american physiological society
PHYSIOLOGICAL REVIEWS

Physiol Rev 101: 1873–1979, 2021
First published April 8, 2021; doi:10.1152/physrev.00038.2020

REVIEW ARTICLE

EXERCISE UNDER HEAT STRESS: THERMOREGULATION, HYDRATION, PERFORMANCE IMPLICATIONS, AND MITIGATION STRATEGIES

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Abstract

A rise in body core temperature and loss of body water via sweating are natural consequences of prolonged exercise in the heat. This review provides a comprehensive and integrative overview of how the human body responds to exercise under heat stress and the countermeasures that can be adopted to enhance aerobic performance under such environmental conditions. The fundamental concepts and physiological processes associated with thermoregulation and fluid balance are initially described, followed by a summary of methods to determine thermal strain and hydration status. An outline is provided on how exercise-heat stress disrupts these homeostatic processes, leading to hyperthermia, hypohydration, sodium disturbances, and in some cases exertional heat illness. The impact of heat stress on human performance is also examined, including the underlying physiological mechanisms that mediate the impairment of exercise performance. Similarly, the influence of hydration status on performance in the heat and how systemic and peripheral hemodynamic adjustments contribute to fatigue development is elucidated. This review also discusses strategies to mitigate the effects of hyperthermia and hypohydration on exercise performance in the heat by examining the benefits of heat acclimation, cooling strategies, and hyperhydration. Finally, contemporary controversies are summarized and future research directions are provided.

cooling; exercise capacity; fatigue; fluid balance; heat acclimation

CLINICAL HIGHLIGHTS

This review examines historical perspectives and recent advances in understanding of the impact of heat stress on human physiological function. Endurance exercise capacity and performance are impaired with a rise in thermal strain, which is determined by the thermal environment. The impairment is primarily mediated by hyperthermia-induced adjustments in cardiovascular, central nervous system, and skeletal muscle function. Failure to replenish excessive body water losses incurred via sweating compromises thermoregulatory capacity and further exacerbates the rise in physiological strain, precipitating fatigue development. The decision to reduce work rate or discontinue exercise in the heat, with or without hypohydration, occurs across a spectrum of physiological and perceptual responses that are task specific. Heat acclimation, along with cooling and hydration strategies, mitigate the deleterious influence of heat stress on exercise performance; however, several aspects of these strategies remain to be elucidated. Along with influencing performance, exercise under heat stress can disrupt homeostatic processes and lead to the development of clinically relevant disorders such as exertional heat-related illness (i.e., muscle cramps, heat syncope, heat exhaustion, and heat stroke) and fluid and electrolyte disturbances (e.g., hypovolemia, hypernatremia, and hyponatremia).

1. INTRODUCTION

Our early ancestors evolved to travel long distances by walking and eventually running in semi-arid environments to scavenge and possibly hunt for protein-rich food sources (1). After leaving the African savannah ~100,000 yr ago, minor physiological adaptations to cold occurred (2); however, humans remain predominantly tropical animals (3). The ability of humans to maintain a stable body core temperature stems from the evolution of several features commensurate with heat dissipation. These features include the multiplication of

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eccrine sweat glands, an elongated body form and reduced body hair favoring convective heat loss, as well as changing from nasal to oronasal breathing to permit greater airflow rates with less resistance and work (4–7). Today, physical activity and exercise are no longer essential to the collection of food, but rather used as a means to maintain aerobic fitness and health, as well as compete, with exercise in the heat becoming increasingly common for several reasons. Mass participation sporting events are growing in popularity around the globe with people looking for more challenging events, which often include exercising under extreme heat (e.g., Marathon des Sables). Climate change is affecting global temperature with the last decade (2011–2020) being the warmest in the 141-yr record and part of a persistent long-term trend (8, 9). The six warmest years have all occurred since 2015, with 2016, 2019 and 2020 being the top three. In 2020, mean global temperature was 1.2°C above preindustrial (1850–1900) levels (9) and is projected to increase by 1.5°C between 2030 and 2052 (10). An increase in the population of major cities is also causing these to become urban heat islands (11). These factors, along with the increased frequency and intensity of heat waves (12), are projected to present international sporting competitions (e.g., Summer Olympics, World Athletics Championships) with increasing restrictions on when, where, and how they may be held (13). Along with elite athletes, recreational competitors also face performance and potential health challenges due to extreme heat exposure. These challenges are associated with the rise in core temperature and loss of body water related to exercising in the heat. This review is therefore focused on providing an integrative perspective of the influence of heat stress and hydration status on physiological responses during exercise, as well as the impact of heat mitigation strategies. While the focus of the review is on exercise per se, in many respects the literature described herein is directly relevant and applicable to physically demanding occupations (e.g., agricultural work, firefighting, military, and mining).

Although it is well established that aerobic performance is impaired when undertaken in hot relative to cool conditions, the mechanisms mediating this impairment remain contentious and differ based on the type of exercise being performed (i.e., incremental, constant work rate, and self-paced). A compromise in hydration status (i.e., body water loss) through increased sweating is well recognized for exacerbating thermal strain (i.e., rise in whole body temperature) and fatigue development under heat stress, yet debate regarding the level of dehydration that can be incurred prior to performance being impacted continues. The approach used to hydrate (i.e., ad libitum or planned drinking) when exercising in the heat also continues to draw discussion and examination. These issues, along with strategies to mitigate the impact of heat stress and dehydration on performance, are examined. More specifically, this review summarizes how body temperature and fluid balance are regulated at rest, describes the changes occurring during exercise in the heat, and explains how this may impact on performance and health. These aspects are expanded on in distinct sections. In sect. 2, a general overview of human thermoregulatory control and the factors that influence heat exchange and heat balance is provided. Different methodologies to assess body core temperature are also addressed, followed by a summary of the major health problems that may occur when thermoregulatory function cannot be maintained in the heat. Section 3 follows a similar pattern for fluid balance and describes the general principles of body fluid regulation. Subsequently, fluid disorders are explained, as well as the methods to determine hydration status. Section 4 summarizes the effects of heat stress on human performance and the underlying physiological mechanisms responsible for the deterioration of exercise performance. Section 5 examines the impact of hydration status on performance in the heat and how systemic and peripheral hemodynamic adjustments contribute to fatigue development. Section 6 describes different approaches to mitigate the detrimental effects of heat stress and exercise-induced dehydration on performance: heat acclimation, cooling interventions, and hyperhydration. Finally, sect. 7 summarizes the findings of the review and outlines contemporary controversies regarding the impact of heat stress and hydration status on aerobic exercise in the heat and suggests avenues of research to advance this field of study.

2. HUMAN THERMOREGULATION AND HEAT BALANCE

Thermoregulation constitutes one aspect of homeostasis and represents the ability of an organism to keep its body temperature within certain boundaries in varying environmental conditions. The fundamental principles and theories regarding temperature regulation will be outlined in this section and placed in the contexts of heat exposure at rest and during exercise. The factors that constitute the human thermal environment will also be examined and contextualized in the framework of heat balance (i.e., interaction between heat gain and loss). These factors include environmental (i.e., ambient temperature, humidity, wind velocity, and solar radiation), task-dependent (i.e., metabolic rate and clothing), and personal (i.e., age, sex, body mass, morphology, and aerobic fitness) parameters. Next, methods to

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determine body core temperature, skin temperature, and thermal strain will be addressed, as will the spectrum of exertional heat illnesses that can develop during exercise in the heat. The intention of this section is to provide a concise overview of the fundamental concepts associated with human thermoregulation and heat balance, so as to contextualize the discussion on health and performance with regards to hyperthermia and dehydration in subsequent sections.

2.1. Body Temperature

Body core (i.e., brain, heart, and other central organs) temperature is typically regulated to ~36.6°C (95% confidence interval: 35.7 to 37.3°C) (14), but may deviate considerably when exposed to extreme conditions. For example, the lowest recorded body core temperature (i.e., rectal) survived by a human is 13.7°C (15). In contrast, during exercise in the heat, well-trained athletes may reach body core (i.e., gastrointestinal) temperatures of 41.5°C without any acute or long-term detrimental effects (16). Body core temperature in humans is the main regulated variable in thermoregulation (17). Core temperature is most commonly determined in the digestive system (e.g., oral, esophageal, gastrointestinal, and rectal) and the head (e.g., ear and forehead), next to its invasive determination in arteries or veins in clinical settings (see also sect. 2.3). Body core temperature is dependent on measurement location as it represents the outcome of local heat balance (18). At rest, the highest body core temperatures are generally observed in the rectum (19). Resting body core temperature is also dependent on age, sex, ethnicity, ambient temperature, dew point, time of day, and month of year (14, 20). For example, a distinct circadian rhythm in body core temperature occurs in humans. After a nadir in the morning between 0400 and 0600 h, body core temperature steadily increases and peaks 1 to 4 h before habitual bedtime (21). The amplitude of this diurnal variation is ~0.5°C in healthy individuals (22), barring any exposure to extreme cold or heat stress, fever, or exercise. Previous studies have also revealed that the elderly have a lower resting core temperature than young adults (23–25). The menstrual cycle significantly alters body core temperature, with an upward shift of ~0.4°C during the luteal phase compared with the follicular phase in premenopausal women (26, 27).

Skin acts as the interface with the environment, but unlike core temperature, skin temperature is not regulated (28) and varies across the body in response to the thermal environment (**FIGURE 1**) (30, 31). Mean skin temperature can nonetheless be categorized as cool (<30°C), warm (30–34.9°C), and hot (≥35°C) (32). The human body itself is generally divided in two main compartments with regards to temperature: the core and the shell (i.e., skin and peripheral tissues). A third compartment is sometimes added to determine changes in body heat content, that of the muscle. Although measuring muscle temperature is invasive, the three-compartment model provides a more accurate estimate of mean body temperature during exercise (33).

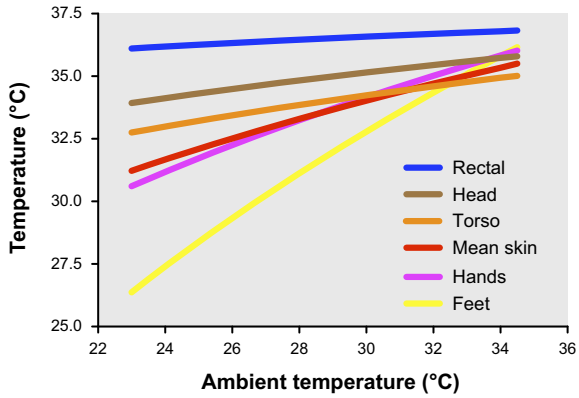

FIGURE 1. Relationship between ambient temperature and rectal, foot, hand, head, torso, and mean skin temperature at rest. These data indicate that changes in core temperature (i.e., rectal) are much smaller than those of the skin and extremities to changes in ambient temperature. Adapted with permission from Olesen (29).

2.2. Behavioral and Autonomic Thermoregulation

Body temperature regulation is accomplished through the parallel processes of behavioral and autonomic thermoregulation. Behavioral temperature regulation operates largely through conscious behavioral adjustments, which when in the heat includes a number of cool-seeking behaviors such as standing in the shade, drinking cold beverages, pouring water over one's head, and wearing light-colored clothing. Adjustments in work rate during exercise in the heat have also been suggested to constitute behavioral adjustments that contribute to regulate body temperature (34). Thermoregulatory behavior decreases the requirement for autonomic responses (35), which operate through physiological processes that are independent of conscious voluntary behavior. These responses include the control of vasomotor (i.e., cutaneous vasodilation) and sudomotor (i.e., sweating) function in the heat, along with metabolic heat production (i.e., shivering) and vasomotor function (i.e., cutaneous vasoconstriction) in cold environments. A negative-feedback system is typically described as the regulatory system mediating autonomic thermoregulatory responses. This physiological control system produces graded responses according to disturbances in a regulated variable: body core temperature. The magnitude of change in autonomic responses is proportional to

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displacement of the regulated variable in relation to its set point. Such control structures are called proportional-control systems. In humans, central (i.e., brain, spinal column, and gastrointestinal tract) and peripheral (i.e., skin) thermoreceptors provide afferent input to thermoregulatory centers located in the hypothalamus, where it is compared with the set point (36, 37). The set point is purely a mathematical concept used to describe the thermal control of effector responses and does not imply a particular neural model of thermoregulation or set temperature. Rather, it describes different recruitment stages within the magnitude of a load error, which is the difference between the input and set point (38). As such, central and peripheral thermoreceptors send information to a central integrator, located in the preoptic anterior hypothalamus (39). This integrator generates a thermal command signal to regulate sweating, skin vasodilation, and vasoconstriction. For example, heat loss mechanisms are activated during a rise in core body temperature, while a decline in body core temperature results in the activation of mechanisms that conserve or produce heat (37). The notion of central integration is supported by data describing the ratio of the contributions from core and skin temperature inputs in controlling sudomotor [4:1 to 20:1 (40–43)] and vasomotor [3:1 to 5:1 (40, 44, 45)] responses. These responses can also be altered by factors such as circadian rhythm, fever, menstrual cycle phase, and heat acclimation (38, 46).

The notion of central integration has evolved, with other regulatory models emerging. These include a model in which heat balance is achieved through heat regulation across a range of heat loads by sensing heat flow to and from the body and defending body heat content through thermoeffector responses (47, 48). Another model suggests that independent thermoeffectors loops coordinate their activities to regulate body temperature around a balance point (28, 49). Although these thermoregulatory control models have merit (50), the current review will rely on the traditional model of a central integrator. Thermoregulation during exercise in the heat is regulated similarly as during rest and influenced by factors such as hydration state and ambient conditions, as well as work rate (i.e., exercise intensity). These factors are discussed in sects. 4 and 5.

2.3. Heat Balance

Human heat balance refers to the equilibrium between the internal rate of metabolic heat production and rate of heat exchange to the surrounding environment via sensible (i.e., convection, conduction and radiation) and insensible (i.e., evaporation) pathways. This equilibrium is expressed as the rate of body heat storage (S):

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

where M is metabolic rate, W is external work rate, C is rate of convection, K is rate of conduction, R is rate of radiation, and E is rate of evaporation. M – W determines the rate metabolic heat gain, whereas C ± K ± R determines the rate of dry heat exchange and E reflects the rate of evaporative heat loss. Four main environmental parameters affect the biophysical properties of human heat balance: ambient temperature, humidity, air velocity, and solar radiation (**FIGURE 2**). In addition to environmental factors, task dependent parameters impact on heat exchange: rate metabolic heat production and clothing; as do personal parameters: body surface area, body mass, sex, age, and aerobic fitness. These factors are briefly addressed below, but for greater depth on the matter the reader is referred to Refs. 51–53.

2.3.1. Environmental parameters.

2.3.1.1. AMBIENT TEMPERATURE. Humans face the challenge of maintaining a stable body core temperature in a world where ambient temperature can reach 54°C in the United States, Africa, and the Middle-East and descend to –89°C in the Antarctic (54). During exercise, ambient temperatures higher than skin temperature led to sensible (i.e., dry) heat gain, whereas lower temperatures lead to heat loss (**FIGURE 3**). Environments with a high ambient temperature and low humidity favor evaporative heat loss, since sweat and moisture from mucosa can more easily evaporate.

2.3.1.2. HUMIDITY. Absolute humidity refers to the amount of water vapor present in the air. In the atmosphere, absolute humidity ranges from near zero to ~30 $g \cdot m^{-3}$ when the air is saturated at ~30°C. The humidity of the air is strongly related to the climate. Hot wet climates are typically found in tropical forest areas and hot dry climates close to deserts. High absolute humidity compromises the capacity to evaporate sweat from the skin because the difference in water vapor (i.e., moisture) between the skin surface and the environment is low.

2.3.1.3. AIR VELOCITY. Standard meteorological wind speed is determined at 10 m above ground and the highest ever air velocity recorded on earth is 113.3 $m \cdot s^{-1}$ (55). At human level (i.e., 1–2 m above ground), air velocity during exercise is dependent on factors such as direction of travel, wind direction and terrain (56). Air displacement across the body results in convective heat exchange, depending on the thermal gradient between the air and the skin. The displacement of air also aids

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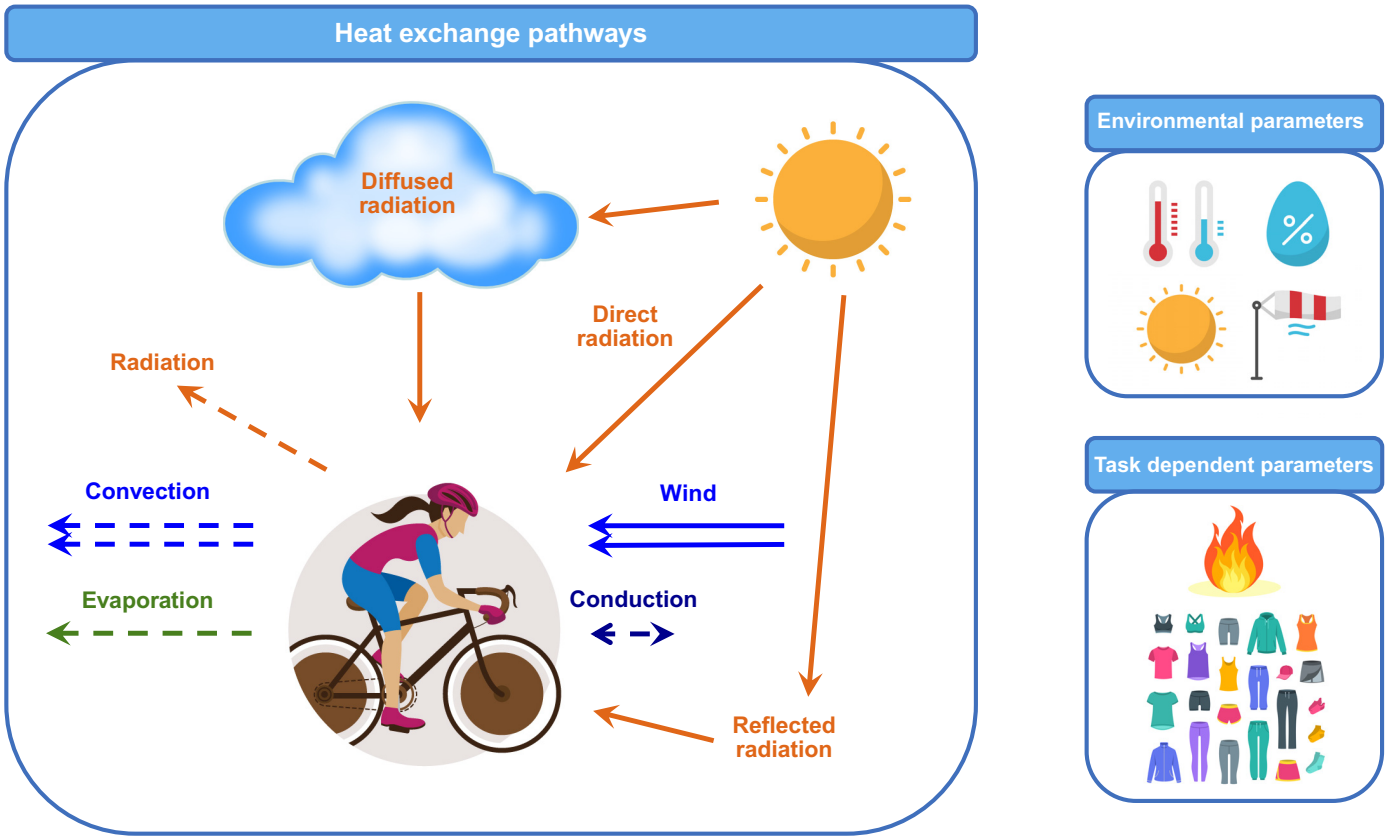

FIGURE 2. Heat exchange pathways and factors influencing human heat balance. Heat exchange to the surrounding environment occurs via sensible (convection: movement of fluids; conduction: direct contact; and radiation: electromagnetic waves) heat gain or loss and insensible (evaporation: vaporization of sweat or water) heat loss. Human heat balance is primarily influenced by environmental (ambient temperature, humidity, air velocity, and solar radiation) and personal or task dependent (metabolic heat production and clothing) parameters. Factors such as age, sex, body mass, and morphology can also influence heat balance.

with evaporative heat loss as it removes the layer of saturated water vapor that may stagnate across the skin.

2.3.1.4. SOLAR RADIATION. Average annual solar radiation directly emitted on the earth's atmosphere is ~1,361 $W \cdot m^{-2}$. The atmosphere absorbs some of this thermal energy such that ~1,000 $W \cdot m^{-2}$ reaches the surface of the earth on a clear day. The amount of solar radiation that reaches the human body depends on the location on earth, time of day, season, and the level of skin area exposed.

The integration of environmental parameters can be used to provide an index of the severity of the thermal environment, with a combination of parameters used to calculate different indexes. A recent meta-analysis identified over 300 thermal indexes, of which 185 were included in various statistical analyses (57). Temperature (98%), relative humidity (RH: 77%), and air velocity (72%) were the most commonly used environmental parameters to calculate the severity of the environment, with solar radiation (45%) and a combination of all four parameters (42%) less often integrated in these calculations. The most commonly used index in sport and exercise is the wet-bulb-globe temperature index (WBGT), which has been advocated by the American College of Sports Medicine (58) and National Athletic Trainers' Association (59) and used by several international sporting organizations (e.g., World Athletics, World Triathlon).

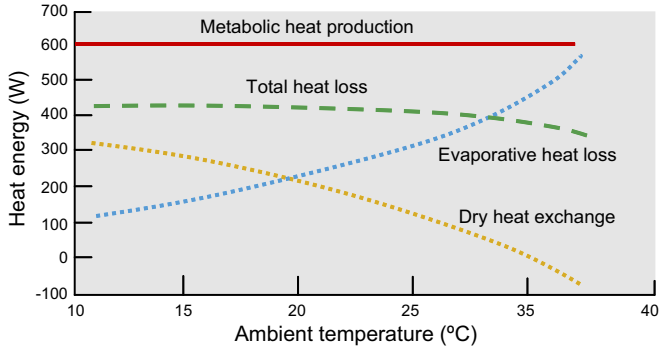

FIGURE 3. Relative contribution of evaporative and dry (i.e., convection and radiation) heat loss during exercise at a constant rate and metabolic heat production at different ambient temperatures. As ambient temperature increases and approaches that of the skin, dry heat loss is reduced and evaporation becomes the primary avenue of heat dissipation.

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2.3.2. Task-dependent parameters.

2.3.2.1. METABOLIC HEAT PRODUCTION. Human metabolism is the sum of resting (~65 $W \cdot m^{-2}$) and exercise metabolism. The oxidation of substrates during exercise contributes significantly to increase body core temperature as only ~20 to 25% (60, 61) of metabolic energy is converted to mechanical work, with the majority released as heat. To compete for the podium in a cycling grand tour, such as the Tour de France, elite cyclists need to deliver a power output of ~6 $W \cdot kg^{-1}$ in the mountains (62), which leads to a sustained (e.g., 15–30 min) heat production of ~1,400 W for a 70-kg male. The highest ever recorded maximal rate of oxygen consumption ($\dot{V}O_{2max}$) is 96.7 $mL \cdot kg^{-1} \cdot min^{-1}$ (63). Although not sustainable for a very long period, this level of oxygen consumption equates to ~2,500 W of metabolic heat production, which underscores the large contribution of exercise-induced heat production to human heat balance.

2.3.2.2. CLOTHING. Clothing acts as a barrier between the skin and the environment, altering heat exchange properties in relation to environmental conditions. The material properties and fit of a garment can affect heat strain during exercise by reducing heat dissipation and promoting heat conservation (64). As such, the insulative properties and water vapor resistance of garments worn during exercise in the heat should be as low as possible. The water absorption capacity of the material should also be low, as sweat trapped in a garment is not evaporated and does not provide cooling (65). Furthermore, the reflective properties of a garment are important in high radiative load scenarios (i.e., direct sunlight). It is not so much the color of the garment that is important, but the reflective properties of the dyes used in the garment (66). Ventilation in the air layer between the skin and garment (i.e., bellows effect) is important for heat loss during exercise in the heat (67). An example of the bellows effect is seen in the desert, where no difference in skin temperature was observed when wearing loose fitting black or white garments (i.e., robes), despite a 6°C difference in the surface temperature of the garments (black: 47°C; white: 41°C) (68).

2.3.3. Personal parameters.

2.3.3.1. BODY SURFACE AREA. Heat generated during metabolism is lost over the surface area of the body to prevent excessive heat storage. Body surface area is calculated using a formula based on height and weight: surface area = 0.20247 × height $(m)^{0.725}$ × weight $(kg)^{0.425}$ (69), which has been validated using three-dimensional scanning techniques (70). A large body surface area is beneficial for evaporative heat loss as the number of active sweat glands is proportional to surface area (71). Dry heat loss is also enhanced by having a larger body surface area when ambient temperature is lower than skin temperature (72). Hence, for a given thermal environment, heat loss potential is greater in those with a large body surface area (51).

2.3.3.2. BODY SURFACE AREA-TO-MASS RATIO. Individuals with a high body surface area-to-mass ratio experience less heat storage during uncompensable heat exposure than those with a lower ratio, due to the larger area for dry and evaporative heat loss relative to body mass (51). The body surface-to-mass ratio declines with increases in body mass and to a greater extent in females (73). Thus for heavy females it is more difficult to release body heat than for equally heavy males. This was confirmed in a study in which a thermal model was used to assess body core temperature in females of different body morphology showing that relatively fat females achieve considerably higher body core temperatures during exercise in a hot/humid environment than their leaner counterparts (74). The model was validated with data from six females with considerable difference in body dimensions during work in the heat (75).

2.3.3.3. SEX. Males and females differ in body size with men generally being heavier, taller and displaying higher $\dot{V}O_{2max}$. However, when standardized for body surface area, metabolic heat production during several tasks is similar between the sexes (76). When standardized for body surface area, however, some sex-related differences remain (e.g., sweat rate). Females have a higher density of activated sweat glands during moderate exercise (77), but sweat rate per body surface area is higher in males during light exercise in humid heat and similar between sexes in dry heat (78). These differences were suggested to stem from females having a higher onset threshold for sweating and better ability to suppress sweating when the skin is wet (78). Generally, however, when males and females are matched for body size and fitness-level ($\dot{V}O_{2max}$), differences in thermoregulation disappear (79–82). As such, there is currently no evidence that females have an inherent disadvantage in thermoregulation when exercising in the heat compared with males of similar age and health status (83).

2.3.3.4. AGE. Aging impacts on both thermoregulatory capacity and fluid regulation (84, 85). Older individuals (>60 y) have a lower resting body core temperature, attenuated cutaneous vasodilatory capacity, less effective sweat response, and decreased thermoreceptor sensitivity compared with younger individuals (84–86). Elderly individuals also have a higher thirst sensation threshold (87, 88), lower total body water (89, 90),

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reduced kidney function (89, 91), and an impaired plasma vasopressin regulation at rest and following dehydration (92, 93). These regulatory functions deteriorate with advancing age (94) and increase the risk of developing hyperthermia and dehydration (84, 85, 93, 95, 96). However, fit older individuals retain a better ability to thermoregulate and can improve thermoregulatory capacity with training (97). In children, thermoregulatory capacity has traditionally been viewed to be less effective than in adults and thermal tolerance inferior during exercise under heat stress due to a higher body surface area-to-mass ratio, diminished sweating capacity, lower mechanical efficiency, and lower cardiac output (98–100). These responses stem from adult-child differences in morphology, as well as endocrine, metabolic, cardiovascular, and thermoregulatory function. However, the view that children are at a thermoregulatory disadvantage has evolved in recent years, with the notion that they may only be at greater risk of severe hyperthermia in extreme environmental conditions (101–103). Methodological considerations such as normalizing physiological responses to body mass and surface area have been proposed when comparing children and adults, to ensure an unbiased comparison of size-dependent responses and that both children and adults are exposed to similar relative heat loss requirements (104).

2.3.3.5. AEROBIC FITNESS. Regular endurance exercise leading to improved aerobic fitness (i.e., $\dot{V}O_{2max}$) has been shown to enhance heat loss capacity. Aerobic training activates cutaneous vasodilation at a lower core temperature and increases skin blood flow for a given core temperature (105–107). The increase in skin blood flow is largely mediated by the expansion of blood volume and greater cardiac output that characterize the trained state (108). Endurance training has also been reported to reduce the internal temperature threshold for the onset of sweating, increase sweat rate at a given core temperature, and increase maximal sweat rate (106, 109–113). Modeling suggests that an exercise training-induced increase in $\dot{V}O_{2max}$ of 12 to 17% should reduce the internal temperature threshold for the onset of sweating by ~0.1°C (114). However, the enhanced sweating function associated with aerobic fitness may also relate to regular endurance training providing a repeated thermal challenge that leads to improvements in thermoregulatory capacity (115). The impact of aerobic fitness on thermoregulation is further discussed in the context of heat acclimation in sect. 6.1.4.

2.4. Core and Skin Temperature Measurement

Body core temperature can be measured at different body locations, depending on the type of equipment and need for accuracy. Blood temperature of the pulmonary artery is considered the gold standard as it best represents average internal human body temperature (116). As a catheter is needed to determine pulmonary artery temperature, body core temperature is often measured at more accessible and less invasive sites, such as the mouth, axilla, aural canal, esophagus, intestine, or rectum. Oral temperature is easy to determine given its accessibility. It is recommended to measure oral temperature under the tongue, as it may vary across different parts of the mouth (117). Factors such as salivation, previous food or fluid intake, gum chewing, smoking, and rapid breathing are known to impact oral temperature (118, 119). Hence, oral temperature may underestimate core temperature, making its measurement less reliable in dynamic conditions or when core temperature is elevated. Assessing axilla temperature takes longer than other body locations as more time is needed to reach an equilibrium. Ambient temperature, local blood flow, underarm sweat, and closure of the axillary cavity are known to impact axilla temperature (118). Axilla temperature typically underestimates core temperature and is less accurate compared with measurements at other body locations (120, 121), especially during fever or elevated vasomotor activity. Aural canal or tympanic temperature is easy to determine given its accessibility. Earwax or dirt in the ear canal, inaccurate placement, and/or the influence of environmental conditions (heat or cold) are known to reduce the reliability of aural canal temperature (122, 123). Moreover, infrared tympanic temperature monitors, which are purported to measure tympanic membrane temperature, more accurately reflect a combination of aural canal and tympanic temperature. Esophageal temperature is measured at the level of the left atrium and provides close agreement with pulmonary artery temperature, as placement of the sensor is close to the aorta. Esophageal temperature rapidly responds to temperature changes (124), making it a preferred method for determining core temperature. The disadvantage of esophageal temperature is its placement, which may cause general discomfort and irritation of the nasal passage (125). Temperature readings may also be affected when ingesting cool fluids or saliva. Gastrointestinal temperature can be obtained using ingestible temperature capsules, which are a valid, reliable, and easily applicable surrogate marker of core temperature (126–130). To allow gastric passage and avoid interference with fluid ingestion (131), capsules are generally ingested ~5 h before measurement. However, it has been shown that even 8 h after ingestion, consuming chilled water (5–8°C) can decrease capsule temperature by 2–6°C (132). This decrease has been attributed to localized cooling of areas (i.e., small and large intestines) in close proximity to the stomach and duodenum.

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In contrast, when fluids are not consumed during exercise, the timing of ingestion (40 min or 24 h) does not appreciably influence gastrointestinal temperature measurement (133). The time between ingestion and measurement (1 to 12 h) also does not appear to affect the difference (0.1–0.2°C) between capsule (i.e., gastrointestinal) and rectal temperature (134). The use of ingestible temperature capsules is especially suitable for field-based conditions, which is important as exercise-induced core temperature elevations are generally higher in field compared with laboratory-based settings (135). Rectal temperature measurement is considered an accurate method for determining core temperature, as long as the rectal thermistor is placed ≥10 cm beyond the anal sphincter (136, 137). Rectal temperature is less sensitive to rapid changes in core temperature, such as observed during exercise, compared with esophageal temperature (116, 138). However, rectal temperature is considered the clinical gold standard for obtaining core body temperature in patients suspected of exertional heat stroke (139). Finally, to determine mean skin temperature, multiple measurements sites should be measured (e.g., chest, upper arm, thigh, and lower leg) (140). The reader is referred to a recent review on skin temperature measurements (141).

2.5. Exertional Heat Illness

Exercise in the heat may increase the risk for developing exertional heat illness (EHI) as heat production often exceeds heat dissipation capacity. While the thermophysiological responses to exercise in the heat are well understood, individual responses vary substantially (142, 143), as does the risk for heat related illnesses. Exertional heat illnesses represent a spectrum of medical conditions related to an increase in body temperature (58, 144). The severity of EHI varies across its continuum, with mild complaints following exercise-associated muscle cramps, to more serious concerns during heat syncope and heat exhaustion, and life-threatening risks during heat stroke (59, 145–147).

Exercise-associated muscle or heat cramps are an early indication of EHI and arise in the form of muscle spasms or cramps, which are experienced as painful contractions and often result in the inability to continue exercising for a short time (148, 149). Muscle/heat cramps typically occur during or after excessive heat exposure, when fitness and heat acclimatization state are relatively low, but training load (150) and exercise intensity (151) are high. Sodium loss due to heavy and/or prolonged sweating is thought to play a significant role in the etiology of muscle/heat cramps, leading to a contracted interstitial fluid compartment and neuromuscular junction hyperexcitability (152–154). However, other studies suggest that neuromuscular fatigue induced by abnormal spinal control of motor neurons is responsible for exercise-associated muscle cramps (151, 155). Although the underlying mechanisms responsible for heat cramps remain contentious, they are likely to be due to a combination of sodium depletion, dehydration, and/or neuromuscular fatigue (156, 157).

Heat syncope, or orthostatic intolerance, can occur when a person is exposed to high environmental temperatures (158). Heat syncope often occurs after prolonged standing, immediately following exercise cessation or after rapid assumption to an upright posture after resting or being seated. It generally occurs during the initial phase of heat acclimatization (i.e., first 5 days), as heat exposure increases peripheral vasodilation and postural blood pooling, diminishes venous return, and reduces cardiac output. Dehydration and the intake of specific medications (i.e., diuretics) may further increase the risk for heat syncope, whereas heat acclimatization induced blood volume expansion may reduce the risk. Athletes can experience lightheadedness but also lose consciousness, which is usually quickly resolved after a period of sitting or lying down to restore cerebral perfusion.

Heat exhaustion is the inability to continue exercising with a core body temperature that typically ranges between 38.5°C and 40°C. Cardiac output cannot be sustained during heat exhaustion due to competing demands for skeletal muscle blood flow, perfusion of vital organs, and heat loss via the skin. Heat exhaustion often occurs in hot and humid conditions and is characterized by heavy sweating, malaise, fatigue, and dizziness. Nausea, vomiting, headache, fainting, weakness, and cold or clammy skin may also be observed (159). As this condition worsens, it is difficult to distinguish it from exertional heat stroke without measuring body core temperature and organ (dys)function. However, critical to the diagnosis of heat exhaustion is a normal mental activity and a stable neurologic status (160). Widespread peripheral vascular dilatation and associated central fatigue and collapse are thought to be responsible for heat exhaustion. Pilgrims with heat exhaustion following multiday desert walking demonstrated tachycardia and high cardiac outputs with signs of peripheral vasodilatation (161). Peripheral vasodilatation attenuates increases in peripheral vascular resistance, which subsequently results in hypotension, cardiovascular insufficiency, and high output heart failure (162).

