a longer duration without impacting their training intensity. We witnessed progressive improvement in our athlete's ability to tolerate the heat over the course of the acclimation. Second, athletes weighed themselves before and after each training ride. We did not specifically prescribe a certain fluid consumption rate. By using this technique, athletes were able to self-monitor and adjust their drinking habits prior to the race in order to avoid excessive unreplaced fluid loss.

Question—Once the tour started, what would you do to minimize excessive heat gain during a stage?

A.L.—While racing, our athletes were only given ice cold beverages. These drinks act as a heat sink and can help to mitigate excessive rise in core temperature. This was a big challenge for us as ice is very hard to come by in most European countries. The fluid that was consumed was specific to each athlete's sweat-sodium concentration. Athletes consumed a ratio of a typical carbohydrate drink, water, and an extra salty beverage to meet their needs. All other water bottles were used to dump over the athlete to ensure that their skin was wet at all times (maximizing evaporative heat loss and simultaneously decreasing endogenous body water loss). As a last step in our defense we used stockings filled with ice placed down the back of the jersey to cool down athletes on very hot days. We generally used these in the hot valleys so that our cyclists' temperatures were not elevated too high prior to their increased effort on the climbs.

Question—Would you do anything different for time trial stages?

A.L.—Time trials are different because, from the first pedal stroke the athletes need to be going as fast as possible. Therefore, a proper warm-up is necessary. We would use ice-vests, or countercurrent water perfusion vests to minimize core temperature rise during this time while still allowing for a warm up. We would also have athletes consume 1–1.5 L of a sodium and carbohydrate beverage before the event. The extra salt would help minimize the typical hypovolemia seen during high-intensity exercise. Also, a bit extra water before the event would reduce the necessity to drink during the event which can negatively impact a cyclists aerodynamic position.

Question—After the completion of a stage how would you prepare the athlete for the following day?

A.L.—As soon as athletes finished a stage we would have them hop into an ice bath or onto the bus for a cold shower to help bring their core temperature down as quickly as possible. Metabolic rates are high even following exercise and very few hotel rooms in Europe have air conditioning. So, in order to make sure the athletes were able to fall asleep quickly and recover, we used cooling head wraps designed for maintaining body temperature in surgical patients. It was remarkable how the athletes fell asleep almost immediately after placing the wrap on their head.

The Application

Athletes participating in road cycling should take precautions prior to races including proper hydration/nutrition before, during, and between races. Pre- and post-stage bodyweights can be taken to ensure that serial reductions in body weight are minimized and rehydration with 150% of fluid loss using glucose and sodium

containing beverages. Knowledge of one's sweat rate is integral to the adequate replacement of fluid during the stage. If races are to take place in hot and humid race locations, all athletes would benefit by undergoing systematic exercise heat acclimatization with a minimal avoidance of heat prior to the race start (except for the days immediately before the race). During racing, tactics such as placing ice down the back of one's jersey may aid in thermoregulation. Prior to particularly high-intensity races (i.e., time trials) individuals should be encouraged to warm-up prior to the event with the use of an ice-vest or other cooling device. Doing so will attenuate rises in body temperature while still allowing for adequate metabolic warm-up.

In mountain biking, athletes should make note of their sweat rate and the expected duration between water refilling stations. With proper planning, riders can then make sure to carry an appropriate volume of fluid on their bike/person. Race directors should be encouraged to have an exertional heat illness treatment plan in place prior to the start of the race. In locations that expect higher temperatures, this plan should include cold water immersion tubs, a method for measurement of rectal temperature, and timely transport of injured athletes to a medical treatment facility.

Triathlon

The Challenges

There are a number of factors exclusive to triathlon that may increase risk of exertional heat illness.

For example, when swimming occurs in warm water and wetsuits are worn, it is likely for internal body temperature to rise during the swim. In this case, the starting cycling body temperature is elevated. Under normal environmental conditions, adequate airflow provides convective and conductive cooling during the cycling leg. However, extreme temperatures and humidity can make thermoregulation during the cycling leg difficult.

Long distance triathlons (i.e., Ironman; 3.86 km swim, 180.25 km bike, 42.20 km run) expose athletes to long duration of exertion. For elite athletes, this distance is covered in between 7:45 and 10 h. Over this time athlete will be exposed to large amounts of solar radiative heat. However, due to the long duration, exercise intensity remains moderate compared to the athlete's maximal capacity. Thus, exertional heat stroke in these events is rare. Other types of exertional heat illness which are related to large volumes of sweat loss, such as exercise-associated muscle cramping, and exercise-associated collapse are higher risk in long distance triathlon.

Due to the long distance, triathlon cyclists typically will wear an aerodynamic helmet during the event. The aerodynamic advantage of the helmet can save an athlete minutes during the 4–5 h bike leg. However, the advantage is gained by decreasing the number of vents on the helmet. In hot environments, this can act as a greenhouse over the athlete's head increasing thermally perceived exertion.

In shorter distance triathlons (e.g., Olympic triathlon; 1.5 km swim, 40 km bike, 10 km run), exercise intensity increases. For example, at the 2016 Olympics the top athletes cycled at velocities of 1.38 min/km (27 mph) and ran at a pace of 3.05 min/km (4:50 min/mile). Due to the high exercise intensity and moderate duration (~2 h), shorter distance triathlons increase the risk of heat stroke.

The Evidence

The risk of overheating on the bike or run following a swim in a wetsuit is minimal when water temperatures are below 26 °C/78.8 °F. Trappe and colleagues demonstrated that core temperature during the swim and cycling legs of a laboratory simulated triathlon was comparable following a swim in a standard swim suit versus while wearing a wetsuit [31]. However, further information is needed because the swim duration was standardized and thus intensity was slightly lower when given the advantage of the wetsuit. Additionally, the water temperature was only tested up to the ITU ceiling of 26 °C/78.8 °F while wetsuits are allowed in non-ITU races up to 28 °C/82.4 °F, the duration of the cycle leg was less than that of an Olympic triathlon, and the air temperature was moderate (25 °C/77 °F) during the cycling.

In long distance triathlons, dehydration and exhaustion can represent between 58 and 72% of medically treated problems. Exertional heat stroke at this distance is rare. However, in shorter distance races, three cases of exertional heat stroke occurred during the observation of one season of triathlon racing in Australia, which all cases occurred in the early season when temperatures were high [43].

Aerodynamic helmets offer a performance advantage to triathlon cyclists maintaining an aero-position for extended periods of time. However, the decreased venting results in hotter temperatures on the skin of the head. This can be perceived but does not result in higher internal body temperatures or reduced work output over short duration (12 km).

The Application

Similar to any long-duration endurance event, triathlon competition results in large volumes of fluid loss. Athletes should understand their specific sweat rates (to cycling and running at their race pace) to aid in proper replenishment during the race. Heat acclimatization is also important for optimal performance in the heat similar to during singular cycling and running events.

Athletes should choose the use of wetsuits and aerodynamic helmets strategically. If water and air temperatures are higher than what has been experienced in preparation, the athlete should err on the side of safety and choose to wear a traditional swim suit and helmet. The performance benefits added by buoyancy and hydro- or aerodynamics of both of these pieces of equipment can easily be negated

by higher body temperature (or the perception of higher temperatures in the case of the helmet).

Lastly, athletes should be familiar with the signs and symptoms of exertional heat illness and adjust race intensity, or discontinue racing should they occur. This can be a difficult decision for a person racing in an elite level race where high levels of exertion and physical pain are part of normal training.

Auto Racing

The Challenges

Auto racers must overcome a variety of physical, psychological, and environmental factors. Motorsport competition necessitates excellent fine motor coordination and great physical and psychological stamina, as races can last over 3 h in high ambient temperatures. Monitoring of racecar cockpit temperatures has shown temperatures ~50° C/122 °F. Inherent in motorsports, drivers travel at very high speeds (e.g., open wheel and stock car drivers can travel at speeds more than 200 mph), requiring repetitive and/or prolonged isometric contractions in response to g-forces. This results to large amounts of physical work and therefore heat production. The clothing and equipment worn by drivers (i.e., fire-retardant suit, boots, gloves, helmets) are necessary for protection, and they create a warm microenvironment and minimize evaporative and convective cooling potential. In addition to many motorsport races occurring during warmer months, drivers experience radiant heat reflection from the engines. It is also important to factor the psychological and emotional stresses when traveling at very high speeds, which is essential to performing optimally and avoiding crashes.

The Evidence

The nature of stress in motorsports is both physiological and psychological. While the relative importance of stressor type is difficult to delineate, motorsport athletes are exposed to similar stresses as other competitive sport athletes. In open-wheel road course testing, motorsport athletes have shown a mean VO_2 of 38.5 mL kg^{-1} min^{-1} and peak VO_2 of 46.5 mL kg^{-1} min^{-1}, equating to approximately an average exercise intensity of ~80% of maximal, or between 9- and 13-fold higher than resting energy expenditure [44]. Heart rates during competitive open-wheel road races have shown to vary between 140 and 160 bpm, similar to normal heart rate values while running an 8- to 10-min mile for conditioned athletes [44]. Similarly, research on stock car racers has shown mean heart rates around 80% of age-predicted maximum [45]. However, this heart rate differs from running in that it may have a higher proportion due to sympathetic nervous activity and less due to physical exertion or muscular

oxygen requirements. Since cockpit temperatures are high and evaporative cooling is diminished from heavy protective clothing, skin temperatures increase over the course of the race and the core to skin temperature gradient decreases. Carlson et al. showed a diminished core to skin temperature gradient in stock car racers (~2.0 °C to ~1.3 °C/~3.6 °F to ~2.3 °F), indicative of significant thermal stress and difficulty maintaining thermal steady state [45].

The Application

Mitigating the effects of cardiovascular and thermal strain is critical for maximizing performance in auto racing. Motorsport athletes should employ strategies to maximize heat acclimatization and aerobic fitness. Fitness programs should focus on not only maximizing aerobic capacity to reduce cardiovascular strain at a given work intensity, but muscular core stabilizers should be trained for endurance to reduce strain associated with repetitive g-forces. Fluid imbalances are a great concern for auto racers as dehydration can decrease cognition and motor skill performance [46], which could considerably increase the risk of an accident. Drivers and teams should develop individualized hydration strategies based on individual sweat rate and concentration. Sweat rates can be determined by assessing pre- and post-race body mass deficits. Sweat-electrolyte concentration can be assessed using the whole-body wash-down technique, which is the current gold standard for assessing sweat composition. Pre-race hydration status can be assessed using methods such as urine specific gravity and urine color. Drivers should also be aware of exertional heat illness symptoms such as dizziness, headache, and excessive fatigue. Motorsport governing bodies can help mitigate risk of illness and injury by considering rule provisions and procedures for modification or suspension of racing when environmental conditions are extreme. It is vital for auto racing governing bodies to continue researching physiological strain associated with auto racing to provide a structure and environment of racing that is sensible for driver health and safety.

Summary

Individual sport athletes and their teams must plan for a multitude of complex and interrelated factors to optimize performance and reduce heat-illness risk during hot conditions. In general, hot conditions can either augment or diminish performance, as research has indicated that short-duration and high-intensity performance can be positively affected, while long-duration performance can be negatively affected in the heat. Although each individual sport presents unique challenges related to its own intensity, duration, mode, and equipment requirements, there are many similarities among sports to consider when strategizing for hot conditions (Fig. 13.1). Common practical strategies to maximizing performance in the heat, including

maintenance of heat acclimatization and fluid/electrolyte balance, altering intensity, and optimizing sport-specific physical preparation, are universal across sports. Athletes, coaches, and medical practitioners associated with individual sport would benefit from employing scientifically sound principles when preparing for hot training and competition conditions. With a wide range of potential interventions available, tailoring strategies specific to both the sport and the individual could be the key determinant in the realization of maximal performance and safety in the heat.

Table 13.3 Top ten strategies for maximizing performance and safety in the heat

10. Be aware of heat-illness signs and symptoms	• Common symptoms of heat illness include disorientation, irritability, dizziness, headache, confusion, and sometimes emotional instability • Those exercising in the heat should be cognizant of common signs of exertional heat illness such as muscle cramps, staggering, profuse sweating, decreased performance, rapid pulse, low blood pressure, and quick breathing
9. Modify clothing and equipment	• Loose-fitting, absorbent, and moisture wicking clothing should be worn when exercising in the heat • Heavy clothing or equipment that can decrease cooling capacity should be minimized during hot and humid conditions
8. Alter intensity and volume	• Reduce intensity during hot and humid conditions, especially if signs or symptoms of heat illness are present • Work with coaches to alter training load (volume and intensity) during oppressive conditions
7. Minimize heat exposure prior to competition	• Warm-up duration and intensity may need to be lowered during hot and humid conditions so that heat storage capacity is not decreased by unnecessary elevation of internal core temperatures prior to exercise • Reduce heat exposure prior to competition to reduce overall fatigue from thermal load and sun exposure
6. Utilize body cooling	• Pre- or per-cooling via ice or water-perfused vests, ice cubes, and towels can be helpful in reducing internal body temperature and maximizing heat loss capacity • Post-training or competition cooling via cold water immersion can be utilized to rapidly return internal body temperatures to homeostatic conditions, speed recovery, and reduce perceived soreness
5. Be physically prepared	• Athletes should gradually progress and maximize sport-specific endurance capacity prior to performing in the heat • Maintaining adequate fitness levels year-round should be a focus as low cardiovascular fitness is a predisposing factor to exercise-induced heat illness
4. Maintain carbohydrate/ electrolyte balance	• While a balanced diet will typically provide adequate electrolytes short to moderate duration activity (i.e., sodium), longer events or those who have high sweat-sodium concentrations (i.e., salty sweaters) may require electrolyte supplementation during competition • Carbohydrate-electrolyte beverages (carbohydrate concentration between 4 and 8%) should be considered for events lasting longer than 1 h and for post-exercise rehydration in order to maintain/replace glycogen stores and promote fluid retention

3. Maintain fluid balance	• A combination of hydration indicators including thirst, body mass loss, and urine color can be useful in assessing hydration status • As athletes acclimatize and become used to training in hot and humid conditions, sweating rate will increase and therefore the need to replace more fluids will increase as well
2. Heat acclimatize	• Athletes should plan for a minimum of 10–14 days of slow and progressive exposure to the heat and humidity for their bodies to physiologically acclimatize • Athletes should strive to maintain heat acclimatization throughout training and competition periods to experience performance benefits related to plasma volume expansion and increased efficiency of heat loss
1. Communicate and plan for the conditions	• Athletes, coaches, and support staff should plan to alter training times to avoid environmentally challenging parts of the day (i.e., mid-day), particularly when close to competition • If hot and humid conditions cannot be avoided (i.e., competition), teams should coordinate and ensure appropriate medical care is available and strategies relating to hydration, body cooling, and pace alteration are established and followed

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Chapter 14
Team Sports

Julie K. Nolan, Courteney Benjamin, J.T.W. Ng, and Yuri Hosokawa

Introduction

While many of the topics discussed with team sports are also true of individual sports (see Chap. 13), there are certainly some aspects of team sports that are quite unique. While team sports often involve activity that may be similar to individual sports, the dynamic of participating in a team setting may directly affect an athlete's susceptibility to heat illness and/or affect performance. For example, there are often significant differences in physical ability and fitness level among each team member which may create an unequal playing environment. Therefore, as the activity itself is often standardized, the difficulty of the activity will not be perceived the same by all team members.

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Psychological Considerations for Team Sports

There are psychological factors that may affect athletes competing in team sports differently than those competing in individual sports. These psychological differences are predominantly related to differences in both intrinsic and extrinsic motivation. While intrinsic motivation involves performing a behavior because of personal interest and enjoyment for that activity, extrinsic motivation is characterized by the influence of outside factors on the willingness to perform the activity [1]. Some of these outside factors that are commonly present in a team setting are influences from a coach or team member (i.e., responding to verbal commands to keep up with another teammate), the pursuit of rewards (i.e., competing with teammates for playing time or a starting position), and avoidance of punishment (i.e., responding to the demands of a coach to avoid extra practice time or conditioning).

In almost all team sport scenarios, the athletes themselves enjoy playing their sport and do so on a voluntary basis. The exception to this would be professional and some school settings in which athletes are required to play a sport as part of their job or enrollment. Therefore, in most circumstances the factors associated with extrinsic motivation are most likely to adversely affect a team sport athlete. In other words, a team sport athlete may feel controlled by outside factors and therefore may be influenced to compete at a level that is unmatched to their physical capabilities. While in some situations this may be beneficial in improving the athlete's skill level, it may also be detrimental (i.e., increased risk for injury) if they are being required to compete at a level not matched for their fitness/ability level. The potential for an adverse effect would be amplified when performing the exercise in the heat.

General Risk Factors Associated with Team Sports

When characterizing how team sports may be affected by the heat, factors such as the intensity level and duration of the sport, the normal climate and time of year the sport is typically played in, and individual factors that are inherent of the sport itself (i.e., equipment considerations, different playing positions) must be considered. There are several risk factors that universally affect exercise in the heat, but based on the aforementioned considerations, individual risk factors may pose more of a threat to some team sports versus others.

These risk factors are typically classified as either intrinsic or extrinsic. Intrinsic risk factors are those that are within the control of the exercising individual and therefore are relatively easy to modify in most situations. Examples that will most often affect team sports include low fitness level relative to other teammates, hydration status if teammates are competing for available water (Chap. 5), and inadequate heat acclimation (Chap. 3). Additional factors specific to the individual include use

of medications that may affect thermoregulation (Chap. 19), presence of fever, and presence of a skin disorder (Chap. 18) [2]. Conversely, extrinsic risk factors are outside the control of the exercising individual. Examples of extrinsic risk factors that will most often affect team sports include high environmental conditions (i.e., high heat and humidity; Chap. 9), athletic equipment that may compromise proper thermoregulation, pressure from teammates or coaches to compete at a higher level, lack of medical personnel for the team, inadequate education and awareness of heat illnesses, and lack of a formal emergency action plan for that team and/or venue. While these extrinsic risk factors cannot necessarily be modified by the athlete themselves, strategies can often be implemented to minimize these risks. The differences in how each risk factor may pose a threat to certain sports are summarized in Table 14.1, and will be addressed in further detail for specific sports throughout the remainder of this chapter (Table 14.1).

Training Smart and Optimizing Recovery

Sleep

An undervalued, yet increasingly popular, topic is the importance of sleep on overall recovery from physical activity (see Chap. 7). Proper sleep patterns are beneficial to an athlete's recovery and subsequent performance [3]. In a team sports setting, both coaches and athletic trainers should stress the importance of proper sleep habits and should allow athletes ample opportunity to practice these habits (i.e., adjusting training/practice times). In the collegiate and professional sports settings, this concept is especially important because late night games and travel across time zones occur frequently and can further adversely affect sports performance [4].

Travel Fatigue

Interstate, international, and even trans-meridian travel is now commonplace for teams of all ages and ability levels [5]. The direct and indirect effects of travel, travel fatigue, and jet lag could potentially be detrimental to performance as well as risk factors for heat-related illness. Travel fatigue can be experienced on any journey and is not exclusive to travel across time zones [6]. It is the summation of physiological, psychological, and environmental factors that can accumulate during a single trip or over the course of a season, negatively impacting an individual's ability to recover and perform [7]. Therefore, travel fatigue is influenced by the distance traveled, frequency of travel, and length of season, and is the result of the demands

Table 14.1 Summary of common risk factors for exertional heat illness and associated risk among games and practices of team sports

	American football		Soccer		Rugby		Lacrosse		Cross-country	
	G	P	G	P	G	P	G	P	G	P
Inadequate acclimatization	Moderate	High	Moderate	High	Moderate	High	Moderate	Moderate	High	High
High exercise intensity	High	High	High	High	High	High	High	High	High	High
Pressure from coaches/team	Moderate	High	Moderate	High	Moderate	High	Moderate	High	High	High
Low fitness level	Moderate	High	Moderate	High	Moderate	High	Moderate	High	High	High
Dehydration	High	High	High	High	High	High	High	High	High	High
High environmental temperatures	Moderate	High	High	High	High	High	High	High	High	High
Athletic equipment	High	High	Low	Low	Low	Low	Moderate	Moderate	Low	Low
Inappropriate rest breaks	N/A	High	N/A	High	N/A	High	N/A	High	N/A	High

Abbreviations: *G* game, *P* practice

Table 14.2 Travel fatigue symptoms

Neurological	Headaches
Psychological	Decreased cognitive performance
	General malaise
	Travel weariness
	Diminished mood states
Immunological	Recurrent illness
Musculoskeletal	Whole body stiffness/soreness
	Decreased active range of motion

of travel itself [8, 9]. TF is cumulative, may become chronic, and can exacerbate symptoms of jet lag disorder [6]. Consequently, it requires ongoing monitoring, detection, and correction by trained medical personnel [7]. Neurological, psychological, immunological, and musculoskeletal symptoms predominate the symptoms of travel fatigue [7] (Table 14.2). In the context of long-haul travels, sleep may be fragmented, often in cramped, non-supine positions with repeated interruptions by lights, noise, stopovers, and connecting flights [6, 9–11].

The direct and indirect effects of travel, travel fatigue, and jet lag could potentially be detriments to performance as well as risk factors for heat-related illness. Jet lag disorder typically does not become significant until three or more time zones are crossed [6]. Athletes tend not to recognize these symptoms as effects of jet lag disorder itself [12]. Recovery can take several days and is generally proportional to the number of time zones crossed. A general rule of thumb states to allow 1 day for every time zone crossed to adapt for eastward travel and 0.5 days for every time zone crossed to adapt for westward travel [5, 12, 13]. Some studies have shown longer adaptation periods for westward travel [14] and shorter adaptations for eastward travel. There is a substantial variability in periodicity between individuals, ranging from 23.9 to 24.5 h [6]. This may explain why some athletes adapt more quickly to eastward travel. A recent study of long-haul eastward travel in professional soccer players suggested that players with greater travel experience could be less affected by the sleep disruption associated with such travel as demonstrated by reduced jet lag and improved function ratings [9].

Travel to a warmer climate among those who have yet to fully acclimate to the heat can serve as a significant risk factor for performance decrement and exertional heat illness. In situations that require a significant climate or time zone change, every effort should be made to arrive with as much time as possible before the first competition. Prevention usually occurs by addressing the other risk factors of heat illness (e.g., recent illness, dehydration, exercise unmatched to fitness), minimizing cumulative heat stress, and having the athletes prepare in advance to peak their physical conditions before the travel. Monitoring wet bulb globe temperature and other weather patterns to modify the exercise load and intensity may also help team staff decide the appropriate amount of physical training that can be completed without posing the athletes at risk for exertional heat illness.

Nutrition

Proper sports nutrition is a well-researched topic that involves a range of dietary strategies to enhance both sports performance and subsequent recovery [15]. However, it is important to note that differences in activity type and individual biometrics may affect these generalized strategies and therefore nutrition plans should be individualized. Generally speaking, athletes should focus on maximizing muscle glycogen stores prior to exercise, which has been shown to delay the onset of fatigue during endurance exercise and improve performance [15]. During activity, carbohydrate ingestion can further improve exercise, especially in events lasting approximately 1 h [16]. However, recent literature has suggested that perhaps fat as a primary energy source during endurance activity may be more beneficial [17]. Finally, recovery post-exercise can benefit from a combination of carbohydrate and protein ingestion. Beverages such as recovery sports drinks and chocolate milk have been shown to be beneficial for proper recovery and have a positive impact on both muscle glycogen and protein synthesis, assuming ingestion occurs during the optimal window of about 45 min to 1 h post training [15]. Coaches and athletic trainers should help to educate athletes on the importance of proper nutrition and, if possible, provide recovery drinks/bars to athletes immediately after training.

Use of Technology

Because of the unique physical characteristics of team sports, global positioning systems (GPS) and other wearable technologies have shown to be extremely valuable to monitor physical workload of an athlete [18, 19]. The information provided by this technology (see Chap. 10) has proved to be quite valuable in monitoring individual training loads and physical demands of specific sports [20, 21], comparing training loads and physical demands among players of different playing positions [20, 22], and assessing overall training load indicators with respect to training outcomes and injury surveillance [23, 24]. Coaches, athletic trainers, and strength and conditioning coaches can all collaborate and use this information to appropriately periodize training based on these individual requirements. This will maximize the physical capabilities of each individual which ultimately lead to the betterment of the team.

Total distances covered over high speeds and total number of high speed runs often decrease with increasing temperature. These findings appear to hold during periods with multiple games in short time periods, even during a main competition phase (i.e., “peaking phase”) of our training when athletes are at or near peak condition. Athletes are also likely to experience upper heart rate zones with increasing temperature. Health implications as well as substitution and game strategies may be altered by the knowledge of this information.

Metabolic Demands

From the perspective of the physical demands associated with sport, team sports are often categorized based on which energy system is predominately required. While most team sports require athletes to be both anaerobically and aerobically fit to be successful, it is important to acknowledge the primary demands of each sport in relation to what extent each energy system is utilized. This will allow for a better understanding of the specific physiological challenges that each sport may pose. The remainder of this chapter will address the implications of heat in different types of physical activities: (1) anaerobic/high-intensity sports (e.g., American football); (2) intermittent sports (e.g., soccer, lacrosse, rugby); and (3) aerobic/endurance sports (e.g., cross-country).

High-Intensity/Anaerobic Sports

Team sports that are considered to be predominately anaerobic often involve many consecutive, short-duration, intense bouts of exercise (i.e., sprinting or blocking) followed by a period of rest. However, there are varying degrees of both the intensity and duration of the rest period. For example, in American football some teams will run a quick-tempo offense in which the rest period between plays is substantially shorter. This situation would require more aerobic fitness to keep up with the faster tempo of the game. Despite the notion that these sports may predominately depend on anaerobic condition, previous research has suggested that a lack of aerobic fitness not only hinders performance, but may also compromise safety with specific regard to heat illnesses [25]. In addition, high-intensity sports are considered to be the most susceptible to heat illness due to a multitude of physiological responses that result in a faster rate of rise in core body temperature [26]. These responses would be even more pronounced in a non-heat and exercise acclimatized athlete when high-intensity exercise is performed in the heat.

The Challenges and Supporting Evidence

Physical Demands

American football is extremely unique because different playing positions require athletes who possess different physical attributes not only to perform the tasks necessary of that position but also to excel at it [20, 27]. For example, skill positions (e.g., quarterbacks, wide receivers, running backs, and defensive backs) tend to be smaller, faster, and more aerobically fit whereas offensive and defensive lineman tend to be bigger, stronger, and more powerful [27]. Linebackers tend to fall between

these two groups for all physical capabilities including size, body composition, strength, speed, and endurance. These variations in physiological demands are in part what makes each athlete great at their respective position.