Heat stroke is the most severe condition in the EHI spectrum and is associated with a core temperature >40°C, central nervous system dysfunction, and multiorgan failure (58, 163–165). Heat stroke is characterized by a reduction in central venous pressure and an insufficient cardiac output to cope with the high thermoregulatory

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demands, which accelerate the rise in core temperature. The combination of circulatory collapse and high core temperature aggravates pathophysiological processes (e.g., inflammatory responses) and can lead to multiorgan failure (166). An alternative pathway may lie with endotoxemia in response to exercise-induced immune and gastrointestinal disturbances (167). Exercise is known to acutely suppress immune function (168–170), and lipopoly-saccharides can leak into the circulation due to increased gut permeability (171–173). Endotoxemia may subsequently trigger a systemic inflammatory response, leading to systemic coagulation and hemorrhage, necrosis, and multiorgan failure. Heat stroke is a life-threatening condition and can be fatal unless promptly recognized and treated. Signs and symptoms are often nonspecific and include disorientation, tachycardia, vomiting, seizures, loss of balance, and coma. In a later stage, rhabdomyolysis, circulatory failure, multiorgan failure, and disseminated intravascular coagulation may occur and could lead to death (58, 158, 160). The risk of adverse outcomes (i.e., morbidity and mortality) increases the longer core temperature remains $>41°C$ and is significantly reduced if core temperature is lowered rapidly (174–176). Aggressive cooling is the cornerstone of heat stroke treatment, and cooling rates $>0.10°C \cdot min^{-1}$ should be targeted to improve prognosis (177). Heat stroke can be distinguished as either classic (passive) or exertional, depending on its cause. Both subtypes result from failure to dissipate excessive heat, but their underlying mechanisms differ. Classic heat stroke usually develops in vulnerable populations such as the elderly or individuals with comorbidities (e.g., obesity, diabetes, hypertension, heart disease, renal disease, dementia, and alcoholism) due to poor heat-dissipation mechanisms or attenuated behavioral mechanisms to reduce heat exposure and heat strain. Classic heat stroke occurs under resting conditions, without involvement of skeletal muscle heat production. In contrast, exertional heat stroke typically affects healthy individuals such as athletes, laborers (e.g., firefighters and agricultural workers) and military personnel, who are involved in strenuous physical activities or exercise in which metabolic heat production overwhelms physiological heat-loss mechanisms, leading to uncompensable heat stress.

Several personal and environmental factors are associated with an increased risk for EHI and exertional heat stroke. A recent systematic review summarized evidence from 42 studies and clustered risk factors into subgroups (**FIGURE 4**) (143). The majority of EHI risk factors were attributable to intrinsic factors pertaining to lifestyle. Athletes experiencing exertional heat stroke often performed similar exercise sessions (i.e., intensity and duration) under comparable environmental conditions, without having any problems or complaints (178, 179). These observations suggest that (temporal) changes in risk factors (e.g., a combination of mild illness, lack of sleep, and previous day heat stress) increase the vulnerability to develop heat stroke on that specific occasion. These insights are important as these risk factors and associated behaviors are modifiable, so communicating this information to athletes, laborers, and military personal working and exercising in hot and humid conditions is of vital importance. The fact that exertional heat stroke remains the third leading cause of death in athletes in the United States, following cardiac disorders and head and neck trauma (160, 180), further emphasizes the need for early recognition of EHI symptoms by healthcare professionals, appropriate equipment to treat heat stroke victims (i.e., ice baths) and education of race participants. For this purpose,

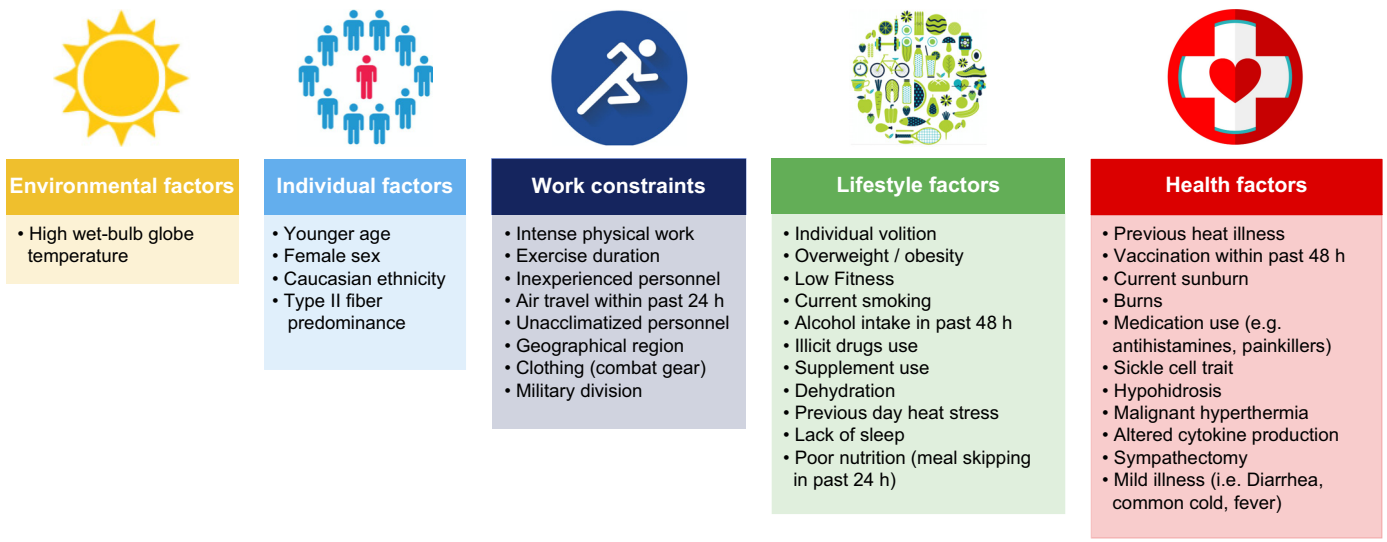

FIGURE 4. Risk factors for exertional heat illness classified in 5 subgroups: environmental, individual, work, lifestyle, and health. Adapted with permission from Westwood et al. (143).

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guidelines and recommendations have been developed for those undertaking athletic events (58, 59, 181) and performing occupational and military tasks (182–184).

3. BODY FLUID BALANCE

3.1. Body Water Balance

Total body water volume represents ~60% of body mass (range: 45 to 75%) (185–188) and is age and sex dependent, with lower values for the elderly and females (186, 189). Body water can be divided into intracellular and extracellular fluid compartments. The intracellular compartment represents ~40% of body mass, whereas the extracellular compartment can be segmented into the interstitial (~15% body mass) and intravascular (i.e., plasma volume, ~5% body mass) subcompartments (186, 190). These fluid compartments are separated by water-permeable cell membranes that allow continuous fluid exchange between compartments. As such, these volumes are not static but represent the net effect of dynamic fluid exchange with varying turnover rates between compartments. Perturbations in fluid balance during exercise or heat exposure modify the net volumes and turnover rates between fluid compartments. However, the continuous exchange of fluid between compartments, driven by osmotic and oncotic gradients and hydrostatic pressure, promotes the maintenance of fluid balance. Human fluid balance is also regulated by the renin-angiotensin-aldosterone system (RAAS) in response to a decrease in blood pressure due to the loss of blood volume. RAAS functions to elevate blood volume and arterial pressure by increasing sodium and water reabsorption in the kidneys. The first stage of RAAS is the secretion of renin into the circulation by juxtaglomerular cells when renal blood flow is reduced. Circulating renin acts to cleave angiotensinogen to angiotensin I, which is then converted into angiotensin II by angiotensin-converting enzyme. Angiotensin II is a strong vasoconstrictor and stimulates the secretion of aldosterone from the adrenal cortex. Aldosterone accelerates the reabsorption of sodium in the kidneys and the retention of water in the circulation, which restores fluid balance. For a comprehensive description of the RAAS, including its molecular working mechanisms, the reader is referred to Refs. 191–193.

Euhydration represents the state of being in water balance, whereas hypohydration is the state of being in negative water balance (i.e., water deficit) and hyperhydration the state of being in positive water balance (i.e., water excess). The processes of losing and gaining body water are referred to as dehydration and rehydration, respectively. Importantly, euhydration is not a static condition but reflected in a dynamic sinusoidal fluctuation of body water loss and gain (194). The daily regulation of total body water and fluid concentrations is a complex process influenced by dietary intake and nutrient availability with the loss of 1% in total body water beyond normal fluctuation typically compensated for within 24 h in free-living individuals (195). This occurs in response to highly controlled processes via which changes in plasma osmolality (i.e., electrolyte-water balance) stimulate body water conservation and acquisition mechanisms. The homeostatic conservation of body water is regulated by the release of arginine vasopressin, an antidiuretic hormone, and the acquisition of water by the stimulation of thirst. The release of arginine vasopressin from the posterior pituitary is triggered by an increase in plasma osmolality of 1–2% or a ~10% reduction in plasma volume (191, 196). The increase in circulating arginine vasopressin activates the reabsorption of water from urine by the kidneys, the main effective regulator of water loss (197). The sensation of thirst is stimulated in response to an increase in plasma osmolality of 5 to 10 mosmol/kgH_2O and decrease in blood volume of ~10% (191, 198–200). The osmolality and volume perturbations required to elicit these compensatory responses depend on the nature (i.e., intracellular vs. extracellular hypohydration) and magnitude of body water losses.

Clinically, hypohydration refers to a state of hypertonic hypovolemia, which follows the net loss of hypo-osmotic body water, causing a rise in extracellular tonicity (201). During such hypohydration, a shift in water from intracellular to extracellular compartments occurs to equilibrate osmolality between compartments, leading to intracellular hypohydration and a rise in plasma osmolality (202, 203). This triggers the osmoreceptor and endocrine mediated cascade to conserve and acquire water. In contrast, extracellular hypohydration occurs when the concentration of body water lost is iso-osmotic relative to plasma (i.e., isotonic hypovolemia), causing a contraction of the extracellular compartment, which cannot mobilize water from the intracellular compartment, leaving plasma osmolality unchanged. The water conservation and acquisition responses associated with extracellular hypohydration are thus nonosmotic and comprise of acute peripheral and renal vasoconstriction, along with the nonhumoral defense of blood volume (202, 204). From a clinical perspective, the magnitude of dehydration in relation to body mass loss is defined as mild (<5%), moderate (5–10%) and severe (>10%) and in relation to osmolality as isotonic (275–295 mosmol/kgH_2O), hypotonic (<275 mosmol/kgH_2O), and hypertonic (>295 mosmol/kgH_2O) (**TABLE 1**) (206, 207). However, from a physical activity perspective lower levels of dehydration have implications on exercise capacity and performance,

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Table 1. ***Classification of dehydration types with potential source of development***

Type of Dehydration	Divergence from Euhydration	Potential Source
Isotonic	Isotonic loss of water and sodium from extracellular fluid No osmotic water shift from intracellular fluid	Gastrointestinal fluid loss (e.g., vomiting, diarrhea) Inadequate fluid and electrolyte (e.g., sodium) intake
Hypertonic	Water loss exceeds sodium loss Osmotic water shift from cells to extracellular fluid	Inadequate water intake Sweat loss Osmotic diuresis (e.g., glucosuria)
Hypotonic	Sodium loss exceeds water loss Osmotic water shift from extracellular fluid to cells	Sweat and/or gastrointestinal fluid loss Water replacement without electrolyte replacement Diuretic therapy following excessive water intake

Adapted with permission from Ref. 205.

such that mild, moderate and severe dehydration can be considered <3%, 3–6%, and >6% body mass loss, respectively (see also sect. 5).

3.2. Body Water Balance during Exercise

Acute exercise produces an increase in systolic and mean arterial pressure, resulting in higher capillary pressure, greater capillary filtration, and an associated net outward fluid shift from intravascular to extravascular compartments (i.e., interstitial and intracellular) (208–210). Exercise-induced increased concentrations of lactate, sodium, potassium, and phosphate increase extravascular osmolality and stimulate intravascular to extravascular fluid shifts (208, 211, 212). In contrast, increases in lymphatic flow enhance fluid shifts from the interstitial to intravascular compartment, and elevated hydrostatic pressures in the contracting muscles stimulate fluid shifts toward the intravascular compartment (213). A balanced state between outward and inward plasma flow may ultimately occur during exercise, which limits the net fluid shift (214). Exercise also stimulates cutaneous water loss through the formation of sweat for evaporative heat dissipation. Sweat rate during exercise is regulated by several factors, including exercise intensity and duration, age, sex, training, and heat acclimatization status, clothing, and environmental characteristics (i.e., air temperature, humidity, wind velocity, and cloud cover).

Sweat is an example of hypo-osmotic fluid loss, so volume depletion caused by excessive sweating results in a proportionally increased plasma osmolality (215). Plasma hyperosmolality acts to mobilize fluid from the intracellular to the extracellular compartment to restore the plasma volume in hypohydrated individuals (216). If the reduction in plasma volume exceeds ~10%, changes in arterial pressure are detected by baroreceptors in the carotid sinus, aortic arch, left atrium, and great pulmonary veins, resulting in the sensation of thirst and secretion of arginine vasopressin to restore plasma osmolality (217). It is important to note that factors associated with exercise can also stimulate arginine vasopressin secretion and thirst sensation (see sect. 3.4). While these processes ensure fluid retention during fluid loss, the opposite responses can be expected during hyperhydration (i.e., decreased arginine vasopressin concentration and increased urine secretion). The loss of body water also occurs through CO_2-O_2 gas exchange and respiratory water loss, which are dictated by exercise intensity. These volumes are compensated for by metabolic water production during metabolism (i.e., substrate oxidation), and their overall impact is quite small (≤1%) (218, 219).

Drinking behavior, drink to thirst or to a fluid replacement strategy, to maintain hydration status and performance during exercise is complex and remains an area of contention within the literature (see sect. 5.6). While drinking to thirst is appropriate in many settings, it traditionally has not been considered a good indicator of body water needs during exercise in the heat at elevated sweat rates, as ad libitum water consumption often results in incomplete fluid replacement or voluntary dehydration (194, 220–226). The notion of voluntary dehydration, however, has been the subject of further analysis (188, 227, 228). Drinking behavior and fluid replacement are influenced by physiological, psychosocial, and environmental factors, experience and expectations, as well as issues related to fluid palatability, food intake, and gastric distension/discomfort (229–231). It has also been suggested that heat acclimation improves the relationship of thirst to body water needs by reducing the time to first drink, increasing the number of drinks consumed per heat exposure, and increasing mean volume per drink (225, 232), reducing voluntary dehydration by ~30% (223, 224, 233). Ultimately, fluid balance during exercise is a dynamic process influenced a several integrative factors. An overview of current hydration guidelines is presented in sect. 5.4.

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3.3. Sodium Balance

Sodium is the major determinant of plasma osmolality, which is an essential regulator of arginine vasopressin secretion and thirst perception. Under normal conditions, plasma sodium concentration is regulated between 135 and 145 mmol·L^{-1} (234). The regulation of sodium, however, must be integrated with the regulation of plasma volume, because changes in water volume alone have diluting or concentrating effects on bodily fluids. Aldosterone, a steroid hormone that is produced by the adrenal cortex, plays a central role in sodium regulation. Changes in plasma osmolality are directly sensed in the adrenal cortex, resulting in aldosterone inhibition or secretion during increased or decreased osmolality, respectively (235). During hyperosmotic dehydration, more water than sodium is lost, resulting in an increase in plasma osmolality (TABLE 1). The inhibition of aldosterone release causes less sodium to be reabsorbed in the distal tubule of the kidney nephron (236). Simultaneously, the increased osmolality causes the secretion of arginine vasopressin, leading to water conservation in the kidneys. The net effect is a decreased amount of excreted urine, with an increase in urine osmolality. These responses are complementary to restore sodium concentration and plasma osmolality.

3.4. Fluid and Sodium Disorders

The evaporation of sweat plays a central role in heat dissipation during exercise and/or passive heat exposure. Although sweating-induced water loss can be counteracted by increased fluid consumption and activation of the RAAS, most individuals do not fully compensate their fluid loss (194, 226, 237–239). Dehydration during prolonged exercise in warm and hot environments impacts on thermoregulatory function and performance (see sect. 5) and can lead to health issues. Acute dehydration is associated with decreased glycemic regulation, worsened mood, blunted blood pressure control, reduced cerebral blood flow during sympathoexcitation, and orthostatic intolerance (240). Although it is generally accepted that a compromise in hydration status of $\geq$2% body mass is detrimental to some aspects of cognitive function (i.e., attention, psychomotor performance and working memory) (241–243), a recent meta-analysis found cognitive performance not to be impaired by hypohydration of ~2% (range: 1.2 to 4.2%) (244). A recent systematic review also reported an inconsistent effect of dehydration within 1 to 3% of body mass loss on cognitive function, with only 13 of 26 studies demonstrating a negative influence on working memory, inhibitory control and attention (245). These findings, however, do not preclude impairment in cognitive function at greater levels of sustained hypohydration. Exercise-induced dehydration may also increase the risk for postexercise hypotension (246), due to decreased cardiac baroreflex sensitivity (247), which may subsequently lead to syncope or collapse after the cessation of exercise (248).

Some athletes intentionally dehydrate for competition. For example, weight category sports, such as wrestling, judo, boxing, taekwondo, and mixed martial arts, apply rapid weight loss interventions before competition weigh-in. A combination of dietary restriction and sweat-induced fluid depletion by heat exposure and/or exercise in vapor impermeable clothing are often employed (249–251). Although precompetition rapid weight loss is an effective way to increase the probability of competitive success (252), rapid rehydration is needed to allow for optimal exercise performance. A systematic review of 4,432 combat sport athletes reported hypohydration levels of up to 10% body mass loss before weigh-in (253). Another study found that on the morning of competition, >40% of combat athletes were hypohydrated based on a urine sample, highlighting the short time allocated to restore fluid balance and the risk associated with entering competition with suboptimal mental and physical performance capacity (254).

Exercise-induced dehydration induces intracellular and extracellular fluid loss in proportion to the loss of water and solutes. Given the hypotonic concentration of sweat relative to plasma (255), the reduction in plasma volume induces an increase in electrolyte concentration (i.e., hypertonic hypovolemia). Hypernatremia is an example of a hypertonic hypovolemic electrolyte disorder and is defined by a plasma sodium concentration $\geq$145 mmol·L^{-1} (256, 257). The prevalence of postexercise hypernatremia is relatively common among endurance athletes (>25%) given its direct relationship with exercise-induced dehydration (258, 259). Hypernatremia was also observed among 30–40% of swimmers performing a short (~1 min) maximal exercise bout, probably due to a shift of hypo-osmotic fluid from the extracellular to the intracellular compartment as exercise duration was too short to induce substantial fluid losses (260). While mild levels of hypernatremia do not lead to serious clinical symptoms, acute and severe levels of hypernatremia (>158 mmol·L^{-1}) are associated with hyperpnea, restlessness, lethargy, and even coma (261).

To avoid the risk of hypohydration, some athletes drink too much during endurance exercise events causing a dilution of circulating electrolytes (i.e., hypotonic hypervolemia). Hyponatremia is defined by a plasma sodium concentration of 135 mmol·L^{-1} or less (262), primarily due to an increase in total body water relative to the amount of total body exchangeable sodium. Hyponatremia can be asymptomatic or symptomatic. Asymptomatic hyponatremia has

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largely been detected in research studies collecting post-exercise blood samples in athletes, whereas symptomatic hyponatremia presents with mild, nonspecific symptoms (i.e., lightheadedness, nausea) (263–265) or more typically with headache, vomiting, and/or altered mental status (i.e., confusion, seizure) resulting from cerebral edema, which may progress to death (262, 266–269). Athletes with symptomatic hyponatremia should be immediately treated with hypertonic saline to reduce brain edema (267, 270, 271). Together with heat stroke, exercise associated hyponatremia is the most dangerous disorder for an athlete. The incidence of asymptomatic hyponatremia varies widely across sporting disciplines (272–274) and is dependent on the type and duration of activity, location of the event, athlete characteristics, and heat or cold exposure. Symptomatic hyponatremia is rare, with 1% incidence in 2,135 endurance athletes (compared with 6% asymptomatic hyponatremia) (275) and 0.1% incidence among 669 ultramarathon runners (276). Smaller individuals and those exercising at a slower pace are more prone to develop hyponatremia (267), probably due to the relatively larger drinking volume-to-plasma volume ratio and increased time to ingest fluid during exercise, respectively. Nonosmotic arginine vasopressin secretion is a key contributing factor to hyponatremia, as elevated arginine vasopressin concentrations stimulate the reabsorption of water from the kidneys, thereby further disturbing the balance between excessive fluid intake and fluid loss. Examples of arginine vasopressin stimuli associated with exercise include nausea/vomiting (277), interleukin-6 release (278), plasma volume contraction (279), hypoglycemia (280). and elevated body temperature (281).

3.5. Hydration Status

The fundamental principles of body water regulation provide the framework for using plasma osmolality as an index of hydration status (186). A reduction in total body water reduces both intracellular and extracellular volume. Since sweat is hypotonic relative to plasma, exercise-heat stress-mediated hypohydration induces plasma hypertonicity and hypovolemia that are proportionate to the water deficit (201). While there currently exists no consensual gold standard to evaluate hydration status (185, 195), plasma osmolality is considered under static hydration conditions to be the most precise and accurate hydration assessment technique (185, 282). However, the measurement of plasma osmolality via freezing point depression or vapor pressure depression osmometry requires invasive (i.e., venipuncture) sampling techniques and expensive analysis equipment. Alternatively, urine concentration reflects the renal response to alterations in plasma osmolality and is typically well correlated with plasma osmolality (195). Urinary indexes of hydration status such as osmolality, specific gravity, and color require measurement techniques that are more cost effective and offer faster response times than plasma osmolality. Urine samples are typically evaluated as first morning void or over a 24-h period, as well as immediately before and after exercise. However, urinary indexes may not accurately reflect hydration status before or following exercise in situations of rapid (re)hydration and isotonic and hypotonic hypohydration (195, 283). Hydration status may also by underestimated from urinary indexes during rapid (re)hydration following hypertonic hypohydration incurred via sweating, as significant urine production may already be occurring before fluid retention responses are well activated. Although urine osmolality analyzed using freezing point depression is considered the most accurate measure of urine concentration, it is subject to considerable (~30%) day-to-day biological variations, even when nutritional intake and exercise are controlled (282). Freezing urine samples may also alter the reliability of chemical analysis techniques (284, 285) as it generates urinary sediments (e.g., endogenous calcium oxalate dehydrate and amorphous calcium crystals) (285). This may indicate why urine osmolality decreases after freezing and thawing when determining it with freezing point depression but not with refractometry (286).

Hydration status is difficult to accurately determine from single samples (287) and thresholds between euhydration and hypohydration difficult to establish. For example, hypohydration has been suggested to occur at a urine osmolality of 586 (288), 716 (289), 830 (290), and 1,052 mosmol/kgH_2O (291). Urine concentration estimated by specific gravity evaluates the ratio of the density of urine to that of pure water at a constant temperature. It can be measured directly via gravimetry and indirectly using the refractive index (i.e., refractometry) or by change in pH of a polyelectrolyte (i.e., reagent strip). Although urine specific gravity via refractometry has been suggested to correlate with urine osmolality (287, 291, 292), others have shown that this relationship is not strong (293). Indeed, urine specific gravity is a technically less accurate method as it is influenced by both the number and molecular mass of solutes such as glucose, protein and urea, temperature, pH, and age, whereas urine osmolality is affected only by the number of particles (294, 295). Reagent strip urine specific gravity was shown to correlate with urine osmolality but not as strongly as with refractometry due to changes in pH (296). In pathological specimens, a weak correlation exists between urine osmolality and urine specific gravity measured by reagent strip in acidic or alkaline urines and those with elevated glucose, bilirubin, urobilinogen, protein, and ketones. Similarly, urine specific

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gravity measured by refractometry correlates poorly with urine osmolality in the presence of hemoglobin, ketones, or bilirubin (296). Among the urinary markers, urine color is probably the least sensitive marker (195).

Another potentially viable and less invasive pathway for monitoring hydration status lies with saliva and tear osmolality, relatively accessible fluids. Saliva osmolality has been suggested to track acute hydration changes during hypertonic hypovolemia as sensitively as urine osmolality (297). However, a lack of correlation between saliva osmolality and urine osmolality and specific gravity has also been shown during multiday events (298). The large inter- and intraindividual variability in saliva osmolality measurements reduce its accuracy (195). Although tear osmolality was shown to increase with dehydration and track alterations in plasma osmolality, as well as provide comparable results to urine specific gravity (299), additional validation research is required. Bioelectrical impedance analysis of body water is a noninvasive technique based on the resistance of a low amperage current (single or multiple frequencies) passed between skin electrodes, whereas the resistant magnitude varies inversely with tissue water and electrolyte content. Although bioelectrical impedance analysis is validated in euhydrated individuals, its application is of less use in dynamic conditions and with dehydrated individuals (186, 300, 301).

Given the difficulty in establishing hydration status due to measurement artifacts and precision discrepancies between assessment techniques, it has been suggested to evaluate and monitor hydration status on a daily basis to establish personalized reference ranges according to a set of relatively simple indexes (**FIGURE 5**) (302–304). These include changes in morning body mass following first morning void, urinary indexes from first morning void, and the sensation of thirst. While the lack of thirst sensation does not necessarily represent euhydration, the presence of thirst is indicative of hypohydration. The regular use of this approach will allow for developing personalized metrics regarding hydration thresholds and provide a more robust diagnosis of hydration status.

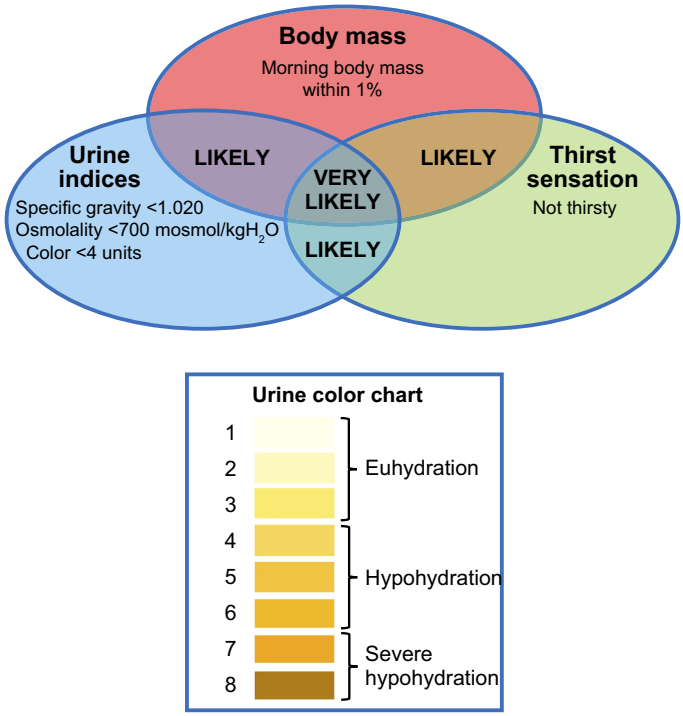

FIGURE 5. Daily hydration status assessment and monitoring diagram. The diagram combines relatively simple markers of hydration status: morning body mass, 1st morning urine void, and subjective thirst. As a single marker fails to provide adequate evidence for hydration status, the combination of 2 markers provides a likely indication of hypohydration and the convergence of all 3 provides a very likely indication of hypohydration. Adapted with permission from Refs. 302, 303.

4. HEAT STRESS AND AEROBIC EXERCISE PERFORMANCE

Prolonged high-intensity exercise is markedly impaired by an increase in the severity of the thermal environment, which is determined by the combination of ambient temperature, absolute humidity, solar radiation, and wind velocity (see sect. 2). The impairment is characterized by the exacerbated development of whole body hyperthermia, relative to exercise in temperate conditions, and the consequent reduction in time to exhaustion during constant work rate exercise (305–309), or progressive decrease in work rate during self-paced exercise (i.e., time trial) (310–317). Maximal aerobic power (i.e., $\dot{V}O_{2max}$), a key determinant of endurance performance (318), is also compromised under heat stress, in line with the severity of the environmental conditions and initial thermal strain (i.e., core and skin temperature) (319–323). The development of hyperthermia-induced fatigue is complex, with performance impairments involving the interplay of several physiological systems (146, 201, 324–327). The foremost mechanisms associated with impaired aerobic exercise under heat stress are linked to hyperthermia-mediated alterations in cardiovascular, central nervous system (CNS) and skeletal muscle function (**FIGURE 6**). The physiological responses associated with these alterations impact on perceptual responses, which can also affect performance by influencing motivation and the willingness to continue exercising in the heat. This section will examine how skin, muscle, and cerebral blood flow are affected during exercise in heat, describe the influence of hyperthermia on central neural drive and skeletal muscle function and metabolism, outline the influence of

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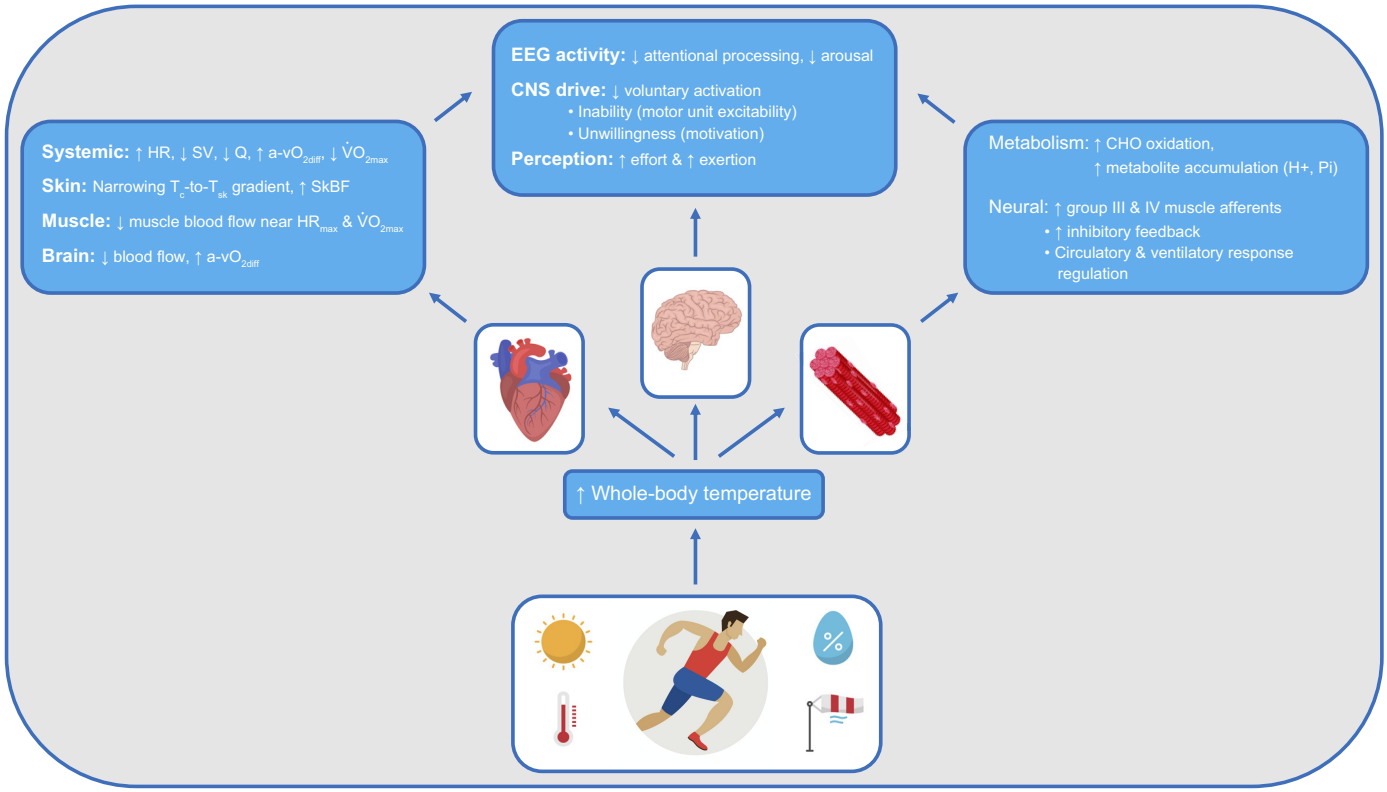

FIGURE 6. Schematic representation of the impact of exercise in the heat on cardiovascular, central nervous system (CNS) and skeletal muscle function associated with fatigue development. The rise in whole body (core, skin and muscle) temperature during exercise in the heat leads to a narrowing core-to-skin temperature (T_c-to-T_{sk}) gradient and a reflex increase in skin blood flow (SkBF), which contribute to increase heart rate (HR). As thermal strain develops, $\dot{V}O_{2max}$ progressively decreases while perceived exertion increases for any given work rate. When prolonged self-paced exercise is performed, the combination of increased thermal, cardiovascular and perceptual strain leads to a reduction in work rate. During constant work rate exercise muscle blood flow is well maintained until HR reaches ~95% of maximum (~90% $\dot{V}O_{2max}$). At this point, cardiac output ($\dot{Q}$) becomes compromised as stroke volume (SV) markedly decreases, resulting in volitional exhaustion, despite an increase in arterio-venous oxygen different (a-vO_{2diff}). Blood flow to the brain is compromised at elevated exercise intensities with hyperthermia; however, the metabolic rate of oxygen is preserved. Despite this preservation, brain activity [encephalography (EEG)] alterations occur in areas associated with the ability to inhibit conflicting attentional processing and the capacity to sustain mental readiness and arousal. Hyperthermia also influences CNS function during maximal voluntary contractions, by suppressing voluntary activation, either through an inability or unwillingness to contract exercising muscles. Occlusion of blood flow during such contractions increases muscle temperature and metabolite concentration, which alter motor unit excitability and impact on the perception of effort. Increased muscle temperature also enhances carbohydrate (CHO) oxidation and metabolite (H^+, hydrogen ions; P_i, inorganic phosphate) accumulation during high-intensity dynamic exercise. The increase in metabolic and mechanical disruption stimulates group III/IV muscle afferents, which provide inhibitory feedback to the CNS and contribute to regulate circulatory and ventilatory response.

thermal strain on $\dot{V}O_{2max}$, prolonged constant work rate and self-paced exercise, and examine the mechanisms linked to fatigue development under heat stress.

4.1. Adjustments in Cardiovascular Function

Lee and Scott (328) postulated over 100 yr ago that circulatory adjustments contribute to limit work capacity in the heat by "drafting blood away from the brain and the muscles to the skin." The cardiovascular response to prolonged aerobic exercise in the heat includes a redistribution of blood to cutaneous vascular beds for the purpose of heat dissipation, coupled with the maintenance of perfusion pressure and oxygen delivery to exercising muscles. As highlighted by Rowell (329), this "forces humans to deal with the two most powerful competing regulatory demands they ever face: the competition between skin and muscle for large fractions of cardiac output." The concept of competition or conflict between regulatory systems has also been suggested to represent commensalism, which is an integrated balance of regulatory control where one circulation benefits without substantially affecting the other (330). Notwithstanding, the development of thermal strain during exercise under heat stress provides a robust challenge to the cardiovascular system, with fatigue linked to adjustments in blood flow to the skin, active muscles, and the brain.

4.1.1. Skin blood flow.

An increase in skin temperature when exposed to environmental heat stress leads to a rise in skin blood flow

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mediated by peripheral (i.e., local endothelial, adrenergic and sensory response) (331) and central (i.e., systemic thermoregulatory response) pathways (332). The rise in skin blood flow acts to transfer more blood from the central circulation toward cutaneous vascular beds, where under favorable thermal gradients dry heat loss can occur. Once sweating is initiated, skin blood flow serves to deliver heat to the skin where it is removed through evaporation. Skin temperature therefore fluctuates in response to the balance between changes in blood flow and sweat evaporation (333, 334). When moderate to high-intensity exercise is initiated, a vasoconstrictor-mediated reduction in skin blood flow occurs, which contributes to provide additional blood to working skeletal muscles (335, 336). Shortly thereafter, cutaneous vasodilation is initiated to aid in the dissipation of metabolically generated heat. Splanchnic and renal vasoconstriction can contribute 600 to 800 mL·min^{-1} of blood to this response (337, 338). The internal temperature threshold at which active cutaneous vasodilation occurs is linked to exercise intensity, with an increase in work rate delaying the threshold relative to rest (**FIGURE 7**) (340–344). The shift in threshold for cutaneous vasodilation during exercise results in a lower skin blood flow for a given core temperature compared with rest. This response is exacerbated when exercising in cool compared with hot environmental conditions, as lower skin temperatures suppress the active vasodilator response to increasing internal temperature, thereby raising the temperature threshold for vasodilation (345). Factors such as time of day (346), menstrual cycle phase in females (347), and plasma osmolality (348, 349) have also been shown to influence the onset threshold for cutaneous vasodilation.r

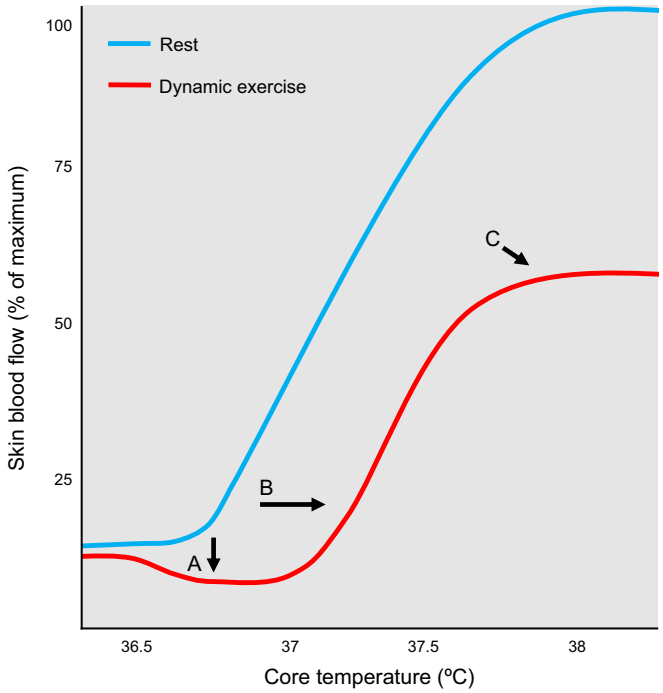

FIGURE 7. Skin blood flow response to hyperthermia at rest and during dynamic exercise. The response is influenced by cutaneous vasoconstriction at the onset of exercise ("A"), a shift in the core temperature threshold for initiating cutaneous vasodilation ("B"), and a leveling off, or plateau, at 50-60% of maximum skin blood flow above a core temperature of 38°C ("C"). Redrawn with permission from González-Alonso et al. (339) and Kenney et al. (330).

The magnitude of the skin blood flow response during exercise-heat stress is mediated by the core-to-skin temperature gradient, with a narrowing of the gradient leading to a reflex increase in skin blood flow (345, 350–352). Central (i.e., visceral and brain) thermoreceptors provide a stronger thermoregulatory drive for increasing skin blood flow at any temperature gradient than peripheral (i.e., skin) thermoreceptors. A 1°C increase in skin temperature accounts for 10 to 30% of the thermoregulatory drive for cutaneous vasodilation and sweating, whereas a 1°C increase in internal temperature mediates 70 to 90% of the response (41, 353, 354). It must be acknowledged, however, that changes in skin temperature are often larger than those of the core during exercise in the heat, providing changes in skin temperature significant influence on the thermoregulatory drive for cutaneous vasodilation and sweating. Under uncompensable heat stress where evaporative capacity is limited and ambient temperature is high, the core-to-skin temperature gradient may be reversed with skin temperature increasing above that of the core. In such conditions, an increase in skin blood flow would lead to an increase in core temperature through convective heat gain (338), effectively compromising thermoregulation (330).

During prolonged exercise in the heat, the rate of rise in skin blood flow markedly decreases after 20–30 min, particularly when core temperature approaches 38°C, reaching a virtual plateau at 50 to 60% of maximum flow capacity (**FIGURE 7**) (339, 351, 355). The reduced rate of rise in skin blood flow is attributable to an attenuation of vasodilator activity, as active vasoconstriction is progressively withdrawn with exercise (356). The plateauing effect occurs in response to the perfusion requirements of exercising muscles (357–359) and circulatory regulation (i.e., cardiac output and arterial blood pressure) (360, 361) taking precedence over thermoregulatory control.

An increase in skin temperature has been suggested to mediate fatigue during submaximal aerobic exercise under stress. The development of fatigue is purported to stem from the greater skin blood flow and cutaneous venous compliance associated with hot skin and the concomitant increase in cardiovascular strain (i.e., increased heart rate and decreased stroke volume

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compromising the maintenance of cardiac output and blood pressure under heat stress) (362–364). This premise is supported by early findings that a rapid lowering of skin temperature restored work capacity following volitional fatigue, in response to a reduction in heart rate (365, 366). A more recent study from Ely et al. (367) demonstrated that ~17% less work was performed during a 15-min self-paced cycling task in 40°C compared with 20°C conditions. The impairment was linked to the higher skin temperature (~5°C) and heart rate (~10 beats·min^{-1}) during the time trial in hot conditions, as end exercise core temperature was similar between trials (~38.2°C). This corroborates previous observations of a ~6.5% lower mean power output in elite cyclists undertaking a 30-min time trial in 32°C compared with 23°C conditions (316). The impairment occurred in conjunction with the maintenance of a higher skin temperature (~6°C) and heart rate (~4 beats·min^{-1}) in the hotter condition, as well as a higher rating of perceived exertion, despite core temperature increasing at a similar rate during the trials (**FIGURE 8**). These decrements in self-paced exercise performance are supported by studies in which volitional exhaustion during constant work rate exercise in uncompensable heat stress (e.g., when wearing encapsulated protective ensemble) coincided with an elevated skin temperature and marked cardiovascular strain, yet relatively low core temperatures (<38.5°C) (368–371) compared with studies without encapsulation (>39°C) (363, 372–374). Others have also demonstrated exhaustion to occur earlier when skin temperature was elevated using a water-perfused suit, despite similar core temperatures and heart rates at exhaustion (363, 364). These observations highlight the impact of a skin temperature-mediated blood volume redistribution to the periphery on cardiovascular function and exercise capacity and performance in the heat, which reiterates the intimate relationship between thermoregulation and circulatory function.

4.1.2. Skeletal muscle blood flow.

Skeletal muscle blood flow requirements during physical activity are linked to exercise intensity, with an increase in oxygen demand matched by an increase in oxygen delivery and perfusion pressure (375–377). The regulation of these circulatory responses is mediated by an increase in muscle sympathetic nerve activity (378, 379) and functional sympatholysis (i.e., inhibition of sympathetically-mediated vasoconstriction in active muscles) (380, 381). Elevated tissue and blood temperatures also contribute to increase active muscle blood flow through metabolic and thermal stimuli inducing the release of erythrocyte-derived adenosine triphosphate (ATP), a potent vasodilator (382–384). Muscle blood flow in trained individuals can reach 3 to 4 L·kg^{-1}·min^{-1} during maximal exercise with a small muscle mass (e.g., forearm and isolated leg) (385). These high levels of blood flow are not attained, however, when performing whole body maximal exercise (e.g., cycling, running and rowing) (386, 387) as the cardiac output required to perfuse active skeletal muscles (estimated as ~15 kg from a total arm and leg muscle mass of 25–28 kg) would exceed 45 L·min^{-1}, which is beyond cardiac pumping capacity, even for elite endurance athletes (378, 388, 389). The limits imposed on the ability to increase cardiac output during whole body maximal exercise are mediated by arterial and cardiopulmonary baroreflexes, as well as muscle metaboreflexes, which increase peripheral

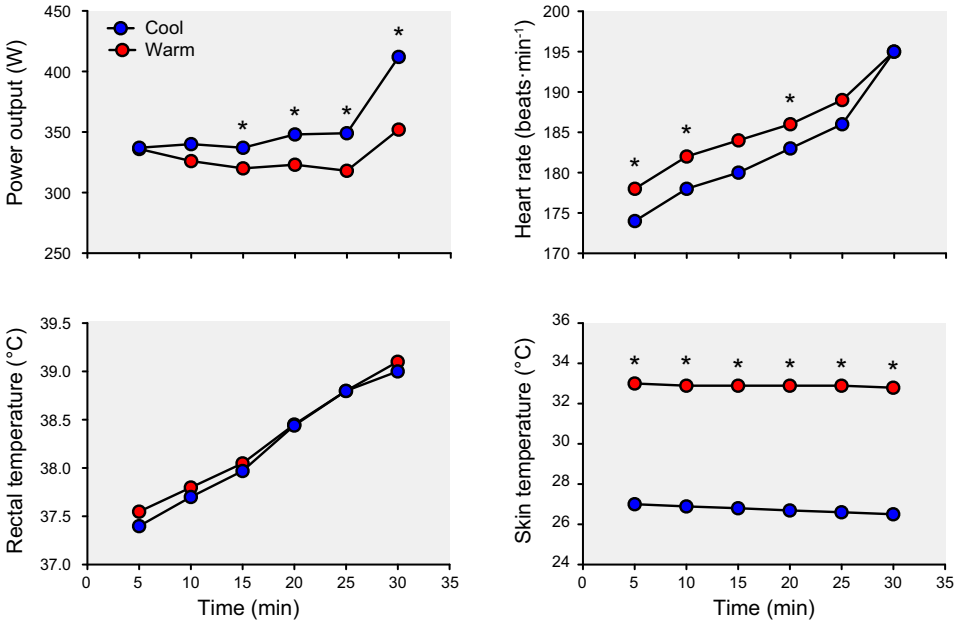

FIGURE 8. Power output, heart rate, rectal, and skin temperature during a 30-min cycling time trial in Hot [32°C and 60% relative humidity (RH)] and Cool (23°C and 60% RH) conditions in elite road cyclists. These data highlight the impact of an elevated skin temperature on exacerbating the cardiovascular response, characterized by an elevated heart rate, despite the maintenance of a lower work rate (i.e., power output) and similar rectal temperature. *Significant difference between conditions ($P < 0.05$). Redrawn with permission from Tatterson et al. (316).

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resistance by augmenting sympathetic activity and restricting hyperemia in the active musculature (339, 390–395). This circulatory control mechanism prevents muscle vascular conductance from reaching unsustainably elevated levels that would significantly compromise the regulation and maintenance of arterial blood pressure.

During submaximal exercise under heat stress, the increased requirement and displacement of blood flow to cutaneous vascular beds provides a significant challenge to the cardiovascular system, which must also maintain skeletal muscle perfusion and oxygen delivery (338, 396). Nevertheless, muscle blood flow can be maintained during prolonged submaximal exercise in the heat (357, 372, 397, 398). In one of the first studies to examine exercising muscle blood flow in the heat, Savard et al. (358) manipulated skin temperature with a water-perfused suit during one-legged knee extension (20–25% $\dot{V}O_{2max}$) and two-legged cycling (50–60% $\dot{V}O_{2max}$). Both exercise modalities were performed during three consecutive 25-min bouts of exercise where no water, hot water (45°C), and cold water (2°C) circulated through the suit, respectively. The authors reported that when additional heat stress was imposed from 25 to 50 min, blood flow to active skeletal muscles was maintained in both exercise modalities. Others have also demonstrated an uncompromised cardiac output and maintained skeletal muscle blood flow during prolonged constant work rate exercise to volitional fatigue under heat stress (357, 372, 398). These observations were taken to indicate that the cardiovascular system is capable of meeting the blood flow requirements of both the skin and active muscles during prolonged submaximal exercise in hot environmental conditions. It must be noted, however, that maximal or near maximal heart rate was not attained at exhaustion in these studies (357, 372, 398), which appears to be the point at which systemic and peripheral (i.e., skin and exercising muscles) blood flow and oxygen delivery decrease.

Indeed, González-Alonso and Calbet (399) demonstrated during brief intensive exercise (i.e., cycling at 80% of peak power output) that volitional exhaustion was associated with a reduction in systemic and exercising muscle blood flow and oxygen delivery and uptake when starting with either high or normal skin and core temperatures. These reductions were exacerbated under heat stress, which accelerated the decline in mean arterial pressure and cardiac output, ultimately deceasing $\dot{V}O_{2max}$. It has also been shown that systemic oxygen delivery is blunted at intensities below $\dot{V}O_{2max}$ by the attainment of a plateau in cardiac output during incremental exercise and a decrease in cardiac output during constant work rate exhaustive exercise in cool conditions (391). Systemic and exercising muscle blood flow, along with oxygen delivery, matched the rise in oxygen uptake from 50 to 90% $\dot{V}O_{2max}$ during incremental exercise. However, beyond 90% $\dot{V}O_{2max}$ a levelling off in oxygen delivery occurred that attenuated the rate of rise in oxygen uptake, despite maximal increases in arteriovenous oxygen difference (a-vO_{2diff}) and heart rate (**FIGURE 9**). These findings were corroborated during supramaximal intensity cycling, and despite the greater metabolic energy requirements of such exercise, plateaus in cardiac output and muscle vascular conductance were noted at similar levels of oxygen delivery to that of maximal exercise (400). These observations indicate an inability of the cardiovascular system to sustain a linear increase in oxygen delivery to exercising muscles, which under heat stress occurs at a lower absolute work rate due to an accelerated decline in cardiac output and mean arterial pressure, leading to a decrease in $\dot{V}O_{2max}$. Although the relatively brief and intense nature of maximal and supramaximal exercise in the heat may be more conducive to reaching cardiovascular limitations, motivated individuals have also been shown to reach such limitations during prolonged submaximal exercise to exhaustion (309, 363, 374, 401). For example, a manipulation of starting esophageal temperature (35.9, 37.4, and 38.2°C) during exhaustive cycling at 60% $\dot{V}O_{2max}$ in 40°C led to a similar final heart rate: 98–99% of maximum (363). The increase in heart rate, along with the decline in stroke volume and cardiac output, was graded in proportion to the magnitude of hyperthermia, with time to exhaustion inversely related to starting esophageal temperature (63, 46, and 28 min). These data highlight the significance of hyperthermia in exacerbating the development of cardiovascular strain during constant work rate exercise to volitional fatigue and the prerequisite increase in maximum or near-maximum heart rate required to reduce systemic oxygen delivery. Conversely, in the absence of cardiovascular limitations where heart rate is not maximal or near-maximal upon reaching volitional exhaustion, a lack of motivation and/or elevated perceived exertion in response to inhibitory afferent feedback appears to mediate the cessation of exercise.

4.1.3. Cerebral blood flow.

The development of hyperthermia during dynamic exercise in the heat is associated with a progressive reduction in cerebral blood flow relative to levels maintained in cooler conditions, whether measured via transcranial Doppler ultrasound [middle cerebral artery mean blood velocity (MCA_V)] (401, 402), the Kety-Schmidt technique (venous drainage) (403, 404), or near-infrared spectroscopy (i.e., tissue oxygenation) (374, 405). The reduction in cerebral blood flow toward resting baseline levels has been

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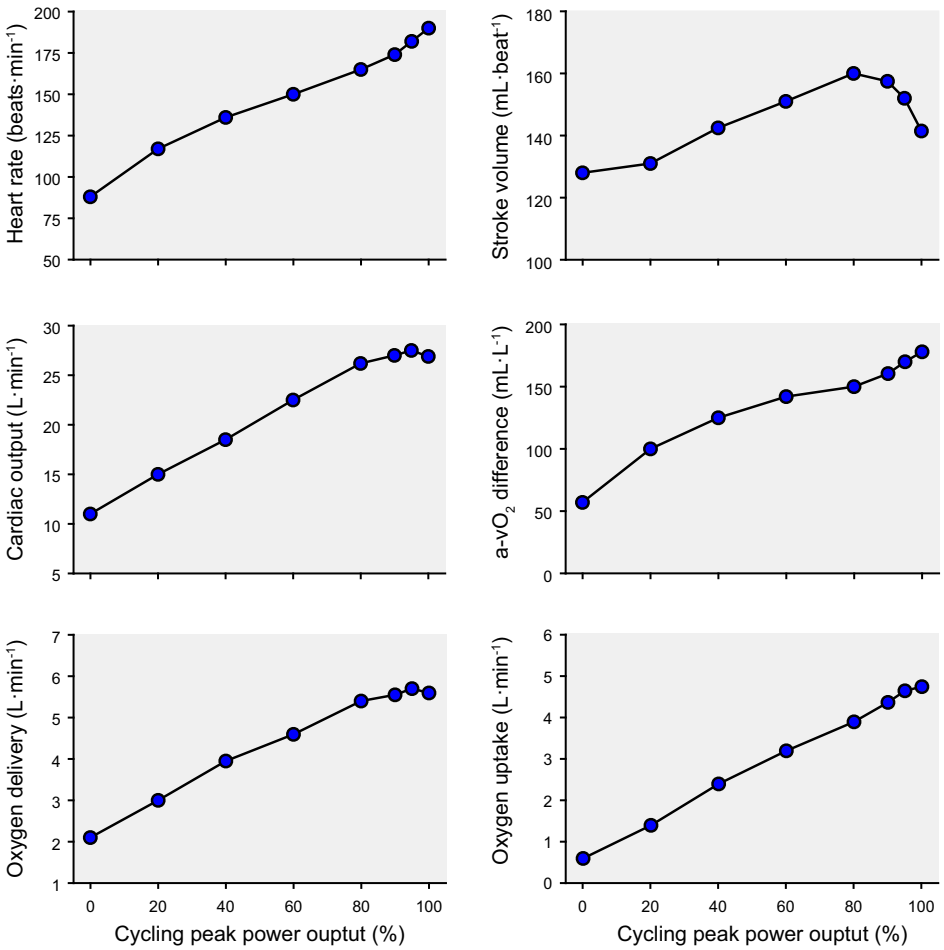

FIGURE 9. Heart rate, stroke volume, cardiac output, systemic arterio-venous oxygen difference (a-vO_{2diff}), oxygen delivery, and uptake during incremental exercise to exhaustion in temperate conditions (20°C) plotted against the relative increase in power output. These data indicate that systemic oxygen delivery is blunted beyond ~90% $\dot{V}O_{2max}$ by the attainment of a plateau in cardiac output, which attenuates the rate of rise in oxygen uptake despite maximal increases in a-vO_{2diff} and heart rate. Redrawn with permission from Mortensen et al. (391).

attributed to an increase in cutaneous blood flow, decreases in cardiac output and arterial blood pressure, as well as hyperventilation-induced hypocapnia [i.e., decrease in arterial carbon dioxide pressure ($Paco_2$)] (401–404). Nybo and Nielsen (401) were among the first to examine cerebral blood flow via transcranial Doppler ultrasound during constant work rate exercise to exhaustion under heat stress. The authors noted that MCA_V decreased in parallel with the rise in body core temperature, whereas a stable elevation in MCA_V was maintained during steady-state exercise in cooler conditions. In contrast, MCA_V was shown to decrease regardless of ambient conditions when performing prolonged (i.e., 45–60 min) high-intensity self-paced exercise, albeit to a greater extent in hot compared with cool conditions (402). The decrease in cerebral blood flow observed during self-paced exercise was suggested to stem from the maintenance of an elevated relative exercise intensity, resulting in a comparable hyperventilation-induced hypocapnia response. While cerebral autoregulation has historically been purported to ensure the maintenance of blood flow to the brain within a range of mean arterial pressures between 60 and 150 mmHg (406), even during moderate intensity exercise (407), it is increasingly becoming apparent that this range is much narrower and within 5 to 25 mmHg of resting values (408, 409). Moreover, it has been shown that intense and exhaustive exercise impairs cerebral blood flow control (410, 411). Thus, given the elevated intensity sustained during self-paced exercise (314, 412) and the progressive increase in relative intensity occurring during constant work rate exercise in hot compared with cool conditions (319, 320), a ventilatory-mediated decrease in $Paco_2$ may influence cerebral vascular tone and alter the relationship between arterial pressure and cerebral blood flow under heat stress (408).

The reduction in cerebral blood flow during strenuous exercise in the heat has been suggested to influence central neural drive to exercising muscles in response to a compromise in oxygen delivery to the brain (401, 413, 414). However, the development of hyperthermia during such exercise is associated with an enhanced cerebral metabolism (405), manifested by a compensatory increase in cerebral oxygen extraction (415). For example, an esophageal temperature of ~39.5°C during

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exercise in uncompensable heat stress, relative to ~38°C in cool conditions, resulted in an ~18% lower cerebral blood flow at the end of exercise (403). The decrease was accompanied by a ~23% increase in a-vO_{2diff} and a ~7% increase in cerebral metabolic rate. The increase in cerebral oxygen uptake was attributed to the Q_{10} temperature coefficient but also to increased levels of stress and mental exertion. In line with this hypothesis, electroencephalography activity measurements indicate that a rise in core temperature and rating of perceived exertion are associated with a decrease in frontal lobe β-activity during exhaustive exercise under heat stress (416–419). Similar findings were reported following the completion of a self-paced time trial in the heat (420) and suggested to represent a suppression of arousal, with potential links to the development of fatigue (417–419). When measured during prolonged self-paced exercise, both α- and β-activity decreased in hot relative to cool conditions across the frontal (F3 and F4) and central (C3 and C4) areas (421). Elevated α-activity in these areas is associated with the capacity to maintain attention, mental readiness and relaxed focus (422–424), whereas β-activity is linked to wakefulness, mental activity and cortical arousal (424, 425). Prolonged high-intensity exercise in the heat therefore seems to induce cerebrocortical activity alterations in areas of the brain associated with the ability to inhibit conflicting attentional processing and the capacity to sustain mental readiness and arousal. Whether these alterations support the premise that an exacerbated reduction in cerebral blood flow under heat stress mediates a performance decrement via central inhibition, particularly given the robust capacity of the cerebral vasculature to extract oxygen, requires further elucidation.

4.2. Central Neural Drive

The role of the CNS in regulating exercise performance under heat stress spans from afferent input influencing the drive or willingness to continue exerting effort (i.e., motivation), to hyperthermia downregulating skeletal muscle recruitment. Brück and Olschewski (426) provided the impetus to investigate these pathways after postulating that body temperature affected certain physiological parameters thought to counteract motivation. More specifically, the authors suggested that increasing levels of hyperthermia influenced the interaction of circulatory, thermal, and muscular discomfort, which progressively reduced the drive to exercise. Nielsen et al. (357, 372) subsequently proposed that hyperthermia per se, rather than circulatory failure, was the critical factor causing exhaustion during constant work rate exercise under heat stress. The attainment of a high core temperature was suggested to influence the CNS by reducing mental drive (i.e., motivation) for motor performance. This conclusion was drawn as daily volitional fatigue during a 9- to 12-day exercise-heat acclimation regimen coincided with a final core temperature of ~39.7°C (372). Interestingly, cardiac output and muscle blood flow were maintained at the point of fatigue, likely in response to heart rate only increasing to ~160 beats·min^{-1}. The authors further demonstrated that the capacity to generate force during a brief (3–5 s) maximal voluntary isometric contraction (MVC) of the knee extensors and elbow flexors was unaltered when measured immediately after exhaustion (372). Conversely, force production was impaired in a subsequent study during a sustained (120 s) MVC conducted after volitional fatigue in the heat, relative to a contraction performed after steady-state exercise in cool conditions (373). The impairment was attributed to hyperthermia-induced central fatigue, a progressive reduction in voluntary activation mediated by the rise in whole body temperature. It was also postulated that an elevated brain temperature might trigger inhibitory signals in temperature-sensitive areas of the hypothalamus to decrease motor activity (425, 427, 428).

Using active and passive heating, others have shown that a progressive increase in core temperature is paralleled by a gradual decrease in voluntary activation and force production during 5 to 30 s MVCs (429–431), with the ability to produce force reincreasing as core temperature returns toward baseline with passive cooling (429, 430). Conversely, local heating of the thigh was shown to impair endurance capacity in the knee extensors during a sustained contraction at 70% MVC through an inability to maintain maximal muscle activation, whereas brief maximal force production was unaffected (432). It was also shown that passive heating to a rectal temperature of 39.5°C did not deleteriously influence torque output during maximal voluntary isokinetic contractions of the knee extensors performed at three different velocities (i.e., 60, 120, and 240°·s^{-1}) (433). Exercise-induced hyperthermia to a tympanic temperature of ~40°C also failed to influence maximal or endurance isokinetic contractions at 240°·s^{-1} (434). The neuromuscular impairments associated with hyperthermia-induced central fatigue therefore seem to occur during static but not dynamic contractions, sustained MVCs in particular, over the time course of whole body hyperthermia, rather than at the attainment of a specific internal temperature.

Although decrements in central neural drive have been linked with the development of hyperthermia (373, 430, 431, 435–437), it has also been shown that voluntary activation and force production capacity are similarly reduced during a sustained (20 s) MVC following self-paced exercise (i.e., 40 km time trial) in hot and cool conditions (438). The postexercise decline in voluntary

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activation accounted for ~20% of the decrease in total force production, indicating that the 0.8°C higher rectal temperature at time trial completion in the heat did not exacerbate central fatigue (438). In a separate study isolating the effect of hyperthermia from exercise, force production capacity of the knee extensors was shown to decline at a faster rate following exhaustive cycling in the heat (rectal temperature: 39.8°C), compared with passive heating (rectal temperature: 39.5°C) (439). Voluntary activation was similarly reduced between conditions, however, with the extent of decline maintained throughout the sustained (45 s) MVC. These studies indicate that the loss of force production capacity originates from both central and peripheral fatigue factors, with the combination of heat stress and prior contractile activity (i.e., exercise) exacerbating the rate of decline. Using transcranial magnetic stimulation, Todd et al. (440) attempted to localize the site of voluntary activation failure by passively heating participants to an esophageal temperature of 38.5°C and assessing brief (2–3 s) and sustained (2 min) MVC performance. Hyperthermia-induced decrements in voluntary torque and cortical activation of the elbow flexors were observed during both contractions, with greater decreases noted during the sustained MVC. It was also noted that peak muscle relaxation rate during the sustained contraction, the steepest rate of decline of torque in the silent period immediately following motor cortex stimulation, was ~20% faster than at baseline (i.e., normothermia). This led the authors to suggest that the greater central fatigue observed during longer contractions may be linked to a failure in voluntary drive to account for temperature-related adjustments (i.e., increase) in muscle contractile function. In essence, although high motor unit firing rates may be transiently attained during brief MVCs, these elevated rates may not be sustained during prolonged contractions (440). To further investigate this premise, Périard et al. (431) actively and passively heated participants from baseline rectal and muscle (vastus lateralis) temperatures of 37.1 and 35.3°C, to 38.5 and 38.7°C, and then on to 39.4 and 39.3°C, respectively. Both active and passive moderate hyperthermia increased peak muscle relaxation rate following transcranial magnetic stimulation when brief (5 s) and sustained (30 s) MVCs were performed. However, only the increase from moderate to severe passive hyperthermia further increased relaxation rate, albeit without decreasing force production capacity to a greater extent than severe active hyperthermia. It was therefore concluded that centrally mediated rates of activation are sufficient to overcome both active and passive hyperthermia-induced increases in peak muscle relaxation, as these fall within physiologically relevant motor unit firing rate ranges (i.e., 10–30 Hz).

Most of the research investigating the impact of heat stress on the central activation of skeletal muscle has focused on neuromuscular responses during maximal voluntary isometric contractions and evoked twitch characteristics following passive and/or exercise-induced hyperthermia (373, 430, 431, 435–441). It is important to note, however, that isometric exercise involves partial to complete occlusion of blood flow to active muscles depending on the intensity of contraction (442, 443), which further increases muscle temperature and stimulates chemoreflexes and mechanoreflexes (444, 445). Afferent stimulation of these reflexes increases muscle sympathetic nervous activity (446), which can alter motor unit excitability, modifying the relationship between central neural drive, motor unit recruitment, and firing rate coding (447, 448). Impairment of skeletal muscle function during isometric exercise under heat stress may therefore relate to a failure in the peripheral transmission of neural drive at any level from cortical activity to sarcolemma depolarization (435). Afferent stimulation resulting from the occlusion of blood flow during a prolonged isometric MVC and the accumulation of metabolites associated with muscular contractions may also influence motivation (449). A concerted effort is required to maintain force production during a prolonged MVC, coupled with a willingness to tolerate discomfort and pain. Minor discomfort is sensed at the onset of a contraction, developing into severe discomfort and pain over time that alters the perception of sensations in the contracting musculature (450). Consequently, mental fatigue, which involves tiredness, limited attention span, and an aversion or decreased commitment to continuing a task or activity (451, 452), may contribute to decrease voluntary muscle activation, particularly after prolonged passive and active heating. Conscious signals originating from both central and peripheral afferent pathways could mediate behavior and reduce motivation to minimize discomfort (453), leading to the abandonment of a task in which the energetic demands (i.e., effort) outweigh the perceived benefits of continued performance (454). A lack of motivation may thus lead to inadequate central neural drive to solicited motor neurons, resulting in a loss of force (455). The loss of force production capacity during isometric exercise under heat stress may therefore represent a psychophysiological phenomenon, wherein an integrated response related to both an inability and unwillingness to exercise regulates performance. Whether such a response also influences prolonged dynamic exercise in the heat remains a topic of contention.

Brain neurotransmitters (i.e., serotonin, dopamine, and norepinephrine) have been implicated in the control of thermoregulation and the potential development of central fatigue (456, 457). On one hand, dopamine appears

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to display ergogenic properties by reducing perceived exertion and thermal discomfort during self-paced exercise at elevated work rates in the heat (458, 459). On the other hand, norepinephrine seems to produce a negative effect on performance (460), while serotonergic manipulation fails to influence the development of fatigue (461, 462). As such, it appears unlikely that a particular neurotransmitter system mediates the delay or onset of fatigue during exercise in the heat (326, 463, 464). Notwithstanding, fatigue is a complex phenomenon influenced by several factors of both central and peripheral origin and it is possible that different neurotransmitter systems interact with thermal, cardiovascular, and metabolic function to modulate fatigue. Further integrative research is required in this area.

4.3. Skeletal Muscle Function and Metabolism

Elevations in muscle temperature lead to alterations in skeletal muscle function and metabolism that affect brief maximal and prolonged submaximal exercise performance differently. An increase in muscle temperature is known to enhance acute explosive exercise performance such as sprinting and jumping (465–467) via improvements in metabolic and contractile function, nerve conduction velocity, and conformational changes associated with muscle contraction (468–470). In contrast, prolonged exercise in the heat increases muscle glycogen utilization and anaerobic metabolism, causing greater accumulation of ammonia and muscle lactate (471–476). Work at high glycolytic rates is also associated with the release of force-depressing hydrogen (H^+) and inorganic phosphate (P_i) ions (469, 477–480). Temperature-induced impairments in sarcoplasmic reticulum function and structural damage compromising sarcoplasmic reticulum calcium (Ca^{2+}) ion regulatory capacity may also influence skeletal muscle force production (469, 481). The metabolic and mechanical alterations occurring within the exercising muscles stimulate group III/IV muscle afferents, which relate the level of perturbation and fatigue to the CNS (482–484). This inhibitory neural feedback has been suggested to influence the perception of exertion and contribute to the development of fatigue during prolonged intense exercise in temperate conditions by decreasing central motor drive (485–488). Along with feedforward regulation from central command (489, 490), group III/IV muscle afferent feedback has been linked to the regulation of autonomic ventilatory and circulatory responses during exercise (491, 492). As such, neural feedback regarding the level of perturbation within the skeletal muscle milieu during prolonged exercise in the heat may exert an inhibitory influence on central motor drive and impact on the regulation of ventilatory, circulatory, and metabolic responses. The extent of this inhibitory influence remains to be determined, however, as it has been suggested to diminish during exercise in extreme environments (e.g., heat and hypoxia), effectively outweighed by direct disturbances to the CNS (e.g., hyperthermia and hypoxemia) (485).

Fink et al. (475) first demonstrated that muscle glycogen utilization was ~76% greater and blood lactate concentration twice as high following intermittent cycling in hot (41°C) compared with cold (9°C) conditions. The increased rate of glycolysis was attributed to a thermal strain-mediated reduction in muscle blood flow and oxygen delivery. A number of other potential mechanisms have since been suggested to augment muscle glycogenolysis through stimulation of phosphorylase activity and other key glycogenolytic and glycolytic enzymes, including elevated muscle temperature (i.e., Q_{10} effect), decreased energy status (i.e., degraded total adenine nucleotide pool and increased free adenosine monophosphate), and enhanced sympathoadrenal response (i.e., elevated circulating epinephrine) (472, 493–497). Conversely, some studies have not observed increased intramuscular glycogen utilization during exercise under heat stress (357, 498–500), which may in some circumstances relate to methodological issues (e.g., preexercise glycogen levels, exercise intensity) (501). Others have also shown that carbohydrate oxidation and muscle glycogenolysis are lowered when the rise in whole body temperature is attenuated during exercise in cooler environments (476, 502) or by heat acclimation (397, 473, 500, 503) and when external cooling is provided (504). This suggests that hyperthermia is a potent modulating factor for increasing carbohydrate metabolism. Notwithstanding, glycogen depletion is not considered a primary factor limiting endurance exercise in the heat (324, 505–507), as high-intensity aerobic exercise may be performed for extended periods (~60 min) without muscle glycogen depletion attenuating performance (508–511). However, protracted exercise performed at a variable work rate results in excessive muscle glycogen utilization (512), and considering that the oxidation rate of ingested carbohydrate is reduced when exercising in the heat (513), it may be speculated that glycogen depletion could be accelerated and performance impacted upon during prolonged aerobic exercise in moderate heat. Moreover, the greater reliance on carbohydrate metabolism when exercising at a given work rate in hot versus temperate environmental conditions is typically associated with markers of increased relative exercise intensity, such as a higher heart rate, blood lactate, respiratory exchange ratio, and rating of perceived exertion, despite systemic oxygen uptake remaining similar (471–474, 500, 514, 515). It is also well established that

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a rise in exercise intensity mediates an increase in glycogenolysis and carbohydrate oxidation and decrease in fat oxidation (516–519). This progressive shift in energy substrate mobilization and utilization during exercise in hot environmental conditions may represent the maintenance of a gradually increasing relative exercise intensity, mediated by a hyperthermia-induced decrease in maximal aerobic capacity (see sects. 4.4–4.6).

4.4. Maximal Aerobic Power in the Heat

Rowell (338) previously highlighted that $\dot{V}O_{2max}$ is unaltered during brief (<15 min) incremental exercise undertaken in hot conditions, as regional vasoconstriction allows for redistributing a sufficient fraction (80–85%) of cardiac output to working muscles. The similarity in response between hot and cool environments stems from the capacity to achieve maximal cardiac output at similar work rates, despite marginally greater levels of whole body hyperthermia in the heat. Indeed, when core temperature remains <38°C and skin temperature does not exceed ~35°C during incremental exercise in the heat, $\dot{V}O_{2max}$ remains similar to cool conditions (314, 319, 520, 521). When preceded by active or passive whole body heating, however, a marked decrease in maximal cardiac output occurs during exhaustive exercise in the heat, which reduces $\dot{V}O_{2max}$ (**FIGURE 10**). The decrement in maximal cardiac output results from a lower stroke volume at the attainment of maximal heart rate at what is typically a submaximal work rate, forcing the cardiovascular system to a premature functional limit (522, 523). The traditional mechanism via which stroke volume was suggested to decrease under heat stress lay with the redistribution and pooling of blood in the periphery (i.e., cutaneous circulation), which decreased central blood volume and concomitantly ventricular filling pressure and end-diastolic volume (338, 352, 524). A more contemporary hypothesis proposes that the reduction in stroke volume is primarily related to shorter ventricular filling time and possibly filling pressure, consequent to a hyperthermia-mediated increase in heart rate compared with when exercising at a similar work rate in cool conditions (i.e., normothermia) (525–529). The tachycardic response in the early stages of exercise in the heat is suggested to stem from a thermoregulatory-mediated rise in cutaneous blood flow (526, 530). As thermal strain develops, the increase in heart rate has been attributed to withdrawal of parasympathetic outflow and increased cardiac sympathetic neural activity in response to baroreflex modulation and/or a hyperadrenergic state (531–536), along with the direct effect of blood temperature on the sinoatrial node (i.e., cardiac pacemaker) (537–542).