While specific anthropometric characteristics may be beneficial to excel at certain playing positions, those same attributes may also predispose athletes to an increased risk of exertional heat illness. A recent review suggested that American football players are presented with unique challenges to thermoregulation due to factors such as the amount of mandatory protective equipment, large body size of players, and the time of year football preseason occurs [28]. A complete review of these challenges specific to the sport of American football can be seen in Table 14.3. Moreover, it has been shown that larger players (i.e., offensive lineman and defensive lineman) are at a further increased risk for exertional heat illness because of greater body mass index, lower aerobic capacity, and the stationary nature of the position [28]. More specifically, linemen have been shown to have a higher sweat rate thereby putting them at an increased risk of dehydration [29], while also exhibiting a reduced heat loss potential due to the static nature of their position [30].

The Equipment

The equipment requirements of American football are necessary to help prevent injury due to the contact nature of the sport; however, protective equipment can also predispose a football player to exertional heat illnesses, especially during the warmer months. This in part is due to the added weight of the equipment, therefore increasing metabolic heat production of the athlete. Additionally, the protective equipment combined with the rest of the football uniform covers almost all of the body surface area (with the exception of the face and sometimes arms). Consequently, the athlete loses the ability to dissipate heat via evaporation. Evaporative heat loss is considered to be the most effective heat loss mechanism, so the athlete is at a significant thermoregulatory disadvantage [31]. This effect becomes more pronounced when exercising at a high intensity and in the heat.

Large Team Size

While team sports in general are often inherent of large player numbers, the size of an American football team is one of the largest among all organized sports. While professional (National Football League) teams are limited to 53 players during the regular season, National Collegiate Athletic Association (NCAA) and high school programs are not limited to the number of players they can hold (NCAA has a limit of 105 before the start of the regular season, but no limit thereafter). This can be seen as a challenge because it puts more responsibility on the team medical and coaching staff to tailor the workout to athletes' individual needs. This is especially problematic in a high school setting where often times there is limited medical supervision.

Table 14.3 Overview of sport requirements and associated challenges of American football

Level	Game type	Duration (min.)	Halftime (min.)	Field dimensions	Substitutions	Equipment	Season	Breaks	Special considerations
Professional	Quarters	60	12	120 yards long, approximately 55 yards wide; end zones are 10 yards deep; hash marks are approximately 24 yards from each sideline	Unlimited	Helmet, pads	August–December	Timeouts, between series, halftime	Various positions should be individually trained; hydration and body cooling should be implemented during breaks
Collegiate	Quarters	60	20	Same as professional except hash marks are 20 yards from each sideline					
High school	Quarters	48	20	Same as professional except hash marks are approximately 18 yards from each sideline					
Youth	Quarters	48	15	Varies by league; dimensions, end zones, and hash marks all variable					

These high player-to-staff ratios highlight the need for preseason staff meetings and education on recognizing the signs and symptoms of exertional heat illness. It is critical that coaches rehearse with the sports medicine staff the procedures for heat emergencies and become competent with skills required to act as first responders (i.e., removal of player from the field and even initiating basic cooling measures).

In addition to the challenges of sufficient medical service, the large team size poses challenges due to the increased number of resources needed to accommodate so many players. Specifically, larger team size may affect the availability of resources such as hydration source, body cooling, and individualized GPS monitoring. Moreover, since breaks between practice drills are usually limited to a couple of minutes, players may not be able to optimally recover during the period and may even be competing for water and body cooling modalities.

Generally, the use of GPS technology was previously described as being a great training and recovery tool with respect to overall workload across practice sessions and games [20–24]. However, research is limited when specifically looking at anaerobic team sports such as American football [20, 22]. Additionally, it may not be financially feasible for teams to purchase these devices, especially if they have a large size team. While it is becoming increasingly popular for professional and well-established Division I NCAA teams to purchase these devices, it may not be possible for others. However, perhaps the best way to not only decrease the risk of exertional heat illness but to also promote enhanced performance in a large team sport is to implement a heat acclimatization program. This program requires no cost and can be implemented for the entire team. The incidence of exertional heat illnesses [32, 33] and deaths resulting from exertional heat stroke [34] are most likely to occur during preseason practices. While standardized mandates exist for both the NFL and NCAA, high school guidelines must be implemented at the state level. Many states have adopted the standardized heat acclimatization guidelines, and it is strongly recommended that all programs implement this process even if it is not mandated by your state [35].

Intermittent Sports

Team sports that are considered to be predominately intermittent involve a relatively long duration of exercise compared to anaerobic sports while also including sporadic bouts of high-intensity exercise bouts (i.e., rapid accelerations and decelerations, changes in direction). The most popular intermittent sports, and subsequently those that are of greatest risk during exercise in the heat, include soccer, rugby, and lacrosse. In contrast to sports that are predominately anaerobic, intermittent sports do not have regular rest periods. Therefore, greater emphasis on aerobic training is important for these athletes. While the physical demands required of various playing positions within intermittent sports are not as widespread as those described

with American football [27], differences within intermittent sports still exist [36–39]. Therefore, an individualized training approach by coaches and strength and conditioning personnel may also be beneficial.

The Challenges and Supporting Evidence

Physical Demands

Although some differences exist among individual sports, the unique physical demands of intermittent team sports are comparatively consistent and have been well studied [37, 39–42]. For example, while rugby was once considered a predominately aerobic sport, recent technological advances showed that rugby places a great deal of stress on the anaerobic energy system [42], similar to that of soccer [40] and lacrosse [39]. Due to the longevity of these matches (60–90 min) coupled with intermittent bouts of high-intensity sprinting [39, 40], high aerobic and anaerobic fitness levels are crucial for an athlete to be successful.

While the match demands of lacrosse have been shown to be most similar to that of soccer [37, 39, 41], men's lacrosse is unique due to the contact nature of the sport (women's lacrosse is noncontact). In addition, men's lacrosse requires more protective equipment which can further predispose them to a heat-related illness. While the degree of contact is not as extreme as rugby, it is still likely that those players who are subject to more collisions have higher metabolic demands placed on them. An overview of similarities and differences among these intermittent sports can be seen in Table 14.4.

Limited/No Rest Breaks

One of the unique challenges that many intermittent sports faces is the limited chance for athletes to rest besides the quarter/halftime break unless the athlete is injured and requires medical evaluation. The game durations are also relatively long in length (60 min for lacrosse to 80–90 min for soccer, depending on level of play) when compared to other team sports. This is further compounded in professional and international soccer matches where there is a maximum of three substitutions allotted for each team for the entirety of the game. Similarly, substitutions in a sport like lacrosse are usually limited to specific playing positions. The intermittent but continuous play also demands the athlete to sustain high performance despite the limited chance for rehydration and body cooling, which have been shown to be beneficial for both reducing the incidence of exertional heat illness and maximizing performance [43–45]. It should be noted that the Fédération Internationale de Football Association (FIFA) recently passed a rule allowing referees to pause the game for a cooling break when the wet bulb globe temperature is above 32 °C/89.6 °F.

Table 14.4 Overview of sport requirements and associated challenges of intermittent sports

	Level	Game type	Duration (min.)	Halftime (min.)	Field dimensions	Substitutions	Equipment	Season	Breaks	Special considerations
Soccer	FIFA	Halves	90	15	110–120 yards long and 70–80 yards wide	Max. 3; no reentry	Shin guards	Varies	Halftime	Various positions should be individually trained; hydration and body cooling should be implemented during breaks
	Professional				110–120 yards long and 70–80 yards wide			Varies		
	Collegiate				115–120 yards long and 70–75 yards wide			August–December		
	High school		80	10	Varies	Unlimited		August–November		
	Youth		30–90 depending on age	Varies	Varies	Unlimited		Year-round	Halftime; sometimes quarters	
Lacrosse (men's)	Professional	Quarters	60	12	110–120 yds. × 53 1/3–60 yds.	Unlimited	Helmet, shoulder pads, chest protector, eye guard	April–August	Between quarters and halftime	Various positions should be individually trained; hydration and body cooling should be implemented during breaks
	Collegiate		60	10				February–May		
	High school		48					Varies		
	Youth		Varies		110 yds. × 53–60 yds. wide			Year-round		
Lacrosse (women's)	Professional	Halves	60	10	110–120 yds. × 53 1/3–60 yds.	Unlimited	Eye guard	May–August	Halftime	Various positions should be individually trained; hydration and body cooling should be implemented during breaks
	Collegiate		60					February– – May		
	High school		50					Varies		
	Youth		Varies		110 × 53–60 yds. wide			Year-round		

Rugby	Professional	Halves	80	10	122.5–133.4 × 74.3 yds.	Max. 7; no reentry	Scrum cap	April–August	Halftime	Various positions should be individually trained; hydration and body cooling should be implemented during breaks
	Collegiate		80		114–144 × 68–70 yds.	Max. 7; no reentry		August–December		
	High school		60–90		122.5–133.4 × 74.3 yds.	No reentry in first half		February–May		
	Youth		60		122.5–133.4 × 74.3 yds.	Max 5; player can return but counts as substitution		Varies		

It has been shown that frequent substitutions may help to resynthesize energy stores (specifically related to the phosphocreatine system), and therefore may preserve high-intensity function later in match [46]. Since lacrosse, rugby, and in some competition levels of soccer allow for unlimited substitutions, utilizing these substitutions may directly enhance performance in the later stages of match play.

Uniqueness of Different Playing Positions

A recent review on the physical demands of rugby highlighted the drastic differences among positional groups with respect to both kinematic and metabolic demands [38]. Specifically, it was found that forwards received the greatest number of collisions, outside backs covered the greatest distance and attained the highest speeds, while overall metabolic costs were highest among other players [38]. While the specific sport requirements of rugby may be unique due to the higher incidence of collisions, comparable conclusions have been made to similar sports such as soccer [36, 37] and lacrosse [39] with respect to recommendations geared towards maximizing performance and safety in the heat. These findings highlight the importance of specific training for positional groups with special attention being paid to the competition requirements of the specific position of that sport.

While some authors [39] have suggested that sport coaches should use player/position specific information to individualize training programs, further research is warranted to more closely explore the relationships among training specificity and injury prevention. This would allow the coaching staff to individually tailor their training sessions and practices to promote optimal training periodization. As a result, this could decrease the risk of both injury- and heat-related illnesses throughout the course of a season, as well as increase performance during games.

Endurance/Aerobic Sports

Endurance-based team sports represent activities that rely on aerobic energy system and involve a long duration of activity. However, the activity is usually continuous and does not include any recurrent bouts of high-intensity exercise. While aerobic training should be the primary focus of these athletes, anaerobic training should not be entirely ignored.

The Challenges and Supporting Evidence

Physical Demands

Although cross-country may be considerably shorter than other endurance events (i.e., marathon, triathlon), it is still considered to predominantly rely on the aerobic energy system. Depending on the level of competition, standard distance for a

Table 14.5 Overview of sport requirements and associated challenges of cross-country

	Level	Distance	Season	Breaks	Special considerations
Cross country (men's)	Collegiate	8–10 km	August–November	None	Athletes not able to be monitored during competition and not able to hydrate and/or cool due to continuous event; hydration and body cooling should be emphasized before competition
	High school	5 km	August–November		
	Youth	3–5 km	Varies		
Cross country (women's)	Collegiate	5–6 km	August–December	None	
	High school	5 km	Varies		
	Youth	3–5 km	Varies		

cross-country race may range from 5 to 8 km. This unique distance poses more of a threat to the athletes with respect to exertional heat illness since cross-country athletes will be able to run and maintain a higher exercise intensity throughout the entire duration of the race, thereby producing more metabolic heat. Additional challenges are described in more detail throughout the remainder of this section, and a summary can be found in Table 14.5.

Race Course Challenges

The race course itself poses unique challenges because it does not allow athletes to be monitored by coaches and/or medical staff for the duration of the activity. From a safety standpoint, these athletes may not have convenient access water and cooling modalities while training or competing. Similarly, runners are in a unique position during competition because they may not be directly influenced by coaches and teammates to modulate their workout intensity.

Pacing

Proper pacing strategies are worth mentioning during cross-country meets given the continuous, relatively long duration nature of the event. There are several proposed influences on pacing strategy and the resulting performance outcome [47]. The majority of pacing research, especially with special consideration to warm-weather environments, has been done on longer duration events (i.e., marathon) [48, 49] where associated performance benefits are likely more pronounced. This is because the main physiological advantages acquired from pacing relates to preserving glycogen stores. However, one study that was recently published highlighted this relationship in a cross-country event and specifically sought to observe differences in pacing among gender [50]. The study did in fact observe differences between males

and females, but due to the likely negligible effect coming from a physiological orientation, they attributed the differences to potential psychological variances in areas such as decision-making, overconfidence, risk perception, and willingness to tolerate discomfort [50].

The unique geographical landscape on which cross-country meets are held offers hints of the potential strategies and different energy systems that may be used in a given race. Runners may give short, hard 50–100 m bursts cresting a hill or after turning a blind corner. Longer, but still intense surges are frequently used at the beginning of races, which often start in an open field but quickly narrow within the first 100 to several 100 m. During other segments, longer, less intense surges may occur over 1–3 min and may occur at multiple portions of the race. Furthermore, runners who give "all out" in the last 400–600 m of a race are often observed, in which motivated athletes may push themselves to exhaustion. When one considers the strategies in dynamic pacing within a given cross-country race, but maybe not trained for, one may be at increased risk of exertional heat illness.

Environmental Challenges

Another unique aspect of cross-country is the potentially large environmental fluctuations that may occur throughout the course of the competitive season. Both collegiate and high-school level cross-country typically span from August to mid-November. Therefore, depending on the specific geographical location, it is not uncommon to have upwards of a 60 °F/16.56 °C environmental temperature difference from the start of the season to the end. What is of most concern is a sporadic temperature increase during the later parts of the season. In the beginning of the season (i.e., August/September), athletes should be acclimatized as high temperatures are normal for that time of year. However, if you get an abnormal spike in temperature later in the season, the athletes likely will respond negatively to the unplanned conditions. As the effect of environmental heat stress has been shown to produce both health and performance decrements in various types of running events [48, 49, 51], this effect would likely be heightened in the event that the athlete is not heat acclimatized.

The Application/Case Example

Practical recommendations to successfully maximize performance and safety in the heat are summarized in Table 14.6.

Table 14.6 Ten practical recommendations to successfully maximize performance and safety in the heat in team sports [3, 4, 15, 18–20, 22–24, 32, 33, 35, 43–45, 51–55]

Recommendation	Best time to implement	Examples/special considerations
Employ a heat acclimatization program	At the very start of the season (preseason)	Heat acclimatization guidelines [35]
Utilize body cooling techniques	Throughout the competitive season; can be done before, during, or between exercise sessions as permitted by the sport	Cold water immersion, cooling vests, cooling fans, cooling units [43–45, 52]
Ensure proper hydration	Daily; before, during, and after exercise	NATA hydration guidelines [53]
Employ appropriate rest breaks during practices	Throughout the season	Special attention should be made at the beginning of acclimatization, during high-intensity drills, and when environmental conditions are high [32, 33, 35]
Encourage off-season training	During the sport's off-season period	Individualized or team-based off-season training program
Modify games/practices based on weather	When necessary	Both absolute weather conditions (high heat/humidity) and relative weather conditions (normal for that time of year) should be considered [51, 54]
Use global positioning system technology during training and games	During normal training and practice sessions and competitive games	Can be used to monitor overall training load, recovery, and differences among player positions [18, 19]
Implement appropriate recovery strategies	Throughout the season following intense practices and competitive games	Proper nutrition, sleep, hydration, and rehabilitation exercises [3, 4, 15, 55]
Ensure proper education and communication among coaches, strength and conditioning coaches, and medical personnel	Prior to the start of the season; routine communication should occur year round	Will aid in optimal training and performance throughout season
Individualize training with focus on periodization	Throughout the season; based on feedback observed from the factors above	Training should be individualized based on player position and fitness level; this will lead to enhanced safety, minimize fatigue and injury, and maximize performance [18–20, 22–24]

Summary

Participating in team sports is one of the most popular activities in the world. As drastic variations in age groups, participation levels, and types of activity exist, so too are the associated risk factors that may compromise both safety and performance, especially when exercise is performed in the heat. However, recent research

involving GPS technology, heat acclimatization guidelines, hydration and body cooling practices, and various recovery strategies have provided valuable recommendations for ways to combat these risk factors. As a result, this information should allow sport coaches, strength and conditioning coaches, and athletic trainers to effectively work together to provide the most optimal training and competition programs, thereby maximizing individual and team performance while minimizing the incidence of exertional heat illness and other injuries.

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Chapter 15
Occupational Settings: Considerations for the Laborer

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The Challenges

Workers in various occupations are exposed to thermal stress, putting these individuals at risk for heat-related illnesses. High air temperature, high humidity, radiant heat sources, direct physical contact with hot objects and strenuous activity in the heat, all play a role into decrements in performance and increased risk for exertional heat illnesses (EHI) [1]. Occupational workers may also face external risk factors that are unique to the occupational setting, including: quota-driven work settings, equipment and protective clothing requirements, long work hours, extended periods in the sun, and a lack of standards for protection. Each of these risk factors increase the risk of EHI, and in many cases, a worker is exposed to multiple risk factors for EHI simultaneously. While some of the risks associated with work in a hot environment can be mitigated with administrative and engineering controls, there is ample evidence to show that many employers do not have adequate safety plans in place [2–6]. Furthermore, certain occupations, such as firefighters and hazardous materials responders, may be exposed to high levels of heat stress during emergency operations. In situations such as these, workers may be required to

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Table 15.1 Examples of classifications of occupations at risk for EHI [7]

Classification by industry	Examples	Environmental heat stress	Heavy equipment	Intense activity	Additional considerations
Agriculture, forestry, and fishing	Agricultural production crops, agriculture services	X	–	X	
	Forestry and lumber	–	X	–	
Mining		X	X	–	Smelting, etc.
Oil and gas extraction		X	X	–	
Construction	Building and general construction	X	X	–	
Manufacturing		X	X	–	Printing press, etc.
Public services and utilities	Transportation	X	X	–	
		–	–	–	
		–	–	–	
		–	–	–	
		–	–	–	
Services		–	–	–	
	Amusement and recreation services	–	–	–	
		–	–	–	
	Public safety (i.e., firefighting, police officers)	X	X	X	

perform in conditions that would otherwise be considered as "unacceptable" and require additional consideration and mitigation strategies.

The type of heat exposure varies depending on the type of industry the laborers work in (Table 15.1).

Exertional Heat Illness Risk Factors

While the workload and environment often induce thermal strain, many workers are also at elevated risk of EHI from the lack of knowledge in prevention and treatment of heat-related injuries. For example, in a study of Latino farmworkers in the USA, workers had a misconception that cooling modalities should not be used after work in the heat [8]. Other examples of EHI risk factors commonly observed in the occupational settings are: lack of heat acclimatization, protective garments, lack of physical fitness, high intensity of work, obesity, preexisting medical conditions, and external motivators to continue working [9].

Environment

Environmental conditions of the work place have a large influence on the risk of someone experiencing EHIs [10]. One of the commonly used environmental indices for outdoor conditions is wet bulb globe temperature (WBGT), which integrates the influence from the air temperature, humidity, wind speed, and solar radiation [11, 12]. Indoor workers may also experience oppressive thermal strain due to the lack of ventilation, radiant heat gain from heat-generating machines, and the use of protective equipment [13]. Further, certain workers such as firefighters and hazardous materials responders may be exposed to high levels of heat stress during emergency operations. In some situations, such as those inherent in firefighting, heat exposure is unavoidable. While protective equipment in these circumstances increases the amount of thermal load, it is also required to prevent severe burns or inhalation of toxic fumes. Therefore, prevention or removal from the thermal hazards is not feasible in some occupational circumstances, and greater efforts should be made to mitigate or lessen the risk for EHIs. Although exertional heat stroke (EHS) incidents are relatively rare during operations, multiple fatal EHS cases have been reported during firefighter training when highly motivated trainees are involved in intensive training and may perform multiple training scenarios in a single day.

Intense Physical Labor

The combination of heavy activity and environmental heat stress in particular creates a dangerous work environment (e.g., crop worker, firefighter, miner). Given the strenuous and continuous nature of the work that laborers are often required to do, many of these individuals can be at risk for EHI. For example, it is not uncommon for crop workers to be harvesting during the hottest times of the day with temperatures over 40 °C/104 °F. Firefighters are required to wear heavy protective equipment and may be exposed to temperatures over 250 °C/482 °F. These workers have also reported a desire to lose weight through increased sweating. This desire to lose weight may increase the risk for dehydration and thus increase the risk of heat-related injuries [8]. The metabolic heat generated from intense physical activity, such as repetitive shoveling in crop fields, lifting, and pulling in hot environments may overwhelm the body's ability to dissipate heat, leading to an increase in body temperature and EHI [8].

Equipment

Laborers may wear uniforms and protective clothing for their own safety in their workplace. While these uniforms are intended to improve safety for the individuals, they may contribute in increasing the amount of thermal strain one experiences.

Chemical, biological, radiological, and nuclear protective equipment, such as those worn by firefighters or military personnel, have been shown to significantly increase the amount of physiological and thermal strain on the body [14, 15]. Selectively permeable fabric has also been shown to result in a higher thermal strain, compared to air permeable ensembles [14]. In the crop fields, 85% of workers have reported wearing long sleeved shirt and 93% wear baseball caps [16]. While these might be beneficial in protecting the skin from rashes or sunburn, increased clothing in these hot environments may diminish the body's ability to dissipate heat [16]. Although protective clothing in the workforce provides considerable protection against the environment in which they are working, it may hinder efficient body heat dissipation. Therefore, special consideration should be paid to the type of equipment required to safely perform a given task.

The type of clothing worn during activities in the heat can also influence the rate of heat accumulation and heat dissipation. For example, the personal protective equipment (PPE) worn by firefighters is imperative to the safety against thermal injuries. However, they are heavy, impermeable, and restrictive, which in turn adds to the thermal, cardiovascular, and perceptual strain on the body [17–20]. Researchers have shown that wearing firefighting gear results in a higher heart rate by 47 beats per minute (bpm) while walking on a treadmill than when doing the same work in a station uniform [20]. Thus, while protective clothing provides essential protection to firefighters, it also adds to the physiological burden of performing the work. When considering worker safety, the physiological burden from environmental condition and PPE should not be neglected.

Lack of Enforced Standards

The lack of scheduled rest breaks and extended work shifts is the leading risk factor for EHIs observed in occupational workers [21]. While Occupational Safety and Health Administration (OSHA) and the National Institute for Occupational Safety and Health (NIOSH) have set rest breaks recommendations in an attempt to protect laborers, their ability to enforce standards and best practice is limited. In migrant farm workers, 34% stated that they have no access to regular breaks, 27% reported having no access to shade, and 26% reported of having no access to medical attention [5]. Not surprisingly, workers also noted that location and cleanliness of the water sources are limiting factors in maintaining hydration status at work [8]. In 2012–2013, there were 20 reported citations for not following the proper heat stress guidelines from OSHA. Of the 20 cases, 13 did not have any policies to identify and assess workplace heat illness risk as described by the National Oceanic and Atmospheric Administration (NOAA) [4]. Furthermore, heat illness prevention recommendations set forth by OSHA were not followed in all 20 cases; with 12 of them not having the heat illness prevention program at all, 7 with inadequate hydration management, and 13 failing to provide shaded rest areas [4]. Only one employer modified worker's work-to-rest ratios to reduce the exertional heat strain and none had heat acclimatization programs to help phase the workers into labor in the heat [4].

The Evidence

Epidemiology

The leading causes of death in workers are: Falls (38.8%), Struck by Object (9.6%), Electrocutions (8.6%), and Caught-in/between objects (7.2%) [22]. However, despite not being listed as a top cause of death in occupational workers, EHIs are still prominent. For example, work-related EHIs were the most common reason for EHI-related emergency room visits in North Carolina from 2008 to 2010 [23]. In light of the logistical challenges in compiling epidemiological data, few studies have explored EHI rates among US workers. A review of data published by OSHA and others demonstrate a declining trend in heat-related fatalities over the past 15 years (Fig. 15.1) [3, 6, 21]. A recent study found heat-related fatalities are estimated to average 1 death per 5 million workers (0.22 per 1 million persons) for US civilian workers [21].The heat-related fatality rate was 32 times higher in males than females [21]. However, in this period of time, only ten documented cased of female fatalities related to heat exposure were reported [21]. When the data are examined by the ethnicity of the worker, workers of African American descent had 1.5 times higher risk of EHI fatality compared to Caucasians, those of Hispanic decent had 3.2 times higher risk than non-Hispanics [21].

A recent critical review of the 2012–2013 OSHA enforcement decisions revealed a total of 84 cases of heat illness that were cited for federal enforcement under paragraph 5(a)(1) "the general duty clause" of the Occupational Safety and Health Act of 1970 in Federal OSHA heat enforcement cases [3, 4]. Due to the lack of a heat-specific standard, OSHA must use this clause, which states that, each employer must "furnish to each of his employees employment and a place of employment which are free from recognized hazards that are causing or likely to cause death or serious physical harm to his employees" [1, 3]. However, of the 84 cases of EHI noted above, 23 of them resulted in death [3], and 19 of the 84 cases involved one or more workers with symptoms of EHI [4]. In this review, 37 cases of EHI occurred in outdoors and 47 were reported indoor, with a local heat source such as laundry equipment or combustion engines [4]. The report also provided various settings in which the EHI cases were cited: solid waste collection, mail delivery, outdoor worksites (ship repair, landscaping, roofing, oil servicing), laundries, and indoor worksites with machinery.

Firefighting is an occupation where heat stress is a ubiquitous concern because firefighters perform strenuous muscular work while wearing heavy, insulated PPE and are often working in high ambient temperatures. Although the annual report of firefighter injuries and fatalities by the National Fire Protection Association (www.nfpa.org) indicate low prevalence of "thermal stress" injuries (5–6%) with EHS fatalities rarely exceeding one per year, it is believed that the many of the physiologic derangements that accompany heat stress contribute to the high rate of fatal heart attacks among firefighters [24–26].