The reduction in $\dot{V}O_{2max}$ occurring during incremental exercise to exhaustion under heat stress following preheating has been extensively studied (320–323, 543). Arngrimsson et al. (319) demonstrated a proportional decrease in $\dot{V}O_{2max}$ in relation to increases in mean body temperature (= [esophageal × 0.87] + [mean

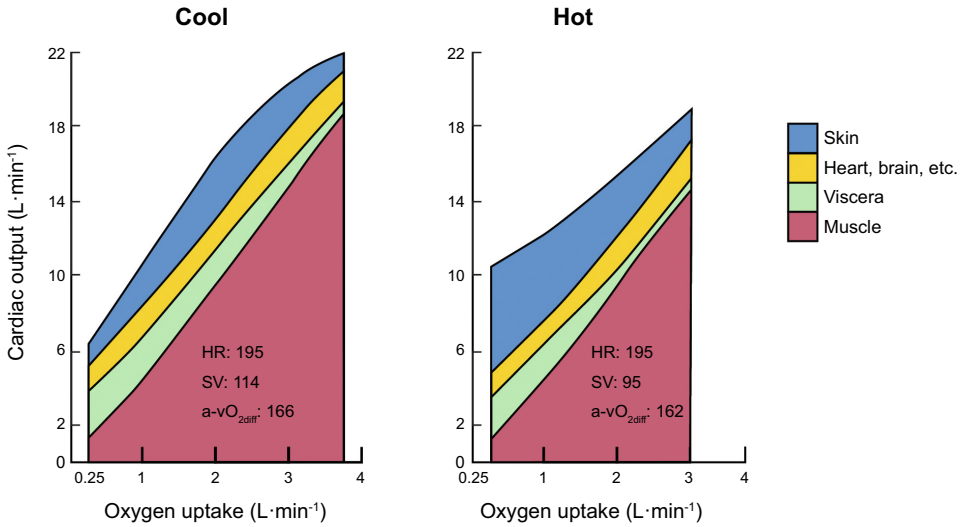

FIGURE 10. Estimated distribution of cardiac output during incremental exercise in temperate (~26°C) and hot (~43°C) environments and its consequent influence on maximal aerobic capacity. Incremental exercise to exhaustion in the heat following whole body heating is associated with a greater redistribution of cardiac output to the skin and an elevated heart rate for any given level of oxygen uptake (i.e., work rate). The elevated heart rate response is influenced by withdrawal of parasympathetic outflow, increased cardiac sympathetic neural activity, and the direct effect of blood temperature on the sinoatrial node. The outcome of this elevation in heart rate is the attainment of maximum heart rate at a lower work rate than in cool conditions, which coupled with a lower stroke volume, leads to decrease in maximal cardiac output, forcing the cardiovascular system to a functional limit at what is typically a submaximal work rate. HR, heart rate (beats·min−1); SV, stroke volume (mL·beat−1); a-vO_{2diff}, arterio-venous oxygen difference (mL·L−1). Redrawn with permission from Rowell (338).

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skin × 0.13]) in both men and women (**FIGURE 11**). These increases in mean body temperature were associated with exercise or passive preheating in ambient temperatures of 35, 40, and 45°C, leading to ~4, ~9, and ~18% reductions in $\dot{V}O_{2max}$, relative to 25°C. Unlike exercise at altitude where $\dot{V}O_{2max}$ is acutely decreased in relation to the severity of the hypoxic stimulus (544, 545), the decrease in $\dot{V}O_{2max}$ under heat stress appears to occur progressively in response to the rise in whole body temperature, not the prevailing climatic conditions per se. It has also been shown that an elevated skin temperature alone does not compromise $\dot{V}O_{2max}$. Utilizing a water-perfused suit, Trangmar et al. (521) manipulated skin (+6°C) and whole body (skin: +6°C, core: +1°C) temperature before participants undertook an incremental cycling test to exhaustion. Relative to a control condition, whole body hyperthermia decreased $\dot{V}O_{2max}$ by ~8%, whereas an increase in skin temperature alone did not affect maximal aerobic power. This observation reinforces the notion that a marked rise in whole body thermal strain is required to elicit a decrement in $\dot{V}O_{2max}$.

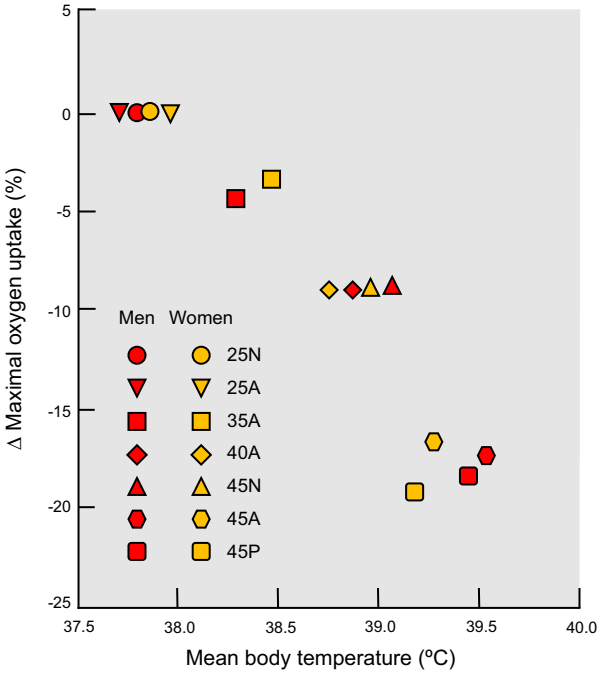

FIGURE 11. Proportional relationship between changes in $\dot{V}O_{2max}$ and mean body temperature at exhaustion in 7 conditions: *1*) 25°C without warm-up (25N); *2*) 25°C with a 20-min warm-up at ~33% of control $\dot{V}O_{2max}$ (25A); *3*) 35°C with warm-up (35A); *4*) 40°C with warm-up (40A); *5*) 45°C without warm-up (45N); *6*) 45°C with warm-up (45A); and 7) 45°C with passive preheating to elevate core temperature to the same extent as 45A (45P), all of which in 50% relative humidity. These data indicate that the reduction in $\dot{V}O_{2max}$ under heat stress is associated with whole body temperature, rather than the prevailing ambient conditions per se. Redrawn with permission from Arngrimsson et al. (319).

4.5. Constant Work Rate Exercise in the Heat

Prolonged constant work rate exercise (e.g., 50–75% $\dot{V}O_{2max}$) in temperate conditions leads to cardiovascular drift, a time-dependent phenomenon characterized by a progressive change or drift in certain cardiovascular responses after 10 to 15 min of exercise (337, 352, 546). This phenomenon is characterized by an increased heart rate and oxygen uptake, decreased stroke volume, and diminished mean arterial and pulmonary pressure, while cardiac output remains constant. Under heat stress, these adjustments in cardiovascular function are exacerbated, with the increase in heart rate strongly correlated to the rise in rectal temperature when exercising at 60% ($r = 0.83$) and 75% ($r = 0.68$) $\dot{V}O_{2max}$ in 40°C and 50% RH (309). The high core temperatures (e.g., 39.5 to 40°C) reached at volitional fatigue (i.e., exhaustion) during such exercise in laboratory settings often coincide with considerable cardiovascular strain (i.e., heart rate >95% of maximum) (363, 396, 418, 439). Previous studies have suggested that fatigue was the result of attaining a "critically" elevated core temperature (363, 372, 373, 401). This concept has been the focus of some discussion (547, 548) with a recent commentary suggesting that using such terminology in a reductionist manner to describe the impact of hyperthermia on the development of fatigue during prolonged exercise in the heat may be misleading (549). Fatigue during exercise in the heat is not caused by a sole factor but by the interaction of several physiological processes, as well as the interpretation of afferent feedback in the CNS (**FIGURE 6**). The elevated heart rate observed at volitional fatigue during constant work rate exercise in uncompensable conditions is associated with an increased a-vO_{2diff} and significant declines in stroke volume, cardiac output, and mean arterial pressure (364, 396, 399), coupled with increases in thermal sensation, discomfort, and perceived exertion (309, 418). The combination of these factors leads to the premature termination of exercise in the heat, with the increase in perceived exertion stemming from an increase in relative exercise intensity (%$\dot{V}O_{2max}$), concomitant to the attainment of $\dot{V}O_{2max}$ at submaximal work rates.

Relative exercise intensity as a determinant of endurance performance is not a novel concept. Gleser and Vogel (550, 551) proposed nearly 50 yr ago that endurance capacity (i.e., time to exhaustion) was a function of relative (i.e., %$\dot{V}O_{2max}$) rather than absolute (i.e., work rate) exercise intensity. By manipulating absolute work rate and inspired oxygen fraction in a series of studies, the authors proposed that prolonged exercise performance decreased exponentially with a rise in %$\dot{V}O_{2max}$. A hypothetical model was developed whereby muscle oxygen demand increased as a function of relative

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intensity, and oxygen delivery as the product of blood flow and oxygen content. In the model, oxygen delivery at lower work rates increased proportionally to meet oxygen demands. At very high work rates, however, a compromise in muscle blood flow occurred that led to insufficient oxygen delivery, causing a rise in anaerobic glycolysis and reduction in endurance time (550). A similar phenomenon appears to occur during prolonged exercise in the heat, with premature fatigue linked to the progressive decline in $\dot{V}O_{2max}$ (**FIGURE 12**). As thermal strain develops during constant work rate exercise in the heat, $\dot{V}O_{2max}$ decreases, an increase in relative exercise intensity and perceived exertion then ensues for a given absolute work rate (320, 453, 552–554). Volitional fatigue during such exercise is therefore dependent on the severity of the thermal strain imposed by the interaction of several factors, including ambient conditions (305, 364), initial body temperature (363), relative exercise intensity (309, 555), and fitness level (309, 370, 555).

Fatigue or volitional exhaustion during constant work rate exercise may also arise through a similar pathway but occur at different physiological endpoints. In the first scenario, motivated individuals exercising at a predetermined work rate (e.g., 60% $\dot{V}O_{2max}$) for a prolonged period may reach maximal or near-maximal heart rate before terminating exercise. Volitional fatigue in such circumstances would relate to the attainment of $\dot{V}O_{2max}$ and a consequent impairment in oxygen delivery to exercising muscles, in response to a compromise in systemic blood flow (i.e., maximal cardiac output) (see sect. 4.1). In the second scenario, despite a similar rate of increase in whole body temperature to the previous scenario, heart rate at the point of volitional fatigue might be well below maximum, which would allow for adequate blood flow and oxygen delivery to active skeletal muscles. Although the attainment of cardiovascular limitations may not represent the primary mechanism mediating the termination of exercise in the second scenario, the progressive increase in thermal and cardiovascular strain may play a role in augmenting the perception of exertion and thermal discomfort, leading to the cessation of exercise. These scenarios indicate that the progressive increase in thermal and cardiovascular strain play a modulatory role in the volitional termination of exercise during constant work rate efforts, albeit with different physiological endpoints.

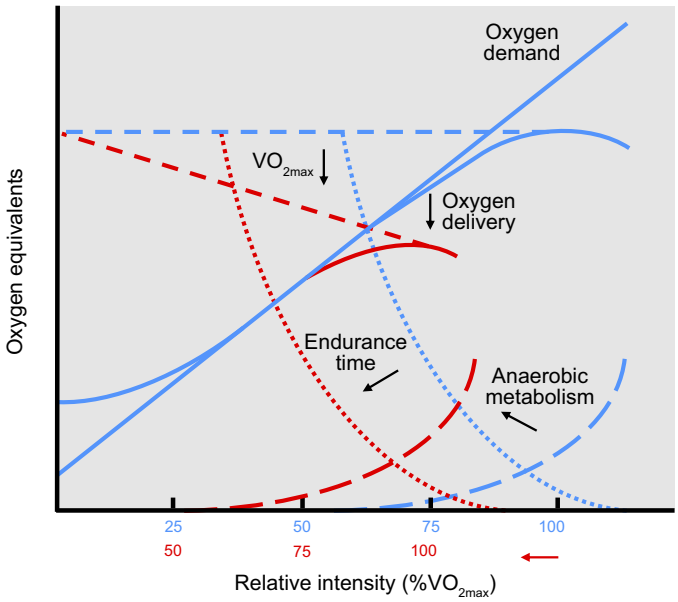

FIGURE 12. Conceptual model of muscle oxygen demand, oxygen delivery, and anaerobic metabolism relative to exercise intensity (%$\dot{V}O_{2max}$) on endurance capacity (i.e., time to exhaustion) during constant work rate exercise in hot (red lines and text) and cool (blue lines and text) conditions. Under heat stress, a gradual increase in thermal strain (i.e., rise in whole body temperature) leads to a progressive decrease in $\dot{V}O_{2max}$, which results in a rise in relative exercise intensity for any given work rate and a greater reliance on anaerobic metabolism, reducing endurance time. Adapted with permission from Gleser and Vogel (550).

4.6. *Self-Paced Exercise in the Heat*

The regulation of prolonged self-paced exercise in the heat has been attributed to the development of thermal strain impacting on cardiovascular function (312, 367, 556), thermal perception (i.e., discomfort) exacerbating perceived exertion (34, 557, 558), and the rate of heat storage influencing locomotor muscle recruitment (317, 559, 560). In most instances, work rate at the start of a prolonged time trial in hot environmental conditions is similar to that of cooler conditions (311–314, 560–563). As a greater level of thermal strain develops in the hotter environment, work rate progressively decreases (**FIGURE 13**). This gradual reduction in work rate is associated with the integration of several factors related to increases in thermal, circulatory, metabolic, and perceptual strain.

In a series of studies, Périard et al. (312–314, 402, 421, 564) demonstrated that a thermoregulatory-mediated rise in cardiovascular strain was associated with a progressive reduction in work rate during prolonged intense self-paced cycling in the heat, relative to when undertaken in cooler conditions. The reduction in work rate under heat stress was significantly correlated with declines in stroke volume, cardiac output and mean arterial pressure during a 40 km time trial (**FIGURE 14**) (312). Heart rate throughout the time trial in the heat (35°C) was also ~8 beats·min^{-1} higher than in cool (20°C) conditions. This elevated cardiovascular response has been shown to exacerbate the decrease in peak oxygen consumption ($\dot{V}O_{2peak}$) measured during the end-spurt of prolonged (45–60 min) time trial efforts

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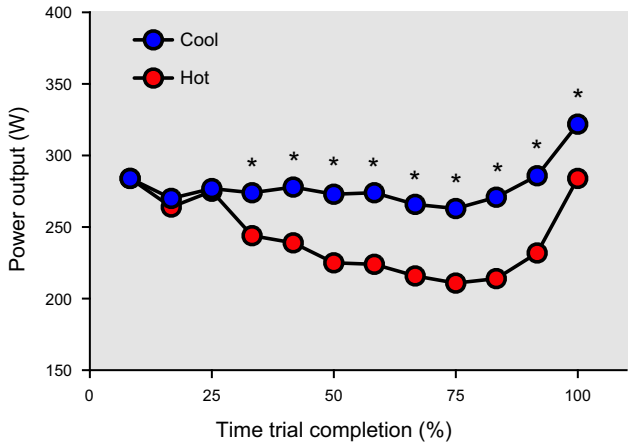

FIGURE 13. Power output during a 40-km cycling time trial in Hot [35°C and 60% relative humidity (RH)] and Cool (20 °C and 40% RH) conditions. These data indicate that work rate (i.e., power output) is similar at the start of self-paced exercise in hot and cool conditions, as the difference in thermal (i.e., core and skin temperature) strain between conditions is relatively small. As exercise progresses, the development of hyperthermia under heat stress mediates a reduction in work rate. *Significant difference between Cool and Hot ($P < 0.05$). Reproduced with permission from Périard et al. (312).

in the heat by ~12%, compared with a similar effort undertaken cool conditions (312, 314, 402). The reduction in $\dot{V}O_{2peak}$ occurs progressively during self-paced exercise in the heat and is accompanied by a gradual decline in absolute work rate (i.e., power output). The latter response has been suggested to occur to maintain relative exercise intensity (i.e., $\%\dot{V}O_{2peak}$) within a narrow range, similar to that of time trial efforts performed in cooler environmental conditions (314). This range widens under heat stress when exercise becomes protracted however, as a disassociation develops between $\%\dot{V}O_{2peak}$, heart rate and perceived exertion. The $\%\dot{V}O_{2peak}$ sustained during self-paced cycling is related to the duration or distance of the event, with time trials of 45–60 min conducted at ~85% $\dot{V}O_{2peak}$ and shorter efforts performed at a greater fraction of maximal aerobic power (313, 314, 412, 565–567). Perceived exertion during such trials in the heat is similar and often higher than in cooler conditions (312, 316, 402, 560). From a performance perspective, the progressive decrease in $\dot{V}O_{2peak}$ and concomitant increase in relative intensity for any given work rate appears to represent the primary determinant mediating the decline in work rate (e.g., power output) observed in hot conditions. This premise is reinforced by data indicating that breathing a hyperoxic gas mixture with fractional oxygen content (FIO2) of 0.45 during the final 25% of a 40 min time trial in the heat (35°C) increased power output relative to breathing normoxic air (FIO2 of 0.21) (564). The improvement in performance was attributed to a hyperoxia-mediated increase in arterial oxygen content and delivery to active muscles, effectively reversing part of the decrease in $\dot{V}O_{2peak}$ and allowing for a greater power output to be maintained. Interestingly however, the extent of the improvement from breathing hyperoxic air was greater in a time trial conducted in cooler conditions (18°C), which suggests that the elevated level of thermal and cardiovascular strain experienced during prolonged self-paced exercise in the heat might partly attenuate the beneficial effect of hyperoxia. Taken together, these findings provide support for the regulation of self-paced exercise involving the conscious interpretation of sensory information relating to effort (i.e.,

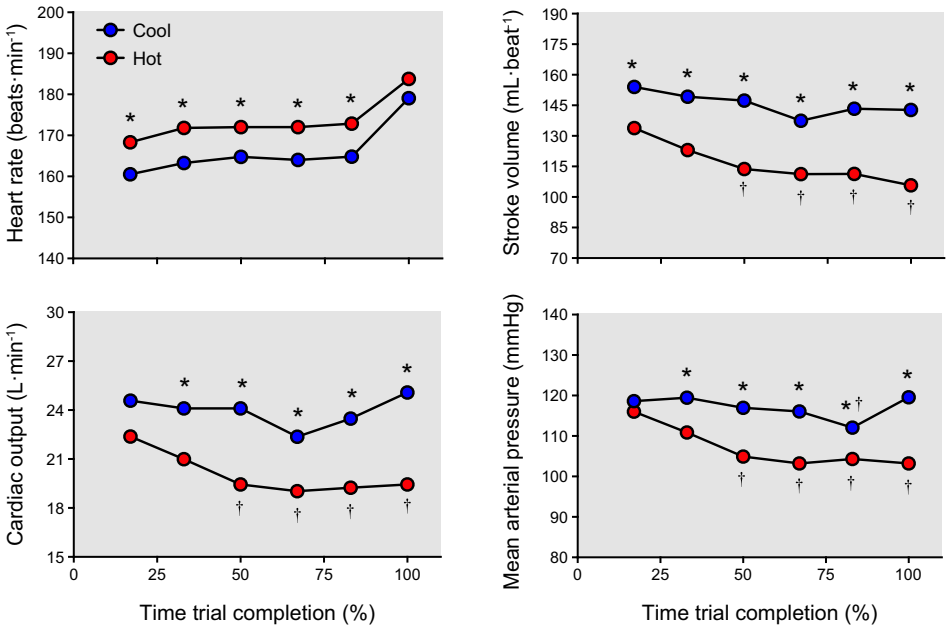

FIGURE 14. Cardiovascular responses during a 40-km cycling time trial in Hot [35°C and 60% relative humidity (RH)] and Cool (20°C and 40% RH) conditions. These data indicate that the development of thermal strain during self-paced exercise in the heat increases the cardiovascular response, despite the maintenance of a lower work rate in the heat. *Significant difference between Cool and Hot ($P < 0.05$). †Significantly lower than 10 min ($P < 0.05$). Reproduced with permission from Périard et al. (312).

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amount of mental and physical energy allocated toward completing a task) and exertion (i.e., level of strain experienced during a task) (552, 568). More specifically, self-paced exercise regulation appears to be an integrative process during which a balance is achieved between an efferent copy of central motor command (i.e., corollary discharge) and afferent sensory input (i.e., physiological signals) originating from peripheral receptors (569–571), which is altered in response to the progressive rise in thermal and cardiovascular strain. The comparative balance between effort and exertion, or the predicted and actual sensory feedback, may therefore allow for sustaining an optimal performance intensity in different environments (e.g., hot, cold, and hypoxic).

In 1916, Lee and Scott (328) wrote that "it is a fact of common experience that a human being in a hot and humid atmosphere feels a disinclination to perform muscular work." Indeed, thermal discomfort is associated with decreased work and athletic performance under heat stress (572). Accordingly, behavioral thermoregulation has been suggested as a mechanism by which self-paced exercise performance may be regulated in the heat. It is proposed that the integration of physiological and psychological influences can formulate a behavior that establishes optimal conditions for heat exchange with the environment (573–575). For example, it is known that heat balance during exercise at a constant work rate is mediated by autonomic responses and the prevailing environmental conditions as metabolic heat production is fixed. In contrast, self-paced exercise allows for behavioral thermoregulatory adjustments to alter or improve the compensability of an environment by adjusting work rate (i.e., metabolic heat production) (557, 574). This coordinated behavioral response is preceded by modifications in subjective thermal perception (i.e., comfort and sensation), primarily driven by skin temperature. Thermal perception is thus purported to play a modulatory role in exacerbating the rating of perceived exertion, with the conscious decision to reduce work rate (e.g., power output or running velocity) occurring primarily to maintain a desired perceived exertion (34, 557, 558, 575). Thermal perception is further suggested to impact on perceived exertion and to reduce work rate only in the early stages of self-paced exercise in the heat, when the increase in body core temperature is minimal, but skin temperature is elevated. Thereafter, when whole body temperature is elevated, factors associated with cardiovascular strain are suggested to modulate perceived exertion and the consequent voluntary reductions in work rate (34). While behavioral thermoregulation represents a powerful mechanism via which conscious decisions contribute to preserve thermal homeostasis (576), Barwood et al. (577) demonstrated that thermal perception, modified by using a menthol spray to induce the feeling of coolness, did not drive changes in work rate during the early stages of a 40-km time trial in warm conditions relative to control and control spray conditions, nor did it affect overall performance. Although work rate in the first few minutes (5–10 min) of prolonged self-paced exercise in the heat is often similar to that of cooler conditions (311–314, 560–563), the progressive decrease in speed or power output during such exercise is typically initiated before a marked rise in thermal strain. As such, work rate in the initial 10–15 min of self-paced exercise may consciously be reduced in response to a thermal perception mediated increase in perceived exertion. Alternatively, experienced individuals may modify work rate and adopt a more conservative pacing strategy to avoid larger decrements in work rate later on during an event, although this approach may not enhance performance (578).

An alternative pathway suggested to mediate the impairment in self-paced exercise performance in the heat lies with anticipatory regulation of muscle recruitment (317, 559, 560). Derived from the central governor model of exercise (579, 580), anticipatory regulation is purported to ensure the prevention of thermal injury by preventing excessive increases in body heat storage and debilitating levels of hyperthermia from developing (581). It is proposed that the moment exercise is initiated, the rate of body heat storage regulates work rate through its influence on perceived exertion. Therefore, work rate decreases to match the maximum tolerable rating of perceived exertion before harmful disturbances can disrupt homeostasis. This hypothesis stems from a study in which power output during a 20-km cycling time trial began to decline in 35°C conditions at a point where core temperature was similar, but skin temperature was ~7°C higher than in a 15°C environment (560). In a follow-up study it was reported that the rate of body heat storage measured in the very early stages (i.e., first 4 min) of self-paced exercise in 35°C conditions was greater than in 15°C and 25°C conditions, leading the authors to surmise that the more rapid reduction in work rate under heat stress was mediated by afferent feedback to the CNS regarding the rate of body heat storage (317). However, the method used by Tucker et al. (317) to calculate the rate of heat storage has been criticized (582), with recent studies demonstrating no association between the initial rate of heat storage and the reduction in self-paced exercise performance in the heat (583, 584). A field-based study has also shown no correlation between the rate of heat storage and 8-km running speed in 17°C and 30°C conditions, with runners capable of accelerating toward the end of the test, despite a rectal temperature >40°C (562). As such, support for the hypothesis that the rate of heat storage during the early stages of exercise mediates changes in self-selected work rate is limited.

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Ultimately, the regulation of self-paced exercise performance involves making decisions based on prior experience, accurate knowledge of a task and an understanding of one's physical abilities, while interpreting and reacting to physiological cues (i.e., sensory information) associated with effort and exertion. Under heat stress, pacing is a process informed by awareness and influenced by factors such as the prevailing ambient conditions, the development of thermal and circulatory strain, the integrity of metabolic processes, and adjustments in skeletal muscle function. Thus self-paced exercise performance in the heat appears to be regulated by interpreting sensory information influenced by increases in thermal, cardiovascular, and metabolic strain.

4.7. Summary

Several factors and mechanistic pathways have been proposed to mediate the impairment in endurance exercise capacity and performance under heat stress. These pathways include hyperthermia-induced *1*) adjustment cardiovascular function, which impact on blood flow distribution, oxygen delivery and heat dissipation, *2*) alterations in central motor drive, which influence muscle activation and force production capacity, and *3*) perturbations in skeletal muscle function, which compromise metabolic and structural characteristics of the muscle and influence autonomic responses. The commonality with all of these responses is the increase in whole body temperature that accompanies prolonged moderate-to-high intensity exercise in the heat and the concomitant link between the development of hyperthermia, sympathetic activity, and circulatory function. These factors appear to cause a gradual decrease in $\dot{V}O_{2max}$ during both constant work rate and self-paced exercise in the heat, which leads to an increase in perceived exertion for any given work rate. The integrative decision to cease exercising (i.e., constant work rate exercise) or to decrease work rate (i.e., self-paced exercise) can occur across a spectrum of physiological and perceptual endpoints, which are exercise, intensity, environmental, context, and participant specific.

5. HEAT STRESS, HYDRATION STATUS, AND EXERCISE PERFORMANCE

Endurance exercise is impaired under heat stress with a compromise in hydration status exacerbating the deleterious effects of thermal strain. Pitts et al. (585) reported over 75 yr ago that dehydration steadily increased rectal temperature, heart rate, and oxygen uptake, while sweat rate decreased and plasma osmolality increased during prolonged marches in hot/dry and hot/humid conditions. As a result, "the subject gradually feels worse and worse, and eventually becomes incapacitated from exhaustion of dehydration, no matter how tough or well acclimatized. Administration of water combats all of these undesirable changes, and in general the more nearly water intake approximates sweat loss, the better off the subjects remains." In his seminal book, *Physiology of Man in the Desert*, Adolph (220) highlighted a few years later that acute dehydration limits the ability to work through a reduction in circulating blood volume and impairment in cardiovascular function. Following on from these early observations and those of others (221, 586–591), it was described that both exercise-induced hypohydration and dehydration impact on thermoregulatory function by reducing whole body sweat rate and skin blood flow, thereby increasing the rate of heat storage (i.e., hyperthermia) and intensifying physiological and perceptual strain. The extent of physiological strain imposed by hyperthermia and dehydration relates to the magnitude of thermal strain and body water loss, as well as the prevailing ambient conditions and mode and intensity of exercise being performed. This section will examine how an elevated whole body temperature and compromised hydration status alter thermoregulatory, cardiovascular, and metabolic function, and how these alterations impact on aerobic exercise performance. The history of recommendations regarding fluid intake during exercise will also be examined and the latest recommendations addressed and contextualized.

5.1. Adjustments in Thermoregulatory Function

Whole body sweat rate during physical activity is determined by exercise intensity and climatic conditions but typically ranges between 0.5 and 2.0 $L \cdot h^{-1}$ with some athletes (~2%) sweating >3.0 $L \cdot h^{-1}$ (592). Gradual reductions in body mass (e.g., 2 to 5%) from water deficit result in marked decrements in plasma (≥10%) and blood (≥6%) volume (593–595). The loss of plasma volume with exercise leads to a state of hyperosmotic hypovolemia that is proportional to the decrement in total body water (596). Although the composition of the precursor secretory fluid contained in eccrine sweat glands is similar to that of plasma, a considerable amount of the ions (e.g., sodium and chloride) within the fluid are reabsorbed as it traverses the duct of the gland, leading to a sweat osmolality approximately half that of plasma (597, 598). The increase in intravascular osmotic pressure resulting from plasma hyperosmolality causes a shift in fluid from the intracellular to the extracellular compartment, which helps defend plasma volume (257). At elevated whole body sweat rates, however, the volume of fluid mobilized from the intracellular compartment into

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the vasculature is insufficient to restore plasma volume and leads to intracellular dehydration.

The influence of dehydration on exacerbating hyperthermia during work in the heat is well established (585, 587–590). In a 1923 review, Marriott (599) synthesized the effects of anhydremia (i.e., reduced fluid content of the blood) on circulatory, metabolic, and thermoregulatory responses, highlighting that "when the blood and tissues become concentrated by water loss the amount of water available for evaporation is diminished and ultimately becomes less than that required for removal of the heat of metabolism. Fever then occurs." Several decades later, in an attempt to determine the link between hydration status, core temperature and sweating, Greenleaf and Castle (600) examined the impact of hyperhydration (+1.2% body mass) and severe (−5.2% body mass) hypohydration on the rectal temperature response during exercise at 50% $\dot{V}O_{2max}$ in temperate conditions. The authors demonstrated increased oxygen uptake, heart rate, and rectal temperature responses in the severe hypohydration trial, along with a reduced sweat rate compared with hyperhydration. The reduction in sweat rate was attributed as the primary pathway exacerbating the rise in rectal temperature (0.1°C per 1% body mass loss) (600). In a subsequent review, Sawka et al. (601) compared the effects of hypohydration on the rise in body core temperature across eight studies and determined that participant characteristics, environmental conditions, and exposure duration influenced thermoregulatory responses. The increase in core temperature during exercise in the heat with hypohydration (2 to 7% body mass loss) appeared to be slightly greater with an estimated ~0.15°C per 1% body-mass loss. Several studies have demonstrated that hypohydration-induced hyperosmotic hypovolemia delays thermoregulatory sweating and cutaneous vasodilation at rest and during exercise and reduces the sensitivity of the relationship between thermoeffector responses and core temperature (348, 349, 602–606). Although both hypovolemia (602, 607–609) and hyperosmolality (610–613) influence these responses, the increase in plasma osmolality appears to be more strongly correlated with the reduction in sweating during exercise-heat stress than the decrease in blood volume (614). Sawka et al. (215) further demonstrated that sweat rate decreased and thermal strain increased proportionally with the severity of hypohydration during prolonged intermittent exercise in the heat (**FIGURE 15**).

An attenuated skin blood flow response for a given level of thermal strain has also been reported (594, 603, 605, 615–617). Nadel et al. (605) demonstrated that a four-day diuretic-induced hypohydration (i.e., iso-osmotic hypovolemia) of ~2.7% body mass loss (17.5% plasma volume contraction) increased the esophageal temperature

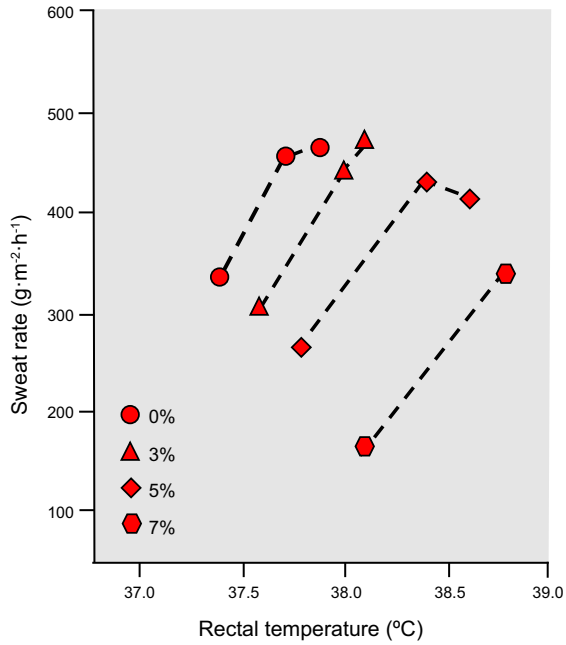

FIGURE 15. Influence of euhydration and 3 different levels of hypohydration (3.1, 5.0, and 6.7% body mass loss) on the relationship between sweat rate and rectal temperature at the completion of 3 (2 for 7% hypohydration) bouts of exercise (10-min rest + 25 min of treadmill walking) in 49 °C and 20% relative humidity. The data indicate a systematically lower sweat rate for a given rectal temperature with increased severity of hypohydration. Reproduced with permission from Sawka et al. (215).

threshold for cutaneous vasodilation (~0.42°C) during 30 min of running in the heat, relative to euhydration and hyperhydration (~2.4% body mass gain). Along with the elevated onset threshold, there was a reduction in cutaneous blood flow for a given temperature with hypohydration (**FIGURE 16**). The authors suggested that the attenuation in cutaneous blood flow contributed to maintain an already compromised venous return, but it also decreased internal (i.e., core-to-skin) heat transfer and exacerbated hyperthermia. These data indicate that arterial and cardiac filling pressure are maintained through baroreflex modulation, at the expense of internal heat transfer, to preserve cardiac output and muscle perfusion (355, 360, 618). These data further highlight that hypovolemia can stimulate circulatory adjustments in the absence of hyperosmolality, although in the context of exercise-induced dehydration, hyperosmotic hypovolemia may further exacerbate these adjustments.

Adjustments in the onset threshold and sensitivity of thermoeffector responses are associated with factors of both central and peripheral origin. Hypohydration and dehydration may reduce the responsiveness of central neural structures regulating evaporative heat loss, with an increase in the onset threshold for sweating associated with a hyperosmolality-induced inhibition of warm-

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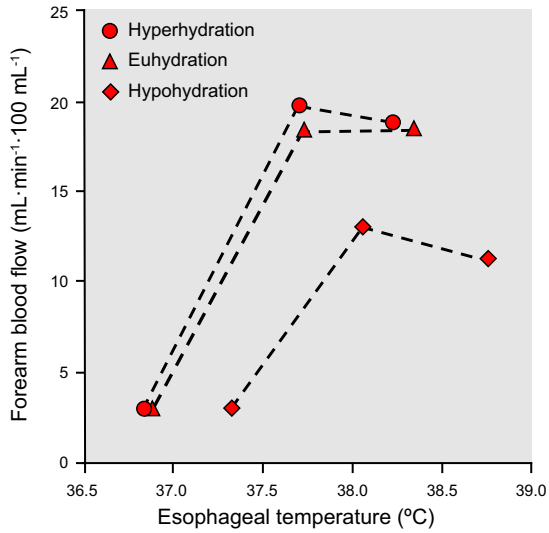

FIGURE 16. Influence of hypohydration (~2.7% body mass loss) and hyperhydration (~2.4% body mass gain) on the relationship between skin (forearm) blood flow and esophageal temperature during 30 min of cycling at 55% $\dot{V}O_{2max}$ in 35°C and 38% relative humidity. The data indicate a lower cutaneous (forearm) blood flow for a given esophageal temperature with hypohydration. Reproduced with permission from Nadel et al. (605).

sensitive neurons within the hypothalamus (i.e., median preoptic nucleus) (619, 620). Osmotically driven peripheral interference with the function of sweat glands may also reduce sweating (600, 621). While the influence of hypovolemia-induced baroreceptor unloading (simulated via lower body negative pressure, head-up tilting, and pharmacological agents) on the modulation of sweating remains controversial (622–627), it may occur under mild to moderate heat stress but is unlikely to occur in relatively acute settings (628). Hypovolemia has further been suggested to increase the onset threshold for cutaneous vasodilation by reducing cardiac preload and altering atrial baroreceptor activity (602), which provides afferent input to the hypothalamic thermoregulatory centers that regulate both cutaneous blood flow and sweating (216). Ultimately, unreplenished (i.e., hypohydration) or inadequately replaced (i.e., dehydration) body water losses during exercise under heat stress reduce blood volume and increase osmolality with the severity of these responses impacting on thermoregulatory capacity.

5.2. Adjustments in Cardiovascular Function

Hyperthermia coupled with progressive dehydration poses a significant challenge to thermoregulatory capacity and cardiovascular control during prolonged exercise, characterized by a reduction in systemic, cutaneous, active muscle and cerebral blood flow. The decline in systemic and regional perfusion is associated with a decrease in cardiac output, increase in total peripheral resistance, and reduction in mean arterial pressure during whole body exercise at intensities above ~60% $\dot{V}O_{2max}$ (594, 595, 616, 618). Conversely, skin, muscle, and cerebral blood flow are uncompromised by the combination of hyperthermia and dehydration during exercise at low intensities or with a small muscle mass (629, 630). The additive effect of dehydration and hyperthermia during exercise is therefore mediated by the severity of each factor. In a study examining different magnitudes of dehydration on thermal and cardiovascular responses, Montain and Coyle (595) demonstrated a linear relationship between the extent of body mass loss and the increase in esophageal temperature and heart rate, as well as the decrease in stroke volume, cardiac output, and skin blood flow (**FIGURE 17**).

The progressive rise in heart rate and decline in stroke volume and cardiac output during aerobic exercise with dehydration in hot (594, 616, 631–633) and temperate (e.g., 22°C) (634) conditions represent fundamental responses associated with dehydration-induced cardiovascular strain. The reduction in cardiac output is primarily related to a decrease in stroke volume, owing to hyperthermia-induced tachycardia and concomitant reduction in blood volume, suppressed venous return, and impaired cardiac filling (525, 526, 528, 529, 594, 635). Maintaining euhydration through fluid ingestion allows for preventing severe hyperthermia and preserving cardiovascular stability (594, 595, 616). Cold conditions (2–10°C) also preserve circulatory function during hypohydrated and dehydrating exercise (~4% body mass loss), as wide core-to-skin and skin-to-air temperature gradients ensure dry heat loss to the environment and minimal heat storage (594, 616, 636). González-Alonso et al. (616) demonstrated that hyperthermia with dehydration leads to cardiovascular instability (i.e., impaired ability to maintain cardiac output and blood pressure) by having participants cycle for 30 min at ~70% $\dot{V}O_{2max}$ in either 2°C or 35°C while *1*) euhydrated with an esophageal temperature of ~38.2°C (normothermic), *2*) euhydrated with an esophageal temperature of 39.3°C (hyperthermic), *3*) dehydrated (~4% body mass) and hyperthermic with a skin temperature of 34°C, *4*) dehydrated and hyperthermic with a skin temperature of 21°C, and *5*) previously dehydrated (i.e., restored blood volume) and normothermic with a skin temperature of 21°C. Hyperthermia and dehydration in isolation were shown not to influence cardiac output or mean arterial pressure during exercise, as their individual effects were similar and increased heart rate by 5% and reduced stroke volume by 7–8% (616). In contrast, the combination of hyperthermia and dehydration increased heart rate by 9% and decreased stroke

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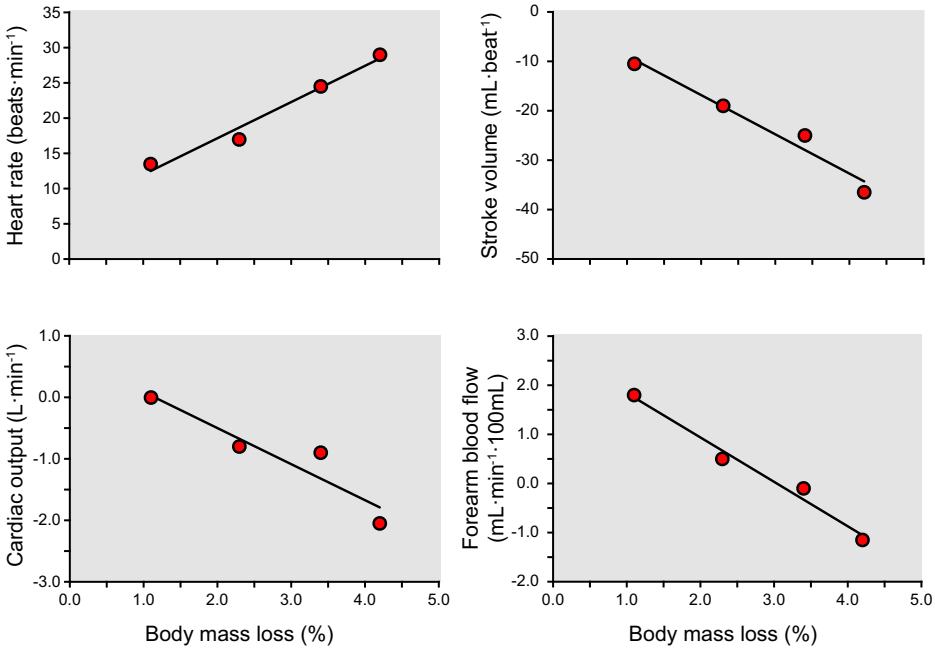

FIGURE 17. Influence of graded dehydration (1.1, 2.3, 3.4, and 4.2% body mass loss) on changes in heart rate, stroke volume and cardiac output from 10 to 110 min and forearm blood flow from 50 to 105 min of cycling at ~65% $\dot{V}O_{2max}$ in 33°C and 50% relative humidity. The data indicate that the magnitude of these responses is proportional to fluid losses experienced via sweating. Reproduced with permission from Montain and Coyle (595).

volume by 20%, resulting in a 13% decrease in cardiac output. Because heart rate was near maximum (~96%) at the end of exercise, the cardiac output generated was the highest possible but inadequate for maintaining cardiovascular function, as evidenced by a decline in arterial blood pressure and increase in systemic vascular resistance. These systemic circulatory adjustments reduce blood flow and impair oxygen delivery to active muscles and the brain, both mechanisms that have been implicated in the development of fatigue during aerobic exercise in the heat.

5.2.1. Skeletal muscle blood flow and metabolism.

The loss of body water through sweating during exercise under heat stress leads to hyperosmotic hypovolemia and increased heat storage due to a decreased ability to dissipate heat. The increase in thermal strain during prolonged whole body exercise is associated with a compromised cardiovascular response, characterized in part by reductions in systemic and active muscle blood flow. Previous studies have demonstrated that exercising muscle blood flow is maintained during prolonged euhydrated exercise in the heat when marked cardiovascular strain is avoided and the capacity to maintain cardiac output and oxygen delivery is preserved (sect. 4.5). In contrast, González-Alonso et al. (618) reported that progressive dehydration (3.9% body mass loss) reduces exercising muscle blood flow and oxygen delivery toward to end of prolonged (135 min) exercise to exhaustion under heat stress, in response to a decrease in perfusion pressure and systemic blood flow. Dehydration also resulted in a greater increase in esophageal temperature (1.5°C) than euhydrated exercise at the same time point, along with a higher heart rate (28 beats·min^{-1}) and lower stroke volume (40 mL·beat^{-1}). Cardiac output was therefore reduced, as were mean arterial pressure and systemic vascular conductance, leading to a ~2 L·min^{-1} reduction in exercising muscle blood flow (**FIGURE 18**). The reciprocal relationship between systemic (i.e., cardiac output) and regional (i.e., exercising limb) blood flow has been demonstrated within various experimental paradigms, including maximal exercise in the heat (399, 521) and hyperthermia-induced local tissue hyperemia (382, 384). Others have also demonstrated a proportional change in exercising muscle blood flow and cardiac output using pharmacological agents to induce changes in limb blood flow (637–641). Similar to maximal exercise under heat stress (399), exercising muscle vascular conductance remains unchanged or slightly increases during whole body exercise with hyperthermia and dehydration (618, 629). This indicates that the fall in exercising muscle blood flow during prolonged exercise is associated with gradual systemic circulatory strain, rather than increased neural vasoconstriction in the active musculature (642). The fall in muscle blood flow and subsequent fatigue during aerobic exercise have been linked to inadequate substrate delivery, energy deficiency, metabolite accumulation (i.e., H^+ and P_i), and elevated muscle temperature, factors that may influence intracellular metabolic processes (643, 644).

When prolonged exercise is undertaken in a euhydrated state with adequate systemic and skeletal

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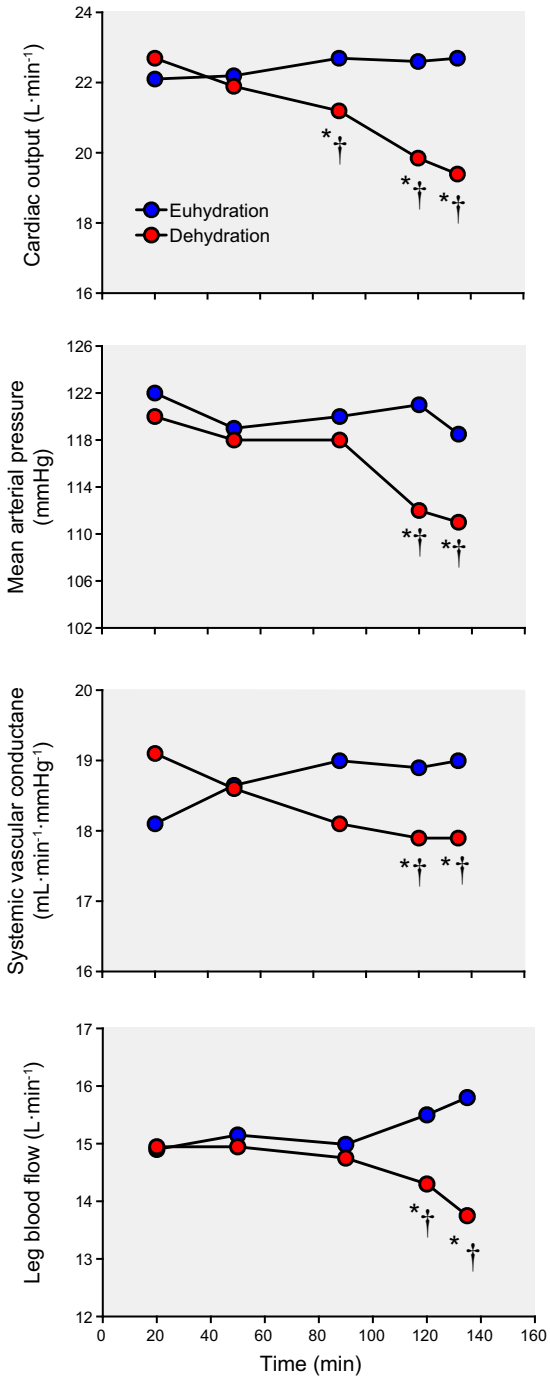

FIGURE 18. Influence of euhydration and progressive dehydration (~3.9% body mass loss) on cardiac output, mean arterial pressure, systemic vascular conductance, and muscle blood flow during exercise at ~60% $\dot{V}O_{2max}$ in 35°C and 45% relative humidity. The data indicate that active muscle blood flow decreases during prolonged exercise in the heat with dehydration in association with a decline in cardiac output and systemic vascular conductance. *Significantly lower than 20 min ($P < 0.05$). †Significantly lower than euhydration ($P < 0.05$). Reproduced with permission from González-Alonso et al. (618).

muscle blood flow, the delivery of substrates and removal of metabolites within the active musculature are closely matched, both in temperate (645, 646) and hot conditions (357, 372). However, net muscle glycogen utilization and lactate accumulation increase during prolonged exercise with progressive dehydration (1–3% body mass loss) in temperate conditions (494–496, 647). Under heat stress, a similar increase in muscle glycogen utilization and lactate production occurs with dehydration (~3.9% body mass loss), along with a decrease in free fatty acid uptake (515). This occurs despite the reduction in exercising muscle blood flow not compromising the delivery of free fatty acids and glucose or lactate removal. The reduction in free fatty acid uptake and increase in carbohydrate oxidation reflects a shift in fuel selection (648), with both hyperthermia and dehydration augmenting glycogen utilization, but hyperthermia potentially being the more potent stimulator. In a recent study, Fernández-Elías et al. (514) had participants cycle for 40 min at a submaximal work rate in temperate conditions in a hypohydrated state (4.4% body mass loss) and in temperate and hot conditions in a rehydrated state (mild hypohydration: ~1.3%). The authors reported that hypohydration increased muscle glycogen utilization by ~35% during exercise in temperate conditions. Intestinal temperature during the hypohydrated trial increased to ~39.2°C, which was similar to the rehydrated trial in hot conditions and higher than the rehydrated trial (~38.5°C) in temperate conditions. Despite an ~11% lower muscle water content at the start of the hypohydrated trial in temperate conditions, glycogen utilization was similar to that of the rehydrated trial in the heat, indicating that hyperthermia and likely muscle temperature are the primary factors stimulating glycogenolysis during intense whole body exercise. As with euhydrated exercise in hot environmental conditions (see sect. 4.5), the physiological and perceptual responses associated with dehydrated exercise at a constant work rate under heat stress are commensurate with the maintenance of a greater relative exercise intensity (494–496, 514, 647). The elevated rate of carbohydrate oxidation (i.e., skeletal muscle glycogenolysis) and diminished rate of fat oxidation during exercise in hot environmental conditions with concomitant hyperthermia, with or without dehydration, may reflect a progressive rise in relative exercise intensity in response to a decrease in $\dot{V}O_{2max}$ (see sect. 4.3). Given muscle glycogen content is a primary determinant of aerobic performance (511, 649–651), the increased rate of muscle glycogenolysis experienced under heat stress may expedite the onset of fatigue during constant work rate exercise. When performing self-paced exercise, work rate adjustments (i.e., reductions) would be required to continue exercising. Collectively, the development of hyperthermia and dehydration during aerobic exercise under heat stress compromise active muscle blood flow and oxygen delivery, and increase the reliance on

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carbohydrate metabolism, both of which may contribute to premature fatigue development.

5.2.2. Cerebral blood flow and metabolism.

Heat stress has been shown to reduce cerebral blood flow during prolonged submaximal and brief maximal exercise (374, 401–405) in response to hyperventilation-induced hypocapnia (i.e., decreased $Paco_2$), mediated by hyperthermia and the maintenance of an elevated exercise intensity, typically >60% $\dot{V}o_{2max}$. At rest, Ogoh et al. (652) demonstrated that whole body passive heating (1.5°C increase in esophageal temperature) increased cardiac output by ~60% but failed to increase intracranial blood flow (i.e., internal carotid and vertebral arteries) as a ~15% reduction was noted. It was suggested that blood flow was distributed to extracranial (i.e., external carotid artery) vascular beds for heat dissipation. It has further been reported during progressive passive heating that cerebral blood flow (i.e., MCA_v) is reduced in response to hyperthermia-induced hyperventilation and the peripheral redistribution of cardiac output (653). The authors further demonstrated that mild hypohydration (1.5% body mass loss), in the absence of hyperthermia, was associated with an elevated cerebral blood flow at rest. In contrast, the reduction in cerebral blood flow appears to be accelerated during dehydrated exercise under heat stress. For example, Trangmar et al. (415) demonstrated during incremental exercise to exhaustion that the combination of dehydration (~3% body mass loss) and hyperthermia (~0.7°C) precipitated the decline in intracranial (i.e., internal carotid and middle cerebral arteries) blood flow without affecting extracranial (i.e., common carotid artery) blood flow. The combination of hyperthermia and dehydration decreased the absolute work rate attained at $\dot{V}o_{2max}$ by ~20%, albeit with equivalent cerebral blood flow values to those without dehydration. The decline in cerebral blood flow during incremental exercise in the heat, with or without dehydration, was accompanied by a compensatory increase in oxygen extraction, which allowed for maintaining the cerebral metabolic rate for oxygen (415). In a follow-up study, prolonged submaximal exercise in the heat was undertaken with and without eliciting exhaustion, in both a euhydrated and dehydrated state (654). Euhydration through fluid ingestion allowed for preserving intracranial and extracranial blood flow during nonfatiguing exercise, with progressive dehydration (~3% body mass loss) and greater hyperthermia (~0.5°C) accelerating the decline in internal carotid artery blood flow and MCA_V and eliciting a decline in extracranial blood flow (i.e., common and external carotid arteries) at the end of nonexhaustive exercise (120 min). During exhaustive exercise, euhydration only delayed the decline in MCA_V, which occurred earlier with dehydration, concomitant with an elevated hyperthermia and tachycardia. As with the previous study, cerebral metabolism was preserved through enhanced oxygen and glucose extraction across the brain (654). This observation is supported by data demonstrating an uncompromised cerebral metabolic rate for oxygen during the transition from rest to moderate intensity exercise (655–657), followed by a rise in cerebral metabolism at intensities approaching maximum (404, 405, 658). These data indicate that dehydration exacerbates cerebrovascular instability during brief exercise to volitional fatigue and prolonged strenuous exercise in the heat by accelerating the decline in cerebral blood flow. The accelerated decline in intracranial blood flow with dehydration is accompanied by a decrease in cerebrovascular conductance stemming from an increase in vasoconstrictor activity, modulated primarily by a decline in $Paco_2$ (415, 654). The reduction in extracranial blood flow during prolonged exercise in the heat with dehydration appears to be influenced by regulatory mechanisms akin to those of the cutaneous circulation (see sect. 4.1.1).

Given the metabolic stability exhibited by the brain, the notion that a compromise in oxygen delivery to the brain might mediate the development of fatigue or impair performance during exercise under heat stress, with or without concomitant dehydration, by compromising central neural drive (401, 404, 413, 414) appears unlikely. Rather, the suppression of exercising muscle perfusion and aerobic metabolism associated with greater thermal strain and cardiovascular instability during exercise-induced hyperthermia and dehydration appear to represent the primary determinants accelerating fatigue development. This premature fatigue is associated with a decline in blood flow and oxygen delivery that requires an increase in oxygen extraction (i.e., $a\text{-}vO_{2diff}$) to support aerobic metabolism in the active musculature. The decline in blood flow is temporally associated with the attainment of upper limits to functional oxygen extraction, which blunts exercising muscle oxygen uptake, and thus whole body $\dot{V}o_{2max}$. Trangmar and González-Alonso (659) developed an elegant conceptual framework outlining the combined influence of dehydration and hyperthermia during maximal and submaximal exercise on regional (i.e., cerebral, skin, and leg muscle) and systemic (i.e., cardiac output) hemodynamics, and the concomitant alterations in aerobic metabolism (**FIGURE 19**). The framework highlights how dehydration-induced reductions in skin blood flow exacerbate physiological strain by increasing thermal strain and how the cerebral metabolic rate for oxygen is preserved and unlikely to contribute to fatigue development, but that a reduction in exercising muscle aerobic metabolism mediates dehydration-induced fatigue development.

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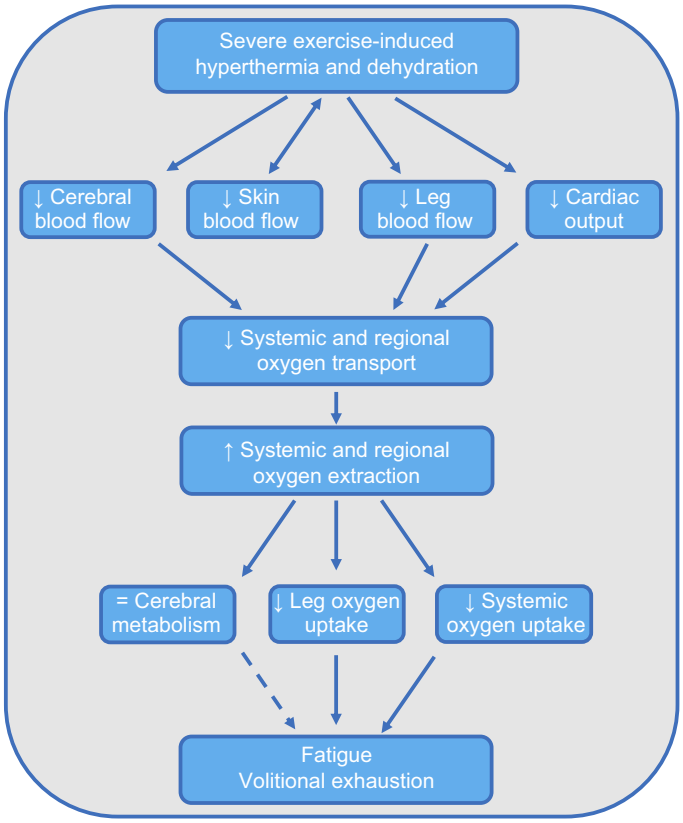

FIGURE 19. Conceptual framework of the impact of dehydration on regional and systemic hemodynamics and aerobic metabolism. The combination of dehydration, hyperthermia, and strenuous exercise markedly reduces cerebral, leg (i.e., muscle) and systemic (i.e., cardiac output) blood flow. Dehydration-induced reductions in skin blood flow exacerbate physiological strain by increasing body heat storage. The reduction in cerebral blood flow is associated with a hyperventilation-induced fall in arterial carbon dioxide pressure. The reduction in cardiac output is due to a progressive decrease in stroke volume in response to an increase in heart rate toward maximum and concomitant to a fall in end-diastolic volume that is not compensated for by the reduction in end-systolic volume. The fall in whole body perfusion requires tissue and systemic oxygen extraction to increase to support aerobic metabolism. Although the cerebral metabolic rate for oxygen is well preserved, and unlikely to contribute to fatigue development during short-duration maximal and prolonged submaximal exercise, reductions in active muscle and systemic aerobic metabolism are likely candidates to mediate dehydration-induced fatigue development. Adapted with permission from Trangmar and González-Alonso (659).

5.3. *Heat Stress, Hydration Status, and Maximal Aerobic Power*

The progressive rise in thermal strain when exercising in increasingly hot ambient conditions is associated with an exacerbated decrease in $\dot{V}O_{2max}$ (319). The magnitude of the decrease is reliant on marked elevations in both core and skin temperature, which in turn drive an increase in physiological strain that suppresses systemic and exercising muscle oxygen delivery (321, 521). When combined with hypohydration, the decrease in $\dot{V}O_{2max}$ under heat stress might be expected to worsen, particularly given a recent meta-analysis reporting that a ~4% reduction in body mass leads to a ~2.5% reduction in $\dot{V}O_{2max}$ in temperate conditions (~20.5°C) (660). Under heat stress however, when hyperthermia is combined with hypohydration, most of the reduction $\dot{V}O_{2max}$ appears attributable to the magnitude of thermal strain. After inducing a ~4% loss in body mass or maintaining euhydration via fluid ingestion, Nybo et al. (321) had endurance-trained participants complete four maximal (~402 W) cycling tests to exhaustion (<8 min): control, hypohydration, hyperthermia, and a combination of hypohydration and hyperthermia (**FIGURE 20**). Thermal strain was carefully manipulated with a water perfused jacket so that starting esophageal and skin temperatures were ~37.5°C and ~31°C in normothermia, and ~38.5°C and ~37°C in hyperthermia. A 16% reduction in $\dot{V}O_{2max}$ was reported with hyperthermia, regardless of hydration status, along with a 52% reduction in exercise time relative to the normothermic and euhydrated trial. In the normothermic and hypohydrated trial a 5% reduction in $\dot{V}O_{2max}$ and 26% decrease in exercise time were noted. The reduction in $\dot{V}O_{2max}$ with hyperthermia was ascribed to a decline in cardiac output, reducing blood flow and oxygen delivery to exercising skeletal muscles (321). The similar reduction in $\dot{V}O_{2max}$ under heat stress, with or without hypohydration, supports previous findings of a ~26% decrease with hyperthermia alone (322) and in combination with hypohydration (4.3% body mass loss) (661), the latter study also reporting a 48% reduction in exercise time. In a separate study, $\dot{V}O_{2max}$ was measured immediately after 60 and 120 min of submaximal cycling in warm conditions without fluid ingestion (2.3% and 3.7% body mass loss, respectively) and after 120 min with fluid ingestion (0.7% body mass loss) (662). A significant reduction in $\dot{V}O_{2max}$ of 8.7% was observed after 120 min without fluid ingestion, in conjunction with greater hyperthermia (~0.3°C), relative to the other conditions in which $\dot{V}O_{2max}$ remained unchanged. Pichan et al. (663) also demonstrated following heat acclimation that hypohydration of 1.3, 2.3, and 3.3% body mass decreased $\dot{V}O_{2max}$ in hot/dry conditions (45°C, 30% RH) by 8.7, 11.0, and 21.7% and in hot/humid conditions (39°C, 60% RH) by 5.6, 8.0, and 20.4%. The environmental conditions (i.e., dry vs. humid) led to a similar decrease in $\dot{V}O_{2max}$, which was more pronounced when hypohydrated by 3% body mass. Altogether, these data indicate that $\dot{V}O_{2max}$ declines markedly upon exceeding the ~3% body mass loss threshold (642, 660) and may only slightly exacerbate the effects of hyperthermia. This suggests that an elevated whole body temperature (e.g., core temperature >38°C and skin temperature >35°C) exacerbates cardiovascular strain and precipitates the

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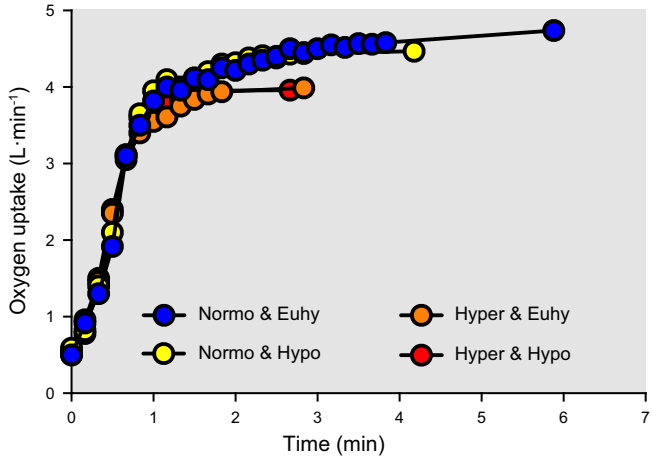

FIGURE 20. Influence of hyperthermia and hypohydration on maximal aerobic and endurance capacity. Oxygen uptake during maximal cycling to exhaustion at ~402 W in normothermic (Normo) and hyperthermic (Hyper: esophageal and mean skin temperature: +1°C and +6°C higher, respectively) states, while euhydrated (Euhy) or hypohydrated (Hypo: ~4% body mass loss). The data indicate hyperthermia with or without hypohydration similarly reduces $\dot{V}O_{2max}$ and that part of this reduction is restored when normothermic but hypohydrated, such that most of the decline in $\dot{V}O_{2max}$ with combined hyperthermia and hypohydration is associated with hyperthermia. Redrawn with permission from Nybo et al. (321).

attainment of $\dot{V}O_{2max}$ to a greater extent than hypohydration of 3–4% during maximal exercise. Whether a similar relationship persists with severe hypohydration (e.g., >6%) remains to be determined. Notwithstanding, hyperthermia and hypohydration/dehydration interact in modulating systemic and regional blood flow (i.e., oxygen delivery), such that their influence is integrative.

5.4. *Heat Stress, Hydration Status, and Prolonged Aerobic Exercise*

The combination of environmental heat stress and a compromised hydration status during constant work rate exercise is characterized by an exacerbated rise in body core temperature; decreased systemic, muscle, and skin blood flow; and increased reliance on muscle glycogen and anaerobic metabolism (215, 595, 605, 618). These responses lead to premature fatigue with several narrative and meta-analytical reviews reporting that a body mass loss exceeding ~2% impairs exercise capacity, particularly when ambient temperature surpasses 30°C (204, 660, 664, 665). In contrast, exercise-induced dehydration to 4% body mass loss does not appear to impact on performance during ecologically valid self-paced exercise tasks (e.g., cycling or running time trials) performed in 20 to 33°C conditions (665, 666). However, hypohydration of 1.7 to 5.6% body mass has been shown to impair aerobic exercise performance in ambient temperatures ranging from 19 to 40°C (660).

To further investigate the influence of hydration status on exercise capacity and performance, we explored the scientific literature and segmented dehydration and hypohydration in body mass deficits of 0.5–1.4, 1.5–2.4, 2.5–3.4, 3.5–4.4, and ≥4.5% across ambient conditions above and below 30°C. In addition to utilizing the reviews highlighted above, a systematic search of the literature was conducted on PubMed (August 2020) to identify aerobic-based constant work rate (excluding incremental/graded $\dot{V}O_{2max}$ tests) and self-paced exercise protocols evaluating the impact of dehydration and hypohydration. To be included in our analysis, studies had to be compared with a euhydration (control) trial and provide environmental condition information (otherwise assumed to be <30°C, i.e., Ref. 667). Studies examining the influence of fluid or beverage composition (e.g., sodium or carbohydrate) were excluded, unless controlled for between trials (i.e., Refs. 668, 669). The observations from our review of the literature are summarized in the sections below. We identified 11 constant work rate studies providing 22 comparisons (TABLES 2 and 3; additional details provided in APPENDIX A) and 33 self-paced exercise studies providing 58 comparisons (TABLES 4 and 5; additional details provided in APPENDIX A). For ease of comparison, all performance outcomes were converted to percent change in time relative to euhydration by equating a 1% change in power output to a 1% change in running time-trial speed or time and a 0.4% change in time during a cycling time trial (709). Decrements in exercise capacity and performance were converted to negative percent changes such that a shorter time to volitional exhaustion during constant work rate exercise and longer time to complete a time trial or less distance completed during self-paced exercise are expressed as negative values. Several performance trials, both at a constant work rate and self-paced, were performed following prolonged exercise (e.g., 60- to 90-min preload). While the exercise preload was not included in the analysis of performance, its implications on physiological responses and performance are important and so it has been acknowledged where relevant.

The separation of dehydration and hypohydration is important when investigating the influence of hydration status on performance as initiating exercise with a particular body water deficit (e.g., 3%: hypohydration) has different physiological implications to reaching such a deficit during the final stages of a prolonged effort (e.g., final 15 min of a 90-min exercise bout: dehydration). It must also be acknowledged that exercise capacity and performance are not suddenly and markedly impaired upon reaching a particular level of dehydration, but progressively affected as dehydration develops. Moreover, while constant work rate exercise allows for isolating independent variables (e.g., fluid intake) in a well-controlled environment to

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Table 2. ***Influence of different levels of dehydration on exercise capacity during constant work rate exercise to exhaustion in ambient temperature conditions below and above 30°C***

Study	Body Mass Loss					Exercise Time, %
	0.5–1.4%	1.5–2.4%	2.5–3.4%	3.5–4.4%	>4.5%	
			Ambient temperature <30°C			
McConell et al. (670)	–	1.8	–	–	–	−24.4
Maughan et al. (671)	–	1.8	–	–	–	−7.9
Fallowfield et al. (672)	–	2.0	–	–	–	−24.6*
McConell et al. (670)	–	–	3.2	–	–	−47.9*
			Ambient temperature ≥30°C			
de Melo-Marins et al. (673)	1.0	–	–	–	–	−2.6
Marino et al. (674)	1.0	–	–	–	–	−1.5
de Melo-Marins et al. (673)	1.3	–	–	–	–	−2.6
Marino et al. (674)	–	1.7	–	–	–	−21.1*

Negative exercise capacity percent values represent a shorter time to volitional exhaustion relative to euhydration. Protocol details provided in TABLE A1. *$P < 0.05$, significantly different from euhydration/control.

examine their effect on dependent variables (e.g., volitional fatigue), it has been argued that this model of exercise lacks ecological validity (229, 666, 710, 711). Endurance athletes competing in real-world settings typically complete a known distance as quickly as possible, rather than maintain a given work rate for as long as possible. As such, self-paced time trials are typically viewed as more representative of a true competitive environment, as athletes can adjust their work rate and regulate their effort. The within-subject reliability of self-paced exercise trials is generally greater than time to exhaustion tests (712–714), although both appear to display similar reliability when the curvilinear relationship between exercise intensity and duration is accounted for (715) and have similar sensitivity (716). Moreover, it has been argued that exercise to volitional fatigue does have some relevance to competitive race scenarios, such as when athletes adopt the pace set by faster competitors until unable to follow (714). Notwithstanding, differentiating the influence of a compromise in hydration status between constant work rate and self-paced exercise provides insight into how fatigue develops when hyperthermic and hypovolemic.

5.4.1. Constant work rate exercise and dehydration.

Dehydration in excess of 2% body mass has been reported to impair constant work rate exercise to volitional fatigue in warmer environments, which TABLE 2 seems to corroborate. A ~19% (range: −24.6 to −7.9%) reduction in endurance time was noted with a body mass loss <2.5% and ambient temperature <30°C. These data represent three studies in which a similar relative intensity (~70% $\dot{V}O_{2max}$) was prescribed, twice to exhaustion (671, 672) and once for 120 min and then followed by an increase in work rate to 90% $\dot{V}O_{2max}$ until fatigue (670). With a body mass loss of 3.2%, a much larger reduction (47.9%) in time to exhaustion was reported and coincided with greater thermal and cardiovascular strain (670). In ambient conditions ≥30°C a 7.0% (range: −21.1 to −1.5) reduction in exercise capacity was observed when body mass losses remained <2.5%. Although no study met the inclusion criteria for dehydration ≥2.5% body mass, one did demonstrate a ~25% decrease in endurance time with a 6.4% body mass loss (717). This study was not included, however, as the euhydration trial was terminated after a predetermined time frame, rather than volitional fatigue. Although available data are generally limited, they indicate that aerobic exercise capacity is largely preserved when body mass loss remains ~1.0% but is impaired when dehydration approaches and surpasses ~2.0%, both in temperate and hot conditions.