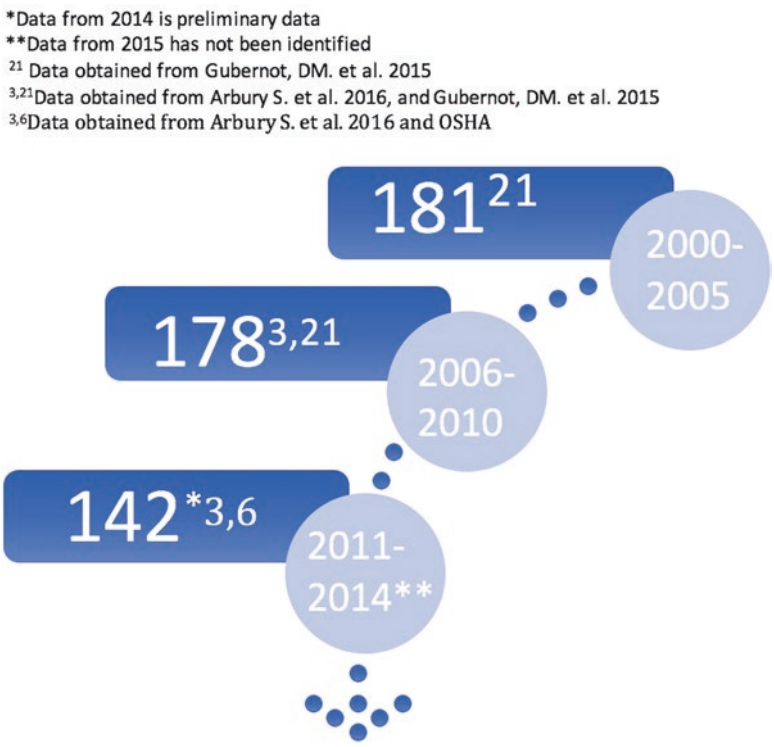

Fig. 15.1 Declining trend of documneted exertional heat related illness cases published in the occupational epidemiological reports over the past 15 years

The Application

Despite the concerning risk of heat-related fatalities among US civilian workers, a lack of prevention strategies is observed nationwide. Astonishingly, a majority (74%) of fatalities occurred within the workers first 3 days on the job [3, 5]. While some states, such as California, have been attempting to improve promotion of EHI prevention and workplace safety, there have been little improvements nationwide [6]. Nevertheless, a study about EHI knowledge and prevention strategies among crop workers in California demonstrates that, despite their leading efforts to improve worker safety above and beyond the OSHA's standards, the translation of the educational effort to their knowledge was limited.

Georgia is also one of the states that has attempted to improve the EHI prevention standards for migrant workers but has seen little success. A study on migrant farm workers in Georgia in 2011 (one of the hottest summers) showed that 70% of workers stated they had EHI-related symptoms in the previous week, and 77% stated that they were not trained on prevention strategies for EHI [5]. Prevention strategies including education on hydration status have been demonstrated to miti-

gate the risk of EHI and therefore, beneficial for the overall health of the worker [27–29]. From a workplace company standpoint, workers education on hydration status may also benefit the performance of the worker and produce work-output and productivity [27–29]. Furthermore, education on EHI of workers has been shown to be effective, however, workers underestimated the time required to heat acclimatize and had low water consumption [30].

Figure 15.2 provides a framework for prevention and mitigation strategies for EHI in outdoor occupational settings. Although the original framework was devised for outdoor workers (such as crop workers and construction workers), it could be adopted for many workers who face the combined challenges of heavy work and high indoor temperatures [31].

Notwithstanding the lack of compliance with the best practices in reported EHS fatality cases, there are efforts aimed at improving worker safety in the states of California, Georgia, and Washington. These states are leading the workplace safety standards by enforcing regulations for employers for drinking water, shade for rest or other means to recover from heat, worker and supervisor training and written heat safety plans, complimenting the national initiative of "Water, Rest, Shade" program. Table 15.2 summarizes the steps involved in the "Water, Rest, Shade" campaign that OSHA launched in 2011 to help mitigate EHIs. The program is designed to help employers put the necessary precautions into place to help reduce the risk for EHI [32, 33]. Providing workers with water rest breaks, and shade is imperative to prevent dangerous rises in the body temperature and provide rest for workers. Implementation of this plan may have an effect on both safety and productivity, however it has not been proven to be effective in the United States. However, sugarcane workers in El Salvador were evaluated before and after the implementation of the "Water, Rest, Shade" program, where the recorded WBGT exceeded 26 °C/78.8 °F for the majority of the days. Following the intervention, water consumption increased 25%, symptoms from EHI decreased, and individual daily production increased daily production increased from 5.1 to 7.3 tons per person per day (Table 15.2) [34].

The program includes education on prevention, recognition, and treatment for both the employer and the employee. In addition, the hallmark of the program is to provide guidance on the progression of heat acclimatization for new employees or employees who are new to the hot working environments (Fig. 15.3) [7, 33].

Pre-cooling intervention is another way to prevent EHIs. Various cooling modalities may be utilized to lower internal body temperature during or between work shifts, which can not only help mitigate the risk for EHIs but also enhance productivity. For example, ice bags may be used on the peripheral arteries, such as the axillae, groin, and back of neck to increase work productivity in the heat [35–37]. Other cooling methods such as hand or head cooling may be applicable for those workers who may not be able to expose their skin due to equipment or uniforms restrictions (e.g., firefighter). While ice bags and these devices are not suitable for EHI treatment, they may aid in attenuating the rise of internal body temperature during daily work activities if they are used before, during, and after work [35, 38]. Although cooling vests have been evaluated and found to have little effect on overall

Fig. 15.2 Prevention strategies to mitigate EHIs (e.g., crop workers, construction workers) Adapted from Lindsley M and Cadorette M, 2015 [31]

Fig. 15.3 OSHA recommended heat acclimatization process [33, 7]

Table 15.2 Ten steps for successful implementation: organizational and individual checklist to mitigate exertional heat illness risk

	Organizational	Individual
1. Medical history	Identify workers with history of heat-or exertion-related illness	Report previous complications during physical activity in heat
2. Recent illness	Workers with recent illness should seek medical care as necessary and notify supervisors prior to work	
3. Work to rest ratio	Adjust the duration of work and rest based upon environmental conditions	
4. Hydration	Worksite should have access to potable water Water should be readily accessible to workers	Workers should hydrate before, during, and after physical work to minimize dehydration Workers should know their sweat rates to gauge on the amount of body fluid loss to prevent from excess dehydration as well as over hydration
5. Heat acclimatization	Extra precaution needs to be provided to during the beginning of the summer months Workers who are new to the working in heat or who recently moved from cooler climate may require additional rest	Workers should recognize that exertional strain is often greater at the beginning of the summer and at the early stage of their training at work
6. Body cooling	When a long heat exposure is expected, there should be a designated space for cooling modality (e.g., rotating ice towels, cold water mist)	Workers should be encouraged to cool their bodies while working or during their rest breaks to minimize the thermal strain
7. Medical supervision	Each worksite should have a designated medical supervisor When feasible, on-site medical supervision is recommended in settings with higher risk of exertional heat illnesses	Workers should know who to report when they suspect exertional heat illness
8. Education	Employer, medical supervisor, and workers themselves should be educated on the signs and symptoms of exertional heat illness Emergency action plan should be developed, shared, and rehearsed by all parties	
9. Clothes and equipment	Employees should provide their workers with appropriate clothing and equipment to minimize excess heat gain while still protecting the worker from potential hazards	Workers should dress appropriately to minimize any potential hazardous exposure but should also be encouraged to remove excess equipment when the scene is safe
10. Wellness	Efforts should be placed to encourage healthy and active lifestyle to the workers	

whole body cooling rates, the cooling effects of these types of devices may be more effective in tempering the rise of body temperature than no cooling [39]. Forearm immersion cooling has also demonstrated a decreased cardiovascular and thermal strain [40]. It should be noted that any of the aforementioned cooling methods that cools a body part (e.g., chest, hands, head) should not be a standalone cooling method in the treatment of EHS; when EHS is suspected, whole body immersion using cold/ice waters should be used.

Recognition and Treatment

As previously mentioned, lack of EHI knowledge and direct supervision by healthcare professional at the work place may lead to missed signs and symptoms of EHI. Knowledge of the typical symptoms of EHI may aid in preventing a catastrophic heat-related injury from occurring in the work place. Symptoms such as dizziness, confusion, agitation, feeling "hot," personality changes, and other central nervous system dysfunction are the common hallmarks of EHI. Prompt recognition and care are imperative to improve the outcome of EHS. It is not uncommon for occupational settings to have safety managers on-site; although a person in this role is less likely to be a licensed healthcare professional, he or she could play a role in recognition and treatment of these types of injuries. A person with training in this area may be able to recognize this as a medical emergency quicker than someone without training. Rapid cooling is essential for an individual who is thought to be experiencing an EHS. Cooling with cold-water immersion (CWI) is the gold standard in treating EHS patients [27, 29]. Rapid cooling to bring the internal body temperature under 38.8 °C/102 °F within 30 min should be accomplished to prevent long-term sequela [29].

Despite the increased public effort on EHS education, there still remains a great misconception regarding the recognition of EHS signs and symptoms [6]. For example, a common misconception about EHS signs includes the absence of sweating. However, there is no evidence to support that a patient will cease sweating; in fact, researchers have found that EHS patients do sweat [8, 10].

Return to Duty

There is a paucity of data on the appropriate timeline for a return to duty (RTD) following an EHI or EHS diagnosis in the occupational world. RTD is complicated and frequently left up to the supervising physician. It is highly advised to follow the RTD criteria set forth by the American College of Sports Medicine, which include items related to end organ healing, blood markers, endurance status, restoration of duty specific skills, and compliance with institutional regulations [41]. However, it

is unclear if athletes' return to play progression will demonstrate the similar success in the laborers' RTD progression.

Case Example 1 *A regional trauma center conducted a disaster drill in late September during hot and humid outdoor conditions. Participants were required to wear a full protective ensemble including a powered air purifying respirator (PAPR) and chemical resistant coverall. The PAPR consists of an impermeable hood that covers the head, neck, and shoulders. Breathing air is delivered to the wearer by drawing ambient air through a battery-driven filter worn at the waist.*

In addition to the medical providers participating in the drill, security personnel donned protective equipment and were stationed at various posts outdoors. One 45-year-old security officer complained of profuse sweating, dizziness, lightheadedness, and fatigue after 45 min. He was removed from the drill and taken to the emergency department. After removing the protective garments, the patient's symptoms and vital signs began to improve although he complained of a mild headache that persisted over the next few hours. His clothes were saturated with sweat. His vital signs and serum blood glucose in the emergency department were normal. The patient cooled passively in the emergency department and was treated with 1.5 L of normal saline before being released.

Despite the low intensity work performed by the patient, he still suffered symptoms of heat illness likely due to the added thermal strain from protective garments. In this situation, the officer was simply removed from the drill site and doffed the protective garments which limited further heat stress. Had there been an actual environmental threat, he would require decontamination before doffing. Similar scenarios could be encountered in workers using pesticides and other chemical applications.

Although all workers who are required to use a respirator must pass the OSHA-required screening examination, the capacity to safely work in protective garments will vary greatly. There is a wide range of fitness and body composition among workers in nearly every occupation. Poor health and preexisting medical conditions may increase risk of EHI during work, especially among those who rarely use protective garments.

Case Example 2 *In 2011, a 23-year-old wildland firefighter (a member of a hotshot crew) died after performing wildland firefighting duties. It was a hot day (dry bulb temperature 40.5 °C/105 °F, wet bulb temperature 23.8 °C/75 °F, 24% relative humidity). The firefighter was working as a member of a crew and had been assisting a saw team (*i.e.*, strenuous work) while wearing protective equipment and leather chaps to protect the legs while clearing trees and shrubs.*

The firefighter was carrying six quarters of water. At 1330 h the crew had lunch together and while all members were hot, tired, and sweaty; no one reported any signs or symptoms of heat-related illness. After lunch, the crews refilled their canteens and returned to work and at 1545 h a supervisor observed the firefighter stumble on a rock slope and asked how he was feeling. The firefighter indicated that he was hot and had a little headache. The supervisor indicated that the firefighter

should rest and the supervisor left to assist another team. When he returned 5 min later to check on the firefighter he found him unresponsive on the trail.

An emergency medical technician (EMT) arrived at the firefighter's side within about 2 min but due to the firefighter's fall, initial care focused on spine injury including placing the injured firefighter on a backboard; and did not focus on heat-related injury. The firefighter was unresponsive to verbal commands but was responsive to pain. A second EMT arrived a couple of minutes later and removed clothing, poured water on the firefighter, and provided shade using a tarp. At this point, his heart rate was 120 bpm and his breathing rate was 12 breaths per minute. At 1610 h, the firefighter stopped breathing and an oropharyngeal airway was placed. Because the firefighter was operating in a remote area, an extraction line had to be cut to get the firefighter to a dirt road where he could be transferred to an ambulance. While he was being extricated, the firefighter went into cardiac arrest. The ambulance left the scene at 1645 h and arrived at the hospital at 1658 h. One-hour post collapse, the firefighter's rectal temperature was recorded at 42.2 °C/108 °F. The Emergency Department continued advanced life support for a short period of time before the firefighter was pronounced dead. The autopsy listed the cause of death as hyperthermia and reported that there was no evidence of clinically significant atherosclerosis or structural heart defects, no pulmonary embolism, and no indication of dehydration or hyponatremia.

This tragic case highlights the dangers faced by firefighters and the challenges faced when performing their duty in remote locations. In this case, fatal hyperthermia occurred due to strenuous work in high ambient conditions despite ample availability of water and work place reminders to work at a "safe" pace. Delayed treatment resultant from remote circumstances and the inability to extract in a timely manner also played an important role in this tragic case.

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Chapter 16
Military Settings: Considerations for the Warfighter

Patrick J. Depenbrock, Shawn F. Kane, and Francis G. O'Connor

The Challenge

The United States military has a long, well-documented struggle with the threat of exertional heat illness (EHI). Even as far back as the Revolutionary War, Molly Pitcher, the heroine of the Battle of Monmouth, is depicted in paintings as she drops water intended for thirsty Colonial soldiers and takes charge of a cannon vacated by her heat-stricken husband. Since that time, we have learned a plethora of lessons from the US military experience. Data from the Pacific and North Africa Campaigns of World War II, Vietnam, and Operations Desert Storm and Iraqi Freedom helped form the early foundation of knowledge for EHI.

The unique nature of the military workforce poses a challenge to commanders and medical officers as they confront EHI. Today, US military personnel routinely work, train, and operate in hot weather climates. Training exercises (including initial entry training like "boot camp" and special badge qualification courses like Ranger School), operational deployments, and the microenvironments of military-unique occupational duties expose personnel to significant heat stress. As a result, EHI prevention, management, and return-to-duty planning is a priority for leaders in both training and operational commands.

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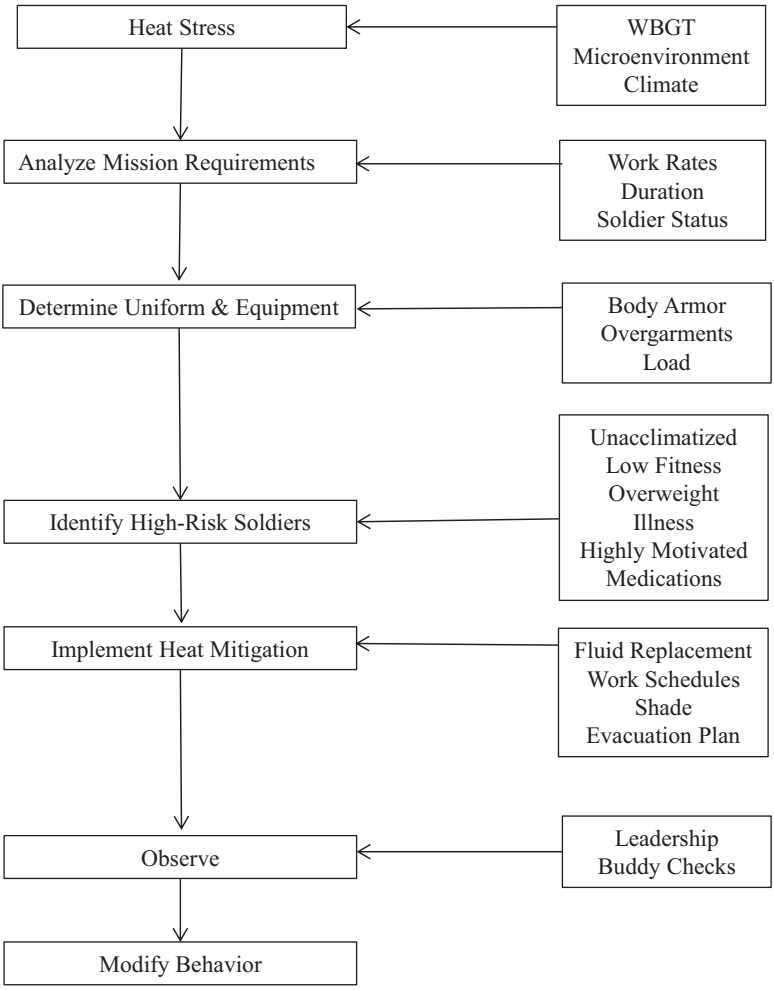

Fig. 16.1 U.S. Army Heat Strain Decision Process [1]. Adapted from U.S. Army Center for Health Promotion and Preventive Medicine. Available at: http://chppm-www.apgea.army.mil/documents/TBMEDS/tbmed507.pdf. Accessed 20 Jul 2016

In addition to individual risk factors considered by athletic trainers (e.g., illness, use of high-risk medications), military commanders and their staff must consider competing demands of mission requirements, motivation of their personnel, specialized protective clothing, body armor, load bearing equipment, limited access to water, occupational microenvironments of tactical vehicles, and others. Fig. 16.1 outlines the relationship of these factors in the military heat strain decision-making process.

The mission requirements of combat may demand intense physical activity, little sleep, and limited access to water, all of which can lead to dehydration and

EHI. Although military personnel train for these scenarios, it is not uncommon that combat scenarios require military personnel to sustain high-intensity work beyond that exhibited in training scenarios [1]. In hot weather training environments, very active soldiers may consume in excess of 12 quarts of water per day. During combat in World War II, very active soldiers conducting desert operations in North Africa required in excess of 16 quarts of water per day. When high water requirements such as these are combined with operational scenarios that limit access to water, military personnel must execute water economy in order to successfully carry out the mission. In North Africa, soldiers fought for 4–5 days on water rations of just 1 quart per day (albeit with impaired combat effectiveness) [1].

Heat stress is also encountered in the microenvironments of the military-unique workplace. Aboard ship, it is not uncommon for dry bulb and wet bulb temperatures to exceed 90 °F/32.2 °C and 81 °F/27.2 °C, respectively [1]. Sailors working in ship engine or boiler rooms are exposed to heat stress conditions so severe that they cannot be tolerated for extended periods. As a result, they work in shifts based on the guidance of Physiologic Heat Exposure Limits (PHEL) published in the Navy Medical Department Publication (NAVMED P)-5010-3. Military personnel who operate close quarters combat vehicles, engage in firefighting, and wear layered protective clothing (especially in hot environments) are also subject to significant heat stress. Protective clothing such as nuclear biologic chemical (NBC) gear and body armor increase heat strain for military personnel, thus they must be factored into fluid replacement and work/rest guidelines. Flight crews encounter heat stress during preflight, engine start, taxi, and standby for takeoff. Reduced cockpit air velocity, thermal burden of protective clothing, and ground delays all impact heat load within the cockpit. The Fighter Index Thermal Stress (FITS) calculation outlined in Air Force Pamphlet 48-151 is used to predict the heat stress within a canopied jet aircraft. Based on this calculation, aircrews can take measures to minimize the impact of heat stress (i.e., avoid exercise 4 h before takeoff, precool cockpits using ground vehicles, limit duration of in-cockpit standby). These measures become critically important in wartime scenarios, when flight crews may be required to fly two or three missions back to back in quick succession with little opportunity to achieve recovery of full body temperature and volume status [1].

The Evidence

EHI has a significant impact on the medical readiness of the US military, particularly during recruit training, and is a common cause of preventable nontraumatic death [2–4]. Over the decade between 2004 and 2014, the Army reported a yearly average of 2–3 fatalities from EHS, the majority of which occurred during physical readiness training and/or testing [5]. Although military basic training recruits who experience an initial episode of EHI are not significantly more likely to be hospitalized for another EHI event during their subsequent military service, the long-term complications of soldiers who fall victim to EHS are more concerning. Compared

to soldiers hospitalized for appendectomies, soldiers hospitalized with EHS demonstrate a higher mortality (30 years later in life) from cardiovascular, liver, renal, and gastrointestinal diseases [6].

The U.S. Army saw a fivefold increase in EHS hospitalization rates between 1980 and 2001. Within this population, gender, race, and geographic home of origin accounted for much of the difference in EHS hospitalization rates [2, 7]. By 2007 this trend continued, as EHS hospitalization incidence rates were noted to have increased seven to eightfold over the previous 20 years [8]. In 2008, a U.S. Army Center for Health Promotion and Preventive Medicine working group found "excessive heat" to be the fourth most important injury challenge facing the U.S. Army and the seventh most common cause of hospitalization for unintentional injury [9].

By 2014 however, the incidence of EHI, including EHS, started to level off across the US military [10]. In 2014, there were 344 incident cases of EHS and 1683 incident cases of other heat injuries (where "other heat injury" was defined by ICD-9 diagnoses of "heat exhaustion" or "unspecified effects of heat"). Compared to the previous 5 years, the annual incidence rate for EHS has remained relatively stable, while the incidence rate for other heat injuries declined by nearly one-third. It is unclear whether this is due to better risk management strategies, education, leadership, or other factors. In Iraq and Afghanistan, US military cases of EHI, including EHS, saw a sharp decrease over the period from 2010 to 2014. This however is commensurate with the declining presence of U.S. forces in those two countries [10].

It is noteworthy that the majority of EHI cases are clustered within two branches of the military. EHS and "other heat injury" rates in the Marines and Army far exceed that of the other services. Of all US military personnel, the youngest and most inexperienced Marines and Soldiers (particularly basic entry recruits training in the southeastern United States) are at the highest risk of heat injury. In 2014, EHS rates in the Marine Corps were 50% higher than that of the Army. To put this in perspective, the Army rates were already more than ninefold greater than those of the Navy and Air Force. This likely reflects the disproportionate number of ground combat arms troops (who are more likely to train in hot weather climates) in the Marine Corps and Army relative to the other services, although other factors may play a role as well.

While the EHS rate was 86% higher among males compared to females, females exhibited a higher rate of "other heat injuries." "Other heat injuries" occurred most commonly in service members younger than 20 years old serving in the Army or Marines who were either recruit trainees or combat arms specialists (e.g., infantry, armor). Previous studies of Marine Corps recruits found age, poor physical fitness, lack of acclimatization, prolonged exertion, lack of sleep, illness, skin disease, history of heat injury, drug use (e.g., ephedra), use of heavy equipment or clothing, and obesity to be risk factors for EHI [11]. Army basic training male recruits who are obese or overweight are more than three times more

Table 16.1 Heat injuries[a] by location in U.S. Armed Forces[b] (2010–2014) [10]

Location of diagnosis	Number	Percent of total (%)
Fort Bragg, NC	1367	10.7
Fort Benning, GA	1352	10.6
Fort Jackson, SC	1275	10.0
Marine Corps Base Camp Lejeune/Cherry Point, NC	626	4.9
Fort Campbell, KY	463	3.6
Fort Polk, LA	414	3.3
Marine Corps Recruit Depot Parris Island/Beaufort, SC	383	3.0
Marine Corps Base Quantico, VA	257	2.0
Fort Hood, TX	254	2.0
Marine Corps Base Camp Pendleton, CA	234	1.8
Okinawa, Japan	230	1.8
Naval Medical Center San Diego, CA	228	1.8
Fort Stewart, GA	209	1.6
Fort Sill, OK	198	1.6
Joint Base San Antonio—Lackland, TX	174	1.4
All other locations	5055	39.7
Total	12,719	100.0

Adapted from Armed Forces Health Surveillance Center. Update: Heat Injuries, Active Component, U.S. Armed Forces, 2014. *MSMR*. 2015; 22(3):17–21

[a]One heat injury per person per 60 days

[b]Active component

likely to sustain EHI than nonobese counterparts during the first 90 days of service [12]. Exposure to the previous training day's heat may also be a factor, suggesting that heat stress is cumulative. A 2005 study of Marine Corps basic trainees found that EHI risk was associated not only with the wet bulb globe temperature index (WBGT) at the time of training, but also with the previous day's average WBGT [13]. The WBGT is an empirical index of environmental heat stress based on the readings of a wet bulb thermometer, a black globe thermometer, and a dry bulb thermometer taken at a location representative of the conditions to which soldiers are exposed [1].

Seasonally and geographically, the majority of heat injuries take place during the heat season (May 1 to September 30) in the southeastern USA, with three Army installations (Fort Bragg, NC; Fort Benning, GA, and Fort Jackson, SC) accounting for nearly a third of all heat injuries (see Table 16.1) [10]. It should be noted that although the heat season is arbitrarily defined May 1 through September 30, and the risk of EHI in susceptible individuals rises as the WBGT increases, EHI and EHS do occur in cool conditions as well [14, 15]. In fact, one study found that more than 17% of EHI at army installations occurred outside the heat season. Hence, leaders should consider the year-round risk of EHI in any risk assessment [16].

The Application

Prevention

Management of heat stress begins with prevention. The United States Military is very clear when it comes to heat injury prevention. The prevention of environmental casualties (heat and cold) is a command responsibility [17, 18]. Commanders are to safeguard the health of all personnel through careful supervision and discipline. This is reinforced through policies published by the Office of the Surgeon General, service regulations, operational doctrine, field manuals, and internal unit policies. Prevention is achieved through the implementation and enforcement of policies that require education of leaders and soldiers, the employment of fitness training principles, and the management of activities based on weather conditions [17]. Fort Bragg NC, which routinely sees the highest number of EHI in the military, requires all military personnel to receive annual training in heat casualty identification, prevention, treatment, and risk mitigation. Commanders are required to maintain an electronic record of this training to enforce 100% compliance. Soldiers are proactively screened for individual risk factors, including the use of high-risk drugs. These include medications that inhibit thermoregulation (e.g., anticholinergics, antihistamines, tricyclics, stimulants, diuretics, antipsychotics, ACE inhibitors, beta-blockers), as well as high-risk supplements (e.g., DMAA). Soldiers with risk factors can then be marked with a tag on their boot laces so they can be monitored during unit road marches, etc. Best practices such as these however, have not always existed. Instead, they have evolved through a deliberate process of risk management.

Risk Management

Risk management is the process through which commanders identify, assess, and prioritize different threats [19]. Once this is achieved, commanders can develop a plan to minimize the impact of negative events. The Office of the Surgeon General routinely provides updated guidance on EHI to assist commanders in the development of their own heat prevention programs. Commanders and their staffs then utilize this guidance to issue their own policies outlining guidance and expectations for the prevention of EHI.

A risk management-based heat injury prevention program is a five-step process that begins with identification of hazards. Once a hazard is identified, an assessment is made of its severity and probability [1, 19]. Controls are then developed and implemented to abate the hazard. The process concludes with supervision and evaluation of the control measure's effectiveness [8]. This process is represented in the U.S. Army's Composite Risk Management Worksheet (DA Form 7566). TRADOC Regulation 350-29 Appendix B includes a prefilled sample worksheet that can prove

very helpful for those new to the EHI Risk Management process [18]. Risk management is then implemented into the U.S. Army Heat Strain Decision-Making Process (see Fig. 16.1) [1]. Despite the critical role that risk management plays in a comprehensive heat injury prevention program, heat injuries will still occur in military operations. As such, commanders will always have to assume a certain level of risk when the probability of EHI is measured against mission requirements [17].