5.4.2. Constant work rate exercise and hypohydration.

A marked body water deficit (i.e., hypohydration) before undertaking exercise influences physiological responses

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Table 3. ***Influence of different levels of hypohydration on exercise capacity during constant work rate exercise to exhaustion in ambient temperature conditions below and above 30°C***

Study	Body Mass Loss					Exercise Time, %
	0.5–1.4%	1.5–2.4%	2.5–3.4%	3.5–4.4%	>4.5%	
	Ambient Temperature <30°C					
Ebert et al. (675)			2.5			−28.7*
	0.5–1.4%	1.5–2.4%	2.5–3.4%	3.5–4.4%	>4.5%	
	Ambient temperature ≥30°C					
Walsh et al. (676)	–	1.8	–	–	–	−30.6*
Cheung and McLellan (677)	–	1.9	–	–	–	−4.4*
Cheung and McLellan (368)	–	1.9	–	–	–	−12.5*
Baker et al. (668)	–	2.0	–	–	–	−33.3*
Cheung and McLellan (677)	–	2.0	–	–	–	−11.9*
Cheung and McLellan (368)	–	2.0	–	–	–	−18.6*
Cheung and McLellan (368)	–	2.1	–	–	–	−14.6*
Cheung and McLellan (368)	–	2.2	–	–	–	−12.4*
Cheung and McLellan (678)	–	2.2	–	–	–	−10.7*
Cheung and McLellan (678)	–	2.2	–	–	–	−18.2*
Cheung and McLellan (677)	–	–	2.6	–	–	−20.5*
Cheung and McLellan (677)	–	–	2.8	–	–	−20.6*
Baker et al. (668)	–	–	3.3	–	–	−61.9*

Negative exercise capacity percent values represent a shorter time to volitional exhaustion relative to euhydration. Protocol details provided in TABLE A2. *$P < 0.05$, significantly different from euhydration/control.

from the onset of exercise and exacerbates the rise in whole body temperature. As such, undertaking constant work rate exercise in a hypohydrated state is clearly detrimental to endurance capacity, regardless of the prevailing ambient conditions (TABLE 3). One study reported that hypohydration of 2.5% body mass yielded a 28.7% reduction in exercise capacity during a simulated hill climb (8% gradient) in 29°C conditions (675). Despite being ~1.9 kg lighter, time to exhaustion was reduced in conjunction with an elevated rectal temperature (~0.5°C) and heart rate (~4 beats·min^{-1}). When ambient temperature exceeds 30°C, a ~17% (range: −33.3 to −4.4%) decrease in endurance time is noted with hypohydration <2.5% body mass and ~34% (range: −61.9 to −20.5%) reduction with hypohydration ≥2.5%. The impact of hypohydration in hotter conditions is quite marked, even with a body mass deficit of 1.8 to 2.0% (368, 668, 676, 677). The studies performed by Cheung and McLellan (368, 677, 678) are noteworthy as participants were dehydrated 15 h before undertaking a walking heat tolerance test to volitional fatigue while wearing protective clothing, such that the environment was uncompensable. Although some participants terminated exercise because of reaching the ethically imposed upper limit for rectal temperature (39.3°C), they reported being very near the point of voluntary termination. Moreover, the average endpoint rectal temperature in each study was ~38.8°C (677), ~38.6°C (368), and ~39.0°C (677) due to the uncompensable nature of the protocol. As such, these studies were included in the current section. The series of studies indicated that even mild hypohydration impairs endurance capacity under uncompensable heat stress and that

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Table 4. *Impact of different levels of dehydration on exercise performance during self-paced efforts with ambient temperature below and above 30°C*

Study	Body Mass Loss					Performance Time (%)
	0.5–1.4%	1.5–2.4%	2.5–3.4%	3.5–4.4%	>4.5%	
	Ambient Temperature <30°C					
Bachle et al. (679)	1.0	–	–	–	–	−1.3
McConell et al. (680)	1.0	–	–	–	–	−0.7
Backx et al. (681)	1.3	–	–	–	–	−0.1
Backx et al. (681)	–	1.7	–	–	–	0.5
Kay and Marino (682)	–	1.8	–	–	–	3.3
McConell et al. (680)	–	1.9	–	–	–	0.7
Bardis et al. (683)	–	2.2	–	–	–	−5.8*
Robinson et al. (684)	–	2.3	–	–	–	1.4*
Daries et al. (685)	–	–	2.6	–	–	1.3
Hillman et al. (686)	–	–	3.0	–	–	−2.0
Daries et al. (685)	–	–	3.2	–	–	2.6
	Ambient temperature ≥30°C					
Bardis et al. (687)	–	1.8	–	–	–	−4.7*
Below et al. (669)	–	1.9	–	–	–	−6.5*
Dugas et al. (688)	–	1.9	–	–	–	−0.6
Dugas et al. (688)	–	2.1	–	–	–	1.0
Perreault-Briere et al. (689)	–	2.2	–	–	–	0.2
Adams et al. (690)	–	2.2	–	–	–	−4.7*
Kay and Marino (682)	–	2.2	–	–	–	0.0
Dugas et al. (688)	–	–	2.9	–	–	−3.6
Perreault−Briere et al. (689)	–	–	2.9	–	–	−0.5
Dion et al. (691)	–	–	3.1	–	–	1.1
Hillman et al. (686)	–	–	–	3.8	–	−5.0*
Dugas et al. (688)	–	–	–	3.9	–	−3.2
Dugas et al. (688)	–	–	–	4.3	–	−2.3

Negative performance percent values represent a longer time to complete a known distance, or less distance completed over a given time period. Protocol details provided in TABLE A3. *$P < 0.05$, significantly different to euhydration/control.

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Table 5. *Influence of different levels of hypohydration on exercise performance during self-paced efforts with ambient temperature below and above 30°C*

Study	Body Mass Loss					Performance Time, %
	0.5–1.4%	1.5–2.4%	2.5–3.4%	3.5–4.4%	>4.5%	
	Ambient Temperature <30°C					
Slater et al. (692)	1.3	–	–	–	–	−0.8
Armstrong et al. (693)	–	1.6	–	–	–	−7.2*
Merry et al. (694)	–	1.8	–	–	–	−5.2
Merry et al. (694)	–	1.8	–	–	–	−2.4
Armstrong et al. (693)	–	1.9	–	–	–	−3.4
Slater et al. (692)	–	2.0	–	–	–	−0.5
Armstrong et al. (693)	–	2.1	–	–	–	−6.7*
Stearns et al. (695)	–	2.1	–	–	–	−4.6*
Casa et al. (696)	–	2.3	–	–	–	−4.8*
Logan-Sprenger et al. (494)	–	2.3	–	–	–	−13.0*
Fleming and James (697)	–	2.4	–	–	–	−5.8*
Fleming and James (697)	–	2.4	–	–	–	−1.2
Cheuvront et al. (698)	–	–	2.9	–	–	−1.0
Cheuvront et al. (698)	–	–	3.0	–	–	−3.2*
Burge et al. (667)	–	–	3.1	–	–	−4.9*
Oliver et al. (699)	–	–	3.2	–	–	−2.8
Stewart et al. (700)	–	–	–	3.8	–	−3.0
Castellani et al. (701)	–	–	–	4.0	–	−5.2*
Kenefick et al. (702)	–	–	–	4.1	–	−1.1*
Kenefick et al. (702)	–	–	–	4.2	–	−3.8*
	Ambient temperature ≥30°C					
Bardis et al. (703)	1.0	–	–	–	–	−2.1*
Berkulo et al. (704)	1.1	–	–	–	–	−1.6
Slater et al. (692)	1.1	–	–	–	–	−0.8
Berkulo et al. (704)	1.3	–	–	–	–	0.3
Slater et al. (692)	–	2.0	–	–	–	−0.1
Cheung et al. (705)	–	2.0	–	–	–	−0.6

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Table 5.—*Continued*

Study	Body Mass Loss					Performance Time, %
	0.5–1.4%	1.5–2.4%	2.5–3.4%	3.5–4.4%	>4.5%	
Wall et al. (706)	–	2.1	–	–	–	0.1
Cheung et al. (705)	–	2.1	–	–	–	2.1
James et al. (707)	–	2.4	–	–	–	−3.1*
Wall et al. (706)	–	–	3.0	–	–	0.1
Funnell et al. (708)	–	–	3.0	–	–	−11.4*
Funnell et al. (708)	–	–	3.0	–	–	−10.1*
Kenefick et al. (702)	–	–	–	4.0	–	−4.9*
Kenefick et al. (702)	–	–	–	4.1	–	−8.7*

Negative performance percent values represent a longer time to complete a known distance, or less distance completed over a given time period. Protocol details provided in TABLE A4. *$P < 0.05$, significantly different to euhydration/control.

short-term aerobic training and heat acclimation fail to enhance exercise-heat tolerance in such conditions, whereas long-term aerobic fitness from regular physical activity is beneficial. The larger reductions in performance capacity noted in the Baker et al. (668) and Walsh et al. (676) studies likely reflect differences in protocol, with participants in both studies running or cycling at 70% $\dot{V}O_{2max}$ for 60–120 min before exercising to exhaustion at 85–90% $\dot{V}O_{2max}$. The higher exercise intensity sustained following the initial bout of exercise to induce hypohydration may have exacerbated the impairment in subsequent performance. Hence, mild to moderate hypohydration reduces exercise capacity in both hot and cool conditions, with exercise performed at higher intensities possibly hastening fatigue development.

5.4.3. Self-paced exercise and dehydration.

The influence of dehydration on self-paced exercise performance appears to be quite equivocal, particularly in cooler environments. During time trial efforts in ambient conditions of <30°C, a similar 0.2% reduction in performance is noted with body mass losses <2.5% (range: −5.8 to 3.3%) and ≥2.5% (range: −2.0 to 2.6%). At ambient temperature ≥30°C, a slightly greater impairment in performance is noted, which is again similar between body mass losses below (−2.2%, range: −6.5 to 1.0%) and above (−2.1%, range: −5.0 to 1.1%) 2.5%. The lack of a persistent effect of dehydration on self-paced exercise performance likely stems from the progressive nature of body water loss and the duration of most efforts being ≤60 min. Of note, Kay and Marino (682) reported similar thermal, physiological, and performance outcomes during 60-min cycling time trials interspersed with sprints efforts in 20°C and 33°C conditions, both with or without mild dehydration (~2% body mass loss). These observations are notable for the similar performance outcome in 20°C and 33°C conditions without dehydration, which contrast previous studies where prolonged time trial performance in the heat was impaired relative to cool conditions (311–314, 560, 718). The lack of difference between hot and cool conditions indicates a potential lack of statistical power ($n = 7$) and/or task familiarity. The findings of Dugas et al. (688) of comparable performances during 80-km cycling time trials across six fluid intake conditions may also have been subject to type II error ($n = 6$). To address this, the authors created two body mass deficit groups (i.e., above and below 2.5%), which highlighted the significant impairment in performance afforded by dehydration. The similar thermal responses observed between conditions may be associated with the ~10.5 $m \cdot s^{-1}$ airflow provided during exercise, which does provide greater convective and evaporative heat loss and more accurately reflects an outdoor cycling setting. Bardis et al. (683) also reported a decrease in performance during a 5-km cycling hill climb in 28–30°C outdoor conditions following 60 min of indoor cycling. Airflow during the hill climb likely matched that of moving speed (~4.7 $m \cdot s^{-1}$), yet performance was impaired. Other studies have also shown that 10- to 20-min time trial performance is decreased in warmer environments with limited airflow after an initial 90 min of submaximal exercise (669, 686). In contrast, Daries et al. (685) reported unaffected 30-min running time trial performances after 90 min of steady-state running in 25°C conditions with an airflow commensurate

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with running speed (~3.8 $m \cdot s^{-1}$), despite 2.6% and 3.0% body mass losses.

Ambient temperature, relative humidity, and airflow (in studies reporting them) were 26.9°C, 55.3%, and 5 $m \cdot s^{-1}$ in the cycling studies and 27.5°C, 48.5%, and 4.0 $m \cdot s^{-1}$ in the running studies identified in **TABLE 4**. This indicates that airflow in the cycling studies was less than encountered when competing in an outdoor environment and may have influenced performance outcomes. Notwithstanding, self-paced exercise performance is relatively well maintained with dehydration <4% body mass, particularly in cooler conditions even when exercise is >60 min, due to the progressive nature of body mass loss. However, some studies may have been underpowered (i.e., small sample size) to draw firm conclusions.

5.4.4. Self-paced exercise and hypohydration.

The impact of hypohydration on self-paced exercise performance is more consistent and pronounced than that of dehydration (**TABLE 5**). A body water deficit <2.5% is associated with a 4.6% (range: −13.0 to −0.5%) impairment in time trial performance in temperate conditions (<30°C) and 3.1% (range: −5.2 to −1.0%) decrement with hypohydration ≥2.5% body mass. In warmer environments (≥30°C), a body mass deficit <2.5% before exercise appears not to influence performance (−0.6%, range: −3.2 to 2.1%), whereas a deficit ≥2.5% leads to a 7.0% (range: −11.4 to 0.1%) impairment. The lower impairment in performance noted with hypohydration under 2.5% may be influenced by four experimental trials in which body mass before exercise was only reduced by 1.0 to 1.3%, which is within the range of day-to-day body mass fluctuations (282, 719). In studies where hypohydration was 2.0 to 2.1% of body mass, intravenous infusion of isotonic saline was used to manipulate hydration state. In the study of Wall et al. (706), intravenous saline infusion was shown to restore blood volume, which probably alleviated the effects of hypovolemia on cardiovascular function, as evidenced by a similar cardiovascular (i.e., heart rate) response to euhydration. In contrast, hypohydration ≥3% yielded consistent performance impairments in ambient conditions of 30 to 40°C (702, 708). Kenefick et al. (702) reported increasingly larger performance reductions with hypohydration of 4% body mass when ambient temperature increased from 10 to 20, 30, and 40°C. Although the influence of hypohydration is less robust during self-paced compared with constant work rate exercise, it does appear to impair performance when exercise is initiated with a body water deficit of 3% or greater in temperate and hot environments.

In summary, caution should be employed when comparing the results of investigations assessing the impact of fluid status on performance outcomes because of differences in water deficit before and during exercise, environmental conditions, exercise task (i.e., constant work rate versus self-paced), and participant characteristics (i.e., age, sex, fitness, and acclimation state). It must also be acknowledged that while laboratory-based studies offer control and provide significant mechanistic insight regarding the impact of hydration status on exercise capacity and performance in the heat, several factors relative to exercising in a real-world environment reduce the validity of certain findings. These factors include familiarization to the stressor (i.e., hypohydration), ability to drink ad libitum, airflow provided during exercise, being blinded to hydration status, and understanding the consequences of hypohydration (229). Notwithstanding, based on the data discussed above it appears that constant work rate exercise is well maintained with dehydration and hypohydration around 1% body mass, even in the heat. However, a ≥2% body mass deficit incurred before or during exercise leads to a marked decrease in exercise capacity over a range of ambient temperatures (20 to 40°C). Although fewer data are available to compare the effects of hydration status on constant work rate exercise relative to self-paced exercise, the influence of dehydration and hypohydration are much clearer in the former. Indeed, the impact of dehydration during time trial exercise is equivocal and performance appears to be preserved up to a 4% loss in body mass in 20 to 33°C environments. Beyond this level of dehydration, the data are ambiguous but indicate a loss of performance. However, hypohydration is associated with a reduction in performance when a time trial is undertaken with a body mass deficit around 3% or larger across a spectrum of ambient temperatures (10 to 40°C). These observations support those of previous reviews (204, 660, 664, 665) and emphasize the impact of a reduction in total body water and blood/plasma volume in augmenting thermal strain and compromising systemic and regional blood flow and oxygen delivery. The data also highlight the importance of convective and evaporative cooling, with studies providing an airflow commensurate with the activity (e.g., running or cycling) associated with less of a performance impairment (685, 688, 689, 691, 694, 706), although several factors interact to regulate performance (**FIGURE 6**).

5.5. Blinded Rehydration

The effect of hypohydration on thermoregulatory, cardiovascular, and perceptual responses during prolonged exercise in the heat forms the mechanistic basis on which performance impairments occur. Conventional methods used to induce different levels of hypohydration (e.g., active or passive heat exposure with fluid

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restriction and diuretic administration) before an exercise task allow for participants to know the hydration status under which exercise will be conducted. This knowledge may partly influence performance (706, 708) through either a placebo (euhydration) or nocebo (hypohydration) effect (720), given that athletes are aware of the impact of hypohydration on endurance performance (721). Several recent studies have therefore attempted to blind hydration status by manipulating total body water via intravenous infusion of isotonic saline (705, 706, 722) or gastric infusion of water with a nasogastric feeding tube (690, 707, 708).

The first study attempting to blind hydration status rehydrated participants following exercise in the heat through intravenous isotonic saline infusion to elicit hypohydration of 0, 2, and 3% body mass loss before a 25-km cycling time trial in the heat with an ecologically valid airflow (9 $m \cdot s^{-1}$) (706). As a result of the infusion, blood volume was restored to baseline levels or above before commencing each time trial and remained elevated during exercise via continuous infusion to match sweat rate. Consequently, performance, physiological, and perceptual responses, including thirst sensation, were similar between trials, although rectal temperature was ~0.3°C higher from 17 km onward in the 3% hypohydration trial. The similarity in responses was attributed to saline infusion preventing greater cardiovascular strain by restoring blood/plasma volume and cardiac filling. Serum osmolality, which drives the thirst sensation (723, 724), was also similar between trials. It has been argued that the sensation of thirst, rather than the loss of body water, modulates performance impairments during aerobic exercise to ensure that brain osmolality remains within homeostatic range (711, 725). To investigate how thirst and hydration status might affect performance, Cheung et al. (705) used intravenous saline infusion after 90 min of dehydrating exercise in the heat to create body mass deficits of 0% and 3% before completing a 20-km time trial in hot conditions with moderate airflow (3 $m \cdot s^{-1}$). Thirst during the time trial was manipulated by altering the sensation of dryness in the mouth by rinsing with water. This approach reduced the sensation of thirst but performance was unaffected, regardless of hydration status and despite hypohydration leading to greater increases in rectal temperature (~0.7°C) and heart rate (~5 beats·min^{-1}). Although the sensation of thirst was reduced with mouth rinsing, it has been shown that oral fluid ingestion increases exercise performance relative to mouth rinsing (726), by stimulating oropharyngeal reflex inhibition of vasopressin secretion and dipsogenic drive (i.e., thirst) (727, 728). Using both intravenous infusion of isotonic saline and oral fluid ingestion to elicit a similar perception of thirst, Adams et al. (722) maintained euhydration and elicited mild dehydration (1.8% body mass loss) during intermittent cycling in warm conditions with moderate airflow (4.5 $m \cdot s^{-1}$). Despite eliciting only mild dehydration, performance was reduced (~8%) in the latter states of exercise compared with euhydration, in conjunction with an elevated rectal temperature (~0.4°C). Previous findings from that laboratory support the notion that mild dehydration or hypohydration impair aerobic exercise performance in the heat (683, 687, 703).

The intravenous infusion of isotonic saline following exercise-induced hypohydration restores blood/plasma volume to varying degrees without affecting plasma osmolality, such that subsequent exercise is performed under an atypical hydration status. To replicate the physiological and perceptual responses associated with exercise-induced hypohydration (i.e., decreased plasma volume and increased plasma osmolality and thirst) while keeping participants naïve to hydration status, James et al. (707) used a combination of oral and intragastric rehydration to maintain (0% body mass loss) and elicit a 2.4% body mass loss before a 15-min cycling time trial in the heat with minimal airflow (~0.35 $m \cdot s^{-1}$). The intermittent exercise protocol performed before the time trial elicited the physiological and perceptual responses commensurate with dehydration (i.e., increased heart rate, rating of perceived exertion, serum osmolality, and thirst and reduced plasma volume). Performance during the subsequent time trial was reduced by ~7.5% with hypohydration, while heart rate and gastrointestinal temperature were similar to euhydration, although a nonsignificant increase in final gastrointestinal temperature of ~0.35°C was noted with hypohydration (707). Given the low facing airflow in this study, it is likely that evaporative heat loss was affected in both the hypohydrated and euhydrated trials (315, 729). It has also been shown during a 5-km cycling time trial in the heat with an airflow of 4.5 $m \cdot s^{-1}$ that hypohydration (~2.2% body mass loss) impairs performance, independently of thirst, when manipulated by intragastric water delivery and drinking small amounts of water (690). The ~6% decrement in performance was associated with a similar heart rate to the euhydrated trial but greater increase in rectal temperature (~0.4°C).

Taken together, these observations indicate that the effects of hypohydration on endurance performance are not entirely mediated by dipsogenic drive, occurring in conjunction with both high and low sensations of thirst. Moreover, it appears that knowledge of hydration status does not exacerbate the impairment in performance stemming from hypohydration. Indeed, the influence of blinded and unblinded hypohydration on self-paced exercise performance in warm conditions with high airflow (5.9 $m \cdot s^{-1}$) was investigated by controlling for thirst and maintaining euhydration (~0.5% body mass loss) or

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inducing hypohydration (~3% body mass loss) via intragastric rehydration and oral ingestion of small amounts of fluid (708). Hypohydration similarly impaired 15-min cycling time trial performance whether blinded (~11%) or unblinded (~10%) to hydration status, with similarly exacerbated alterations in heart rate, rating of perceived exertion, gastrointestinal temperature, serum osmolality, thirst, and plasma volume, relative to euhydration. This reinforces the observations and interpretation of previous studies that hypohydration equivalent to ~3% of body mass is detrimental to performance, despite participants knowledge of hydration status.

5.6. Hydration Guidelines

The influence of hydration status on aerobic performance and the approach used to hydrate (i.e., ad libitum or planned drinking) during exercise are highly debated topics. It is not the intention of this section to participate in this debate or to address optimal fluid composition (i.e., carbohydrate and electrolyte), as previous papers have discussed this in detail (204, 725, 730, 731). Instead, this section outlines the evolution of hydration (i.e., fluid replacement) recommendations and contextualizes the most recent guidelines to exercise performance in the heat.

On one hand, performance impairments have been shown to occur when body mass losses exceed ~2% during exercise in well-controlled laboratory settings under temperate conditions (667, 669, 670, 672, 692, 698, 732, 733). Avoiding such losses with a planned fluid intake strategy has been suggested to optimize performance by preserving thermoregulatory and cardiovascular function (59, 146, 201, 204, 734–736). On the other hand, some studies have shown endurance performance to be uncompromised in temperate conditions despite a ~2% body mass loss (680, 684) and reported that the fastest finishers in endurance events (e.g., marathon and triathlon) often experience the greatest body mass losses (e.g., ~10%) (275, 737, 738). As such, it has been proposed that exercise performance may be maximized by drinking according to the dictates of thirst (i.e., ad libitum) (665, 666, 739, 740). This notion is based on the thirst mechanism being optimized to preserve serum/plasma osmolality within normal range and protect intracellular volume, thus preserving homeostasis (741). Although contention exists regarding the impact of hypohydration and dehydration on aerobic performance in cold and temperate conditions, there is general agreement that a compromise in hydration status is detrimental to endurance exercise in warm and hot conditions (see sects. 5.3 and 5.4). Recommendations regarding fluid replacement for optimizing performance and reducing the risk of thermal injury are therefore typically aimed at individuals exercising for protracted periods of time in hot outdoor environments. These recommendations have evolved since first introduced ~50 yr ago, to account for the sport or activity (i.e., duration and intensity), setting (i.e., recreational or competitive), individual (i.e., training and acclimatization status), and environmental conditions.

Interest regarding the impact of hydration status and fluid ingestion during athletic events emerged in the 1960s but mostly focused on marathon running. Following its inception in the first Olympic Games in 1896, drinking and eating during a marathon were discouraged for a variety of reasons, ranging from pride to digestive discomfort (742–744). While research regarding the effects of dehydration on work performance was advancing in occupational and military settings (220, 221, 223, 224, 585, 588, 590, 745), studies focusing on athletic performance appeared later and mainly investigated the impact of severe body water loss on heat dissipation and hyperthermia. For example, Pugh et al. (746) reported that the winner of a marathon in temperate conditions experienced a 6.7% loss of body mass and final rectal temperature of 41.1°C. Such elevated rectal temperatures had been reported previously in shorter (e.g., <10 km) warm, humid races (747). Of interest, however, was that average water intake during the marathon was ~420 mL and the concomitant body mass deficit ~5.2% (746). It was concluded that the capacity to dissipate heat limits performance and that successful runners have elevated sweat rates. To avoid the detrimental effects of dehydration, Wyndham and Strydom (748) subsequently suggested to drink 300 mL of water every 20 min from the beginning of a marathon (748). This suggestion was derived from the significant linear relationship ($r = 0.67$) established between the magnitude of dehydration (beyond 3% body mass loss) and final rectal temperature during a 32-km run. A similar correlation ($r = 0.58$) between dehydration (~6% body mass loss) and rectal temperature was originally observed following the Boston Marathon by Buskirk and Beetham (749), a relationship that improved ($r = 0.66$) when controlling for work rate (i.e., speed × body mass). Costill et al. (750) contextualized the issues surrounding hydration and marathon running at the time by stating that "while there appears to be significant value in the replacement of body fluids during prolonged, severe running, the rapid fluid loss, limited rate of gastric emptying, current competitive rules, and feeding habits during the marathon makes this practice largely ineffective." These issues, although identified in the context of marathon running, are inherent to most endurance sports performed within the heavy-to-severe intensity domains in a structured competitive environment (e.g., cycling, race-walking). Notwithstanding, these early observations

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provided the impetus for developing hydration guidelines regarding fluid intake during exercise.

The American College of Sports Medicine published its first position stand regarding the prevention of heat injuries during distance running in 1975, recommending to frequently ingest fluids during competition (751). The position stand was expanded in 1984 to consider community joggers, fun runners, and elite athletes participating in distance running with the recommendation to drink 100 to 200 mL of water every 2–3 km (752, 753). These recommendations latter evolved to drink early and often during exercise to replace all body mass lost via sweating or consume the maximal amount of fluid tolerable (754). Adjustments to this position were then made indicating that to maintain performance an individualized fluid replacement plan should be adopted to prevent excessive dehydration (>2% body mass loss) and pronounced alterations in electrolyte balance (734). In 2000, the National Athletic Trainers Association published their first statement on fluid replacement during physical activity advising that fluids should be consumed to offset sweat and urine losses and maintain body mass loss <2% by consuming 200–300 mL every 10 to 20 min (755). This recommendation was advocated for several years (59, 158) and recently amended to indicate that health and performance are optimized when body mass losses are limited to 2% or less, without gaining body mass through water ingestion (756). The caution against gaining body mass was added following the first case of symptomatic hyponatremia linked to endurance exercise (757) and the subsequent increase in documented cases (265, 266, 758) (see also sect. 3.4). In contrast to statements emphasizing the prescription of fluid intake based on body mass loss, the International Marathon Medical Directors Association suggested in 2003 that athletes drink 400–800 mL·h^{-1} ad libitum, with faster and heavier runners racing in warm conditions drinking at the higher rate, and slower runners competing in cool conditions at lower rates (759). These recommendations were updated 3 yr later, with the advice that runners understand their individualized fluid needs but defer to physiological cues to increase (i.e., thirst) or decrease (i.e., increased urination, bloating, and weight gain) fluid intake when running (741). It was further indicated that running in extreme heat (>38°C) may require fluid intake beyond the dictates of thirst.

Following on from the evolution of these recommendations and expanding research regarding the impact of hydration status on performance and whether to drink to a plan, or to thirst, balanced and practical hydration recommendations have emerged to incorporate a broader spectrum of sports and physical activities. These place greater emphasis on the context in which exercise is performed than avoidance of a particular level of dehydration (i.e., %body mass loss). For example, to reduce physiological strain and preserve optimal performance, a panel of experts recommended in a consensus statement on training and competing in the heat that body mass loss from water deficit should be minimized during prolonged intense exercise in the heat, within the constraints of the competition setting (e.g., fluid availability and event characteristics) (181). It has further been suggested that high-intensity exercise eliciting elevated sweat rates, along with activities >90 min in the heat, should be accompanied with a planned hydration strategy, whereas drinking to thirst may be sufficient to offset fluid losses during low-intensity exercise of shorter duration (<90 min) in cooler climates (760). A meta-analysis examining the impact of fluid intake strategy on endurance exercise in warm conditions concluded that planned and ad libitum drinking yielded similar performance outcomes, with body mass losses of ~1% and ~2%, respectively (739). The slightly greater loss of body mass with ad libitum drinking is a common observation given that when provided with access to fluids, humans replace <75% of body water losses during exercise (194, 226, 237–239). Nevertheless, the findings of the meta-analysis indicate that the two hydration strategies should be viewed as complementary with the decision to use a particular one based on several factors, including exercise duration, fluid availability, food ingestion, ambient conditions, heat acclimatization status, and various logistical factors. A position statement from Sports Dieticians Australia on nutrition for exercise in hot environments highlighted that the divergence in total fluid intake between planned and ad libitum drinking may increase with exercise duration, particularly under heat stress, and opportunities to access fluid during real-life sporting events were more limited than in laboratory studies (304). To address both the practicality and value of fluid intake during a competitive event, it was suggested that individualized fluid replacement plans be informed by prior assessment of fluid balance, perceived thirst, gastrointestinal tolerance and performance metrics in similar settings to competition and adjusted according to real-time assessment.

Finally, to accurately determine the impact of hydration status on athletic performance, careful establishment of a euhydrated baseline is required. Given the day-to-day fluctuations in body mass (≤1%) attributable to variations in total body water (282, 719), at least 3 consecutive days of measurements should be performed nude, following first morning void and after ingesting fluids (1–2 L) the evening prior (204). Total body water loss provides an estimate regarding how much dehydration may impact on physiological and perceptual responses, within the context of an exercise task in particular environmental conditions. For example, in cold and

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temperate conditions, a greater level of dehydration (e.g., 2 to 4% body mass loss) is better tolerated during prolonged high-intensity exercise, even with an elevated body core temperature, due to attenuated physiological and perceptual responses.

5.7. Summary

A rise in core temperature and loss of body water via sweating are natural consequences of prolonged moderate to high-intensity exercise. When undertaken in the heat, exercise-induced hyperthermia and dehydration are augmented, with excessive body water loss reducing blood volume and increasing osmolality. This response influences thermoregulatory capacity by reducing the sensitivity of thermoeffector responses to a given core temperature, further increasing thermal strain. The rise in whole body temperature augments skeletal muscle glycogenolysis and anaerobic metabolism and coupled with hypovolemia progressively compromises systemic and regional (i.e., cutaneous, active muscle, and cerebral) perfusion and oxygen delivery. These adjustments lower $\dot{V}O_{2max}$ and accelerate the development of fatigue during constant work rate exercise, primarily by attenuating oxygen delivery and uptake in exercising skeletal muscles. Although likely unrelated to modifications in cerebral perfusion, adjustments in central neural drive may also impact on the ability to continue exercising when hyperthermic and dehydrated. Performance impairments during self-paced exercise relate to similar hemodynamic adjustments intensifying physiological and perceptual responses. While hyperthermia is unavoidable during prolonged high-intensity exercise in hot environmental conditions, ensuring euhydration before exercise and replacing sweat losses during exercise contribute to mitigate the compounding effect of hyperthermia and dehydration. The choice of which approach to utilize, drink to thirst or plan to drink, should be dictated by the nature of the exercise task (i.e., intensity, duration), ambient conditions, availability of fluids, and an understating of one's fitness and acclimation status.

6. MITIGATING THE IMPACT OF HYPERTHERMIA AND DEHYDRATION

Section 4 described how the increase in thermal strain during aerobic exercise under heat stress intensifies physiological and perpetual responses to detrimentally impact on performance. In sect. 5, it was demonstrated that the loss of body water during prolonged exercise can further intensify these responses and exacerbate performance impairments. This section discusses the potential pathways via which reductions in performance stemming from hyperthermia and dehydration may be alleviated. Firstly, human adaptation to repeated heat exposure (i.e., heat acclimation) is discussed, as are some of the contentious issues regarding the adaptive process and consequent benefits. Secondly, the potential for external and internal cooling methods to improve performance is outlined, along with the time frame (i.e., before and during exercise) for using different approaches and their physiological underpinning. Finally, the effects and pathways via which hyperhydration influences exercise performance in the heat are described.

6.1. Heat Acclimation

The development of thermal strain when physically active in the heat intensifies the physiological and perceptual responses associated with exercising at a given work rate, leading to impaired endurance capacity. Progressive dehydration as a result of excessive sweat loss in the heat exacerbates this impairment by further reducing evaporative heat loss and increasing cardiovascular strain. However, repeated exposure to heat stress, such as during natural heat acclimatization or laboratory-based heat acclimation, induces adaptations that allow for better fluid balance and increased cardiovascular stability. Although elicited in different settings, heat acclimatization and heat acclimation induce similar physiological adaptations and are often used interchangeably (761, 762). These adaptations include increased total body water and expanded plasma volume, reduced heart rate, increased stroke volume and better sustained cardiac output during exercise, increased myocardial function (in animal models), enhanced skin blood flow and sweating responses, improved skeletal muscle metabolism, and increased thermal tolerance when exercising at a given work rate (146, 761, 763–768). TABLE 6 outlines the functional benefits associated with the heat acclimation/acclimatization that improve performance and comfort in the heat when exercising at a given work rate, as well as reduce the risk of exertional heat illness and thermal injury (146, 763). This section will further outline the time course of human heat adaptation, explain the different approaches that can be used to induce heat acclimation, detail the performance benefit conferred by acclimation when exercising in hot and possibly cool conditions, detail the process of sudomotor and vasomotor adaptation, describe the process of total body water and plasma volume expansion, and examine whether permissive dehydration enhances the adaptive process.

6.1.1. Time course of heat adaptation.

Heat acclimation is considered the primary intervention one can adopt to reduce physiological strain and

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Table 6. ***Physiological adaptations and functional consequences associated with the heat acclimation phenotype in humans that improve performance at a given work rate and increase maximal aerobic power***

Adaptation	Consequence
Core temperature	Reduced
Rest: decreased	
Exercise: decreased	
Sweating	Improved
Onset threshold: decreased	
Rate: increased	
Sensitivity: increased	
Skin temperature	Reduced
Exercise: decreased	
Skin blood flow	Improved
Onset threshold: decreased	
Sensitivity: increased	
Fluid balance	Improved
Thirst: improved	
Electrolyte losses: reduced	
Total body water: increased	
Plasma volume: increased	
Cardiovascular stability	Improved
Heart rate: lowered	
Stroke volume: increased	
Cardiac output: better sustained	
Blood pressure: better defended	
Skeletal muscle metabolism	Improved
Muscle glycogen: spared	
Lactate threshold: increased	
Muscle and plasma lactate: lowered	
Muscle force production: increased	
Whole body metabolic rate	Lowered
Acquired thermal tolerance	Increased
Heat shock proteins expression: increased	
Cytoprotection: improved	

This table was adapted with permission from Sawka et al. (146).

optimize performance in anticipation of exercising in hot environmental conditions (181). The adaptive capacity of humans to environmental heat stress has been recognized for centuries, with reports of Europeans relocating to East and West Indian climates initially being adversely affected by the environment but over time habituating and living comfortably (769). This habituation process included behavioral modifications (e.g., reduced work rate, rescheduled work periods, and use of shelters) and potential blood adaptations allowing for "a pretty good state of health" (769). In 1884 Jousset (770) expanded on these observations by providing a comprehensive overview of the responses associated with heat acclimatization, suggesting that the human organism adapted to hot climatic conditions by modifying either its constitution or function. The author highlighted that a lowering of heart rate and improved maintenance of blood pressure were fundamental adaptive responses. The hematological adaptations occurring following prolonged sojourns in different climates were later described by Barcroft et al. (771) with an account of "blood volume increasing as the temperature rose and decreasing as it fell." A comprehensive understanding of the essential adaptations to heat stress at the time was summarized by Sundstroem (772), including those related to body temperature, heat exchange, metabolism, blood distribution, heart rate, blood pressure, endocrine, and nervous system function. The time frame and magnitude of heat adaptations, along with the mechanistic pathways via which they developed, were greatly elucidated during the last century by research conducted in military and occupational settings (e.g., mining) (773–779). This knowledge was expanded on with more recent research findings, some of which within a sporting context (563, 780–791).

Heat acclimation is a highly individualized process and dependent on several factors such as the active or passive nature of the regimen, the duration, and frequency and number of heat exposures, along with the environmental conditions in which it occurs. Acquisition of the heat acclimation phenotype occurs relatively quickly with a substantial fraction of adaptations in certain physiological parameters developing within the first week of heat exposure (e.g., plasma volume expansion and decreased heart rate) (761, 792, 793). However, regimens of 14 days or longer are recommended to achieve maximal adaptations and the associated benefits (**FIGURE 21**) (765, 794). This recommendation aligns with the biphasic model of heat acclimation proposed by Horowitz et al. (795–797) in which the kinetics of adaptation are mediated by interactions between central thermoregulatory autonomic outflow and effector organ responsiveness. The initial short-term phase (1–5 days) is characterized by a decreased effector organ output-to-autonomic signal ratio, whereby increased efferent activity overrides impaired peripheral responsiveness to produce adequate effector output. In the second longer term phase (>21 days), the effector organ output-to-autonomic signal ratio is increased, as both central and peripheral adaptations enhance physiological efficiency and reduce the requirement for increased excitation. The extensive work by Horowitz and colleagues (797–807) using an animal model (i.e., rats) over protracted periods (up to 60 days) of continuous heat exposure (34°C) has provided a breadth of mechanistic insights. As a result, the genomic responses, molecular signaling

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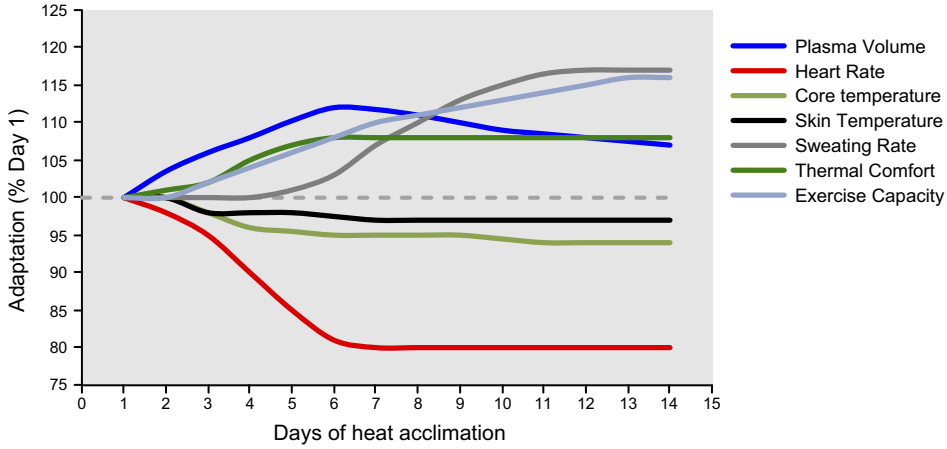

FIGURE 21. Generalized time course of adaptations related to exercise-heat acclimation. Within a week of acclimation plasma volume expansion occurs and heart rate is reduced during exercise at a given work rate. Core and skin temperatures are also reduced when exercising at a given work rate, whereas sweat rate increases when in uncompensable conditions. Perceptually, the rating of thermal comfort is improved. As a result of these adaptations, aerobic exercise capacity is increased. During the second week of heat acclimation some adaptions can further develop or reach a plateau. The magnitude of these adaptations is dependent on the active or passive nature of the regimen, the duration, frequency and number of heat exposures, and the environmental characteristics. Adapted with permission from Périard et al. (765).

and epigenetic mechanisms associated with the acquisition of heat acclimation homeostasis have begun to emerge. This includes insight into the neuroplasticity of the thermoregulatory system (e.g., changes in temperature-thresholds for activating heat dissipation effectors), the increase in hypothalamic and cardiac cytoprotective molecules (e.g., heat shock proteins and hypoxia-inducible factor-1α), alterations in the expression (i.e., faster activation and suppression) of genes involved in cross-tolerance (e.g., ischemic-reperfusion), and cardiac remodeling of the myosin isoform profile increasing contractile efficiency (i.e., greater pressure generation and slower contraction and relaxation velocities) (763, 808, 809). These insights, however, emphasize the gap in knowledge between the mechanistic understanding of long-term heat adaptations in animals and that of human physiological heat acclimation, particularly as it relates to long-term biphasic acclimation. Moreover, although a generalized categorization of short (<7 days)-, medium (8–14 days)-, and long-term (>15 days) heat acclimation has been proposed for humans (781), the heterogeneity between heat acclimation regimens necessitates a classification more appropriately based on the adaptive stimulus provided by a chosen regimen, such as the cumulative adaptation impulse (810). To date, however, a representative index of the adaptive stimulus provided by heat acclimation and the consequent level of adaptation has yet to be developed and utilized.

The physiological adaptations associated with heat acclimation are transient in nature and steadily decay without consistent heat exposure. It has been proposed that the adaptations developing most rapidly (e.g., decreased heart rate) during the acclimation process are also those that decay most quickly (773, 811). Given the diversity of heat acclimation protocols and the relatively few studies that have examined the rate at which adaptations deteriorate, there is contention regarding the rate of decay for heat acclimation. It was suggested that 1 day of exercise-heat exposure is required for every 5 days spent outside of the heat to maintain adaptation (773, 812) or that 1 day of heat acclimation is lost for every 2 days spent without heat exposure (813). In a recent meta-analysis of 21 studies, it was concluded that the rate of decay for the main adaptations following heat acclimation (i.e., lowered heart rate and core temperature) is ~2.5% per day without heat exposure and that there are insufficient data to make a good estimate for the decay in sweat rate (814). As such, in the 2 wk following heat acclimation, a well-acclimated individual may lose 30–35% of the heart rate and core temperature adaptations if regular heat exposure is removed. Notwithstanding, there is support for the notion that aerobic fitness and regular exercise in cool conditions contribute to maintain adaptations or reduce the rate of decay (773, 815).

6.1.2. Heat acclimation approaches.

Heat adaptation occurs following a series of prolonged daily or semidaily exposures to an environment that elevates whole body temperature, increases skin blood flow, and elicits profuse sweating. Differences in endogenous (i.e., metabolic heat production) and exogenous (i.e., ambient and radiative temperature, humidity, and air velocity) thermal loads allow for creating or utilizing environments suitable for different heat acclimation and acclimatization approaches. As highlighted in **FIGURE 22**, these include self-paced exercise, constant work

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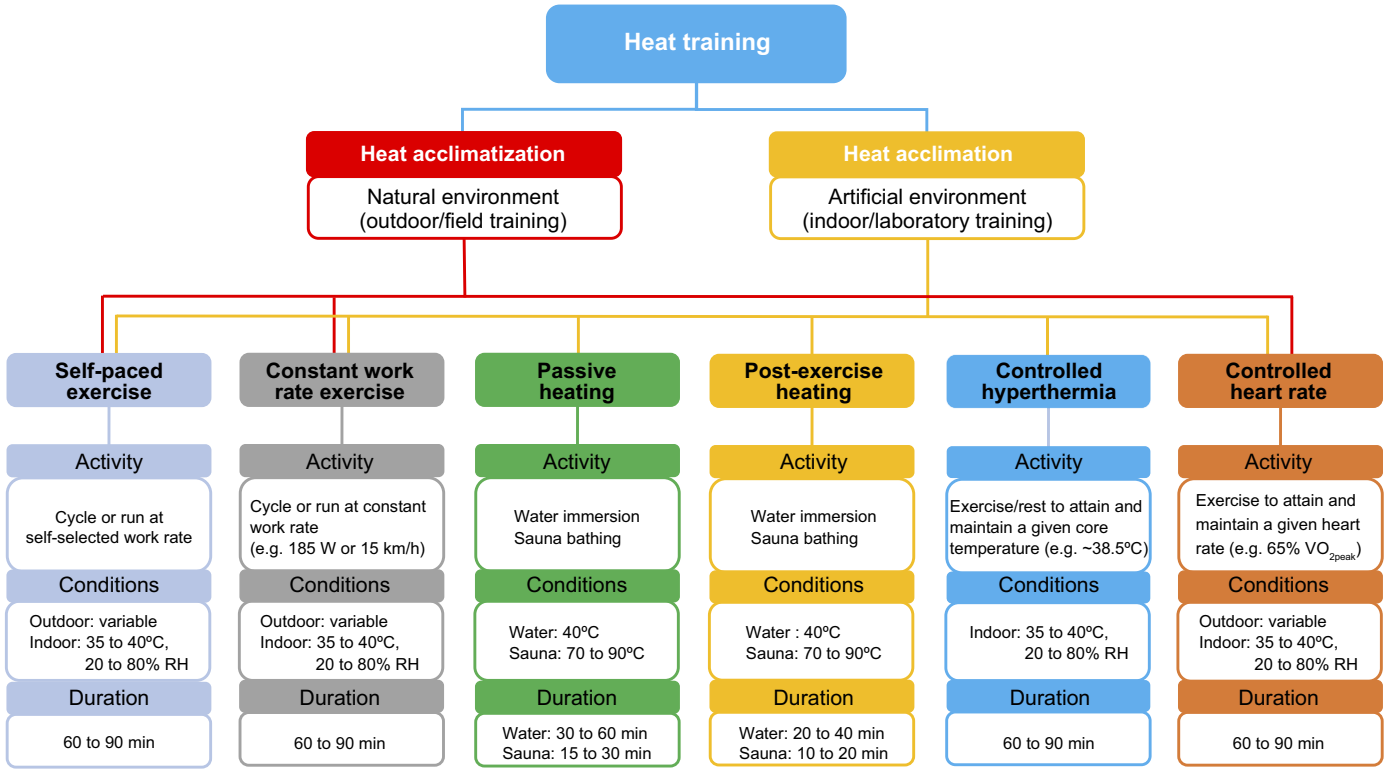

FIGURE 22. Schematic overview of the methods used for heat acclimation and heat acclimatization, with examples for activity selection, environmental conditions and duration. Manipulation of temperature and humidity is recommended in accordance with the anticipated environmental conditions to be encountered during upcoming competitions. RH, relative humidity. Adapted with permission from Daanen et al. (814).

rate exercise, passive heating, postexercise passive heating, controlled hyperthermia, and controlled heart rate heat acclimation. Although all of these approaches can be utilized in artificial environments (i.e., indoor or laboratory settings) to heat acclimate, only the self-paced exercise model can be employed with relative ease in a natural outdoor environment to acclimatize. Constant work rate and controlled heart rate protocols can also be used to heat acclimatize but with greater difficulty due to the logistical constrained (e.g., terrain) associated with maintaining a given work rate or heart rate.

The self-paced exercise-heat acclimatization approach was originally developed by the military to ensure the safety of large and diverse groups of recruits during basic training and to prepare unacclimatized soldiers for rapid deployment to hot environments (816, 817). In an athletic context, self-paced heat acclimatization offers a sport-specific means of inducing adaptation in several individuals exercising together but self-regulating their effort according to fitness level and the parameters of the training session and prevailing ambient conditions. It is therefore often used with team-sport athletes (818–822) and has been successfully utilized to enhance performance in trained cyclists following an outdoor training camp (563, 783). The potential shortcomings of this approach lay with the difficulty in standardizing the inter- and intraindividual stimulus for adaptation within and between training sessions, stemming from the self-regulated nature of the regimen. Notwithstanding, self-paced exercise-heat acclimatization provides an individual with the ability to control their effort based on the integration of objective measures such as heart rate and time, as well as subjective perceptual cues like the perception of exertion, thermal comfort, and sensation.

Similar to self-paced heat acclimatization, the constant work rate approach was developed within military and occupational (e.g., mining) settings (779, 792, 823–826). It provides significant improvements in work capacity under heat stress through enhanced thermoregulatory, cardiovascular, metabolic, and cellular adaptations (112, 372, 398, 473, 827, 828). It has also been shown to improve time trial performance (785, 787) and intermittent sprinting (829) in the heat. Because the endogenous (i.e., work rate) and exogenous (i.e., ambient conditions) thermal loads are fixed during constant work rate exercise, it has been suggested that a progressive reduction occurs in the forcing function driving adaptation, as physiological strain decreases with acclimation, potentially attenuating the adaptive response (768, 830). This limitation was accommodated for in a recent study examining the effects of prolonged (5 wk) exercise-heat acclimation (cycling at 60% $\dot{V}O_{2max}$),

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whereby ambient temperature was set to 35°C (30% RH) in the first week of training and increased by 1°C in subsequent weeks (787, 831). The authors reported that rectal temperature was elevated to ~38.5°C within 35–40 min and increased to ~39.6°C at the end of all 60-min training sessions, providing a consistent thermal impulse for adaptation. Constant work rate heat acclimation has also been shown in a meta-analysis to provide a similar magnitude of improvement in exercise performance to that of controlled hyperthermia heat acclimation (794). A direct comparison between approaches also found that controlled hyperthermia did not provide greater adaptations than constant work rate exercise (832, 833). It appears, therefore, that constant work rate heat acclimation is a viable and potent means of inducing adaptations to the heat.

Controlled hyperthermia, or isothermic heat acclimation, was developed by Fox et al. (830, 834, 835) in an attempt to define the stimulus for adaptation based on the degree and duration of thermoregulatory imbalance (i.e., elevation of body temperature), rather than the characteristics of the climate in which participants were exposed. This approach is purported to provide a forcing function that increases in proportion to adaptations by manipulating endogenous and/or exogenous thermal loads to attain and maintain a target core temperature, typically around 38.5°C (768, 781). Numerous studies have demonstrated that controlled hyperthermia heat acclimation leads to several hallmark adaptations, including a lower heart rate and core temperature during exercise, higher sweat rate, and improved aerobic exercise capacity in the heat (833, 834, 836–841). It has also been reported that the expansion of plasma volume may be sustained during controlled hyperthermia heat acclimation, as it allows for maintaining a consistently elevated physiological strain throughout the adaptive process (842). These findings remain to be replicated, however, as the early expansion of plasma volume generally regresses during prolonged heat acclimation (826, 843–845). Notwithstanding, the controlled hyperthermia model of heat acclimation offers a safe and effective way of inducing adaptation due to the constant monitoring of core temperature. From a practical perspective, however, it may lack real-world application and relevance to athletes and coaches training with heart rate, rather than with body core temperature.

An alternative approach to controlled hyperthermia was recently proposed with the adaptive stimulus regulated by manipulating work rate to maintain a given heart rate (765). With this approach, the level of strain attained and sustained within each acclimation session corresponds to the heart rate associated with a relative intensity specific to exercise in cool conditions (e.g., % $\dot{V}O_{2max}$). Hence, the absolute work rate required to maintain a given heart rate increases as acclimation develops, providing a constant stimulus for adaptation. For example, the power output required to hold a heart rate equivalent to 65% $\dot{V}O_{2max}$ during the last 75 min of a 90-min session in 40°C and 40% RH increased by ~25 W (15%) throughout a 10-day protocol (846). Controlling heart rate also allowed for maintaining an average rectal temperature of ~38.4°C for those final 75 min. Other studies have utilized the controlled heart rate model to induce heat acclimation and demonstrated a lowered core temperature and heart rate during exercise at a given work rate in the heat, increased sweat output and reduced sweat sodium concentration, plasma volume expansion, improved cerebral perfusion, enhanced heat dissipation (i.e., evaporative heat loss), and improved time trial performance under heat stress (113, 846–849). Given that the maintenance of a greater relative exercise intensity during exercise-heat acclimation may hasten the adaptive process (794), the controlled heart rate approach appears to offer both a practical and time-efficient manner by which to heat acclimate.

Passive heat acclimation (i.e., without an exercise component) involves regular exposure to a high exogenous heat load provided by a hot bath (e.g., ~40°C), environmental chamber (e.g., >45°C), or sauna (e.g., 70–90°C) for 30 to 90 min, depending on the medium. The approach has been shown to yield adaptations commensurate with exercise-heat acclimation, such as a decrease in heart rate and rectal temperature and an increase in sweat rate during exercise at a given work rate in the heat (850–853). Passive heat acclimation has also been shown to lower the onset threshold for sweating and increase sweat sensitivity (854, 855), initiate hyperthermia-induced ventilation at a lower core temperature (105), and improve skeletal muscle contractility (i.e., twitch amplitude) and force production capacity without altering central activation or peripheral neural activity, evidence of enhanced skeletal muscle function (788). Although passive heat acclimation has been shown to increase $\dot{V}O_{2peak}$ in temperate conditions (854), changes in endurance performance under heat stress have not been evaluated and it remains undetermined whether this approach can lead to improvements in prolonged exercise capacity. Based on the principles of adaptation theory (768), it has been suggested that exercise-heat acclimation regimens replicating the work rate and environmental conditions in which competition will occur may be more effective at developing sport-specific adaptations that contribute to improve performance (765, 856). However, in highly trained individuals, thermoregulatory heat adaptations may suffice to enhance performance in the heat, due to an already well-developed aerobic capacity.

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In an effort to maintain a strong training impulse and induce heat acclimation, passive heat exposure is now frequently being used immediately after exercise in cool conditions. This approach has the benefit of allowing athletes to complete regular training without heat stress affecting training quality, coupled with acquiring the adaptations related to chronic heat exposure. Post-exercise passive heating for 30 to 45 min can be achieved via sauna exposure (789, 857) or hot water immersion (791, 858–861) as it induces an increase in whole body temperature beyond that achieved with exercise in cool conditions. The adaptations associated with this approach are akin to those of more traditional heat acclimation protocols. In a series of studies, Zurawlew et al. (791, 859–861) demonstrated that 6 consecutive days of postexercise (i.e., 40 min run at 65% $\dot{V}O_{2max}$) immersion in 40°C water lowered core and skin temperature, heart rate, perceived exertion, and thermal sensation in both endurance-trained and recreationally active individuals during a 40 min run in the heat. Part of these adaptations were also retained for 2 wk after completion of the regimen (861). From a performance perspective, postexercise water immersion has been shown to improve 5-km treadmill performance by ~5% in hot but not cool conditions (791). In contrast, a 32% increase in run time to exhaustion in cool conditions was noted in fit individuals after heat acclimation via postexercise sauna bathing, which is equivalent to a ~2% improvement in 5-km running time trial performance (857). Although the evidence is limited, it appears that passive heating following exercise in cool conditions provides a practical approach to heat acclimate in those that may have difficulty adopting more traditional exercise-based regimens, or those that wish to maintain training quality (862, 863).

6.1.3. Performance benefits in hot conditions.

The benefits of heat acclimation on exercise performance in the heat are well established. For example, exercise-heat acclimation has been shown to increase $\dot{V}O_{2max}$ by ~4% when undertaken in 49°C following whole body preheating (543) and by 8 to 10% in 38°C (785, 847). Heat acclimation has also been shown to enhance endurance exercise capacity by ~23% (i.e., time to exhaustion or task failure) and performance by ~7% (i.e., self-paced time trial) under heat stress, with medium to long-term acclimation providing more robust improvements than short-term regimens (794). In a heat acclimatization study with well-trained cyclists, Racinais et al. (563) demonstrated that three cycling time trials (43.3 km) undertaken in hot outdoor conditions (~36°C) were initiated at a similar power output to that of pre- and postacclimatization time trials performed in cooler conditions (~9°C). The authors reported a marked decrease in power output following the onset of exercise in the heat on the first day of training in hot (34°C) and dry (18% RH) outdoor conditions, leading to a ~16% lower mean power output than in the cool trials (**FIGURE 23**). However, after 1 wk of training in the heat, the decrement was partly recovered with mean power output only ~8% lower than that of the cooler trials and almost fully restored (i.e., ~3% lower) after 2 wk of training in the heat. Interestingly, heart rate was similarly elevated throughout the time trials in the heat and slightly higher than in the cooler trials, which supports the notion that a similar relative exercise intensity (i.e., $\%\dot{V}O_{2max}$) is maintained during self-paced exercise in hot and cool conditions (312, 314, 864), with heat acclimation/acclimatization increasing the absolute intensity (e.g., power output) that can be sustained. Other potential adaptations conferred by heat acclimation that could benefit endurance performance include a reduction in oxygen uptake (767, 865) and blood and muscle lactate accumulation (473) during submaximal exercise in the heat, as well as an increase in lactate threshold (785, 838). The mechanisms responsible for these adaptations remain unclear but could stem from the increase in total body water enhancing lactate removal through increased splanchnic circulation (866) or through increased cardiac output and decreased metabolic rate delaying lactate accumulation (500, 865). Heat acclimation has also been shown to reduce muscle glycogen utilization during submaximal exercise in the heat (397, 500, 503), in part due to a reduction in plasma epinephrine (473). Collectively, these adaptations provide the integrative framework upon which endurance performance is improved when heat acclimated.

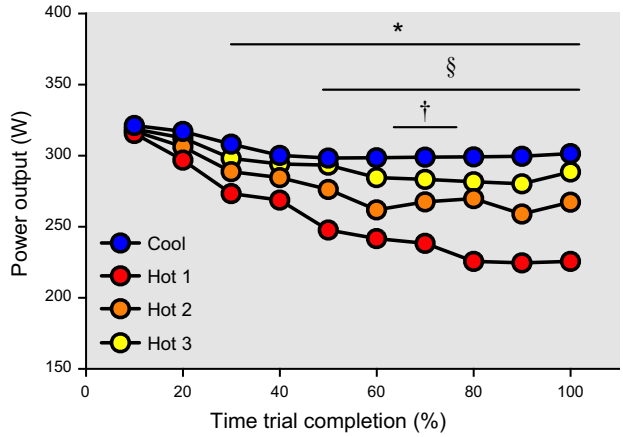

FIGURE 23. Power output during 43.4-km cycling time trials performed in Cool (~8°C; average of trials performed before and after heat acclimatization) and Hot (~37°C) conditions on *day 1* (Hot 1) and after 6 (Hot 2) and 14 (Hot 3) days of training in the heat. These data indicate the magnitude of improvement that can occur in time trial performance with heat acclimatization in trained cyclists. *§†Cool time trial power output significantly higher than Hot 1, Hot 2, and Hot 3, respectively ($P < 0.05$). Redrawn with permission from Racinais et al. (563).

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6.1.4. Performance benefits in cool conditions.

Observations of performance enhancement in cool conditions following heat acclimation have sparked debate as to whether heat adaptations provide a benefit to exercise performance in cooler conditions (867, 868). For example, one group demonstrated significant improvements in $\dot{V}O_{2max}$ (~180 mL·min^{-1}) and 60-min time trial performance (~16 W) in cool conditions following 10 days of constant load heat acclimation (785), while another reported no such improvement, despite similar changes in $\dot{V}O_{2max}$ (~200 mL) and 30-min time trial performance (~6 W) following controlled heart rate heat acclimation (847). Several other studies have reported that training in the heat enhances different aspects of aerobic performance in cool conditions (13 to 23°C), such as $\dot{V}O_{2max}$ (543, 869), power output at $\dot{V}O_{2max}$ (838, 840, 870), lactate threshold (785, 838, 840), and endurance performance (840, 857, 871). These improvements have been reported following a variety of short- to long-term heat acclimation regimens using passive, active, and controlled hyperthermia protocols in participants of different fitness levels and within different phases of their competitive season. Similarly, team-sport athletes participating in preseason (820), in-season (819), and off-season (818) training camps have been shown to experience improvements in performance in cool conditions following a period of intensified training in the heat. Cardiovascular, hematological, thermoregulatory, skeletal muscle, and cellular adaptations stemming from heat acclimation have been suggested to provide the ergogenic stimulus for enhancing performance in cooler environments (785, 872, 873). However, similar to the contention regarding the effects of altitude training on endurance performance at sea-level (874–876), the transfer of benefits stemming from training in the heat to performance in cool environments (i.e., cross adaptation) is difficult to accurately determine given the heterogeneity between the studies highlighted above.

The absence of a control group in several studies precludes the ability to adequately distinguish between the effects of heat acclimation and those of training per se on performance enhancement in cool conditions. In a study that included both hot and control (i.e., cool condition) training groups, it was shown that 30-min time trial performance, $\dot{V}O_{2max}$ and peak power output increased only in the heat acclimation group when testing was undertaken in the heat (847). In a separate study, 2 wk of outdoor training in hot (34°C) or cool (<15°C) conditions failed to improve 43.30km time trial performance in cool conditions in either group, despite an overall improvement in $\dot{V}O_{2max}$ in cool conditions, predominantly within the control group (877). Following 28 sessions over 5–6 wk of heat (35–40°C) or control (<15°C) training, Mikkelsen et al. (787) showed that 15-km time trial performance in cool conditions (14°C) was improved by ~6% in both groups, with $\dot{V}O_{2max}$ and peak power output remaining unchanged. Taken together, the benefits of training in hot compared with cool conditions for improving performance in cooler environments appear to be unclear, potentially due to nature and intensity (i.e., moderate to intense) of the training regimens used to induce exercise-heat acclimation. Moreover, training per se, even in cool conditions, leads to adaptations commensurate with heat acclimation.

Indeed, even when undertaken in cool conditions, endurance training reduces physiological strain and increases exercise capacity in the heat, with aerobically trained individuals exhibiting several characteristics of the heat-acclimated phenotype (e.g., lowered resting heart rate and core temperature) (878, 879). A recent meta-analysis examining the impact of heat mitigation strategies on lowering core temperature at the start of exercise, attenuating the rate of rise in core temperature during exercise, and improving endurance performance determined that the most influential strategy was being aerobically fit, followed by heat acclimation, precooling, and fluid ingestion (880). Sotiridis et al. (881) recently demonstrated that 10 sessions of aerobic training in temperate conditions (24°C) elicited a ~10% improvement in $\dot{V}O_{2max}$ under heat stress in untrained but not trained individuals, with both groups exhibiting a lower resting rectal temperature and increased whole body sweat rate during exercise in the heat. The lower resting core temperature of fit individuals has been proposed to stem from a training-induced increase in blood volume enhancing core-to-skin heat conductance through increased cutaneous blood flow (882). Ravanelli et al. (849) also demonstrated that 8 wk of aerobic training in 23°C conditions led to reductions in resting and end-exercise core temperature in previously untrained individuals, as well as an increase in whole body sweat rate during uncompensable exercise-heat stress, adaptations that were further enhanced following 6 days of heat acclimation. The authors suggested that adaptations related to aerobic training, rather than fitness per se (i.e., $\dot{V}O_{2max}$), mediated these thermoregulatory improvements when exercising at a fixed rate of heat production per kilogram of total body mass (115). The independent effect of training, fitness, and acclimation status on thermoregulatory responses during uncompensable heat stress remains difficult to determine, however, as these factors are intimately linked. The addition of a control group to heat acclimation research studies is also complex, as exercise performed at a given work rate in cool conditions will elicit a lower thermal and

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cardiovascular response than in the heat, whereas exercise performed to produce a given thermal or cardiovascular response will lead to a lower work rate being sustained in hot compared with cool conditions. The nature of the exercise stimulus (i.e., absolute work rate and metabolic rate) along with the thermal and cardiovascular responses would therefore differ, which to a certain extent is the goal of adding a control group. The decision regarding what type of stimulus to provide a control group should be based on the research question, with the inclusion of both matched exercise stress and physiological strain groups likely providing valuable insight. Ultimately, the addition of at least one control group to heat training studies would allow for better identifying the extent to which heat stress and/or training stress provide the adaptive benefit for enhancing performance in hot as well as cool conditions.

6.1.5. Sudomotor and vasomotor adaptations.

An improved sweating response is considered a hallmark indicator of heat acclimation with both central and peripheral adaptations contributing to the improvement. Centrally, a shift in the internal temperature threshold for the initiation of sweating occurs in response to a heat acclimation mediated reduction in core temperature (106, 112, 883). The onset threshold for sweating occurs at a lower internal temperature but following a similar change in absolute temperature (884). Peripherally, adaptations occur at the level of the sweat gland with an enhancement of both secretory capacity and sensitivity (835, 885–888). These adaptations stem from enhanced cholinergic sensitivity along with an increase in size (i.e., hypertrophy) and efficiency of eccrine glands (889, 890). An increased resistance to hidromeiosis has also be reported, such that higher sweat rates can be sustained (830, 891). The composition of sweat is also influenced by heat acclimation with electrolyte (e.g., sodium, chloride and potassium) concentration decreasing in response to an increase in their conservation (891–895). The mechanistic pathways via which sweat electrolyte conservation occurs remain unclear, although sodium conservation has been linked to aldosterone-mediated sodium ion reuptake within the reabsorptive duct of the sweat gland (896–898).

Assuming that prevailing ambient conditions allow for evaporation, the enhanced sweating response associated with heat acclimation results in improved evaporative cooling and decreased skin temperature, reducing thermoregulatory skin blood flow requirements. A decrease in skin temperature may also reduce cutaneous venous compliance and allow for a redistribution of blood volume from the peripheral to central circulation (233, 899). Historically, heat acclimation was thought to decrease the internal temperature threshold for cutaneous vasodilation without altering the slope of the blood flow-internal temperature relationship (i.e., sensitivity) (106, 900, 901). However, Lorenzo and Minson (902) showed that heat acclimation does improve cutaneous vascular sensitivity to locally applied acetylcholine, an endothelium-dependent vasodilator, without altering maximal skin blood flow. More recently, Barry et al. (903) demonstrated that heat acclimation lowers the change in mean body temperature required to activate heat loss thermoeffector responses (i.e., cutaneous vasodilation and sweating). The change in mean body temperature needed to trigger skin sympathetic nerve activity did not simply reflect the reduction in resting core temperature associated with heat acclimation, but occurred after a smaller change in mean body temperature. It remains to be determined, however, if the reduction in onset threshold for skin sympathetic nerve activity reflects earlier vasomotor or sudomotor activation, as recordings reflect sympathetic outflow to both the cutaneous vasculature and eccrine sweat glands (904). The mechanistic pathway via which heat acclimation improves the neural control of body temperature remains unresolved but may relate to augmented thermal sensitivity of peripheral warm receptors, as well as an increased thermosensitivity and/or plasticity of central (i.e., hypothalamic) neurons (903).

6.1.6. Total body water and plasma volume expansion.

An increase in total body water (i.e., intra- and extracellular fluid) following repeated exposures to heat stress is a classic response associated with the heat acclimation phenotype (771). During the first week of heat acclimation total body water can increase by 2–3 liters (~5–7%) (826, 842, 843, 905) with expansion mostly occurring within the extracellular fluid compartment (i.e., intravascular and interstitial space) (842, 905). The increased retention of fluid within the vascular space (i.e., plasma volume expansion) has long been considered a primary adaptation supporting cardiovascular stability during exercise-heat stress through improved cardiac filling (146, 216, 338, 591, 766, 843, 906–908). Plasma volume expansion is purported to enhance vascular filling and atrial pressure (i.e., preload), thereby decreasing heart rate and increasing stroke volume and arterial blood pressure during exercise at a given work rate in the heat (591, 826, 907). This response is akin to the expansion of blood volume (i.e., plasma and erythrocyte) that occurs with an increase in fitness following aerobic training, which is considered an important contributor to improvements in performance via larger stroke volume and greater ventricular filling (878, 909). Recent studies, however, have reported that 5-day passive (910) and 10-

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day exercise-heat (911) acclimation have minimal effects on left-ventricular volumes, function, and systemic hemodynamics at rest and during exercise. It must be noted that postacclimation echocardiography measurements in these studies were undertaken at similar core temperatures and heart rates as preacclimation, along with a similar plasma volume (1–3% expansion). Additional research is thus required to clarify the link between blood/plasma volume expansion and cardiac function during exercise under heat stress in the heat acclimated state. Framing improvements in cardiovascular stability in the broader context of integrative heat acclimation adaptations is important, as other factors can influence cardiac function. For example, a reduction in resting core temperature contributes to the attenuated elevation in core temperature and thus heart rate during exercise at a given work rate in the heat following heat acclimation (912). Plasma volume expansion also increases the specific heat capacity of blood (913), which improves heat transfer from the core to the skin, potentially allowing for a reduction in the skin blood flow response (146).

The expansion of plasma volume is mediated by the retention of fluid in response to increases in plasma colloid (i.e., protein) and crystalloid (i.e., electrolyte) osmotic pressures (209, 826, 844, 914, 915). Intravascular protein content increases via acclimation-induced albumin synthesis (916, 917), coupled with a decrease in cutaneous blood flow (209) and capillary permeability (915, 918) that allow for protein to remain within the intravascular space. The oncotic effect created by the net increase in intravascular protein content during heat acclimation causes a shift in fluid from the interstitial to the intravascular space (844, 907, 914), with each gram of albumin osmotically attracting ~15 mL of fluid (919). In parallel, the conservation of sodium chloride through the increased secretion of aldosterone (920, 921) during heat acclimation helps maintain extracellular fluid osmolality and in turn to conserve or expand extracellular fluid volume (216, 257, 842, 905). The expansion of plasma volume following heat acclimation varies between 2 and 16% (765, 794) and appears within the first few days of heat exposure (907, 922) with erythrocyte volume typically remaining unchanged (923). The latter response may be influenced by the relative brevity of most heat acclimation interventions, as longer periods of training (4–6 wk) are required to expand red blood cell volume and total hemoglobin mass (924). In a recent heat acclimation study conducted over 5 wk, it was reported that a 7.6% plasma volume expansion was accompanied by a 4.2% increase red blood cell volume and 3.2% increase in total hemoglobin mass (831). The expansion of plasma volume was correlated, albeit moderately ($r = 0.49$), with the increase in total hemoglobin mass, leading the authors to speculate that there may be an erythropoietic compensatory response occurring, secondary to the expansion of plasma volume. A subsequent study utilizing a similar 5-wk heat acclimation training approach reported a 4.6% increase in total hemoglobin mass and 4.8% expansion of plasma volume, with red cell volume and blood volume remaining unchanged (925). A moderate correlation between changes in plasma volume and changes in hemoglobin mass was also reported ($r = 0.54$). The pathway via which this is purported to occur is associated with the hypothesis that the kidney functions as a "critmeter" to adjust hematocrit within normal values (e.g., 45%) by regulating red blood cell volume and plasma volume to stabilize arterial oxygen content (926). Although an attractive premise, additional research is required to elucidate the magnitude and time course of the erythropoietic response to exercise-heat acclimation.

The rapid expansion of plasma volume during heat acclimation was traditionally viewed as transient phenomenon, with a small contraction typically occurring after 1 wk of acclimation, despite continued heat exposure (826, 843–845). This phenomenon has been suggested to be an experimental artifact stemming from a failure to maintain a constant adaptation stimulus, due to utilizing the constant work rate heat acclimation approach. By clamping core temperature at 38.5°C during 16–17 days of controlled hyperthermia heat acclimation, Patterson et al. (842, 905) induced an increase in plasma volume of ~13% that remained expanded for the duration of the regimen. The authors also reported an expansion of interstitial fluid that decreased slightly from the midpoint (11–15%) to the end (6–9%) of heat acclimation, suggestive of a ubiquitous expansion of the extracellular compartment. Despite these observations, contention remains as to the extent plasma volume can be expanded and maintained during heat acclimation. Recently, 5 wk (28 sessions) of constant rate exercise heat acclimation with a weekly increase in ambient temperature (1°C) to maintain the adaptation impulse yielded a ~7% expansion of plasma volume (831). Unfortunately, plasma volume was not measured throughout the acclimation process (e.g., midpoint), but the extent of increase is less than that observed by Patterson et al. (842, 905), despite core temperature increasing to ~39.6°C during each 60-min training session (831). Additional research is therefore required to more clearly elucidate the time course of expansion and retention of plasma volume to a given thermal impulse.

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6.1.7. Heat acclimation with permissive dehydration.