Heat Mitigation Strategies

Once commanders identify and assess hazards, they can establish and implement controls to mitigate the risk associated with the hazard. Adherence to acclimatization protocols, work-rest cycles, and hydration guidelines improves heat tolerance [20, 21]. Below are just some of the examples of heat mitigation strategies used by military commanders and their units.

Acclimatization

Heat acclimatization refers to a series of biological adaptations made by the body in order to reduce the physiologic strain imposed by heat. These adaptations include activation of the renin-angiotensin-aldosterone system, salt conservation, expansion of plasma volume, and increased resistance to rhabdomyolysis. Reduced core temperature, early enhanced sweating response, early cutaneous shunting of blood, and lower body heat production improve a soldier's thermal comfort. Lower heart rate, improved thirst, and reduced salt concentrations in urine and sweat improve a soldier's exercise performance [1, 22].

Heat acclimatization takes place when a soldier experiences repeated heat exposures that are sufficiently stressful to elevate body temperature and provoke a sweating response. Heat acclimatization requires 2 weeks of daily heat exposure for the average soldier, where daily heat exposure entails a minimum of 2 h in the outdoor hot weather environment [1, 22]. These 2 h can be broken up into 1-h intervals. During this period of heat exposure, a soldier must engage in cardiovascular exercise in order to optimize the physiologic adaptations of acclimatization. The attached table contains a list of acclimatization strategies for military personnel preparing for deployment or elite training schools that will take place in warm weather climates (see Table 16.2) [1, 22].

Work-Rest Cycles

Hot weather guidelines regarding work-rest cycles and fluid replacement should be initiated once the WBGT index exceeds 75 °F/23 °C. In general, as the WBGT increases, either the physical work intensity should be decreased, or the ratio of

Table 16.2 Heat acclimatization strategies for warm weather deployments or elite training schools [1]

1. One month prior
• Optimize physical fitness in current climate
• Set aside between 4 and 14 "acclimatization days"
• Mimic deployment/training environment on "acclimatization days"
– In warm weather climate, acclimatize in heat of the day
– In temperate climate, work out in a warm room wearing sweat suit
• Ensure adequate heat stress on "acclimatization days"
– Induce sweating
– Slowly work up to 100 min/day of continuous exercise
– Add intensity (e.g., load, pace) once able to exercise comfortably for 100 min/day
• Learn to fuel and hydrate
– Do not wait for thirst mechanism before hydrating
– Do not skip meals
– Drink ½ quart of fluid for each pound lost during 100 min of exercise in heat
2. On arrival
• Allow 2 weeks of progressive heat exposure and physical work
• Acclimatization requires a minimum of exposure of 2 h per day
• Acclimatize in the heat of the day
• Incorporate cardiovascular endurance exercise (rather than strength training) into acclimatization
• Start slowly and gradually increase exercise intensity (e.g., slow unit runs lasting 20–40 min should be feasible by third day)
• Train in the coolest part of the day
• Use interval training or work/rest cycles
• Increase heat and training volume as tolerance permits

Adapted from U.S. Army Center for Health Promotion and Preventive Medicine. Available at: http://chppm-www.apgea.army.mil/documents/TBMEDS/tbmed507.pdf. Accessed 20 Jul 2016
Notes: (1) Very fit soldiers can acclimatize in 1 week, while less fit soldiers take up to 2 weeks. (2) Monitor the least fit soldier (who suffers greater heat strain) and the most motivated solider (who may overdo physical activity). (3) Be alert of salt requirements, particularly in first week of acclimatization (i.e., don't skip meals). (4) Be alert of water requirements, as heat acclimatization increases sweating rate (i.e., heat acclimatized soldiers dehydrate faster if they do not consume fluids)

work-to-rest should be decreased (Table 16.3) [1]. Protective gear such as body armor or NBC clothing increases heat strain by interfering with the dissipation of heat from the body [23]. As such, the index should be corrected as follows: add 5 °F/2.8 °C to WBGT for use of body armor; add 10 °F to WBGT index for use of NBC clothing. Under certain extreme conditions, such as when WBGT index >90 °F/32 °C, work should be suspended [1]. Reserving moderate to high intensity work (425–600W) for nighttime when the WBGT index is lower is one way units can optimize work efficiency and reduce water requirements. Desert operations have demonstrated that working at night reduced water requirements by 30% (from 10 quarts per day to 7 quarts per day) [1].

Table 16.3 Fluid replacement and work/rest guidelines in warm weather [1]

Heat category	WBGT index (°F)	Easy work (250W)		Moderate work (425W)	Hard work (600W)		
		Work/rest	Water intake (qt/h)	Work/rest	Water intake (qt/h)	Work/ rest	Water intake (qt/h)
1	78–81.9	No limit	½	No limit	¾	40/20	¾
2	82–84.9	No limit	½	50/10	¾	30/30	1
3	85–87.9	No limit	¾	40/20	¾	30/30	1
4	88–89.9	No limit	¾	30/30	¾	20/40	1
5	>90	50/10 min	1	20/40	1	10/50	1
Easy work		Moderate work			Hard work		
– Weapon maintenance – Walking on hard surface @ 2.5 mph, <30 lb load – Marksmanship training – Drill and ceremony		– Walking on loose sand @ 2.5 mph, no load – Walking on hard surface @ 3.5 mph, <40 lb load – Calisthenics – Patrolling – Individual movement (e.g., low crawl and high crawl) – Defensive position construction			– Walking on hard surface @ 3.5 mph, ≥40 lb load – Walking on loose sand @ 2.5 mph with load – Field assaults		

Adapted from U.S. Army Center for Health Promotion and Preventive Medicine. Available at: http://chppm-www.apgea.army.mil/documents/TBMEDS/tbmed507.pdf. Accessed 20 Jul 2016
Notes: (1) Work/Rest times and fluid replacement volumes apply to average size, heat acclimatized soldier in battle dress uniform performing up to 4 h of work. Fluid needs may vary based on individual differences and exposure to sun. (2) Add 5 °F, 10 °F, and 20 °F to WBGT index in humid climates if wearing body armor, NBC with easy work, NBC with hard work respectively. (3) Hourly fluid intake and daily intake should not exceed 1½ quart and 12 quarts, respectively

Fluid Replacement

Knowledge of daily water requirements in hot weather environments is useful for mission planning. In hot weather climates, soldiers will consume between 3 and 12 quarts per day during military training depending on their level of activity and sun exposure [1]. Thirst does not reliably motivate personnel to consume adequate fluids for the replacement of sweat loss [1]. As a result, leaders must enforce mandatory canteen water breaks. Leaders ensure adequate salt consumption by supervising meal compliance. Soldiers are taught to monitor hydration through evaluation of urine color/volume (e.g., Riley water card, Ogden card) and body weight [1].

Pre-event Cooling

Cold water extremity immersion has been implemented in military trainee populations as a potential measure to prevent EHI. Arm immersion in cool or cold water has demonstrated effectiveness as method of reducing body temperature [24].

The highly vascularized nature of the forearms with their high surface to mass ratio makes it a convenient location to cool peripheral blood before it returns to the body's core [25]. One system of arm immersion cooling (AIC) was implemented during summer months at the U.S. Army Ranger School. Soldiers used the large trough filled with cold water following high-intensity events and during rest periods. A retrospective study of more than 10,000 Ranger candidates found that AIC did not affect the incidence rate of EHI, but it was associated with reduced EHI severity and reduced medical cost per casualty [26]. Pre-event cooling may also affect performance. In one study that examined the effect of pre-event cooling on performance, the use of an ice-cooling vest for 20 min prior to exercise demonstrated improved running performance [27].

Microclimate Cooling Systems

A microclimate cooling system is a cooling undergarment that uses ice packets or cooled air/liquid (circulating in tubes over the skin) to remove body heat. Microclimate cooling systems are effective in alleviating heat stress and extending exercise capabilities in soldiers wearing protective clothing [1]. Microclimate cooling systems have been successfully used in armored vehicles like the M1A1 tank, but the need to retrofit vehicles to accommodate the cooling systems and their power source has limited fielding [1, 25]. Microclimate cooling systems have been used on a trial basis for dismounted soldiers, but are not in routine operational use.

Mist Fans

Mist fans and commercial cooling towels are other methods of heat mitigation used by some military units. Their effectiveness, however, is questionable. One small randomized control trial of 35 active, military-age men at Fort Benning, GA demonstrated that compared to passive cooling, neither mist fans nor cooling towels were an effective means of reducing core temperature following physical activity [28]. Thus, there is a need for additional research in the area of novel heat mitigation strategies for military populations.

Early Diagnosis

The decentralized nature of most military training and operations necessitates that noncredentialed healthcare providers (e.g., medics, corpsmen) and nonmedical personnel (e.g., Soldiers, Marines, Airmen, Sailors) perform a majority, if not all, of the patient care for heat casualties. This requires that medical officers educate nonmedical personnel in the identification of warning signs, treatment, and evacuation of heat

Table 16.4 Exertional heat illness definitions by Army Medical Department for purpose of medical profiling [29]

Diagnosis	Definition
Heat exhaustion (HE)	A syndrome of hyperthermia (core temperature at time of event usually ≤40 °C/104 °F) with physical collapse or debilitation occurring during or immediately following exertion in the heat, with no more than minor central nervous system (CNS) dysfunction (such as headache, dizziness). HE resolves rapidly with minimal cooling intervention
Heat injury	HE with clinical evidence of organ (for example, liver, renal, stomach) and/or muscle (for example, rhabdomyolysis) damage without sufficient neurological symptoms to be diagnosed as heat stroke
Heat stroke (HS)	A syndrome of hyperthermia (core temperature at time of event usually ≥40 °C/104 °F), physical collapse or debilitation, and encephalopathy as evidenced by delirium, stupor, or coma, occurring during or immediately following exertion or significant heat exposure. The HS can be complicated by organ and/or tissue damage, systemic inflammatory activation, and disseminated intravascular coagulation
Heat stroke without Sequela	HS with clinical signs and symptoms that resolve within 2 weeks following the heat exposure event
Heat stroke with Sequelae	HS with evidence of cognitive or behavioral dysfunction, renal impairment, hepatic dysfunction, rhabdomyolysis, or other related pathology that does not completely resolve within the 2 weeks following the heat exposure event
Complex heat stroke	HS that is recurrent, or occurring in the presence of a non-modifiable risk factor, either known (for example, a chronic skin condition such as eczema or burn skin graft) or suspected (for example, sickle cell trait or malignant hyperthermia susceptibility)

Adapted from Army Regulation 40-501 Chapter 3-45 1 Dec 07/RAR 23 Aug 10

casualties. This entrustment of early clinical decision-making to noncredentialed healthcare providers and nonmedical personnel represents a paradigm shift that is reflected in EHI command policies and treatment protocols throughout the military.

The Army Medical Department (AMEDD) uses various definitions of EHI used for the purpose of medical profiling (Table 16.4) [29]. These diagnoses are not working diagnoses, as they are not established until after the patient has reached a definitive level of care with appropriate laboratory diagnostic capabilities. Compared to heat exhaustion (HE) and heat injury (HI), heat stroke (HS) carries the significant burden of morbidity and mortality. As such, medical officers put significant emphasis on EHS awareness and early diagnosis. Due to the limited availability of medical resources in the field environment, military personnel must have a high index of suspicion and a low threshold for assessing rectal temperatures and initiating aggressive cooling techniques in potential EHS cases [30]. This strategy is the optimal route to minimizing EHS fatalities. In field training scenarios, medics and corpsmen are often the first responders. Fig. 16.2 represents the schematic for field assessment and treatment of heat casualties by Army/Air Force Medics [1]. Highly motivated military personnel may push their bodies to physiologic extremes in hot weather training, therefore it is vital that personnel be

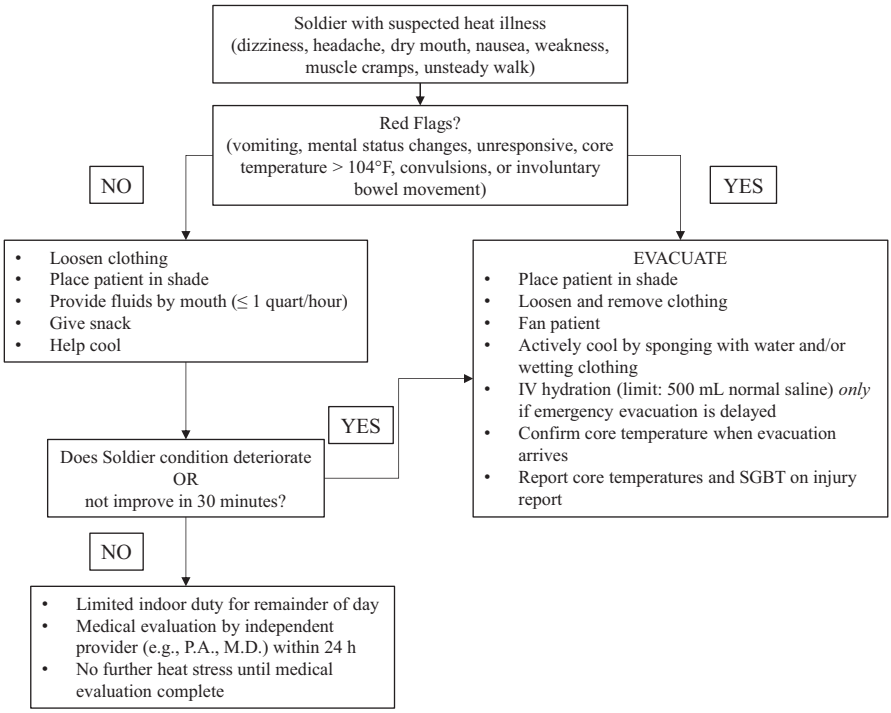

Fig. 16.2 Field treatment of heat casualties by U.S. Army/Air Force Medics [1]. Adapted from U.S. Army Center for Health Promotion and Preventive Medicine. Available at: http://chppm-www.apgea.army.mil/documents/TBMEDS/tbmed507.pdf. Accessed 20 Jul 2016

aware of pre-collapse symptoms such as fatigue, irritability, dizziness, irrational thought, and the inability to continue. In addition to traditional warning signs, these pre-collapse symptoms should be considered triggers to initiate medical care [31]. This should begin with an assessment that includes measurement of internal body temperature. Rectal temperature is the only consistently reliable and valid way to measure internal body temperature in the field [32]. However, for logistical reasons or reasons due to patient noncompliance, a first responder may not be able to obtain a rectal temperature in the field. After a cardiac cause is ruled out, any service member who displays altered mental status in the right environmental and activity conditions (which usually, but may not necessarily, include warm weather) is considered an EHS until proven otherwise. Inability to obtain an internal body temperature measurement should never be a reason to delay treatment. The survival of untreated EHS drops precipitously to less than 50% after the first 30 min [33]. Thus, heat stroke is a heat emergency. No one will ever be questioned for rapidly cooling a patient with symptoms consistent with an EHI [34].

Treatment

A service member participating in strenuous hot weather activity with altered mental status and a documented elevated rectal temperature of greater than 104 °F/40 °C requires rapid cooling and evacuation. The US military follows the same treatment principles of "cool first, transport second" as used by trainers who treat heat injuries in a competitive sports environment. Cold water immersion is unquestionably the gold standard for the rapid and safe cooling of suspected heat stroke patients [35]. The CHAMP/Marine Corps Marathon (MCM) Hyperthermia Algorithm (available at https://www.usuhs.edu/sites/default/files/media/mem/pdf/mcmalgorithms2011.pdf) is an excellent protocol used by staff providing medical coverage to the annual MCM and Army Ten Miler (ATM) road races in Arlington, VA [36]. Military field training exercises, however, pose a different challenge. In a military field training exercise, the movement of military personnel can be geographically dispersed and separated by topographical obstacles. This diminishes the likelihood that a credentialed medical provider will be involved in the earliest stages of casualty care. Additionally, there are the logistical challenges of transporting and maintaining ice water immersion tubs throughout these geographically dispersed locations. These challenges, however, are mitigated with a comprehensive, risk management-based medical support plan that delegates responsibility and emphasizes routine casualty response training.

At Fort Bragg NC, an installation with the highest incidence of EHI in the military, all brigade-size training events with a moderate to high risk for EHI (e.g., land navigation, marksmanship, battery firing exercises, timed road marches, unit runs greater than 5 miles, organized training in remote areas of post) during the heat season require prior coordination with the installation military hospital emergency department. Additionally, all training events with a moderate to high risk for EHI during the heat season require strategic deployment of rapid cooling stations (RCS) throughout the training area. Each RCS is trained on the use of ice sheets and is supplied with a five-gallon drink cooler filled with a 50/50 water/ice mixture, two sheets, two towels, and a rectal thermometer. Field litter ambulances, which are strategically prepositioned to support training, are also equipped to provide rapid cooling as they transport EHI casualties to a higher level of care [37].

Treatment protocols and techniques may differ slightly between military installations. For example, aid stations at the US Army Ranger School at Fort Benning, GA wrap EHS patients in ice sheets supplemented by the use of cold water running from a hose folded into the sheets. In desert environments where ice is not available, military personnel can construct field-expedient cool water immersion baths by digging a plastic-lined pit in a shaded area. Surface evaporation and contact with the cooler subsurface sand cools the water [1]. Another field-expedient method of cooling involves the placement of a heat casualty inside a body bag filled with cold water from a shaded water tank or subsurface water pit. Whichever cooling method is used, rapid identification and treatment of a heat casualty by a well-rehearsed team whose members are well-versed in their responsibilities is important.

Rehearsals are the key to every military operation, and caring for EHI casualties is no different [38]. All of these options offer acceptable cooling rates in remote settings or during transport to definitive care. It is strongly encouraged that fixed medical facilities located in high-yield casualty training areas establish and manage their own cold water immersion cooling stations.

Return-to-Duty Considerations

Return to play (RTP)/return-to-duty (RTD) decision-making can be a complex and demanding process. Although the final decision is most commonly left in the hands of the providing physician, input is required from the athletic trainer, physical therapist, coach, and the athlete. In the military, RTD is additionally impacted by mission requirements as dictated by the Commander and the operational tempo.

The cornerstone assessment in the RTP/RTD decision requires a fundamental understanding of anatomic and functional healing. EHI RTP/RTD is especially challenging because we have an incomplete understanding of the pathophysiologic processes involved in the development of, and recovery from, this disorder [39, 40].

Despite the frequency of EHI, current civilian and military RTP/RTD guidelines are largely based on anecdotal observation and caution [40, 41]. At this time, there exist no evidence-based EHI RTP/RTD guidelines or recommendations for returning athletes or warfighters. Most guidelines are commonsense recommendations that require an asymptomatic state and normal laboratory parameters, coupled with a cautious reintroduction of activity and gradual heat acclimatization. Current suggestions for the return of EHS victims to full activity range from 7 days to 15 months [42]. This lack of consistency and clinical agreement can negatively affect athletes, soldiers, and military readiness, as medical providers can only to guess as to the best RTP/RTD solution for each individual. Additionally, whereas current guidance states that EHS casualties may return to practice and competition when they have reestablished heat tolerance, there exist no evidence-based tools to assess when the body's thermoregulatory system has returned to normal [40].

The lack of clear evidence-based guidance has allowed some physicians to clear athletes or warriors for return to participation following EHS without considering exercise heat tolerance deficits, neuropsychological impairments, altered fitness status, or acclimatization status [43–45]. RTP/RTD after EHS should involve a carefully planned, and incrementally progressive, physical challenge under close supervision. Current research indicates that most individuals will eventually recover fully from EHS; indeed, this occurs in the vast majority of cases when the athlete is treated promptly with aggressive cooling strategies (i.e., ice-water immersion) [39, 43, 46]. Although definitive, evidence-based RTP/RTD guidelines do not presently exist, the current recommendations are summarized in the following sections.

Current Civilian Recommendations

In the authors' opinion, the consensus RTP guidelines set forth by the ACSM are clear, succinct, and provide a rational process for guiding athletes who have experienced an exertional heat illness. Current recommendations from the ACSM for returning an athlete to training and competition are as follows: [42]

1. Refrain from exercise for at least 7 days following release from medical care.
2. Follow up about 1 week post-incident for a physical examination and lab testing or diagnostic imaging of the affected organs, based on the clinical course of the EHS incident.
3. When cleared for return to activity, begin exercise in a cool environment and gradually increase the duration, intensity, and heat exposure over 2 weeks to demonstrate heat tolerance and to initiate acclimatization.
4. If return to vigorous activity is not accomplished over 4 weeks, a laboratory exercise-heat tolerance test should be considered.
5. Clear the athlete for full competition if heat tolerant between 2 and 4 weeks of full training.

Current Military Recommendations

Army physicians are guided by medical fitness standards as outlined in Army Regulation (AR) 40-501, while Navy physicians who care for Sailors and Marines are guided by the Secretary of the Navy Instruction (SECNAVINST) 1850.4E. The military services do not share consensus recommendations on returning warfighters to duty after experiencing an EHI [41]. In fact, the individual service recommendations are more diverse and varied than those in the civilian sector. An ACSM roundtable was convened at the Uniformed Services University of the Health Sciences (Bethesda, MD) on October 22 and 23, 2008, to address this issue of variability with both military and civilian experts. Specifically, the conference sought to (1) discuss the issue of returning victims (athletes and soldiers) of EHI to either play or duty and (2) develop consensus-based recommendations. The conference convened over 20 recognized EHI experts from both civilian and Department of Defense sports medicine communities. The conference comprised seven 1-h EHI topic blocks, including definitions and basic epidemiology; pathophysiology; recognition and treatment; the role of thermal tolerance testing in recovery and return to play/duty; the role of genetic and biomarkers in recovery and return to play/duty; prevention of an ensuing incident of EHS; and current civilian and military guidelines for return to play/duty [32]. Conference agenda and presentations are available at http://www.usuhs.mil/mem/champ.html; a publication is pending detailing these proceedings. The conference results were used as a foundation to develop new guidance that is presently used by the AMEDD [47], and are further elaborated upon at http://champ.usuhs.mil/chclinicaltools.html (Table 16.5).

Table 16.5 Progression recommendations for the soldier with heat stroke (HS), or heat exhaustion (HE), heat injury (HI), pending Medical Evaluation Board (MEB)[a] [47]

Restrictions[b]	HS without complication	HS with complications	Complex HS or HE/HI pending MEB
Complete duty restrictions	2 weeks	2 weeks minimum; advance when clinically resolved	2 weeks minimum; advance when clinically resolved
Physical training and running/walking/swimming/bicycling at own pace and distance not to exceed 60 min per day No maximal effort; no fitness testing; no body armor; no chemical/biological training; no road marching	1 month minimum	2 months minimum	Pending MEB
Gradual acclimatization No maximal effort fitness training; no fitness testing; no chem/bio training. May road march at own pace/distance with no more than 30 lbs	1 month minimum	2 months minimum[c]	N/A
Continue gradual acclimatization May participate in unit fitness training; chem/bio training up to 30 min; road march at own pace/distance with no more than 30 lbs up to 2 h	N/A	Pending completion of 30-day heat exposure requirement, if not accomplished during prior profile[c]	N/A

Adapted from U.S. Army, Heat Illness MEB and Profile Policy. Washington, DC: Headquarters, U.S. Army Medical Command; 2009. Policy Memo 09-039

[a]Consider expert consultation (contact champ@usuhs.mil for military cases)

[b]Soldiers manifesting no heat illness symptoms or work intolerance after completion of profile restrictions can advance and return-to-duty without an MEB. Any evidence of heat illness symptoms during the period of the profile requires an MEB referral

[c]HS with Sequelae return to full duty requires a minimum period of heat exposure during environmental stress (Heat Category 2 during the majority of included days)

Emerging Concepts in Return to Play

As evidenced by the lack of consistency in RTP/RTD guidelines in both military and civilian communities, much work remains to be accomplished in this critically important area. Increasingly, however, a consistent theme has been recognized as these important clinical decisions are made; each EHS event is unique in its etiology and management, and therefore requires a highly individualized approach to RTP/RTD. Research is currently underway to further to define criteria for the return of thermotolerance, develop tools that may assist in return to play in difficult cases, and evaluate those who may be at risk for future events.

Summary

EHI has a significant impact on the medical readiness of the United States military. The military places a strong focus on heat injury prevention. Military treatment protocols focus on the early identification and rapid cooling of suspected EHS. Prior planning ensures that rapid cooling capabilities are available in the field environment. In order to assist military providers in the management of EHI cases, the Army has standardized its medical profiling for EHI by establishing recommendations for follow-up, RTD, and referral indications for medical board evaluation.

Case Example *A 30-year-old male Army officer collapsed approximately 200 m from the finish line of a 12-mile road march. He was a resident physician in training attending the fall season Expert Field Medical Badge (EFMB) course. The officer had a body mass index of 29 kg/m^2, had no significant medical issues, and was taking no medicines or supplements. One month prior to the start of the 10-day course, he abruptly increased his exercise from jogging 6 miles per week to road marching 18–20 miles per week. On the morning of the EFMB road march, the participants were awakened early in the morning after 3 h of sleep to begin the event. Each participant carried a 45-pound ruck sack with two 1-quart canteens filled with water. At the time of the event, the WBGT was 64 °F/17.8 °C. The officer collapsed at approximately the 2 h and 53 min mark (event standard is 3 h). An ambulance was dispatched from the finish line. No cooling system was on board the ambulance. The patient was immediately transported to the on-base medical center where rectal temperature was 107 °F/41.7 °C at approximately 20 min post collapse. While in the emergency room, he became unconscious and was intubated. He was exposed and placed in ice sheets. After 50 min he was cooled to a core temperature 102 °F/38.9 °C, approximately 70 min post collapse. He was given a 1 liter bolus of normal saline, admitted to the intensive care unit (ICU), and placed on a ventilator. Blood urea nitrogen and serum creatinine revealed a mild prerenal azotemia on admission. Liver transaminases spiked to four times their normal value on day 2. Serum creatine phosphokinase peaked at the upper level of normal on day 2. The patient remained in a coma for 10 days. He was kept in the ICU for 22 days and discharged directly from the ICU. The officer began limited exercise approximately 60 days post-incident due to difficulty returning to exercise. He graduated from residency 6 months off cycle and was able to complete his 4-year active duty service obligation as an Army family physician.*

Table 16.6 Ten steps for successful implementation

1. Establish a comprehensive exertional heat injury (EHI) prevention policy with command support
2. Ensure personnel undergo annual training to educate them on awareness, prevention, early diagnosis, and treatment of EHI
3. Ensure personnel train regularly as first responders and rapid cooling station team members (if used, should be familiar with operation of indwelling rectal thermistor)
4. Empower first responders to make early decisions to initiate rapid cooling (patient exposure, application of ice sheets, cold water bath immersion)
5. Begin acclimatization at least 1 month prior to traveling to a warm weather climate for deployment or elite military training school
6. Conduct risk management matrix for each training event to determine likelihood of EHI occurrence (e.g., low risk, moderate risk, high risk)
7. For events with moderate- to high risk for EHI, ensure rapid cooling stations are pre-deployed at event training site and/or along physical training routes
8. For large-unit training events (i.e., 1500 or more personnel) with moderate- to high risk for EHI, coordinate with local hospital emergency department in advance
9. Ensure evacuation platforms (e.g., field litter ambulance, MEDEVAC helicopter) are coordinated in advance and equipped to conduct rapid cooling in transport
10. Standardized EHI recovery profiles and return-to-duty recommendations help ensure uniform standard of care across the military healthcare system

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Part V
Individual Issues

Chapter 17
Exertional Heat Illnesses

Rebecca M. Lopez and John F. Jardine

Introduction to Exertional Heat Illnesses

Aside from maximizing performance in the heat, it is important to be aware of medical conditions that may occur as a result of exercising in a hot and/or humid environment. The negative effects of exercising in hot conditions date back to ancient times, when "sunstroke" was to blame in farmers, military men, and sport [1, 2]. Prevention from heat illness and the use of cold water to treat heat stroke can be traced back to thousands of years ago [1, 2]. Despite the history behind this, exertional heat illnesses still occur fairly often, and we have ways to go in terms of the prevention, recognition, and treatment of exertional heat illness. Exertional heat illnesses may occur due to dehydration, electrolyte depletion, heat strain, fatigue, or an accumulation of heat storage due to an inability to adequately dissipate body heat. There are also various extrinsic and intrinsic risk factors that may predispose an athlete to an exertional heat illness [3, 4] (Fig. 17.1). For instance, intrinsic risk factors may include characteristics of the athlete, such as level of physical fitness and/or heat acclimatization, body composition, or recent illness, such as a fever or stomach upset. Extrinsic factors are more organizational factors, such as improper work to rest ratios, lack of access to fluids during exercise, or exercising during the warmest part of the day.

It is imperative for athletes and coaches to be aware of the potential risk factors for exertional heat illness and take the necessary steps for prevention. Although the causes of the different exertional heat illnesses may vary, general prevention

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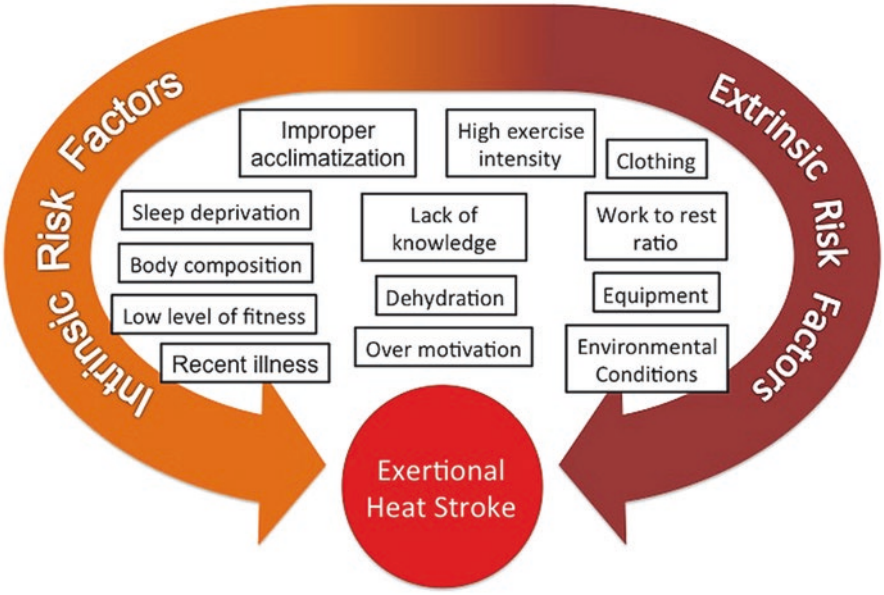

Fig. 17.1 Extrinsic and intrinsic risk factors of exertional heat illness (Figure courtesy Douglas J. Casa, PhD and Miwako Suzuki, KSI Summer Fellow; Korey Stringer Institute)

strategies include: a gradual progression to exercise in the heat to ensure adequate heat acclimatization, exercising during the coolest times of the day, ensuring proper hydration before, during and after exercise, and modifying activity (i.e., shorter duration, decreased intensity, no equipment) when the environmental conditions are harsh [1]. In particular, exercising when there is a high ambient temperature together with a high relative humidity poses the greatest risk. High relative humidity results in a decreased ability to evaporate sweat from the skin's surface, which is the most effective way of body heat dissipation.

In this chapter, we will discuss the different exertional heat illnesses and provide the ways in which they can be prevented, recognized, and treated. Guidelines for when an athlete can safely return to activity following an exertional heat illness will also be discussed. In order to understand the medical concerns associated with exercising in the heat, this chapter will also include the physiology and the latest research on exertional heat illnesses and provide real world examples of exertional heat illnesses to allow the reader to clinically apply the information presented.

The Physiology

Body temperature is dependent on the balance between heat production from metabolism and heat loss (or gain) to the surroundings. Exercising in the heat causes a net heat gain from metabolism and the environment. Increasing body temperature,

or hyperthermia, may lead to a gamut of heat-specific illnesses. Heat illness is a spectrum of diseases with seemingly different definitions for different populgations. To standardize all disease definitions, the World Health Organization (WHO) has published a classification system for diseases known as the International Classification of Diseases (ICD). The most recent version, ICD-10, medically classifies ten heat disorders. Four of these disorders (heat cramps, heat syncope, heat exhaustion, and heat stroke) will be discussed here as medical diagnoses (Table 17.1). These four are the most common disorders seen in athletes and other physically active populations (laborers, firefighters, soldiers).

Heat Cramps

Though given an ICD code as a medical diagnosis, this term is a misnomer, as heat does not directly trigger muscle cramping. It is the physiologic response to heat exposure that ultimately leads to muscle cramping. As such, a more appropriate term of exercise-associated muscle cramping (EAMC) has been proposed [3, 5]. EAMCs were always thought to be prompted by fatigue, excessive sweating, and a significant sweat-induced whole-body sodium deficit [6]. Recent experimental and observational data suggest that cramping is multifactorial and the neuromuscular system is involved in its mechanism [7]. The cause of EAMCs may also vary from person to person, and there is still more to learn regarding the causes of EAMCs. In general, EAMC is clinically recognized as debilitating muscle cramps at the large muscle groups in the limbs or abdominal wall after high intensity exercise. Treatment starts with resting in a cool environment. It should also involve hydration with attention to replacing sodium losses with sports drinks or other sources of sodium (e.g., bouillon cubes), without drinking to excess. Oral hydration should be attempted first, with intravenous (IV) hydration reserved for intractable cramping or in individuals who are vomiting. Stretching and massage of the muscle is the most effective way to reduce discomfort. Protracted cramping should prompt assessment of serum sodium levels to assess for hyponatremia. EAMC predictors include a prior history of EAMC, faster race times, and previous muscle injury or damage [8]. Individuals that are "salty sweaters" (i.e., salt marks on shirt or cap) may be predisposed to EAMC if sweat sodium losses are not replaced with fluid and/or diet. Prevention of EAMC should start with attention to these risk factors. It should also involve matching daily salt and fluid intake with expected losses during intense exercise or activity. Knowledgeable athletes may predict their losses based on previous experiences. Additional sodium may be added to the diet in anticipation of expected losses, especially in athletes with a previous history of cramping. As always, conditioning for the specific stresses of the activity and heat acclimatization practices may help prevent muscle cramps. Cramp-prone athletes should keep a cramp log to document events that preceded EAMC [7]. Identified trends can be targeted when considering return to play. Training and conditioning in the preseason should also be examined. It is not uncommon for athletes to experience EAMC

Table 17.1 Exertional heat illnesses: prevention, recognition, treatment and return to play

Exertional heat illness	Prevention	Recognition	Treatment	Return to play
Exercise associate muscle cramps (heat cramps)	• Heat acclimatization • Exercise acclimatization • Adequate hydration and nutrition	• Involuntary muscle contraction, often affecting the lower leg musculature	• Ice, massage, and light stretching • Remove from exercise until EAMC resolves	• Once EAMC resolved, as tolerable • Determine the cause of EAMC (i.e., muscle fatigue versus sodium deficit) • Supplement with salty snacks (pretzels, chips) if caused by sodium depletion
Heat syncope	• Heat acclimatization • Exercise acclimatization • Adequate hydration and nutrition • Avoid sudden stoppage of exercise	• Sudden collapse/ fainting during exercise in the heat	• Rule out a more serious condition (i.e., cardiac arrest, EHS) • Elevate legs, remove from hot environment • Hydrate once conscious	• Determine the cause of heat syncope and make modifications as needed (i.e., heat acclimatization, proper hydration) • Can RTP as tolerable as long as other, more serious conditions have been ruled out
Heat exhaustion	• Heat acclimatization • Exercise acclimatization • Adequate hydration and nutrition	• Dizziness, nausea, pale/ faint, vomiting, headache • Elevated body temperature due to exercise but not >105 °F/40.6 °C	• Remove excessive equipment/ clothing • Move to cooler area • Replace fluids if possible • Keep out of activity same day	• Determine cause of heat exhaustion and make modifications as needed (i.e., adequate hydration and electrolytes in diet, heat acclimatization) • Gradual return to exercise

(continued)

Table 17.1 (continued)

Exertional heat illness	Prevention	Recognition	Treatment	Return to play
Exertional heat stroke	• Heat acclimatization • Adequate work to rest ratios • Modify exercise intensity and/or duration in extreme weather conditions • Adequate hydration, sleep, and nutrition • Avoid exercising when ill (i.e., fever, infection, gastrointestinal illness)	• Body temperature >105 °F/40.6 °C • Altered mental status (i.e., confusion, irritability, unconscious) • Collapse, vomiting, dizziness	• Aggressive cooling on-site via cold-water immersion or other effective cooling technique (i.e., dousing with cold water, rotating cold towels) • Remove from cold tub once temperature reaches • 102 °F/38.9 °C • Transport to hospital emergency department	• Needs physician clearance • Normal blood work • Determine cause of EHS and make modifications (i.e., heat acclimatization, proper hydration, appropriate exercise intensity and rest breaks) • Gradual RTP exercise progression while being monitored for recurring signs/symptoms

Abbreviations: *EAMC* exercise-associated muscle cramp, *RTP* return to play, *EHS* exertional heat stroke

during the first competitive game. Therefore, conditioning sessions and team practices should gradually increase in time and intensity leading up to the competitive season to prevent the EAMC often seen during the first couple of games of the season.

Heat Syncope

Exercise-associated collapse (EAC) is a condition that results from a postural hypotensive event. It typically occurs when an athlete finishes an event and the "pumping" action of the exercising muscles ceases, causing decreased venous return to the heart. Heat syncope is a collapse or fainting episode similar to EAC that may occur in heat-unacclimatized persons who stand for a prolonged time in the heat, or suddenly stand after prolonged sitting in the heat. In the heat, the body is taxed to provide blood to exercising muscle as well as to the periphery for thermoregulation. Coupled with the physiologic vasodilatation of peripheral vessels to disperse heat, the body experiences a relative hypovolemic state causing hypotension and collapse. Signs and symptoms may include lightheadedness, tunnel or darkening vision, and pale, sweaty skin. Initial treatment for any patient with sudden collapse is attention to airway, breathing, and circulation. The differential diagnosis of

syncope is vast; other causes, specifically cardiac dysrhythmias, must be considered. One such cardiac anomaly to be ruled out is Brugada Syndrome. Characterized by specific electrocardiogram (ECG) abnormalities, it is associated with an increased risk of sudden cardiac death. The cause of Brugada Syndrome is linked to an abnormal sodium channel [9], which is known to be sensitive to temperature changes. There are reports of heat-illness induced Brugada Syndrome [10]. Therefore, an ECG should be performed for all patients who experience multiple episodes of syncope. Treatment starts with moving the patient to a shaded or cooler area. The patient should be laid supine with the legs elevated above the level of the heart. Fluids can be given orally. The patient should avoid sudden standing until fully recovered. The patient will likely only require supportive care. If not recovered after 20 min, other causes of syncope should be considered, and the patient should be evaluated in the emergency room. Prevention comes with heat acclimatization, which will eventually increase the plasma volume to accommodate the competing needs of blood supply to both the periphery and working muscles. Athletes prone to EAC should avoid prolonged standing in the same position in the heat or standing suddenly from rest (especially after recovering from exercise in the heat).

Heat Exhaustion

Heat exhaustion is the inability to continue exercise or physical activity in the heat due to inadequate cardiac output [3]. It usually occurs in the setting of dehydration though this need not be present for the diagnosis. Heat exhaustion is the most common heat-related illness observed in physically active populations. The signs and symptoms of heat exhaustion are nonspecific. They include dizziness, fatigue, lightheadedness, headache, abdominal cramps, nausea, vomiting, diarrhea, and myalgias. The patient's skin is typically pale and cool with profuse sweating. Those afflicted will present to care with a complaint of difficulty continuing their activity in the heat. Clinically, their internal body temperature is <105 °F/40.6 °C with no significant central nervous system (CNS) dysfunction [3, 11]. Treatment of heat exhaustion first involves removing the patient from the heat to a shaded or cool (i.e., air-conditioned) area. Excess clothing and/or equipment should be removed. The patient should be placed in a supine position with their legs elevated above the level of their heart. The patient with heat exhaustion may benefit from cooling though the mode of cooling is less important as it is for patient comfort and not life-saving as in exertional heat stroke (EHS). A tub may be used for cold-water immersion although dousing with cold water or evaporative modalities may be sufficient. As these patients are often dehydrated, rehydrating the patient may be helpful. If not nauseous or vomiting, oral hydration with chilled water or sports drinks is sufficient. IV fluids should be reserved for those unable to take fluids orally. Patients who completely recover with these treatments may be discharged from the treatment area. Those who fail to improve after 1–2 h of appropriate treatment should be transported to an emergency department for additional observation and testing.

Patients treated for heat exhaustion should not return to exercise on the same day. Instead, there should be a gradual return to activity and modifiable risk factors should be addressed during the progression.

Exertional Heat Stroke

Of the four diagnoses discussed in this chapter, heat stroke is most serious as it is a life-threatening emergency and may cause serious morbidity or mortality if not promptly treated. Heat stroke in physically active individuals is more appropriately termed EHS to differentiate it from classic non-exertional heat stroke that occurs in individuals at the extremes of age, usually in the setting of an environmental heat wave. Though both may be disabling or fatal if not recognized and treated appropriately, only EHS will be discussed here. EHS is defined by an internal body temperature above 105 °F/40.6 °C and CNS dysfunction [3]. CNS dysfunction may manifest as an altered mental status, irritability, emotional liability, unsteady gait, seizures, or coma. It is important for coaches to be aware of the altered cognitive status an athlete with EHS may present with and be prepared to prevent further injury. An EHS patient's behavior may vary from being unconscious to acting erratically and aggressively [12]. The sooner the cooling process begins, the better the prognosis. Morbidity and mortality in EHS occur as a result of multi-organ system injury or failure associated with an elevated internal body temperature.

The pathogenesis of EHS is an inability of the body to compensate for heat stress. The heat load incurred by exercising in the heat must be dissipated to maintain a constant body temperature. In addition, increased cardiac output during exercise shunts blood from the viscera to the periphery, leading to intestinal ischemia and resultant hyperpermeability. This allows for leakage of endotoxins from the intestines into the blood stream. This endotoxemia may then cause hemodynamic instability and possibly death [13]. Concurrently, the body responds to the heat stress via a cytokine-mediated inflammatory response to protect against tissue injury and to promote tissue repair. Following heat stress, cellular protection during the acute phase response is mediated by initiating the production of stress proteins, such as heat shock proteins [13]. Endotoxemia may also exaggerate the acute inflammatory response leading to an increased production of cytokines and nitric oxide, which can interfere with thermoregulation and precipitate hyperthermia, hypotension, and heat stroke [14]. Renal dysfunction/failure, liver dysfunction/failure, and coagulopathy develop secondary to hyperthermia and circulatory collapse [13]. This multi-organ system failure makes EHS ultimately life threatening.

The key to the successful treatment of EHS is simple: to lower the body temperature of the patient and minimize the duration of hyperthermia as rapidly as possible. The length of time the internal body temperature is above the critical threshold (105 °F/40.6 °C) dictates morbidity and the risk of death from EHS [3] (Fig. 17.2). The goal in these patients is to lower the core body temperature to <102–103 °F/38.9–39.4 °C within 30 min of collapse [16]. Current evidence suggests that cold water

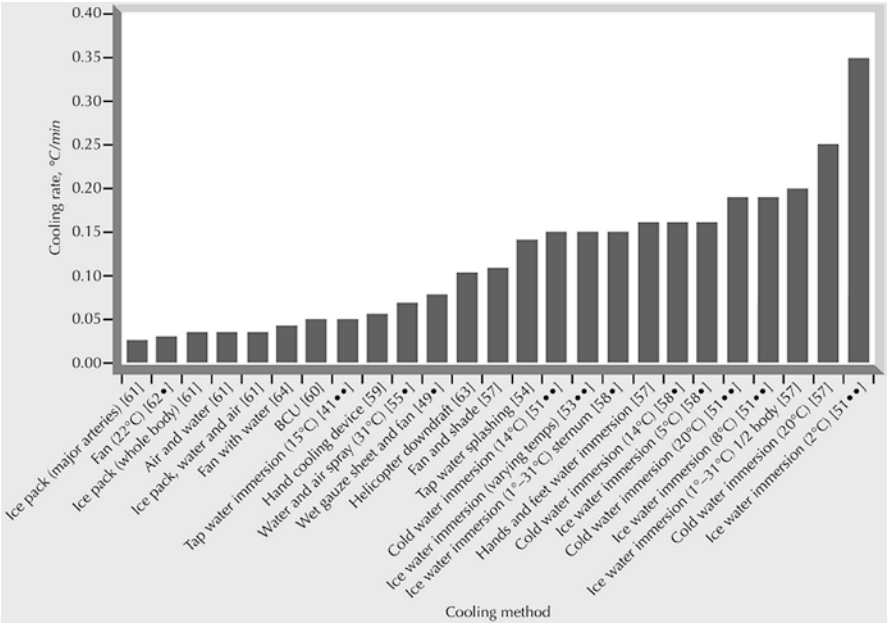

Fig. 17.2 Cooling rates of various cooling modalities. Figure depicts experiments with healthy hyperthermic athletes and heatstroke victims [15]. References in the figure are associated with the original article. Reprinted with permission from Casa DJ, Armstrong LE, Ganio MS, Yeargin SW. Exertional heat stroke in competitive athletes, Current Sports Medicine Reports, Vol. 4/ Number 6, pages 309–317, http://journals.lww.com/acsm-csmr/Fulltext/2005/12000/Exertional_Heat_Stroke_in_Competitive_Athletes.6.aspx, © 2005, with permission from Wolters Kluwer Health, Inc.

has the best cooling capacity to achieve rapid treatment [3]. Cooling rates associated with various cooling modalities have been extensively evaluated [17] (Fig. 17.3). Cold-water immersion (CWI) has cooling rates superior to any of the other studied modalities and is considered the gold standard for treatment of EHS [17]. This modality includes immersion of the patient in very cold (35–50 °F/1.7–10 °C) water, covering as much of the body surface area as possible with constant and aggressive stirring of the water to keep bringing cold water to the skin surface and an iced or wet towel wrapped around the top of the head [17]. Use of a 50-gallon stock tank or wading pool makes this an inexpensive and practical method of cooling. Guidelines for implementing CWI for a patient with EHS have been published in a National Athletic Trainers' Association position statement [3] (Table 17.2). In certain situations, the use of an immersion tub is not feasible or practical. In such circumstance, the use of cold showers, tarp-assisted cooling, dousing patients with cold water, or rotating cold water-soaked towels should be considered. With an aggressive cooling modality, it is imperative to remove the patient from cooling when their body temperature reaches 102 °F/38.9 °C in order to ensure hyperthermia is decreased but to also avoid hypothermic overshoot.

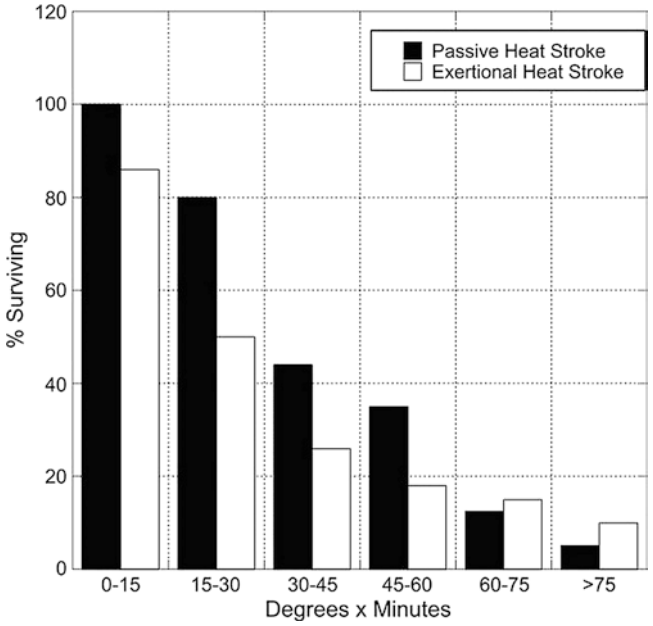

Fig. 17.3 Relationship between severity of hyperthermia (passive versus exertional heat stroke) and rat survivability [18]. Reprinted with permission from Casa DJ, Kenny G, Taylor N; Immersion Treatment for Exertional Hyperthermia: Cold or Temperate Water?, Medicine and Science in Sports and Exercise, Vol. 42/No. 7, pgs 1246–1252, http://journals.lww.com/acsm-msse/Citation/2010/07000/Immersion_Treatment_for_Exertional_Hyperthermia_.3.aspx, © 2010, with permission from Wolters Kluwer Health, Inc.

Hyponatremia

Although hyponatremia is not considered to be an exertional heat illness, it is included in this chapter because of the similarities in some of the signs and symptoms between hyponatremia and heat illness. It is also imperative to educate athletes about hyponatremia as it is a potentially fatal condition. Exercise-associated hyponatremia (EAH) is defined by blood sodium concentrations below 135 mmol/L^{-1} [19]. These lowered sodium levels can occur during or up to 24 h after exercise and often result from excessive sweat sodium losses, overdrinking of water, or a combination of both [19]. Some of the signs and symptoms of EAH may include lightheadedness, dizziness, nausea, vomiting, and headache [19]. Severe EAH can result in altered mental status (such as confusion or disorientation) as well as coma, so it is important to differentiate between EAH and a heat illness such as heat exhaustion or EHS, as the treatment is vastly different. As severe EAH can be life threatening, treatment involves immediate transport to an emergency department.

Table 17.2 Guidelines for implementing cold water immersion for a patient with exertional heat stroke [3]

1. Initial response. Once exertional heat stroke is suspected, prepare to cool the patient and contact emergency medical services
2. Prepare for ice-water immersion. On the playing field or in close proximity, half-fill a stock tank or wading pool with water and ice (make sure there is sufficient water source) (a) The tub can be filled with ice and water before the event begins (or have the tub half-filled with water and keep three to four coolers of ice next to the tub; this prevents having to keep the tub cold throughout the day). (b) Ice should cover the surface of the water at all times (c) If the athlete collapses near the athletic training room, a whirlpool tub or cold shower may be used
3. Determine vital signs. Immediately before immersing the patient, obtain vital signs (a) Assess core body temperature with a rectal thermistor (b) Check airway, breathing, pulse, and blood pressure (c) Access the level of central nervous system dysfunction
4. Begin ice-water immersion. Place the patient in the ice-water immersion tub. Medical staff, teammates/coaches, and volunteers may be needed to assist with entry to and exit from the tub.
5. Total-body coverage. Cover as much of the body as possible with ice water while cooling (a) If full-body coverage is not possible due to the tub size, cover the torso as much as possible (b) To keep the patient's head and neck from going under water, an assistant may hold him or her under the axillae with a towel or sheet wrapped across the chest and under the arms (c) Place an ice/wet towel over the head and neck while body is being cooled in the tub (d) Use a water temperature under 15 °C/60 °F
6. Vigorously circulate the water. During cooling, water should be continuously circulated or stirred to enhance the water-to-skin temperature gradient, which optimizes cooling. Have an assistant stir the water during cooling
7. Continue medical assessment. Vital signs should be monitored at regular intervals
8. Fluid administration. If a qualified medical professional is available, an intravenous fluid line can be placed for hydration and support of cardiovascular function
9. Cooling duration. Continue cooling until the patient's rectal temperature lowers to 38.9 °C/102 °F (a) If rectal temperature cannot be measured and cold-water immersion is indicated, cool for 10–15 min and then transport to a medical facility. A rectal temperature should be obtained as soon as possible in order to track progress (b) An approximate estimate of cooling via cold-water immersion is 1 °C for every 5 min and 1 °F for every 3 min (if the water is aggressively stirred). For example, someone in the tub for 15 min would cool approximately 3 °C or 5 °F during that time
10. Patient transfer. Remove the patient from the immersion tub only after rectal temperature reaches 38.9 °C/102 °F and then transfer to the nearest medical facility via emergency medical services as quickly as possible

Reprinted with permission from Casa DJ, DeMartini JK, Bergeron MF, Csillan D, Eichner ER, Lopez RM, et al. National Athletic Trainers' Association Position Statement: Exertional Heat Illnesses, Journal of Athletic Training, Vol. 50/No. 9, pgs 986–1000, © 2015

The Research

Exercise scientists have been conducting research on exertional heat illnesses for years. This research has led to the current information and the guidelines that clinicians often refer to when looking to optimize performance in the heat.

The exertional heat illness that seems the most difficult to figure out is EAMCs. The exact cause of EAMCs has not been clearly identified. Furthermore, researchers tend to disagree on the likely causes. In the literature, there are two schools of thought: (1) those who believe EAMCs are caused by muscle fatigue, and (2) those that believe EAMCs are caused by electrolyte depletion during exercise [20].

In the muscle fatigue and overload theory, it is believed that an EAMC is caused by a sustained abnormal spinal reflex activity occurring as a result of muscle fatigue [20, 21]. The muscle fatigue is believed to increase excitatory muscle spindle activity and decrease the inhibitory golgi tendon organs within the muscle. As a result, a fatigue athlete would experience this involuntary muscle cramping during exercise.

The other theory behind EAMCs is that a loss of electrolytes due to either excessive sweating or excessive loss of electrolytes in sweat causes the involuntary cramping [20, 22]. In particular, the role of sodium in regulating fluids between cells and compartments within the body is believed to play a factor. Some athletes are believed to be "salty sweaters" in that they lose excessive sodium in their sweat, which may predispose them to cramping during exercise in the heat [22]. The environment in which the muscle cramping occurs can also play a role. For instance, some believe if the environment is mild and an athlete is experiencing EAMC, it is likely due to muscle fatigue. Conversely, if it is hot and humid and an athlete is exercising for an extended period of time (i.e., a tennis tournament), then the EAMCs are likely due to sodium depletion [22].

There have been some randomized controlled trials that have attempted to replicate the muscle cramping seen with exercising individuals; however, the mechanism by which the muscle cramps are induced in the laboratory is different than what occurs on the field. As a result, the research on EAMCs is still conflicting and lacking concrete evidence.

EHS has probably been studied the most of the exertional heat illnesses, particularly in recent years. Although many of the EHS deaths have occurred in younger athletes [23], some of the recent epidemiological data suggests a low incidence of EHS in youth and high school sports [24–26]. The discrepancy between the EHS deaths and the rate of EHS could be due to under-reporting of EHS in the youth and high school setting.

Much of the research on EHS has focused on the prevention, recognition, and immediate treatment of EHS [17, 27–33]. More recently, some of the research has shifted to recovery and return to exercise following EHS [34–39]. As a result, various organizations such as the American College of Sports Medicine (ACSM) and the NATA have created position statements and consensus statements with guidelines for the prevention, recognition, treatment, and return to activity following EHS [3, 40–44].

Regarding prevention of EHS, it is known that EHS is multifactorial [3, 33]. There are various risk factors that may predispose an athlete to EHS that were identified through various case series of actual EHS victims, where the predisposing factors or events that led to EHS were documented [33, 45, 46]. Some of these factors can be specific to the individual, such as lack of heat acclimatization, low physical fitness, or body composition, while others can be organizational or external risk factors, such as when coaches demand athletes to exercise at a higher intensity than they are accustomed to, exercising at the hottest times of the day, lack of access to fluids [3, 33].

The recognition and diagnosis of EHS relies on an accurate method of body temperature assessment. Several studies have found that the only method to accurately measure internal body temperature among exercising individuals on-field is rectal thermometry [27, 29]. Only medical professionals (i.e., physician, athletic trainer, emergency medical technician) should be allowed to obtain a rectal temperature measurement in cases of a collapsed athlete suspected of having EHS. In the presence of both an elevated rectal temperature and CNS dysfunction, the exercising individual should be cooled aggressively via CWI [3, 31, 41, 47–49]. Once the EHS victim is cooled to about 102–103 °F/38.9–39.4 °C, he or she should be transported to the nearest hospital for further examination.

Once the athlete is released from the hospital, it is important to ensure full recovery prior to returning to exercising in the heat. The recovery and return to activity following EHS is the area of the research that is lacking the most. General recommendations suggest waiting between 7 and 21 days before returning the athlete to exercise [3]. The athlete should be cleared by a physician and exhibit normal blood laboratory results prior to returning to exercise [3, 39, 44]. It is also imperative to determine if the athlete has become heat intolerant as a result of the EHS. This can be determined by monitoring the athletes (i.e., heart rate, internal body temperature) during a gradual, progressive return to exercise [38]. Although the existing return to activity guidelines have been very generalized, recent reports of individual cases [34, 37] have highlighted the importance of an individualized return to exercise process specific to the athlete's needs, the extent of the EHS suffered, and the presence of any lingering signs or symptoms. It is also imperative that the exercise progression closely mimics the specific type, intensity, and duration of the exercise the athlete is seeking to return to.

The Application

Prevention

The research related to exertional heat illnesses can easily be applied to the industrial, military, and athletic settings. Whether this information is utilized for an exercising individual or applied by a coach for an entire team, proper precautions can

help prevent or decrease the risk of heat illnesses. One of the most effective methods of preparing athletes for exercising in the heat is heat acclimatization. This process of getting the body accustomed to exercising in a hot environment allows an exercising individual to physiologically adapt to this stressor. Heat acclimatization consists of increasing the duration and intensity of exercise in the heat across days. Complete acclimatization to a hot and/or humid environment often takes between 10 and 14 days. Guidelines [42] for proper heat acclimatization have been in place for some time now; however, some high school associations have been slow to make the policy changes (http://ksi.uconn.edu/high-school-state-policies/heat-acclimatization-policies/).

Other organizational factors can aid in the prevention of exertional heat illnesses. Coaches can take simple steps, such as setting practice times for the coolest part of the day, modifying the amount of equipment worn for practices, and having access to cold fluids during exercise in the heat. Educating athletes on the importance of taking care of their bodies will also help with the prevention of exertional heat illnesses. Emphasis should be placed on ensuring athletes are getting adequate sleep, eating a nutritious diet, and drinking sufficient water throughout the day (not just during exercise).

Weighing athletes before and after practices can provide information about hydration habits and fluid needs. It is generally accepted that athletes will lose some weight during exercise due to sweat losses; however, athletes should prevent losing more than 2% of their body weight in order to prevent decrements in performance and an exertional heat illnesses. If an athlete gains weight during exercise, he or she is consuming too much fluid during exercise, which can also be dangerous. Therefore, body weights before and after exercise help determine whether they consumed enough or too much fluids during exercise, informing the athlete how much fluid needs to be replaced before the next practice to optimize recovery (i.e., drink 16 ounces of water or sports drink for every 1 pound of body weight lost during exercise).

Recognition and Immediate Treatment of Exertional Heat Illnesses

The recognition and immediate treatment of exertional heat illnesses is best left to healthcare professionals (i.e., athletic trainers, physician assistants, physicians). However, oftentimes the coaches are the first ones on the scene when an exertional heat illness occurs. Teammates should also be aware of the signs and symptoms of heat illness so that they can alert the coach or medical staff if one of their teammates is struggling. Determining the severity of the heat illness (i.e., ruling out EHS) is of utmost importance, as EHS is a potentially fatal medical emergency. Teams should have a venue-specific emergency action plan in place, so that everyone knows what to do in case of a heat emergency. Having cold tubs on the field or easily accessible

should be standard procedures for any practice or event occurring in a hot, humid environment. In cases where a cold tub is unavailable, an alternative cooling method (e.g., locker room shower, tarp-assisted cooling modality, or ice cold towels) should be prepared ahead of time. When healthcare professionals are on-site, a valid temperature device (i.e., rectal thermometer) should be available to accurately diagnose an EHS.

Case Example *Cold-water immersion has been used exclusively at the Falmouth Road Race. The Falmouth Road Race (FRR) is a 7-mile race held annually on the third Sunday of August in Falmouth, Massachusetts. Because of the favorable environmental conditions and relatively short distance, runners sustain a high intensity pacing throughout the race. There are more than 12,000 runners ranging from elite to novice status. An overall incidence of 2.13 ± 1.62 EHS cases per 1000 finishers has been reported* [49] *making Falmouth a unique opportunity to observe large-scale treatment of EHS. CWI has been used for the treatment of EHS since the inception of the race in 1973. In 2011, 274 cases of EHS over an 18-year period of the FRR were reviewed. Despite the high occurrence of EHS at this event, no deaths have been reported* [49]. *There have been an additional 111 cases of EHS with no deaths reported (unpublished data). The medical coverage for the race includes three course medical tents with a larger finish line medical tent; all equipped with CWI tubs to assure a victim is treated within minutes. Tubs are pre-filled with water and ice to maintain the water temperature as cold as possible. Due to the success of CWI, > 90% of patients treated in the medical tents are discharged home from the racecourse by physicians overseeing their care. Of those transported to the hospital, none required admission and were discharged from the emergency department. A study of fatal episodes of EHS among military personnel revealed that lack of medical triage was a common factor in all fatalities* [33]. *The success of treating EHS at the FRR is due to the rapid recognition and treatment of EHS. Furthermore, medical volunteers are trained to recognize the signs and symptoms of heat illness prior to the day of the race, and they use rectal thermometry to obtain an accurate assessment of internal body temperature. Medical coverage along the course also allows for expedient transport to a treatment area within minutes. Pre-filled tubs allow for immediate on-site cooling in keeping with the "cool first, transport second" guideline. The FRR provides an example of optimal treatment for patients with EHS.*

Summary

In conclusion, athletes exercising in the heat should be aware of the potential impact the environment can have on their exercise performance and safety. Adequate training and knowing the steps to take to prevent an exertional heat illness is the key to safely and effectively perform in a hot, humid environment. Prevention of heat illness should include a gradual increase in the exercise duration and intensity across

7–14 days to ensure proper heat acclimatization. Knowing individualized fluid needs will help ensure adequate hydration and a balanced diet with sufficient electrolytes to prevent muscle cramping and heat exhaustion. Lastly, organization factors such as exercising during the cooler parts of the day, modifying the amount of equipment worn or the exercise intensity on days of extreme temperature and humidity, and ensuring adequate rest breaks can help prevent EHS. If an exertional heat illness does occur, it is essential to have medical staff on-site for prompt recognition and treatment, particularly for EHS which is a life-threatening condition. Once the exertional heat illness resolves, it is imperative to determine the cause(s) and make any necessary modifications in order to confirm a safe return to play.

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Chapter 18
Other Medical Conditions of Concern During Hot Weather Exercise

Nathaniel Nye, Rebecca L. Stearns, and Francis Tran

Introduction

Humans, like other homeothermic species, rely on various physiologic mechanisms to maintain a relatively constant body temperature. The ability to thermoregulate allows life-sustaining cellular biochemistry to proceed optimally despite fluctuating physical workloads and environmental conditions. In fact, thanks to our unsurpassed ability to thermoregulate and dissipate heat, a well-trained and acclimatized human can outrun any land animal, even horses and hyenas, over long distances in warm climates [1]. However, certain medical conditions (or medications used to treat them) can compromise these adaptations, leading to heat intolerance (see Tables 18.1 and 18.2).

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Table 18.1 Overview of medical conditions increasing risk of exertional heat illness [2–4]

Type of condition	Examples
Disorders of skin and sweating	Sunburn, large burn scars, psoriasis, miliaria rubra, cystic fibrosis, ectodermal dysplasia, chronic anhidrosis, therapeutic X-ray radiation, Type 1 diabetes mellitus [4]
Metabolic/ thermoregulatory disorders	Malignant hyperthermia, insomnia (sleep deprivation), febrile conditions (common cold, streptococcal pharyngitis, mononucleosis, etc.), hyperthyroidism
Cardiovascular disorders	Heart failure, cardiomyopathy, myocarditis, hypertension (especially due to medications used to treat hypertension)
Functional factors	Low physical fitness, lack of acclimatization, fatigue
Multifactorial	Obesity, dehydration, advanced age, previous heat stroke (depends on what factors contributed to previous episode)
Unknown mechanism	Sickle cell trait

The Physiology

Effective thermoregulation, or heat tolerance, depends upon the ability to achieve a balance between heat gain and heat loss. Heat may be gained either endogenously, as a by-product of metabolism, or from the external environment. Metabolic heat production is a function of the basal metabolic rate plus the work load. Approximately 80% of all energy released through substrate metabolism in our cells is lost as heat, while only 20% is converted to effective processes such as muscular contraction [3]. In other words, for every five calories burned, one calorie becomes useful energy, while the other four are converted to heat and must be dumped into the environment in order to maintain homeostasis.

Normal Physiologic Mechanisms for Cooling

In order to capitalize on these modes of heat transfer for cooling, the body must make several physiologic changes when exercising, especially in the heat. Most evidence supports the theory that these thermoregulatory processes are governed centrally in the brain, specifically in centers within the hypothalamus [3, 6]. Temperature-sensitive neurons in the skin and core transmit signals to the hypothalamus, which "compares" these inputs against a programmed temperature set point (approximately 37 °C/98.6 °F), and then drives appropriate physiologic adjustments to maintain the body temperature within a narrow range (approximately 36–40 °C/96.8–104 °F).

When an individual begins to exercise, metabolic heat begins to accumulate, and within seconds to minutes, cardiovascular and cutaneous responses begin to increase heat transfer into the environment. Cardiac output increases, hastening the flow of blood carrying heat away from the core. Blood flow to the gastrointestinal tract becomes less important (i.e., digestion is of low priority during exercise, as muscles

Table 18.2 Medications increasing risk for heat illness [2, 3, 5]

Medication class	Examples	Used to treat	Proposed mechanism
Antihistamines	Diphenhydramine (Benadryl®), Loratadine (Claritin®)	Seasonal allergies, insomnia	Impaired sweating (anticholinergic side effect)
Anticholinergics	Meclizine (Antivert®), Tolterodine (Detrol®), Dicyclomine (Bentyl®)	Vertigo, bladder spasm, irritable bowel syndrome	Impaired sweating
Phenothiazines	Thioridazine, Chlorpromazine, Promethazine (Phenergan®), Prochlorperazine	Schizophrenia, psychotic disorders, nausea/vomiting	Disrupted hypothalamic function, impaired sweating (anticholinergic side effect)
Mood stabilizer	Lithium	Bipolar disorder	
Anticonvulsants	Lamotrigine (Lamictal®), Topiramate (Topamax®), Gabapentin (Neurontin®), Acetazolamide	Epilepsy, migraine headache prophylaxis, altitude sickness, bipolar disorder, neuropathic pain	
Tricyclic antidepressants	Amitriptyline (Elavil®), Nortriptyline (Pamelor®)	Depression, neuropathic pain, migraine headache prophylaxis, post-concussion syndrome, insomnia	Increased heat production, possibly altered central thermoregulation
Beta blockers and calcium-channel blockers	Metoprolol (Lopressor®), Atenolol, Propranolol	Hypertension, post-MI mortality reduction, tachyarrhythmias	Impaired compensatory increase in cardiac output
Diuretics	Hydrochlorothiazide (HCTZ), Furosemide (Lasix®)	Hypertension, heart failure	Dehydration and depletion of electrolytes (e.g., sodium, potassium)
Decongestants	Pseudoephedrine (Sudafed®), Ephedrine	Common cold, cough, nasal congestion	Increased heart rate and blood pressure
Stimulants	Caffeine, ephedra, pre-workout stimulants, methylphenidate (Ritalin®), cocaine, ecstasy, methamphetamine	Performance enhancement, ADHD, narcolepsy, eating disorders	Increased heart rate and blood pressure, increased activity, impaired sweating
Alcohol	Beer, wine, liquor	Socially or as anxiolytic	Diuresis, impaired vasomotor reflexes

burn stored glycogen and fat) and is restricted, a process called splanchnic vasoconstriction. This shunts blood toward the skin, where blood vessels become dilated (cutaneous vasodilation) in an effort to promote heat transfer. Concurrently, sweat production increases to achieve greater evaporative heat loss. Finally, the increase in

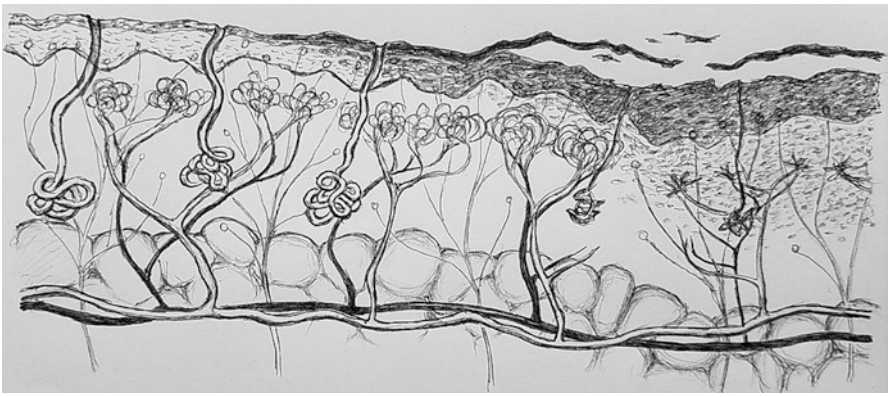

Fig. 18.1 Healthy skin contains many functioning capillary beds and eccrine sweat glands. When the skin is burned, these dermal structures are damaged and function is impaired

respiratory rate supports the greater metabolic demand for oxygenation and ventilation, while providing an additional benefit of increased heat loss through exhalation of hot, humid air.

Under many circumstances, the body is able to successfully leverage these cooling mechanisms to compensate for increased metabolic heat production. However, until physical exertion ceases, the body temperature will generally "plateau" at a somewhat elevated but safe level (38–40 °C/100.4–104 °F). This is termed compensable heat stress. Yet it is not hard to imagine how, especially with prolonged or high levels of exertion, these cooling mechanisms can be overwhelmed, even when functioning normally. When this occurs, fatigue usually takes over and protects against exertional heat illness [6]. Yet, when prolonged exertion occurs in the setting of existing medical conditions (or medications) that impair these cooling mechanisms or override the protective fatigue response, it can be a recipe for disaster.

The Evidence

Disorders of Skin and Sweating

The skin is the largest organ in our body and serves as the interface between the complexities of the internal and external environments. By sensing and adapting to environmental conditions, skin protects the body and greatly contributes to homeostasis. When the internal body temperature exceeds the hypothalamic set point, a sympathetic reflex triggers generalized vasodilation and secretion of fluid via eccrine sweat glands. When there is a disturbance in either the control of sweating or in the actual glands themselves, hyperthermia may ensue. The inability to sweat is termed anhidrosis and can originate from direct skin injury and scarring,

inflammatory skin disorders, neurologic disorders, genetics, medications, or may be idiopathic (as in chronic idiopathic anhidrosis) [7].

Trauma and burns variably impair vasodilatory and sweat gland functions depending on severity of the injury (see Fig.18.1). Burns may result in mild cellular injury and edema, moderate injury with blistering, or complete destruction of the skin and necrosis of the sweat glands. Even mild sunburns cause decreased ability to sweat. Scarred or grafted skin is fibrotic and lacks functional sweat glands. When extensive areas are affected by traumatic injury, burns, scarring, or skin grafting, it can significantly hinder thermoregulation [8, 9].

Inflammatory skin disorders disrupt skin function and impair heat exchange. Psoriasis is a chronic skin disease with many subtypes, but typically produces red plaques with silvery scale on the knees, elbows, scalp, and/or trunk. It is relatively common, affecting approximately 2–3% of the general population and often manifests in the third decade of life [8, 9]. Atopic dermatitis (eczema) is also common, and if not well controlled, may affect large surface areas. Skin with any active inflammatory rash is dysfunctional in terms of heat exchange.

Disorders of the central and peripheral nervous systems may disrupt the vasomotor and sudomotor reflexes which mediate heat exchange. The most important of these conditions is diabetes, which results in peripheral neuropathy and thus impairs vasomotor and sudomotor control. Research has shown compromised heat dissipation in type 1 diabetics due to decreased sweat rate during exercise [10]. Central nervous system diseases are rare in physically active populations, but disorders such as Parkinson's disease, progressive supranuclear palsy, multiple sclerosis, and stroke (cerebrovascular accident) can cause localized or generalized anhidrosis [8, 9].

Genetic disorders such as ectodermal dysplasia and cystic fibrosis (CF) can also affect the sweat glands and thermoregulation. Ectodermal dysplasia is a rare genetic disorder that causes variable disturbance in the production of hair, nails, teeth, or sweat glands [10]. CF is an autosomal recessive disease that affects transmembrane ion conduction. Individuals with CF have thick, viscous mucus (impairing respiration/ventilation) and cannot regulate the amount of sodium lost in sweat. In high temperatures or increased physical activity, increased sweating causes large quantities of sodium to be lost, potentially leading to hyponatremia (manifesting as confusion, seizure, and/or coma), especially when combined with over-hydration with hypotonic fluid [11].

Disorders of Metabolism and Central Thermoregulation

Akin to an automobile engine being revved up for increased power output, the metabolic "engine" of the body responds to multiple different physiologic controls, or "gas pedals." These include a basal metabolic rate, volitional exercise, the inflammatory response to infection, and endocrine function (e.g., adrenaline, thyroid hormone, cortisol). And just as an automobile engine at full throttle will soon overheat

and shut down, disorders of thermoregulation and metabolism may lead to injury or even death, especially in the setting of exercise.

At first glance, it is simple reasoning that individuals with a current fever, such as with a viral respiratory infection, are at increased risk for heat illness with exertion. However, the underlying physiology is actually quite complex. In short, fever results from an increase in cytokines such as IL-1α and TNF-α, which raise the hypothalamic temperature set point. The basal metabolic rate increases in response, creating hyperthermia even without exercise. For those with current febrile illness, the additional stressors of physical exertion in high ambient temperatures may further elevate the body temperature, raising risk for exertional heat illness. It is important to note that infection/inflammation not only contribute to but result from hyperthermia. The multi-organ failure resulting from exertional heat stroke (EHS) has been suggested to be the result of a systemic inflammatory response syndrome [3, 12], which may be triggered by endotoxin leaking into the circulation when splanchnic vasoconstriction during prolonged exercise causes hypoxic bowel injury.

A unique and important physiologic "gas pedal" is seen in individuals with malignant hyperthermia susceptibility (MHS). Malignant hyperthermia (MH) is a severe, life-threatening hypermetabolic reaction to volatile anesthetic agents and succinylcholine. It is caused by mutations in the gene for the calcium channels responsible for muscle contractions, such as ryanodine receptor-1 (RYR1) [13]. When a person carrying the gene mutation is exposed to anesthetic agents, an uncontrolled cycle of generalized muscle contraction occurs, resulting in muscle rigidity, rhabdomyolysis, and extreme hyperthermia, often resulting in death if not treated promptly using dantrolene. The clinical and physiological similarities between MH and EHS have long been recognized, raising the question of whether individuals with MHS are also at increased risk for EHS. A retrospective study from the French Armed Forces found genetic evidence of MHS in 45% of EHS victims, and multiple case reports of EHS in individuals with MHS have been published in the past two decades [13]. In order to prove a causal link, however, MHS would have to be demonstrated before the onset of heat stroke. Unfortunately, this type of human research is extremely difficult to accomplish, and though these two conditions are associated, a direct causal link between MHS and EHS remains elusive. Recently, an expert panel highlighted the increasing number of case reports of an MH-like syndrome which shares many features of EHS, MH, and exertional rhabdomyolysis (ER), but is unique [14]. It presents much like MH with core temperature >40.5 °C/104.9 °F and muscle rigidity, but is triggered by exertion rather than inhalational anesthetics, occurs in individuals with MHS, and responds well to treatment with dantrolene. Unfortunately, it is usually diagnosed retrospectively after being treated presumptively as EHS.

The thyroid gland is widely recognized for its importance in driving metabolic rate, but an overactive thyroid (hyperthyroidism) is relatively rare. Hyperthyroidism is characterized by an increase in the thyroid hormones triiodothyronine (T3) and

thyroxine (T4) and greatly affects thermoregulation [15]. These thyroid hormones may push the internal body temperature above the hypothalamic set point by increasing the basal metabolic rate, oxygen consumption, and general adrenal function. Furthermore, elevated T4 levels are associated with decreased production of heat shock proteins. As a result, those afflicted are relatively intolerant to warm temperatures, even at rest.

Cardiovascular Disorders

The demands on the heart are greatly increased when moderate to intense exercise is combined with heat stress. Environmental heat exposure increases the inotropic status (rate and strength of cardiac contraction) to compensate for decreased central venous return. For those with existing cardiovascular disease (CVD), the increased circulatory demands from exercise and heat stress may worsen cardiac function, resulting in heat intolerance or cardiac decompensation [16].

In the era of modern medicine, people are living longer despite being "sicker" thanks to advanced medical care and surgical procedures. Concomitantly, running and endurance sports are increasing in popularity among all segments of the population, including senior citizens. Indeed, aerobic exercise can be very beneficial for those with heart disease and is often prescribed by physicians. There are many "master's" runners on today's roads and trails who may have varying degrees of heart disease, from well-controlled hypertension to ischemic cardiomyopathy and heart failure. Without a doubt, it is critical to understand the role of cardiac disorders in risk for exertional heat illness.

In 2014, an important study reported the relative prevalence of serious cardiac events versus serious events related to EHS during all endurance races in Tel Aviv, Israel between 2007 and 2013 [17]. Out of 137,580 runners participating in the races, there were two serious cardiac events (one myocardial infarction, one supraventicular tachyarrhythmia), neither of which was life threatening. On the other hand, there were 21 serious cases of EHS, 2 of which were fatal and 12 life threatening. At first pass, this seems to indicate that heart disease may be a relatively minor risk factor for adverse outcomes in endurance athletes. However, there is a chicken-and-egg factor to consider. It was not known how many of the participants had underlying CVD, and it is possible that some of those who suffered EHS may have been among those with CVD. In other words, though EHS had been labeled as the cause of death or adverse event, underlying CVD may have been an important precipitating factor. Even though most serious health events at endurance races are not ostensibly cardiac in nature, further research needs to be done in this area, as it appears that CVD is an important risk factor for exertional heat illness [17, 18].

Interplay Between Risk Factors

As medicine continues to improve and people continue to live longer, a person's age becomes more and more important as a factor in heat intolerance. Studies of persons aged 60 years and older have shown an increased risk of exertional heat illness due to multiple factors. Increasing age is associated with decreased sweat production. Furthermore, aging brings a decrease in skin blood flow due to impairments in the sympathetic nervous system signaling, vascular smooth muscle hypertrophy, and thinning of the dermis leading to a decrease in capillary density [19]. And perhaps most importantly, the ability to increase cardiac output in response to exercise is impaired, in part due to decreased maximum heart rate and increased ventricular wall stiffness. These age-related changes are worsened in the presence of chronic diseases such as atherosclerosis, hypertension, heart failure, pulmonary diseases, kidney disease, and obesity. Taken together, age-related changes may interfere mildly to critically with the body's ability to counter a rising internal body temperature, with or without physical activity, in hot environments [20].

Adding to the many other known health risks of obesity, it also increases the risk of exertional heat illness. Obesity may increase the incidence of fatal EHS by as much three and a half times. The contributing mechanisms include a decreased density of sweat glands (i.e., number of sweat glands does not increase as skin is stretched), and a decreased ratio of surface area to body mass [21, 22]. Convective and radiant heat transfer are also impaired by the depth created by the adipose tissue between heat-generating muscle and the skin surface. These impairments are compounded when other chronic medical conditions are present. With respect to obese individuals, risk can be mitigated by weight loss, as well as by optimizing other factors such as hydration, sleep, acclimatization, and proper cycles of exercise and rest.

Functional Disorders

Modifiable or functional disorders include those within one's control and represent opportunities to improve heat tolerance. A couple of these factors are low physical fitness and lack of heat acclimatization. Low physical fitness is an important and common factor among EHS victims [23, 24]. In the laboratory, VO_2max has shown a stronger direct correlation with heat tolerance than any other known biometric measure [23]. Therefore, all individuals entering a scenario of increased risk for exertional heat illness (e.g., those entering preseason in the fall or spring sports) should be physically prepared to handle the predicted workload in a cool environment prior to exposure to a warm environment.

When exposed to changing environmental and exercise conditions, the healthy human body makes various physiologic adaptations, collectively termed heat acclimatization, over the course of about 10–14 days. This results in improved physiologic responses to heat stress and reduced risk for exertional heat illness. Many documented EHS deaths have occurred during the first 2 weeks of sports

participation in a warm environment [25]. The NCAA, the National Athletic Trainers' Association (NATA), and the NFL all have policies regarding preseason heat acclimatization practices [26]. The most compelling result of these policies is that since the 2003 NCAA guidelines were first implemented, there have only been two deaths from EHS during the preseason period [27]. Prior to this, the NCAA was averaging about one death every year, suggesting that this policy has saved approximately 15–30 athletes' lives [28]. Similar success has been demonstrated within the high school model since its implementation.

Mechanism Unclear

Sickle cell trait (SCT) raises the risk of exertional illness (e.g., ER, exertional collapse) and, though the mechanism remains unclear, merits particular discussion. SCT is common, affecting 8% of African Americans, and has been shown to carry an increased relative risk (RR = 37) of sudden death among NCAA football players [29] and in military recruits (RR = 27.6) [30]. A 2012 study [31] evaluating 31 years of data from the United States Sudden Death in Athletes Registry found that 87% of SCT deaths in athletes occurred when the ambient temperature exceeded 80 °F/26.7 °C, and 74% occurred in southern or border states during summer or autumn. It is important to note that SCT is not a single entity, but there are many genetic variants of SCT, and there are likely other genes which, if inherited along with SCT, may increase the risk of exertional illness [32]. Not all individuals with SCT are at the same level of risk, but science has not yet progressed enough to allow further risk stratification.

The cause of death in athletes and military recruits with SCT remains controversial, but likely involves microvascular occlusion (either caused by sickling, resulting in sickling, or both), followed by extensive rupture of muscle and red blood cells causing hyperkalemic death [33]. These cases generally present as a non-sudden conscious collapse, followed by a fulminant illness which often has features of ER, EHS, or both [33, 34]. However, neither ER nor EHS is universally present in SCT-related collapse. While all of these etiologies do overlap, these events are termed exertional collapse associated with sickle cell trait (ECAST). Because of data showing increased relative risk of death, all NCAA athletes and most US military recruits are required to be screened for SCT. Notably, the US Army does not require SCT screening for its recruits, rather adhering to a policy of following exertional injury precautions in all recruits and soldiers. A recent, landmark study [32] found no increased risk of death in soldiers with known SCT compared to those without the trait, when all were subject to exertional injury precautions.

Athletes and recruits with SCT should be counseled that sudden death is very rare in both SCT-negative and SCT-positive athletes, but there is a possible increased risk of death with SCT compared to those without SCT. They should also be counseled on the importance of maintaining adequate hydration and avoiding extremely intense, repetitive exercise, especially when the individual is not acclimatized to the heat or exercise. By doing so, they can minimize risk of death.

The Application

Role of the Pre-participation Physical Exam

The pre-participation exam (PPE) is the first opportunity to address any health-related risk factors prior to heat exposure or exercise. It is imperative to consider predisposing factors for exertional heat illness and address them prior to any issues arising during physical exertion where medical care may or may not be imminently available. A standardized PPE form has been endorsed and published by the American Academy of Pediatrics, American Academy of Family Physicians, American College of Sports Medicine, American Medical Society for Sports Medicine, and other societies [35]. The PPE form includes a review of current and past medical conditions and current medications and supplements, which may be helpful in identifying predisposing risk factors for EHS. However, this form includes only one question on exertional heat illness: "Have you ever become ill while exercising in the heat?" With any history of exertional heat stroke, it is imperative to gain a full history of the treatment that was provided, hospitalization details, laboratory test results, and any return to activity challenges/timeline. The NATA recommends that questions regarding heat acclimatization be included in the PPE [36]. After the PPE is completed, it is also imperative to educate the individual regarding modifiable risk factors, such as appropriate fitness, heat acclimatization, hydration, and modifying exercise sessions in hot environments.

Practical Risk-Reduction Strategies with Respect to Medical Conditions

Exertional heat illness risk reduction for specific medical conditions will vary based on the type of activity and setting. While for all scenarios risk reduction will be surrounded by first identifying individuals at higher risk, educating them on mitigating options, and then implementing these prevention strategies, the resources and ability to implement these strategies vary greatly (see Fig. 18.2).

Mass Participation Endurance Races

Mass participation endurance races probably represent the most challenging circumstances to identify pre-event medical conditions that could increase exertional heat illness risk. This is compounded by the lack of mandatory pre-event medical evaluation or demonstration of previous successful completion of a similar event. Mass participation events typically involve a wide range of individuals from professionals to students, younger and older populations, highly trained athletes and weekend warriors, who all may present with a wide variety and degree of risk factors. As many as 19% of race participants continue to compete despite the presence

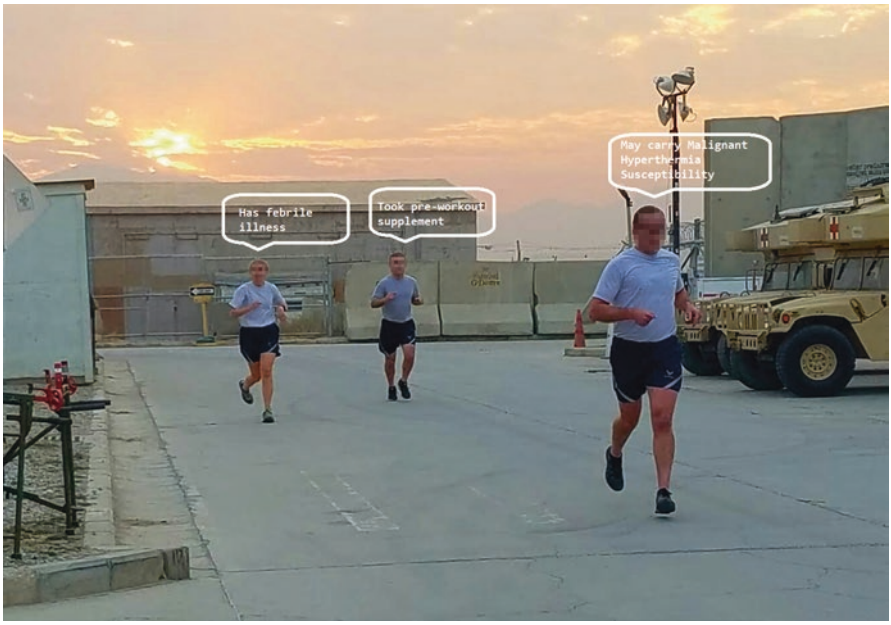

Fig. 18.2 There is potential for high-risk conditions in any athlete, and vigilance is required

of an infectious disease [37]. Recently, a pre-race survey was used to identify acute pre-race illnesses and selectively educate those with positive answers on how to reduce their risk for serious medical events. Race organizers saw a dramatic decrease in the number of registered runners who did not start the race after the education, as well as overall lower incidence of runners who did not finish the race. These effects were probably not solely due the pre-race survey and education; however, it demonstrates a potentially effective method to raise participants' awareness about their risk of exertional illness [37].

It is critical for athletes to understand the value of a pre-race PPE, and for healthcare providers to be well versed on the PPE in order to prevent race-day medical tent admissions. Event medical directors and staff should consider pre-race screening and targeted education prior to competitive events to reduce the incidence of serious and catastrophic injuries.

Team Sports

Team sports are generally more closely monitored than mass participation endurance sports, and therefore many risk-reduction strategies are more practical. When coaches or athletic directors require a comprehensive PPE, many at-risk individuals are identified and educated regarding the best strategies to reduce their risk of a heat-related illness. For the majority of athletes, in whom the PPE reveals no red flags, it is then the responsibility of the sponsoring institution (e.g., high school,

college, league) to provide appropriate and adequate medical care to their athletes. This will ensure that not only the immediate care is available, but prevention strategies are also in place to reduce the overall risk. Many prevention strategies are available and readily implemented in team settings, which include heat acclimatization, adjustment of work-to-rest ratios, proper hydration strategies, body cooling, and environmental monitoring using the wet-bulb globe temperature with appropriate exercise modification. Communication between coaches and medical staff is critical whenever athletes with known increased risk of exertional heat illness are participating.

Military

Similar to team sports, the military has a great degree of control when it comes to pre-participation screening. In addition, specific roles and assignments within the military may greatly influence the risk profile of an individual. However, with deployment (especially in combat) or in some training scenarios, there is potential for lack of access to immediate medical care or the inability to modify physical demands/external conditions. Screening, therefore, becomes more critical and likely more conservative than other examples discussed here. This being said, many of the same prevention or preparation strategies can be used in the military, which include but are not limited to: education on heat-related illnesses, heat acclimatization, improvement in physical fitness, hydration, and body cooling strategies. Of great interest in the biotechnology realm is the ability to remotely monitor individuals' physiologic status in real-time, thereby allowing cessation of exercise prior to any onset of a heat-related illness. While this technology is still evolving, it presents an incredible advancement in the ability to send military men and women into the field but also halt them before placing themselves or their unit at further risk.

Common Misconceptions

There are a myriad of misconceptions regarding medical conditions and their influence on thermoregulations and heat tolerance. A few of the most common misconceptions and truths are discussed in Table 18.3.

Case Example *A 30-year-old male collapsed at the finish line upon completing a 10-km community road race. The ambient temperature was 70 °F/21.1 °C with 64% humidity. He immediately went into convulsions, and a medical team responded. He was completely unresponsive, tachycardic (171 bpm) and hyperthermic, with a rectal temperature of 40.2 °C/104.4 °F. He was covered with bags of ice, intubated, and administered intravenous (IV) fluids within 20 min of collapse. He was immediately taken to a nearby hospital and admitted to the intensive care unit, where he continued to be cooled and received aggressive IV hydration. Laboratory evaluation*

Table 18.3 Common misconceptions about exertional heat stroke [24, 28, 38–40]

Misconception	Truth
Only the unfit succumb to EHS	There are a great number of fit athletes that experience EHS, largely believed to be due to their ability to sustain high exercise intensity for a prolonged period of time, therefore producing a large amount of metabolic heat [24]
Taking fever-reducing medications will help to treat EHS	Cold water immersion is the gold standard for treatment of EHS. Because the mechanism of heat production during EHS is very different from that of a febrile illness, fever-reducing medications will not have a significant impact on reducing body temperature [28]
A previous EHS episode(s) will invariably place an individual at higher risk for another EHS	Outside of other confounding medical conditions (e.g., malignant hyperthermia susceptibility), if an individual is treated aggressively and immediately, long-term complications are rare and many EHS victims return to their previous activities without further difficulty [38–40]
Risk factors for classic heat stroke (CHS) and EHS are the same	Classic and exertional heat stroke are often discussed collectively or interchangeably, resulting in misconceptions regarding EHS. The most basic difference is the populations usually affected; CHS affecting young children and the elderly, and EHS affecting adolescents and active adults. Young children and the elderly commonly have developmental factors or medical conditions that predispose them to CHS but not EHS, such as limited decision-making skills (e.g., dementia), and limited independent mobility. Infants and the elderly may also have an incomplete or weakened thermoregulatory system, but this will raise risk for both CHS and EHS

showed hyperkalemia (K = 6.4 mmol/L), severe coagulopathy, and moderate acute injury to the liver and kidneys. With supportive care, his laboratory values normalized and he was able to be discharged home in stable condition 5 days after his collapse.

Eight weeks later, the athlete had full resolution of symptoms and his lab values had normalized. He performed a maximal exercise challenge test based upon an incremental cycling protocol at room temperature, and the results showed moderate exercise intolerance (lactate threshold reached early at 75W, only 29% of his age-predicted level), but rectal temperature remained normal during exercise. Further history and physical exam revealed no abnormality to explain his initial collapse or persistent exercise intolerance. He reported no significant past medical history (in particular, no personal or family history of malignant hyperthermia). The athlete was also a semi-professional rugby player with no prior difficulty with exercise. He was subsequently found on genetic testing to have an RYR1 mutation, known to be associated with malignant hyperthermia. He was counseled on prevention of exertional heat illness and course of action in the event of symptom occurrence and was allowed to gradually return to sport [13].

Though MHS is relatively common (approximately 1 in 3000 [41]*), athletes and soldiers are usually unaware that they carry the genetic mutation. Exertional heat illness in these individuals may occur with lower workloads (intensity or duration) or lower environmental heat stress than would generally be expected. This case*

illustrates that these individuals may even succumb to exertional heat illness in conditions they had previously tolerated without problem. While it is generally impractical to modify outdoor activities based solely on the possibility of a participant with MHS, it is essential for coaches, leaders, and healthcare providers to be aware that MHS exists in the community and to keep exertional heat illness on the differential diagnosis whenever CNS dysfunction occurs in the setting of exertion.

Table 18.4 Ten steps to successful implementation [34, 35, 37, 38, 40]

1. Pre-participation physical examination—identify and educate high-risk individuals; consider online pre-race screening and education in mass participation events [35, 37]
2. Ensure good communication between coaches/leaders and medical staff
3. Educate athletes regarding risks of using supplements and medications
4. Allow for 10–14 days of heat acclimatization before increasing physical exercise in the heat
5. Develop and implement appropriate hydration strategies, with special attention to those with medical conditions which contribute to heat intolerance
6. Ensure rest is allowed at appropriate intervals; use decreased intervals between rest periods for those with medical conditions described in this chapter
7. Develop and implement plans for monitoring environmental conditions (e.g., wet-bulb globe temperature), including appropriate exercise modifications based on measurements
8. Consider using remote physiologic status monitoring technology
9. In cases of previous exertional heat stroke, consider heat tolerance testing after blood markers return to normal levels
10. Once heat tolerance is confirmed, follow a gradual return to sport under guidance of multidisciplinary medical team, if no contraindications found [34, 38, 40]

Disclaimer The opinions contained herein are those of the authors and do not represent the position or policy of the United States Air Force or the Department of Defense.

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Chapter 19
Drugs and Supplements

Rachel K. Katch, Michael S. Smith, Michele LaBotz, Luke N. Belval, and Yuri Hosokawa

Introduction

Many drugs and supplements are marketed to exercising individuals as having potentially ergogenic, or performance-enhancing, properties. In recent years, research indicates that younger athletes and exercising individuals are increasingly experimenting with these substances to both improve appearance and trying to improve their overall exercise performance [1]. This chapter will examine the physiology and research behind some of the most common drugs and supplements seen in sport to examine their potential for enhancing, or hindering, exercise performance in the heat.

Drugs and Supplements with Fluid Balance Mechanisms

Since hypohydration has a profound impact on exercise performance and health, maintenance of euhydration is paramount to optimizing athletic outcomes [2]. There are many drugs and supplements in today's market that can alter the body's fluid

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balance, potentially enhancing or decreasing exercise performance. This segment will focus on drugs and supplements that exert changes on the body's fluid balance during exercise.

Enhance Performance in the Heat

The Physiology

One mechanism that has been established to mitigate the impact of hypohydration on exercise performance is the idea of hyperhydration, or fluid loading prior to activity, to diminish the absolute fluid losses from sweat. However, excess fluid consumption alone results in increased diuresis, and prolonged hyperhydration is difficult to maintain. The use of glycerol, sodium, potassium, and creatine monohydrate (creatine) has been suggested by researchers and clinicians as potential mechanisms for achieving hyperhydration.

Glycerol is a three-carbon alcohol that naturally appears as the backbone to fatty acids [3]. When taken in isolation, glycerol distributes evenly across the body's fluid compartments and exerts an osmotic pressure. Specifically, glycerol is filtered and reabsorbed rapidly at the kidney, while being excreted fairly slowly [4]. Concurrently, water is reabsorbed in the nephron facilitating an increase in total body water. This increase in total body water has been shown to last for several hours and is of a greater magnitude than water-, or sodium-induced hyperhydration.

An additional strategy that has been used to combat hypohydration during exercise in the heat, especially with prolonged duration, is to rehydrate with carbohydrate (CHO) and electrolyte containing fluid (Table 19.1) [5]. One way to replace the nutrients lost is through the use of sports drinks, which typically contain 6–8% CHOs along with small amounts of sodium and potassium [5]. Carbohydrate stores are relatively limited and can be acutely manipulated on a daily basis by dietary intake or even exercise, and provide a key fuel for the brain and muscular work, especially over longer duration activities [5]. Sodium is important for maintaining fluid balance in the body during exercise in the heat. It is the most common electrolyte in circulating blood and in sweat, and significant sodium losses can occur with exercise in the heat, potentially leading to hyponatremia if blood sodium levels fall below 135 mmol/L [5]. Sodium content of sweat varies between 20 and 70 mEq/L (460–1610 mg/L), and is dependent upon genetics, heat acclimatization (decreases sodium content in sweat), and sweat rate (high sweat rates increase sweat sodium content) [5]. Sweat rates during exercise in the heat often range from 1.0–1.5 L/h in adolescents and adults, but have been documented up to 3 L/h in American football players [5]. Preadolescents 9–12 years old have lower sweat rates of 300–700 mL/h [5]. Additionally, the difference between intracellular and extracellular potassium concentrations (150 mEq/L as compared to 3.5–5 mEq/L, respectively) is critical for maintaining cell function while exercising in the heat [5]. Potassium loss in sweat is much lower than that of sodium with 4–7 mEq (156–274 mg) of potassium

Table 19.1 Hypothetical supplemental sodium and potassium recommendations for various duration sports [2, 6]

Sport	Duration	Average sweat rate ($L \cdot h^{-1}$)	Average sweat electrolyte losses ($mEq \cdot L^{-1}$)
Basketball	~40 min	1.6	Sodium: 37.52
			Potassium: 5.36
American football	~60 min	2.14	Sodium: 74.9
			Potassium: 10.7
Soccer	~90 min	1.46	Sodium: 76.65
			Potassium: 10.95
Tennis	Best of three sets	1.6	Sodium: 56.0–168.0[a]
			Potassium: 8.0–24[a]
Half-marathon[b]	13.1 miles	1.49	Sodium: 104.3
			Potassium: 14.9
Full ironman triathlon[b]	140.6 miles	Bike: 0.81 Run: 1.02	Bike sodium: 182.01 Bike potassium: 26.00 Run sodium: 174.93 Run potassium: 24.99

Sweat rates ($L \cdot h^{-1}$) based off average male sweat rates during summer practice/competition; average sweat sodium concentration ($35\ mEq \cdot L^{-1}$); average sweat potassium concentration ($5\ mEq \cdot L^{-1}$). *Recommendation values presented are generalized. For individual recommendations, specific sweat rates, sodium losses, and potassium losses should be calculated for the duration of exercise.*

[a]1-h vs. 3-h

[b]Sodium and potassium concentration losses based on average finishing times

lost per liter of sweat; however, it is important for cell function to be able to replenish potassium as well as sodium during exercise [5].

Creatine is one of the most widely used and studied dietary supplements for improving anaerobic performance and increasing lean body mass [7, 8]. However, there has been ongoing debate in the literature regarding the effect of creatine on performance in the heat. Creatine ingestion results in an increased fluid volume within the skeletal muscle cells; however, no evidence has been found to support the concept that this supplementation hinders the body's ability to thermoregulate or negatively effects fluid balance [9]. In fact, due to the increase in total body water during exercise, especially in the heat, it is speculated that creatine could mitigate dehydration, lowering internal body temperature and reducing heart rate [10].

The Evidence

Glycerol-induced hyperhydration has been examined in a variety of endurance event settings. Coutts et al. found that hyperhydration lessened the performance decrements that occurred between warm and hot environments when glycerol was consumed prior to an Olympic distance triathlon [11]. Similarly, Kavouras et al. observed increased plasma volume and greater exercise time to exhaustion using 1 g/kg of body mass of glycerol supplementation with water before exercise [12].

Wingo and colleagues investigated the effects of glycerol-induced hyperhydration on mountain bike performance and observed no physiological performance improvement in comparison to hydration alone [13]. On the contrary, Goulet et al. found that glycerol-induced hyperhydration improved cycling peak power output and endurance capacity even when only 1/3 of fluid losses were replaced [14].

Researchers have also observed an attenuated rise in heart rate and rectal temperature during exercise testing in the heat following 7 days of glycerol and creatine (another osmolyte that maintains cell volume and fluid balance) supplementation [15]. Goulet et al. performed a meta-analysis of studies that examined endurance performance following glycerol supplementation [4]. They found that glycerol-induced hyperhydration increased fluid retention by 50% and increased endurance performance by 2.62 ± 1.6% in comparison to water-induced hyperhydration [4]. However, it should be noted that only four studies were included in the analysis. Another interesting application of glycerol is its potential for increasing the efficiency of post-exercise rehydration. Van Rosendal et al. investigated intravenous (IV) fluid administration with glycerol supplementation following 4% dehydration in comparison to IV administration alone [16]. While the group that received glycerol had the greatest restoration of plasma volume, there were no performance differences observed in a subsequent 40 km cycling time trial [16].

In regard to the use of CHO and electrolytes, multiple studies have demonstrated the benefit of CHO ingestion during activity when that activity lasts longer than 60 min [17]. Carbohydrate concentrations above 4% appear to slow gastric emptying [17], which may delay fluid and CHO absorption, and increase risk of stomach upset with exercise. Water, CHO, and sodium absorption is enhanced when sports drinks contain higher CHO concentrations (up to 10% glucose), glucose and fructose in combination, or when the beverage has relatively low osmolality (<270 mOsm/kg) [17]. Some studies have demonstrated that CHO receptors within the mouth activate reward sensors in the brain, which may improve exercise performance without actually ingesting anything [17]. However, this practice does not address fuel and hydration needs, particularly for the endurance athlete.

Additionally, a review of the literature by Jager et al. found that creatine supplementation increases muscle phosphagen levels by approximately 10–40%, and improves performance in high intensity, intermittent activities [7]. When used during training, creatine supplementation has been shown to promote gains in performance and/or lean body mass, with weight gain being reported as the only significant side effect [6]. In regard to creatine supplementation and exercise in the heat, multiple studies [9, 18] concluded that there was no evidence to suggest that supplementation hinders the body's ability to dissipate heat or negatively impacts total body fluid balance. In fact, despite many anecdotal claims of its danger, it appears that creatine supplementation could have positive influences on body fluid balance, and has been shown to reduce the rate of perceived exertion when training in the heat [10]. Specifically, a meta-analysis found there was no evidence that creatine supplementation hinders the body's ability to dissipate heat or negatively impacts an athlete's body fluid balance, and that controlled experimental trials of athletes exercising in the heat resulted in no adverse effects from creatine supplementation at recommended dosages [9].

The Application

The use of a particular drug or supplement should be based on individualized needs during exercise, and always after consulting a healthcare professional. While it is apparent that in some endurance settings glycerol can improve performance, the National Collegiate Athletic Association (NCAA) bans the use of glycerol as it can also be used as a masking agent for blood doping. Effective January 1, 2018, however, the World Anti-Doping Agency (WADA) states that the magnitude of glycerol-derived effects is regarded as minimal, therefore, glycerol has been removed from WADA's prohibited list [51].

In activities lasting longer than 60–90 min, individuals should develop a plan to adequately maintain blood glucose, typically using CHOs. There is some trial and error in determining the type and amount of CHOs that work best for the individual athlete, so a CHO supplement strategy should be determined well ahead of any competition. General guidelines for the suggested intake of CHOs should be based on the individual's body size and the duration and intensity of the activity [5]. The sodium content in most "standard" commercial sports drinks serves to stimulate thirst and increases volume intake, but is not enough to replace sodium losses that accumulate with activity in the heat. Loss of sodium may become problematic, especially during endurance/ultra-endurance events. In addition, when sodium losses accumulate over multiple outings, as might occur in tournament situations or with multiple practices per day, specific sodium supplementation may be necessary. For individuals who sustain heavy salt losses, some sports drinks marketed for endurance activity contain higher sodium levels than traditional sport drink products. Another alternative is to add 1/8–1/4 tsp table salt per serving of a standard sports beverage (1 tsp table salt = 2.3 g sodium).

The typical recommendation for creatine supplementation consists of a loading phase of 20–25 g · day^{-1} for 2–7 days, immediately followed by a maintenance phase of 2–5 g · day^{-1} for the remainder of supplementation [8]. Based upon the evidence cited in the above sections, creatine supplementation has been widely studied and determined to be a potential ergogenic substance during sport with little to no side effects, even when exercising in the heat. However, with the lack of the Food and Drug Administration oversight in the production of supplements, athletes, coaches, and sports medicine personnel should make sure that the benefits and potential side effects are well known before use, and that the recommended dosage of creatine supplementation is followed.

Decrease Performance in the Heat

The Physiology

Although banned from many sports competitions, diuretics are widely used. It has been shown that dehydration and decreased plasma volume is a risk factor for suffering exertional heat illness [19]. Diuretics affect potassium homeostasis in exercising muscle (i.e., intracellular potassium and the resting membrane potential of the cell both decrease), and the resultant hypokalemia can lead to muscle cramps

Table 19.2 Common recreational drugs

Recreational drugs	
• Alcohol	• Amphetamines
• Nicotine	• Benzodiazepines
• Marijuana	• Ecstasy
• Opium	• Cocaine
• Lysergic acid diethylamide	• Heroin

and cardiac arrhythmias secondary to electrolyte shifts/losses [20]. With the main purpose of diuretics being to excrete salt and water, the risk of dehydration during exercise is heightened, thus increasing the risk of suffering exertional heat illness.

There are also a multitude of recreational drugs, both legal and illegal, utilized in the athletic setting and everyday life (Table 19.2). One drug that causes deleterious shifts in total fluid volume is alcohol. The consumption of alcohol and drugs by student athletes is a very concerning problem in today's world, especially in the collegiate setting due to increased physical demands combined with a unique social environment [21]. Alcohol is a powerful diuretic with just 1 g of ethanol consumed producing an excess of 10 mL of urine caused by the inhibition of vasopressin [22]. Furthermore, alcohol has also been shown to contribute to peripheral vasodilation, which could further exacerbate dehydration due to increases in fluid loss through evaporation while exercising [22]. Taking all of this evidence together, the physical strain experienced during and after alcohol consumption may result in added risk of suffering exertional heat illness.

The Evidence

Many research articles and guidelines [19, 20, 23] mention diuretics as an intrinsic risk factor for exertional heat illnesses. Specifically, Fonseca et al. [23] investigated the impact of heat exposure on eight individuals with hypertension. These individuals were also on diuretics and/or an angiotensin-converting-enzyme inhibitor [23]. Researchers found that exposure to a heat stress condition seemed to cause an imbalance in their redox status and an unregulated inflammatory response in cytokine interleukin-10; a cytokine also involved in exertional heat illnesses [23]. Additionally, Claremont et al. [24] assessed the impact of diuretic-induced dehydration on exercise heat tolerance in seven participants. As a result of the diuresis, plasma volume decreased 15.3% (SE ± 1.3), while heart rates during exercise increased 20–25 beats/min when compared to the control condition [24]. Although significantly greater increases ($P < 0.05$) in rectal and muscle temperatures occurred during exercise in the heat trial, mean skin temperature was lower due to decreased peripheral blood flow to the skin (800 mL/min less) [24]. Diuresis had no impact on sweat rate during exercise (heat = 0.80 L/h vs. control = 0.80 L/h), and body heat dissipation appeared limited mainly by a reduction in peripheral blood flow [24]. The diuretic-induced dehydration was found to increase the sensitivity of some subjects to the exercise-heat stress, producing symptoms of heat exhaustion [24].

In regard to alcohol, research [22] has shown that consuming alcohol has deleterious impacts on both anaerobic and aerobic performance in the heat. Once an individual reaches an alcohol intoxication threshold of 20 mmol/L, endurance performance is decreased in both animals and humans [22]. Additionally, Kalant et al. [25] found that alcohol had a severe impact on an individual's ability to thermoregulate, especially during exercise in the heat. In animal models, in a higher ambient temperature, ethanol induced a hyperthermic state and increased mortality. This impairment in thermoregulatory function was so great, that it was compared to a subject experiencing a change in body temperature set-point, as you would see with the onset of a fever [25].

The Application

Although banned for use in some sports, diuretics are still used by many for the management of medical conditions. The literature has shown that diuretics can be harmful to an individual exercising, especially in the heat, due to their propensity to cause electrolyte and water imbalances. Whether the use of diuretics is due to trying to cut weight, flush out other banned substances, or because of hypertension or another illness, it is important to understand the risks of this drug on the exercising body in stressful environments. The marked dehydration following diuretic intake exerts a detrimental effect on the cardiovascular and thermoregulatory systems of the body during exercise and can lead to exhaustion, irregular heartbeat, heart attack, and death [20]. Consultation with a healthcare professional should always take place prior to exercise.

Lastly, although alcohol is not a banned substance, individuals partaking in alcohol consumption prior to exercising in the heat should be aware of its potential detriment.

Case Examples

Case Example 1: *Creatine Monohydrate One of these unfortunate situations occurred in 1997, when the NCAA and other federal agencies investigated the deaths of three college wrestlers who died over a 5-week period* [26]. *The three wrestlers were Billy Jack Saylor, of Campbell College in North Carolina; Joseph LaRosa, of the University of Wisconsin at La Crosse; and Jeff Reese, of the University of Michigan* [26]. *What these wrestlers had in common where: (1) they died following grueling workouts in the heat to lose weight for competition and (2) they were all using creatine as a supplement* [26]. *Reese, 21, died of heart and kidney failure on December 9th, after exercising for 2 h while wearing a rubber suit in a room heated to 92 °F/33.3 °C; LaRosa, 22, died of heat stroke on November 21st, after riding a stationary bicycle in a steam-filled shower while wearing a rubber suit; and Saylor, 19, died of a heart attack on November 9th,*

after riding a stationary bicycle and refusing liquids for several hours [26]. *The deaths were first attributed to weight loss practices; however, with high creatine levels found in the blood stream of the three wrestlers it called into question creatine's potential involvement with the deaths* [26]. *The conclusion of the investigations, however, determined that it was exertional heat stroke and not creatine that ultimately lead to the deaths of these three individuals* [26].

Case Example 2: Recreational Drugs *On August 9th, 2009, emergency medical services were dispatched to Grambling State University for an unconscious 20-year-old male basketball athlete* [27]. *The basketball team had just run a 4-mile, timed, outdoor punishment run where the athlete collapsed and became unconscious* [27]. *On August 26th, 2009, the athlete passed away from complications of exertional heat stroke* [27]. *When the blood toxicology report was available, it showed that the athlete had both marijuana and alcohol (10 mg/dL) in his system at the time of the collapse* [27]. *Although the athlete's doctor is quoted saying that these substances were found in the athlete's system, they, "more likely than not," did not cause the athlete's death, it was also reported that the role these substances play in exertional heat stroke could not be ignored. While the cause of the exertional heat stroke was certainly multi-factorial, the ultimate cause of death was a lack of appropriate cooling to quickly lower Henry's body temperature.*

Drugs and Supplements with Central Mechanisms

Enhance Performance in the Heat

The Physiology

Caffeine is the only commonly used substance found to have a potential performance-enhancing central mechanism, meaning changes occur via the brain and spinal cord as a result of consumption. Caffeine (1,3,7-trimethylxanthine) is found naturally in various plants (e.g., coffee beans, cocoa beans, tea leaves, cola nuts) and artificially made in the laboratory [28, 29]. It is the most widely consumed pharmacologic and psychoactive substance in the world, and also the most consumed substance in the sport setting to treat fatigue and enhance alertness and energy [28]. The use of caffeine-containing energy drinks has increased substantially over the last few years and is currently the most common way caffeine is ingested by sports participants [28]. The NCAA has an allowable urinary level of less than 15 μg/mL, which would require a 70-kg person to drink at least five regular sized cups of coffee to approach illegal levels.

Caffeine is quickly absorbed from the gastrointestinal tract into the circulatory system once ingested. It appears in blood within 5–15 min and typically reaches its peak plasma level within 30–60 min, although interindividual differences exist [30]. Caffeine affects functions throughout the body including the central nervous system (CNS), the cardiovascular system, as well as metabolic, renal, and muscular function. The positive and negative impact of caffeine can be explained through three

predominant mechanisms: (1) the antagonism of adenosine receptors, particularly in the CNS, (2) increased mobilization of intracellular calcium, and (3) the inhibition of phosphodiesterases [30].

The Evidence

Common side effects of caffeine can occur at any dose independent of exercise activity, and include symptoms such as insomnia, gastrointestinal problems, anxiety, mood disorders, and headaches; however, these effects occur much more frequently at doses greater than 9 mg/kg of body mass (Fig. 19.1) [31]. There is some disagreement on the exact cardiovascular side effects of caffeine, but it is known that high doses of caffeine can lead to palpitations and cardiac arrhythmias (e.g., atrial fibrillation, supraventricular tachycardia, premature ventricular contractions) [30]. In addition, some studies have shown that caffeine can cause an increase in blood pressure through its vasoconstrictive properties [30]. In the heat, one study [32] found that when given a 3 mg/kg of body mass caffeine dose 1 h prior to exercise and another 3 mg/kg of body mass dose 45 min into a 2-h submaximal bike ride, followed by a 15-min performance trial, leg pain was reduced by 27% when the trial was performed in the heat (33 °C/91.4 °F) compared to a cool environment (12 °C/53.6 °F). Noteworthy during this research, pain perception was 74% higher in the hot versus cool environment, and the consumption of small amounts of caffeine was able to partially attenuate that increase [32]. Lastly, the literature is mixed regarding caffeine's ability to increase exercise endurance performance. Some studies report that 5, 6, or even 9 mg/kg of body mass of caffeine prior to exercise did not improve performance in the heat [32], while others have concluded that 6 mg/kg of body mass of caffeine was ergogenic and increased exercise endurance performance in the heat [32].

The Application

There is little doubt that caffeine intake, even when used in small doses, confers a performance benefit in athletics across a wide range of activities including sprint events, performance events, and power events in normothermic environments. However, the side effects seen at increased doses of caffeine may decrease athletic performance in some individuals [29]. While caffeine has not been demonstrated to induce a diuretic effect, it has been shown to potentially increase sodium lost through sweat [31].

When examining thermoregulation in exercising individuals, it does appear that caffeine has a mild thermogenic effect during exercise [31]. Athletes, coaches, and sports medicine personnel should be aware of not just the ergogenic benefits of caffeine but also the potential side effects such as stomach irritation, insomnia, and increased heart rate. Finally, given the interindividual variability of effects of caffeine on performance, athletes should use caffeine in a non-com-

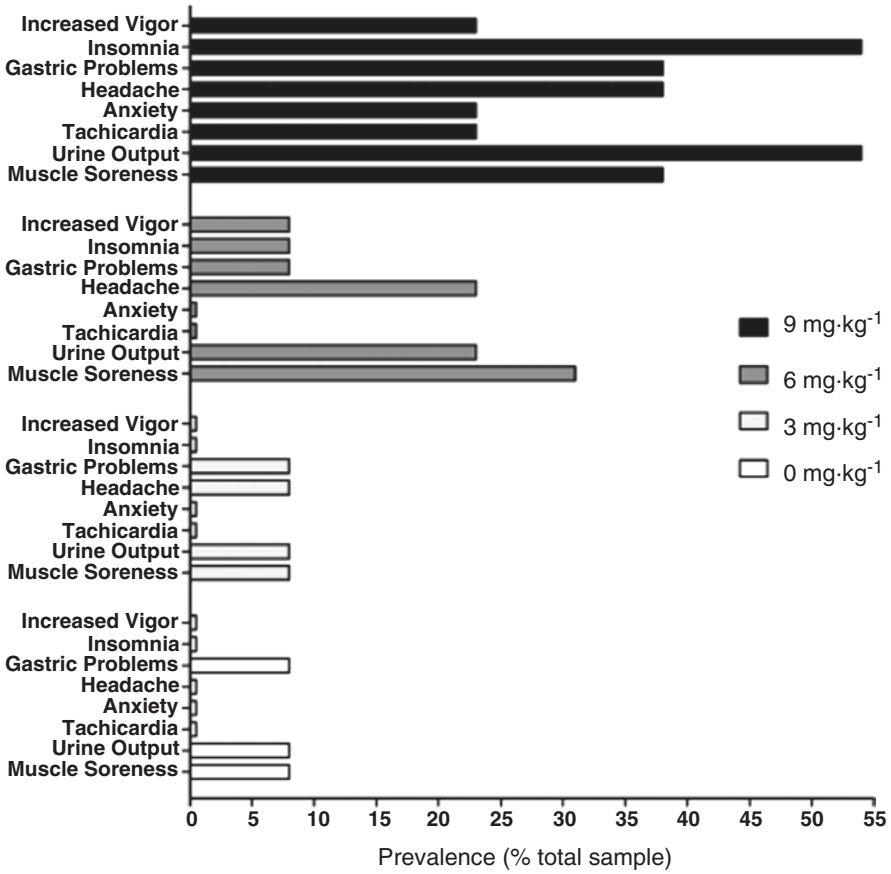

Fig. 19.1 Dose-response on side effects of caffeine ingestion. Data are presented as percent of prevalence [31]. Reproduced from Mora-Rodriguez R, Pallares JG. Performance outcomes and unwanted side effects associated with energy drinks. Nutrition Reviews. ©2014, Vol 72, S1, pages 108–120, by permission of Oxford University Press

petitive environment first to see what their response will be prior to using it in a competition [28].

Decrease Performance in the Heat

The Physiology

Amphetamines have been widely used by athletes to potentially increase performance through largely unknown mechanisms. Specifically, ecstasy (3,4-methylene dioxymethamphetamine [MDMA]) is a popular drug of abuse in today's society, and there have been reports of severe hyperthermia, altered mental status, and

autonomic dysfunction associated with its use [33]. Within the body, mitochondrial uncoupling proteins (UCPs) are the most prominent direct mediators of facultative thermogenesis [34]. Specifically, UCP3 has a potential involvement in the regulation of human thermogenesis and metabolism, and this mechanism is completely overridden when MDMA is exposed to the system [34]. In many clinical case reports, it has been demonstrated that MDMA and methamphetamine often induce catastrophic hypermetabolic hyperthermia that leads to skeletal muscle breakdown and death, suggesting that pathological mitochondrial uncoupling and heat production within the muscle may contribute to human fatalities [34].

Stimulants such as methylphenidate (e.g., Ritalin and Concerta) and dextroamphetamine-AMP (e.g., Adderall) are the most common pharmacologic treatments for the treatment of attention deficit hyperactivity disorder. [35]. Although the precise mechanisms underlying the action of these medications are not completely understood, they appear to increase the availability of dopamine, which could account for their therapeutic effects [35]. The medication may also mask signs and symptoms of fatigue due to this increase in dopamine, and allow for a longer duration of exercise with elevated temperature in excess of 40 °C/104 °F and increased heart rates, predisposing an individual to exertional heat illness [35].

Another drug that has harmful impacts on the thermoregulatory system is ephedrine. Ephedrine is a stimulant that is structurally similar to amphetamine and is derived from *ephedra sinica* (also known as Chinese ephedra or ma huang), which is the chemical component that produces the stimulant and thermogenic effects of ephedra [36]. Ephedrine's ability to increase energy expenditure and promote weight loss occurs through a few mechanisms [37]. It stimulates alpha-1, alpha-2, and beta-1 adrenergic receptors to increase the release of epinephrine and norepinephrine, which in turn has an effect on both the cardiovascular system and central nervous system [37]. The thermogenic effects of ephedrine occur via its effect on fat and glucose metabolism through its interaction on beta-3 receptors, with additional thermogenic effects occurring due to interactions with beta-1 and beta-2 receptors [37]. These same mechanisms are also responsible for the unintended consequences of ephedrine, which we will explore briefly in the application section.

A group of drugs that can have deleterious impacts during exercise in the heat, and are the most widely prescribed antidepressant medication in the United States, are selective serotonin reuptake inhibitors (SSRIs) [38]. Thermoregulation is a complex system of various afferent and efferent pathways, with the main neurotransmitters involved being serotonin and dopamine [38]. Specifically, serotonin neurotransmission mediates body temperature, while a serotonin agonist decreases the internal temperature primarily during rest [38]. When this delicate balance of neurotransmitters is not working appropriately, the hypothalamic set temperature could be impacted, increasing the risk for suffering an exertional heat illness [38]. Additionally, SSRIs have been found to reduce serum sodium concentrations, and in turn, thirst, leading potentially to accelerated dehydration during exercise in the heat [39].

Hydroxy-methyl-glutaryl (HMG) CoA reductase inhibitors (statins) are the most effective medications for managing elevated concentrations of low-density lipoprotein cholesterol (LDL-C) [40]. Resultantly, they are among the most prescribed

drugs in the United States and world, as approximately 28% of US adults ages 40 and older have received a statin prescription [40]. Statins inhibit HMG CoA reductase, which is the rate limiting enzyme in cholesterol production in the liver. Inhibition of HMG CoA reductase thus prevents production of mevalonic acid and, consequently, cholesterol. Interestingly, the production of mevalonate is also triggered in response to heat stress, and increased production of mevalonate is associated with greater heat tolerance in cells [41]. Therefore, in theory, utilizing statins could potentially place an individual at risk of suffering exertional heat illness due to blocking mevalonate's natural ability to provide greater heat tolerance.

The Evidence

One study investigated the impact of MDMA and methamphetamines on UCP3 knock-out mice in the heat [34]. They found that UCP3 is a candidate thermogenic mediator in intact animals, and when UCP3 is knocked-out, the mice had a severe hypermetabolic hyperthermic response to MDMA and methamphetamines, leading to death [34]. Additionally, Morozova et al. [42] investigated the internal body temperature and oxygen consumption of control and amphetamine-treated rats running on a treadmill with an incrementally increasing load. They found that the administration of amphetamine slowed down the internal temperature rise; however, it was found that the muscle temperature prediction model revealed the muscle temperature at the end of exercise was significantly higher in the amphetamine-treated group [42]. Thus, amphetamines were determined to potentially mask or delay fatigue by slowing down the exercise-induced internal temperature rise; however, this could impact the thermoregulatory system resulting in potentially dangerous muscle overheating [42].

In regard to ADHD medication's impact on exercise in the heat, Watson et al. [43] investigated the impact of dual dopamine/noradrenaline reuptake inhibitor on performance, thermoregulation, and the hormonal responses to exercise. Nine healthy endurance-trained athletes were recruited and participated in four exercise trials ingesting either a placebo or 2 × 300 mg bupropion prior to exercise in temperate (18 °C/64.4 °F) or warm (30 °C/86 °F) conditions [43]. Trials consisted of 60-min cycle exercise at 55% maximal workload, followed by a timed trial performance [43]. Researchers found that in the experimental group, rectal temperature and heart rate were significantly higher than in the control group (control 39.7 ± 0.3 °C/103.5 ± 0.5 °F, experimental 40.0 ± 0.3 °C/104 ± 0.5 °F, $p = 0.017$; control 178 ± 7 beats min^{-1}, experimental 183 ± 12 beats min^{-1}, $p = 0.039$) [43]. Researchers concluded that bupropion use enabled subjects to maintain a greater time trial power output in the heat with the same perception of effort and thermal stress reported during the placebo trial, despite the attainment of a higher internal body temperature [43].

Roelands et al. [44] examined the effect of a dopaminergic reuptake inhibitor on exercise capacity and thermoregulation during prolonged exercise in temperate and warm conditions. Eight healthy and well trained cyclists were either given a placebo or methylphenidate (Ritalin; 20 mg) prior to exercising in the same conditions as Watson et al. [43, 44]. Results showed that the time trial was completed 16% faster in the experimental group (38.1 ± 6.4 min) than in the control (45.4 ± 7.3 min; $p = 0.049$) [44]. In the heat, rectal temperature was significantly higher at rest ($p = 0.009$), and throughout the time trial in the experimental group ($p < 0.018$), reaching values above 40 °C/104 °F [44]. Additionally, throughout the experimental trial, heart rates were significantly higher ($p < 0.05$) [44]. Researchers concluded that the use of a dopamine reuptake inhibitor clearly improved performance when exercising in the heat; however, it caused hyperthermia without any change in the perception of effort or thermal stress potentially increasing the risk of exertional heat illness [44].

Furthermore, prior to its removal from the market, it was found that ephedrine-containing products made up less than 1% of all herbal supplement sales, but were responsible for nearly 64% of all recorded adverse events seen with herbal supplements [45]. Adverse events reported from the use of ephedrine involved many body systems and included myocardial infarction, hypertension, cardiac arrhythmias, strokes (both hemorrhagic and ischemic), seizures, paranoia, psychosis, and possibly death [36].

There are a few studies conducted on temperature-related deaths in individuals taking SSRIs [46, 47]. Page et al. [46] investigated the risk of high ambient temperature on patients taking SSRIs (i.e., patients with psychosis, dementia, and substance abuse). She found that there was an overall 4.9% increase in risk of death per 1 °C/1.8 °F increase in ambient temperature above a threshold of 18 °C/64.4 °F [46]. Additionally, Page et al. [46] found that SSRIs could alter body temperature regulation via anti-dopaminergic action and impair sweating via anticholinergic action. More studies, however, are needed to determine the impact of SSRIs on exercise in the heat.

Lastly, there are no studies to our knowledge that have researched the impact of statins on thermoregulation in human subjects; however, there have been some plant-based studies with interesting results [41]. Many components of the mevalonate pathway in animal cells are structurally and functionally similar to those of plant cells, and environmental insults like heat, sunlight, and water stress lead to the synthesis of mevalonate-derived lipids such as isoprene [41]. In plants, isoprene's increased synthesis when exposed to environmental insults, particularly heat stress, has been associated with enhanced thermotolerance [41]. Since the mevalonate pathway is highly conserved in all living cells, it is hypothesized that the ability to activate the synthesis of some isoprenoids and/or sterols in order to favorably respond to adverse stimuli may additionally exist in mammalian cells [41]. However, the impact statins have on exercise in the heat has yet to be fully discovered in human subjects.

The Application

Amphetamine is known to impact the thermoregulatory system by altering both heat production and heat dissipation [42]. It has also been shown that when exercising in high ambient temperatures, amphetamines increase the temperature at which exhaustion occurs creating a risk of developing exertional heat stroke [42]. All forms of amphetamines, including ecstasy, are banned by the WADA during training and competition, and are illegal to use in everyday life as well.

Medication taken for the treatment of ADHD may be ergogenic for performance during exercise in the heat; however, it puts an individual at risk due to diminishing their perceived exertion allowing them to participate in an activity longer despite increasing thermoregulatory strain. With ADHD being one of the most common neuropsychiatric disorders of childhood with almost 10% of children in the United States being diagnosed [48], individuals taking ADHD medications should be aware of their potential increased risk of exertional heat illnesses during exercise, especially in the heat, and should consult their physician prior to conducting any form of exercise.

Ephedrine-containing compounds have been banned in the United States since 2004. However, prior to its ban, numerous side effects were reported that affect athletic performance. In regard to cardiovascular complications, arrhythmias, myocardial infarction, and cardiac arrest have all been reported in athletes using ephedrine-containing compounds [36]. The sympathomimetic effect of ephedrine impairs the body's ability to dissipate heat properly, which is particularly important to athletes who are at risk of exertional heat illness [36]. The relationship between ephedrine and its ability to impair the normal thermoregulatory ability of the body causing an increased risk of exertional heat illness led to it being banned by Major League Baseball.

With the lack of knowledge of SSRI's impact on performance in the heat, it is imperative that any individual taking SSRIs should consult with a physician prior to exercising, especially in extreme environments. With an increase in the number of active individuals being prescribed SSRIs, it is very important for individuals to know the benefits and the risks associated with their use.

Lastly, statins are a widely sold drug with over 25 million people around the world receiving statin therapy [49]. Although there is limited evidence that suggests the potential influence of statins on thermoregulation, statins have been linked to myopathies during exercise, specifically rhabdomyolysis. Consultation with a physician should always occur prior to exercise, especially when exercise will be conducted in an extreme environment such as heat.

Case Example 3: Selective Serotonin Reuptake Inhibitor *Selective serotonin reuptake inhibitors have been linked to several cases of exertional heat stroke* [38]. *In one case, a patient who suffered from depression was being treated with fluoxetine HCL* (e.g., *Prozac) and lithium carbonate* [38]. *While engaging in mild intermittent work for 4 h under hot (37 °C [98.6 °F]) and dry (15% relative humidity) conditions, the subject lost consciousness, and was in a hyperthermic state upon reaching the emergency department* [38]. *She was also found to be suffering from disseminated*

intravascular coagulation, and a year later still suffers from residual cerebellar symptoms due to her exertional heat stroke potentially caused by drug-induced heat intolerance [38].

Case Example 4: 3,4-Methylenedioxymethamphetamine *Another situation that demonstrates the potentially dangerous mixture of drugs and heat occurred when a 20-year-old woman was brought to the emergency department by paramedics unresponsive and cyanotic with agonal respirations* [33]. *According to her boyfriend, she ingested two tablets of MDMA for recreational purposes, and within 4 h, her vital signs were as follows: blood pressure 105/77 mmHg, heart rate 160 bpm, and rectal temperature 41.7°/107 °F* [33]. *After suffering a grand mal seizure, she deteriorated into cardiac arrest and cardiopulmonary resuscitation was initiated* [33]. *The patient died 4.5 h after initial presentation of symptoms* [33]. *It is important to note that the hyperthermia in this case was not caused by exercise, but from the drug itself* [33]. *If exercise in the heat was to occur, these symptoms would most likely be intensified.*

Case Example 5: Statins *There have been several reported cases of athletes on statins experiencing rhabdomyolysis (i.e., severe muscle injury resulting in muscle fiber death and potential renal failure) after intense exercise such as marathon running* [49], *and exercise in the heat is an established risk factor for rhabdomyolysis. Taken collectively, these findings raise the question of whether chronic statin therapy impairs heat tolerance during sustained exercise, but to the best of our knowledge and literature review, the impact of statins on the body temperature response to exercise is not known.*

Case Example 6: Attention Deficit Hyperactivity Disorder Medication *In 2009, a 15-year-old high school sophomore named, Max Gilpin, collapsed while running punishment sprints at the end of a preseason practice in the middle of August heat* [50]. *Max died 3 days later of septic shock and multiple organ failure, resultant from an exertional heat stroke* [50]. *After the coach, Jason Stinson, was put on trial for criminal negligence, the defense utilized the medical examiner's testimony stating that Max was potentially ill the day of practice, and that his use of Adderall, as the primary cause of his exertional heat stroke* [50]. *Stinson was not charged with criminal negligence; however, it is impossible to know what role Adderall played in Max's exertional heat stroke death. While the cause of the exertional heat stroke was certainly multi-factorial, the fact that Max was not appropriately cooled was ultimately the reason he died from the heat stroke.*

Summary

Although some drugs and supplements are ergogenic, it must be noted that the improper or illicit use of any drug and/or supplement can be harmful to the body, and in some circumstances, cause lifelong disability or death. A list of banned and allowed subtances by the WADA and NCAA are summarized in Table 19.3.

Due to the lack of quality regulation, individuals should use caution when utilizing drugs and supplements, especially when exercising in a stressful environment such as the heat. The utilization of these drugs and supplements should be done on an individualized needs basis, and always in conjunction with a healthcare professional trained in the appropriate use of these substances.

Table 19.3 World Anti-Doping Agency (WADA) and National Collegiate Athletic Association (NCAA) in-competition banned and allowed substance list [51, 52]

	WADA		NCAA	
Substance	Category	Concentration	Category	Concentration
Glycerol	Banned	Any concentration	Allowed	–
Sodium, potassium, carbohydrates	Allowed	–	Allowed	–
Creatine monohydrate	Allowed	–	Allowed	–
Diuretics	Banned[a]	Any concentration	Banned	Any concentration
Alcohol	Banned	>0.10 g/L blood alcohol	Banned[b]	Any concentration
Caffeine	Allowed	–	Banned	Any concentration
Amphetamines (ecstasy)	Banned	Any concentration	Banned[c]	Any concentration
Selective serotonin reuptake inhibitors	Allowed	–	Allowed	–
Ephedrine	Banned	>10 μg/mL urine content	Banned	Any concentration
Stimulants (Attention Deficit Hyperactivity Disorder)	Banned	Any concentration	Banned	Any concentration
Hydroxy-methyl-glutaryl CoA reductase inhibitors (statins)	Allowed	–	Allowed	–

[a]Exceptions are available [51, 52]
[b]Only banned in rifle competition
[c]Amphetamines are a controlled substance in the United States

Table 19.4 Ten steps to successful implementation of drugs and supplements in sport and physical activity

1. Individuals participating in sport and physical activity should be educated on the potential benefits of drugs and supplements on performance in the heat
2. Individuals participating in sport and physical activity should be educated on the potential negative side effects of drugs and supplements on performance in the heat
3. Individuals participating in sport and physical activity should be educated on the signs and symptoms of usage or withdrawal of the above drugs and/or supplements
4. Medical personnel and administrators in charge of events involving physical activity should have policies in place for providing support and intervention to individuals showing signs of inappropriate drug and/or supplement use
5. Administrators should have policies in place for individuals to seek confidential help and/or guidance from a medical professional on appropriate drug and/or supplement use for their specific needs
6. Medical personnel and administrators in charge of events involving physical activity should have an emergency action plan in place describing the actions to be taken in the case of exertional heat stroke, cardiac arrest, respiratory arrest, exertional heat illness, and any other emergency injury or illness due to drug and/or supplement use
7. Medical staff, coaches, and athletes should be educated and aware of the list of approved and banned drugs and supplements for individual sporting competitions and organizations
8. Sports medicine staff should be educated in the correct dosage of an approved drug and/or supplement for an individual's specific performance needs
9. If possible, sports medicine staff and administrators should have policies in place that implements confidential drug and supplement screening to individuals suspected of abuse
10. If possible, sports medicine staff should be provided a drug and/or supplement history of the individuals participating to provide recommendations, and treatment in case of an emergency

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