Physical activity performed under heat stress relies primarily on evaporative cooling to dissipate metabolically generated thermal energy, which can lead to varying levels of dehydration depending on the compensability of the thermal environment and fluid intake. The fluid secreted in sweat is initially drawn from the interstitium and then from the intravascular and intracellular compartments (593, 927–931). This sequence occurs as hypo-osmotic sweat, relative to plasma, increases osmotic pressure within the extracellular fluid compartment, resulting in the transmembrane flow of water from the intracellular compartment. The loss of body water and fluid shift between compartments activates regulatory processes to maintain blood volume, plasma osmolality and blood pressure. As such, restricting fluid intake (i.e., permissive dehydration) during heat acclimation has been proposed to provide a thermally independent stimulus to enhance the adaptive process by further challenging fluid regulatory responses (781, 810, 812). The magnitude of dehydration imposed during such regimens should be well regulated, as large body water deficits result in greater heat storage and physiological strain (220, 595, 604, 616, 618), which could impair the adaptive process (846). In the animal model, adjustment in cardiovascular and thermoregulatory responses compensate for small changes in hydration status but are altered at severe levels of hypohydration (>10% body mass loss) (932). Severe hypohydration has thus been shown to dampen long-term heat adaptation in rats at the phenotypic and genomic levels through attenuated thermoeffector responses (i.e., onset and gain) (933) and altered hypothalamic gene expression (934). The dampened adaptative response was associated with continuous exposure (30 days) to 34°C and 35% RH with severe hypohydration. While these findings provide novel insights into the impact of severe hypohydration on the long-term adaptive process to environmental heat stress in rats, the regimen in which this occurred differs greatly to those adopted by free-living, exercise-trained humans. Indeed, it has been suggested that eliciting a body mass loss of 2–3% within each acclimation session may induce beneficial adaptations in humans (810, 842, 935), as this level of dehydration initiates fluid conservation and stimulates thirst (196, 239, 724, 936, 937), without compromising the adaptive response by the overly impacting on thermoregulatory and cardiovascular function.

Patterson et al. (842, 884) were the first to investigate the impact of permissive dehydration on the adaptive response to chronic heat stress. A 3-wk controlled hyperthermia protocol was employed wherein physically active individuals experienced a body mass loss of ~2.5% within each 90-min exposure. On *day 8*, an ~11% increase in plasma volume, ~5 beats·min^{-1} decrease in resting heart rate and ~0.20°C reduction in resting core temperature were observed, as well as an increased sweat rate (~0.22 L·h^{-1}) and decreased exercising heart rate (~10 beats·min^{-1}) and core temperature (~0.40°C) during constant work rate semirecumbent cycling. The adaptations were sustained until the end of acclimation on *day 22*, prompting the authors to conclude that the expansion of plasma volume can be sustained during long-term heat acclimation, provided the stimulus for adaptation is maintained (842). In a follow-up study, it was reported that heat acclimation does not confer preferential protection against plasma volume loss when exercising in humid heat, with a greater hemoconcentration noted on *days 8* (~3.4%) and *22* (~4.6%) of acclimation, relative to *day 1* (i.e., baseline) (905). It was suggested that the greater plasma loss was advantageous, as it supported greater sweat secretion and facilitated post-exercise plasma volume restoration due to an increase in plasma osmolality, and to a lesser extent the oncotic pressure gradient, which modulated a rapid influx of fluid within the intravascular space after transitioning from an exercising to a resting state. Garrett et al. (938) also reported a plasma volume increase (~4.5%) along with reductions in core temperature (~0.3°C) and heart rate (~14 beats·min^{-1}) during exercise-heat stress after only 5 days of controlled hyperthermia heat acclimation with restricted fluid intake (~2.1% daily body mass loss). Although these observations offer insight into fluid regulatory processes during heat acclimation, the lack of a euhydrated control group limits the scope of evidence upon which firm conclusions regarding permissive dehydration promoting a sustained expansion of plasma volume can be drawn.

Studies in which a control group was employed to examine the role of hydration status on the adaptative response to heat acclimation offer conflicting results. A short-term (5 days) controlled hyperthermia heat acclimation regimen using a crossover design in which euhydration (~0.3% body mass loss) or permissive dehydration (~1.8% body mass loss) were induced demonstrated a trend for plasma volume expansion to be greater with dehydration (8 vs. 4%) (935). Although final heart rate was ~9 beats·min^{-1} lower during exercise-heat stress after dehydrated heat acclimation, similar changes in exercising core and skin temperature were noted. In contrast, Neal et al. (870) reported that under similar levels of thermal strain (i.e., 10 days of controlled hyperthermia heat acclimation), dehydration (~2.7 vs. ~0.6% body mass loss) did not influence the induction or decay (7 days) of heat adaptations in trained individuals. While heart rate, sweat rate, and core and skin

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temperature were improved following heat acclimation, the magnitude of these responses was equivalent between the euhydrated and dehydrated regimens in this crossover design study, as was plasma volume expansion. The similar expansion of plasma volume was attributed to the comparable increase in aldosterone concentration noted between regimens, along with plasma osmolality in the dehydration regimen not surpassing the ~2% threshold required to stimulate renal water conservation (204). Others have also shown in recreational athletes that 3 days of exercise-heat training with ~1.4% (euhydration) or ~2.4% (dehydration) body mass losses do not influence plasma volume expansion or adjustments in thermal, cardiovascular and perceptual responses (939). Interestingly however, sweat rate increased to a greater extent (~150 $mL \cdot h^{-1}$) following dehydrated heat acclimation, which is somewhat surprising given the restricted stimulus to induce adaptations (i.e., three sessions separated by 48 h).

Using a 5-day controlled hyperthermia (38.5°C) approach with intervals and steady-state cycling in separate groups of endurance-trained athletes, Pethick et al. (940) reported that euhydration (~0.2% body mass loss) and dehydration (~2.2%) led to similar decreases in resting core temperature (0.3°C) but failed to improve 20-km time trial performance in the heat. An expansion of plasma volume was observed (~3.6%), however, only when data were aggregated with a control group added after the initial experimentation. The control group exercised at 75% of maximum heart rate in 22°C, which corresponded to the average heart rate sustained by the experimental groups undertaking controlled hyperthermia heat acclimation. Accordingly, work rate was not matched and the manner in which the target heart rate was achieved differed between individuals and within each training session. Of note, despite daily whole body temperature being much lower in the control group, plasma volume expansion (~5.2%) was similar to the euhydrated (~4.8%) and slightly greater, albeit not significantly, than the dehydrated (~1.7%) group. The expansion may have originated from an increase in training load, with the authors acknowledging that training intensity and volume were greater than recently experienced (940). These data do not support the notion that dehydrated heat acclimation enhances fluid regulatory processes, but rather, that training per se may provide an adaptation stimulus beyond that imposed by heat stress and dehydration. In a recent 10-day crossover design study in which recreationally trained individuals exercised at a heart rate equivalent to 65% $\dot{V}O_{2max}$ (i.e., controlled heart rate heat acclimation), euhydration (~0.6% body mass loss) was shown to be more beneficial than dehydration (2.9% body mass loss) at inducing adaptation, as evidenced by an enhanced sweat rate, decreased skin temperature and improved self-paced exercise performance in the heat (846). Conversely, dehydration consistently impaired the exercising component of heat acclimation at the targeted heart rate and limited adaptations beyond day five of the intervention. Although heat acclimation resulted in a significant expansion of plasma volume (~4% on *day 5*), no differences between or within either intervention were identified. The lack of difference occurred despite the ~3% reduction in daily body mass exceeding the ~2% decrease required to stimulate fluid regulatory responses (204).

Taken together, the potential for permissive dehydration or restricted fluid consumption to enhance the adaptive response to chronic heat exposure remains contentious, with minimal evidence to suggest that moderate changes in hydration status influence heat acclimation induction (227, 941). Additional studies are therefore required to elucidate whether this practice is beneficial, deleterious, or even trivial to the adaptive process and to endurance performance. Given the potential for a dehydration-mediated compromise in training intensity (i.e., lower sustained work rate) during exercise-heat acclimation, consideration should be given to performing higher intensity exercise at the onset of each session, or in separate cooler sessions altogether, if the goal is to provide a training stimulus based on load factors. Future studies may also seek to determine the effectiveness of dehydrated interventions against appropriately work-matched euhydrated controls in a crossover design approach, with a standardize degree of daily body mass loss and postintervention rehydration guidelines.

6.1.8. Summary.

Heat acclimation is an integrative process that elicits a host of physiological adaptations impacting on thermoregulation, fluid balance, cardiovascular function, and metabolism. Several active and/or passive approaches can be employed to induce these adaptations, with regimens of over 10 days purported to optimize the adaptive response. Heat acclimation has traditionally been used to enhance performance in the heat, although recent investigations have proposed that the benefits of chronic heat exposure may also contribute to improve aerobic performance in cool conditions. Contention remains, however, as to the impact of exercise-heat acclimation on performance in cooler environments, with the role of training per se potentially interfering with the response. Uncertainty also surrounds the premise that a constant forcing function throughout the heat acclimation process

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allows for maintaining the expansion of plasma volume and whether permissive dehydration enhances fluid regulatory processes.

6.2. Cooling

Exercise performance in the heat is impaired due to the complex interplay between increases in thermal strain (i.e., core and skin temperature), thermal perception (i.e., sensation and (dis)comfort), fluid disturbances (i.e., dehydration), and cardiovascular, CNS, and skeletal muscle function (see sect. 4). Over the past decades, different cooling techniques have been developed with the aim of offsetting exercise-induced increases in core body temperature and improve thermal perception. Cooling interventions can increase heat storage capacity before exercise (i.e., precooling) and attenuate the increase in core body temperature during exercise (i.e., per-cooling). Meta-analyses have shown that cooling interventions can successfully improve aerobic exercise performance in the heat (942–945), but their effectiveness is highly variable across techniques and study protocols. Performance benefits from cooling appear to be the greatest during exercise in high ambient temperatures (>30°C) and/or conditions in which prolonged exercise is performed (945). The current section summarizes the characteristics of available cooling techniques, discusses the principal underlying mechanism(s) underpinning their effectiveness, and reviews the (dis) advantages associated with their use in practice. Subsequently, the optimal application of pre- and per-cooling interventions is examined, followed by a comparison of the benefits of various cooling interventions for different types of activities (i.e., sprint vs. intermittent vs. endurance exercise). Finally, methodological and practical considerations are addressed to enable the translation of findings from laboratory studies to real-world application.

6.2.1. Cooling techniques.

Cooling techniques can be divided into internal and external cooling (**TABLE 7**). Internal cooling aims to alleviate thermal strain due to lowering core body temperature and creating a heat sink via cold fluid or ice ingestion. External cooling techniques, such as cooling garments, cold water immersion, or fanning, aim to reduce thermal strain via an increase in the core-to-skin temperature gradient and an improved thermal perception. Internal and external cooling techniques can be used in isolation but also in conjunction as the attenuation of heat strain with a combination of techniques may induce a greater net effect, both physiologically and perceptually (946, 947).

6.2.1.1. INTERNAL COOLING AND THERMAL RESPONSES. Cold beverages have a recommended temperature between 10°C and 24°C (734, 755), whereas ice slurry beverages (<0°C) consist of millions of very small ice particles submerged in a liquid. The small particle size of the ice slurry provides a larger surface area for heat transfer, with additional energy required to phase change from ice to water (334 $kJ \cdot kg^{-1}$) (948). This phase change is threefold larger than that of warming cold water to mean body temperature, which underlines the effectiveness of ice at providing a greater heat sink. Furthermore, the liquid of iced beverages allows heat transfer via conduction by facilitating contact between the ice particles and body tissue of the digestive tract (i.e., mouth, esophagus, stomach, and gut). Hence, ingestion of cold or iced beverages can directly influence core temperature, as energy is required to warm the ingested fluids to body temperature, resulting in a lower core body temperature (948). Thermal comfort and thermal sensation are also improved (949, 950), as the cold/iced beverages stimulate thermoreceptors in the mouth and gut (574, 951). An additional benefit of internal cooling techniques is their contribution to hydration status before and/or during exercise. In contrast, skin temperature is only slightly affected following internal cooling strategies, with minor reductions due to vasoconstriction.

Although ice slurry ingesting can effectively lower core temperature, some studies suggest that evaporative heat loss is attenuated during the initial phase of exercise in hot (33°C) and dry (24% RH) ambient conditions (949, 952), which may contribute to accelerate exercise-induced elevations in core body temperature. Furth-ermore, consumption of large volumes of ice beverages may induce physical complaints such as gastrointestinal distress, nausea, and/or “brain freeze”/headache. It is, therefore, recommended to practice internal cooling strategies during training sessions and simulated competition to determine the optimal volume and beverage temperature tolerated by individual athletes. In addition to the ingestion of cold fluids, several studies have explored the possibility to influence internal (i.e., mouth and gut) thermal perception as a strategy to improve exercise performance. As such, menthol has been used as a mouth rinse (953) or additive to existing cooled beverages (954). Menthol is known to induce a sensation of freshness, coolness, and nasal patency via stimulation of the transient receptor potential melastatin 8 channel (955), which serves as a cold receptor (956). The high density of cold-sensitive thermoreceptors on the tongue and mucous membranes of the oropharyngeal cavity, therefore, induce a larger effect compared with a similar dose

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Table 7. ***Classification and characteristics of distinct cooling techniques***

Cooling Technique	Examples	Precooling	Per-Cooling	Key Advantages	Potential Disadvantages
External Cooling					
Cooling garments	Cooling vest Ice vest Cooling packs Ice towel Neck collar	✓	✓	Most effective per-cooling strategy Available in different types and sizes Garments can be adjusted to sport type Phase change materials can be adjusted to melting-point specific cooling temperatures (i.e., 6°C, 15°C, 21°C) Easy to apply and implement	Additional weight may hamper use for per-cooling Large differences in cooling time and rate across garments Sport rules and regulations may prohibit use during competition
Cold water immersion	Whole body immersion Partial water immersion	✓		Most effective precooling strategy Covers a large part of the body	Difficult to implement in field-based settings Special equipment is needed (e.g., bath, ice) May lower muscle temperature below optimal physiological state
Fan use	(Cold) air fanning Water spray + fanning	✓		Easy to apply and implement	Only applicable to static conditions Less effective in humid environments Electricity or batteries required
Menthol cooling	Cream Gel Spray		✓	Easy to apply and implement	High concentrations may induce adverse health effects (i.e., skin irritation) Limited to no effect on performance outcomes Effect only perceptual, no cooling power provided
Internal Cooling					
Cold/iced beverage ingestion	Cold water ingestion Ice slurry ingestion	✓	✓	Direct effect on core body temperature Contributes to maintain fluid balance Easy to apply and implement	Potential gastrointestinal discomfort
Menthol cooling	Beverage Mouth rinse	✓	✓	Can easily be added to cold water or ice slush ingestion	Unknown

on the trunk (957). Menthol mouth rinsing has been shown to improve running (953) and cycling (958) performance in the heat, in conjunction with lower ratings of perceived exertion. In addition, a larger expired air volume was reported in the menthol condition, suggesting that menthol may induce a greater drive to breathe or lower airway resistance. Further studies are warranted to clarify the underlying mechanisms of improved exercise in the heat following menthol mouth rinsing. A 3°C menthol aromized beverage improved 20-km time trial performance in the heat (30.7°C, 78% RH) by 9% compared with a control condition in which the same beverage (volume and temperature) was ingested without menthol additives (959). Hence, the application of menthol as a perceptual internal cooling strategy can improve athletic performance and thermal sensation (960, 961).

6.2.1.2. EXTERNAL COOLING AND THERMAL RESPONSES. Cooling garments and cold water immersion aim to lower heat strain and attenuate the rise in core temperature by increasing heat transfer from the core to the periphery (962). This is achieved by reducing skin temperature and increasing the core-to-skin temperature gradient. A large body surface area is needed to achieve sufficient heat transfer, so cooling garments

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typically target the torso (~24% of body surface area) (70), whereas cold water immersion to the upper chest targets ~90% of the body. The decrease in skin temperature with external cooling is also associated with a reduction in cutaneous blood flow (963), which allows for central blood volume to be better maintained, thus attenuating cardiovascular strain and contributing to improve exercise performance in the heat.

Examples of cooling garments include cooling vests (5–10°C) and ice vests (0°C), but local cooling interventions can also be used such as cooling collars or ice towels. Cooling garments need to be activated in a freezer, refrigerator, or ice water before their use. Reductions in core body temperature, skin temperature, and heart rate have been reported following the use of most, but not all, commercially available cooling garments during precooling (942, 964). The absolute cooling capacity of the specific garment is key, in combination with the body surface being covered (as large as possible), appropriateness of the fit to the user (to allow conduction of heat), and the wear time or time that the cooling effects last (preferably >20 min). The use of cooling garments during exercise (per-cooling) may not lower core temperature per se (942), but the preservation of a large core-to-skin temperature gradient may be beneficial to enhance performance and attenuate the rate of rise in core temperature.

Neck cooling and head cooling are limited by their small body surface area (1% and 8%, respectively (70)), so cooling garments covering these body sites do not impact on physiological parameters such as core body temperature and heart rate (965). On the other hand, cooling the neck and head can reduce skin temperature and disproportionally improve thermal sensation and thermal comfort via stimulation of the thermoreceptors (966). Some studies have reported that per-cooling with neck and head garments can improve self-paced and constant work rate exercise performance in the heat under laboratory conditions (967–969).

Local and whole body cold water immersion are effective strategies to extract heat from the body during pre- and/or postcooling (942, 970). Water temperatures of 15 to 25°C are typically adopted, but lower temperatures may be applied for postcooling. Very cold water immersion of a large body area may be perceived as uncomfortable, which may reduce exposure time and impact. Although colder water was believed to induce greater physiological changes over a longer period of time, severe skin vasoconstriction may prevent core body temperature from decreasing during cold water immersion. Indeed, a recent study found similar cooling rates using 14°C and 26°C water due to an attenuated vasoconstrictor drive at 26°C (971). Nevertheless, the mixture of warm "central blood" with cool "peripheral blood" will reduce core body temperature after termination of immersion in very cold water (i.e., <5°C due to the after drop effect) (972). A potential caveat of cold water immersion is the reduction of muscle temperature leading to reductions in muscle power, force and velocity (973), and a loss of dexterity (974). Thus application of local or whole body cold water immersion should be dependent on the type of exercise that needs to be delivered.

Increasing airflow using a fan can enhance heat loss via convection and evaporation during exercise in the heat. Indeed, a curvilinear relationship was found between wind speed and heat storage in cyclists (**FIGURE 24**) (315). Increases in wind speed were associated with a lower heat storage, with a plateau occurring beyond a wind speed of 25 $km \cdot h^{-1}$. Reductions in core and skin temperature, sweat rate, and heart rate have been found with increasing air velocities when exercising at a constant work rate (315, 975), whereas thermal sensation is improved (976). Furthermore, an improved exercise performance was found at higher air velocities (315, 975). Adding water spray or skin wetting to fanning may further enhance heat dissipation (i.e., evaporation) (977), especially in hot and dry environments. It is important to note that the benefits of increasing airflow only pertain to exercise in the laboratory or work performed in an indoor setting, as airflow cannot be artificially influenced during outdoor competition.

External menthol application involves spraying a solution on the skin or clothing, or applying a cream or gel to the skin. External menthol application does not affect core and skin temperature but improves thermal sensation similarly to internal menthol cooling (960). Some (978, 979), but not all (980, 981), studies suggest that high menthol concentrations (>1%) may induce undesirable effects, such as vasoconstriction, reduced skin blood flow, delayed onset of sweating, and a greater

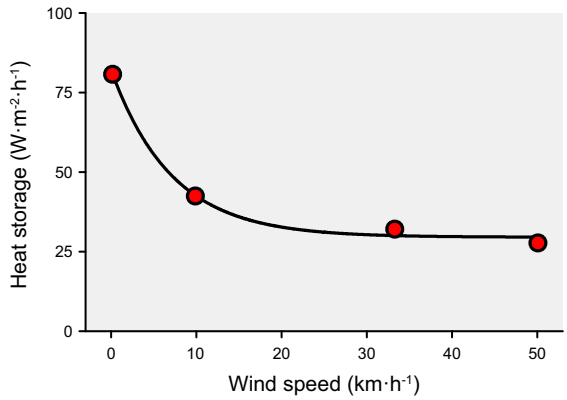

FIGURE 24. Calculated heat storage expressed per hour during exercise at 60% of peak power output at different air velocities. Heat storage at 0 $km \cdot h^{-1}$ was significantly different from all other conditions, whereas the 10 $km \cdot h^{-1}$ condition was only higher compared with the 50 $km \cdot h^{-1}$ condition. Redrawn with permission from Saunders et al. (315).

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increase in core temperature. A recent meta-analysis reported a lack of performance benefits for external menthol cooling (960), but large differences were found across studies. Future studies are warranted to assess whether a specific dose, application technique (e.g., spray versus cream versus gel) and location of menthol application may improve performance.

6.2.2. Cooling and performance.

6.2.2.1. PRECOOLING. Precooling interventions aim to lower thermal strain before exercise, using internal and/or external cooling strategies. Precooling-induced reductions in core temperature lead to an increased heat storage capacity (942). The application of precooling is not a novel approach. Initial studies, published 65 to 85 yr ago, investigated the effects of cooling on cardiovascular dynamics, oxygen uptake, and heat exposure tolerance time (982–984), mainly from an occupational health perspective. Studies published in the late 1970s and early 1980s were the first to assess the impact of precooling on exercise performance using cold water swimming (13–15°C) and cold air exposure (0°C) (985–987). Cold water swimming did not improve time to exhaustion and $\dot{V}O_{2max}$, but this may have resulted from extreme cooling as some participants became hypothermic (985). Cold air exposure did improve time to exhaustion (987) and work rate (986) during cycling exercise in mild ambient conditions (18°C). From the mid-1990s onwards, many studies evaluated the physiological and psychological benefits of precooling and found that whole body cooling [i.e., cold water immersion (949, 988), and cold air exposure (986, 987, 989)], partial body cooling [i.e., cooling vests (990, 991), and cooling packs (992, 993)] and internal cooling techniques [i.e., cold water and ice slurry ingestion (949, 964, 994)] were effective at increasing endurance performance in the heat.

The magnitude of performance improvement following precooling is highly variable and depends on the *1*) exercise protocol (i.e., constant work rate vs. self-paced exercise), *2*) nature of the exercise performed (i.e., sprint vs. intermittent vs. endurance), *3*) cooling dose provided (i.e., cooling intensity and duration and body surface area covered), *4*) perceived reductions in heat strain, and *5*) ambient conditions (i.e., compensability of the environment) (942, 970). Several meta-analyses have summarized the benefits of precooling on exercise performance (942–945). The most recent overview identified data from 45 experimental trials (995). Precooling improved exercise in the heat in all but two trials, with an average performance enhancement of 4.7%. Cooling technique-specific improvements varied from 2.0% to 7.1%, with cold water immersion being the most effective precooling strategy (**FIGURE 25**). These findings suggest that whole body vigorous cooling is superior to localized and/or less vigorous cooling techniques at improving exercise performance in the heat.

6.2.2.2. PER-COOLING. Per-cooling interventions aim to attenuate the increase in core temperature during exercise. The relevance of per-cooling is reinforced by the fact that the benefits of precooling interventions typically fade ~25 min after the onset of exercise (996). Beyond this time point, core temperature does not differ any more between the precooling and control condition (989, 997, 998), suggesting that the benefits of precooling are predominantly derived during the early phase of endurance exercise. Furthermore, exercise intensity (i.e., work rate), and therefore heat production and thermal strain, are much higher during exercise as compared with warming-up, emphasizing a potential larger ergogenic benefit of per-cooling versus precooling. On the other hand, not all cooling techniques (i.e., cold water immersion) can be applied for per-cooling due to practical (i.e., accessibility, additional mass, and power cord) or regulatory limitations during competition (999).

Several systematic reviews and meta-analyses have assessed the performance benefits of per-cooling (943, 995, 1000–1003). Although there is consensus that per-cooling interventions improve performance in the heat, the effects differ across study designs and cooling techniques. For example, there is debate as to whether per-cooling improves time to exhaustion, as systematic reviews and meta-analysis reported inconsistent outcomes (1002, 1003). Ruddock et al. (1002) only included cooling techniques that would be practical for athletes to use during competition. Four studies were included, encompassing neck cooling, palm cooling, and cold fluid ingestion, but no clear performance benefit was found. In contrast, Stevens et al. (1003) adopted less strict inclusion criteria and pooled findings from nine per-cooling studies, including cold fluid ingestion, fanning and water spraying, menthol mouth rinsing, cooling garments, neck cooling, and palm cooling. The authors reported that only one of nine studies found no performance improvement, so concluded that per-cooling could effectively improve time to exhaustion. Self-paced exercise protocols such as time trials may be more suitable to assess the benefits of per-cooling, given their better reliability and ecological validity (1004). Indeed, per-cooling was found to be effective during self-paced exercise studies in all systematic reviews and meta-analyses (1002, 1003). These findings reinforce the performance benefits of per-cooling during competition.

A recent study quantified the ergogenic effects of per-cooling among 270 athletes from 26 studies. It was observed that exercise performance improved by 5.3% (995). Cooling technique-specific improvements

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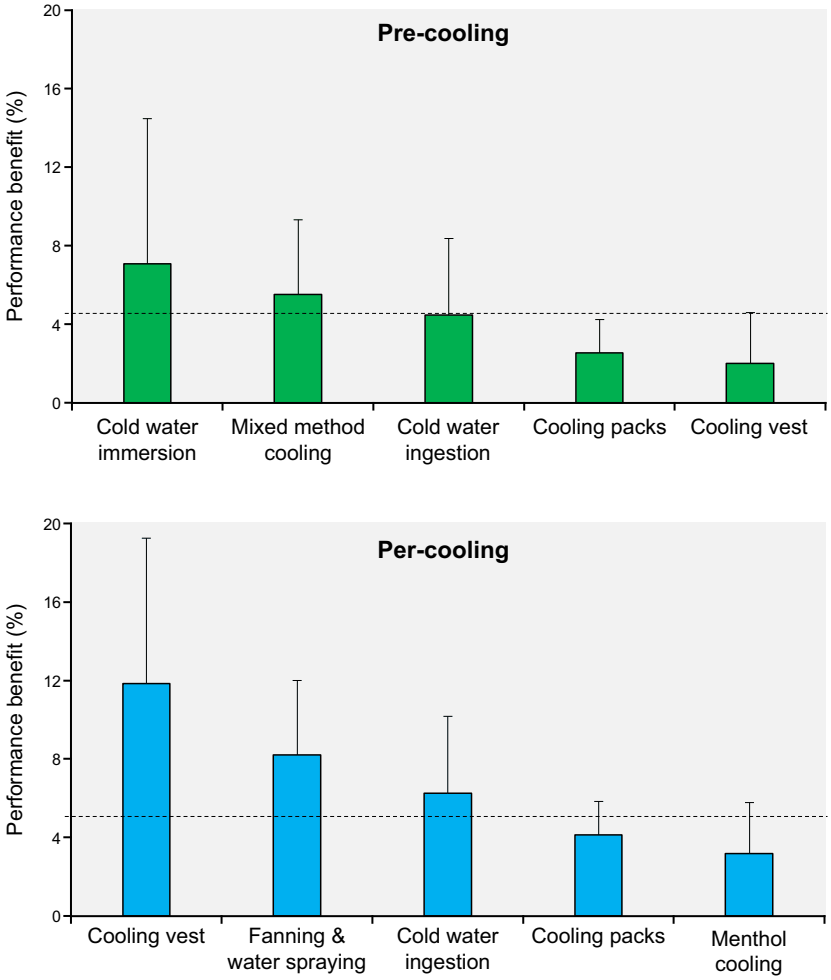

FIGURE 25. Relative performance improvements for pre-cooling (*top*) and per-cooling (lower panel) interventions during exercise in the heat. The dashed line represents the average performance benefit for pre-cooling (4.7 ± 4.7%) and per-cooling (5.3 ± 6.5%). Cooling strategy-specific data are presented as average ± SD and extracted from Bongers et al. (995).

varied from 3.2% to 11.9%, with the use of cooling/ice vests being the most effective per-cooling strategy (**FIGURE 25**). Previous studies also assessed the effects of per-cooling on psychological and physiological outcomes, demonstrating improved ratings of perceived exertion, thermal perception, and skin temperature (1003), but no effect on peak core temperature, heart rate, and sweat rate (942, 1003). Few studies have assesed the combined effects of pre-cooling and per-cooling to explore whether the effects are superior to the use of a single cooling strategy. Exercise performance improved by 5.6% (range: −1.7% to +23%) in the combination condition but did not differ from the independent application of precooling or per-cooling (943). A potential explanation for the lack of difference may relate to the duration of the exercise protocols that were used (<1 h) in combination with the high frequency of cooling interventions during exercise (i.e., cold water ingestion) (1003). Hence, future studies should explore whether the combination of pre- and per-cooling yield additional benefits for endurance exercise lasting >1 h in a sport-specific context.

6.2.2.3. MAGNITUDE OF PERFORMANCE BENEFIT. The benefits of cooling are not uniform across athletes engaging in different types of activities and sports. Based on the relative isometric and isotonic components of exercise, athletes can be classified as endurance (e.g., running, cycling, race walking, triathlon), intermittent (e.g., field hockey, football, tennis, beach volleyball), power (e.g., sprint, BMX), or skill (e.g., sailing, archery). In general, endurance athletes seem to benefit most from cooling (995). Meta-analyses have shown that precooling induces greater improvements in exercise performance in endurance athletes than intermittent athletes (944, 945). Likewise, per-cooling interventions induce greater benefits during aerobic compared with anaerobic exercise, with cold beverage ingestion and external cooling providing the greatest benefits for aerobic performance and whole body cooling garments being favorable for anaerobic exercise (1001). The difference in performance improvement between endurance and intermittent exercise likely relates to the duration and cumulative heat gain occurring during exercise. As thermal strain is often greater in endurance versus intermittent exercise, the window for improvement is likely

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larger. Nevertheless, it is important to reiterate that both endurance (6.4%) and intermittent athletes (3.0%) have been shown to benefit from cooling interventions (995).

Power athletes (e.g., sprinters, jumpers) do not benefit from cooling, and exercise performance may even be deteriorated following precooling (944, 945). The lack of cooling benefits in such athletes is multifactorial. First, exercise duration is short (typically <1 min), so the impact of thermal strain is unlikely to limit performance. Second, it is methodologically difficult to provide evidence for small improvements (i.e., <1 s) in sprint performance, as a large sample size and specific (high-resolution) measurement equipment are needed. Third, muscle temperature is a key determinant for skeletal muscle contractility, anaerobic metabolism, and sprint performance (466, 467, 1005). Cooling interventions may (in)directly impact muscle temperature and attenuate performance in power athletes. As such, cooling interventions are not recommended for power athletes.

The benefits of cooling in skill-based sports are less well studied, despite the presence of elevated thermal strain in some sports (1006). Mixed outcomes have been reported in studies assessing the impact of cooling interventions on psychological and cognitive outcomes. For example, cold pack application to the head improved working memory and rapid visual processing following passive heating but had no effect on pattern recognition memory (1007). Another study reported that application of cold packs to the head preserved working memory capacity but not visual memory in the heat (435). Other studies showed no benefit of cooling interventions on cognitive capacity (1008) or only in specific subdomains (1009, 1010). These inconsistent outcomes suggest that the effect of cooling on cognitive function is dependent on the intervention (i.e., timing, type, frequency, and duration of cooling) (1011), type of task (1012), and the factor investigated (e.g., cognitive tasks, working memory, visual memory, executive function, and auditory function) (1013). More research is needed to determine which type (pre- vs. per-cooling) and technique (i.e., internal versus external) of cooling can be beneficial to skill-based athletes.

6.2.3. From laboratory to field conditions.

Most studies assessing the benefits of cooling interventions have been performed in laboratory conditions using moderately to well-trained young individuals. A large variety of experimental protocols have been used to assess performance benefits (e.g., time to exhaustion and self-paced exercise) in different modes of exercise (e.g., cycling and running), with most studies performed in hot ambient conditions (>30°C). The large majority of studies only included young men. These study limitations may hamper translation to field conditions on several levels. First, the translation of findings from amateur to professional athletes may be associated with lower benefits as the window for improvement is smaller given their greater aerobic fitness. On the other hand, no clear association between fitness (i.e., $\dot{V}O_{2max}$) and the effects of precooling on performance have been identified (945). Second, cooling benefits may be different for veteran compared with young athletes, as exercise performance, and thus heat production, declines with advancing age (1014), whereas thermal sensation and perception is impaired (86). Third, most meta-analyses aggregate data independently of exercise protocols, which induces heterogeneity to the overall effect of cooling, such that the benefits of cooling in specific settings (i.e., activity, exposure time, exercise intensity) may be either over or underestimated. Fourth, the performance benefits of cooling are less clear in temperate ambient conditions (945) and largely unknown in cool temperatures. Of note, even in 10.5°C conditions a high proportion (15%) of amateur runners experienced hyperthermia (core temperature >40°C) during a 15-km road race (1015). Fifth, the interplay between ambient temperature, absolute humidity, solar radiation, and wind speed is highly dynamic in field conditions and thus not comparable with most ambient conditions adopted in laboratory studies (i.e., high temperature, moderate humidity, no solar radiation, and low wind speed). The typically low airflow provided during laboratory-based studies appears to be particularly influential and lead to an overestimation of the ergogenic benefits of precooling. For example, Morrison et al. (1016) demonstrated that combining cool water immersion (24°C) precooling with an airflow of ~4.8 $m \cdot s^{-1}$ during exercise in warm conditions (30°C) did not enhance performance or decrease thermal and cardiovascular strain more than providing airflow alone. Additional studies are therefore warranted to validate the performance benefits of pre- and per-cooling under realistic ambient conditions, including appropriate airflow. Sixth, it is unknown how heat acclimation status impacts the benefits of cooling strategies during exercise in the heat. Seventh, sex differences in body characteristics and exercise-induced increases in core temperature and sweat rate may contribute to distinct benefits of specific cooling strategies in male versus female athletes (83, 1017). Taken together, these restrictions indicate that laboratory findings cannot be directly extrapolated to field conditions, as certain cooling techniques and protocols may not be as effective as initially demonstrated. Future studies should therefore further examine the implementation of cooling strategies in field conditions.

Recent studies have evaluated the use of cooling interventions by elite athletes competing in World

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Championship events under hot and humid ambient conditions. During the 2015 International Association of Athletics Federations World Championships in Beijing (China), 307 athletes from 5 event categories (i.e., field, sprints, middle distance, long distance and decathlon/heptathlon) participated in a survey (1018). Only 52.4% of the participants reported that they planned on using at least one precooling strategy, 10.4% planned on using two strategies and 4.9% three strategies. Male athletes (52%) and those competing in middle distance races (70.3%) more often applied cooling strategies. A similar study was performed with 69 professional cyclists before the 2016 Union Cycliste Internationale World Championships in Doha (Qatar) (1019). Almost all cyclists planned some form of precooling before the individual or team time trial (96.4% and 98.6%, respectively). Although 74% of participants indicated that they would precool before the road race, a large difference in the use of precooling was found between male (57%) and female (96%) cyclists. Findings from these two studies highlight the heterogeneity in the use of cooling strategies by professional athletes. Whereas only half of track and field athletes used prearranged precooling strategies, almost all cyclists planned on using a precooling strategy ahead of the time trial. Differences in education on the (perceived) benefits of cooling, but also available resources and the type of sport (e.g., sprinters vs. endurance athletes), may have contributed to these differences. Given the increasing evidence regarding the performance benefits of cooling, a further increase of the prevalence of cooling strategies during competition in hot and humid environments is expected. Whether similar trends are applicable to the use of per-cooling is currently unknown and should be explored in future studies.

6.2.4. Summary.

Cooling interventions improve exercise performance in the heat. There is a large variety of cooling techniques available, and a selection can be made based on the effectiveness, feasibility, and sport-specific regulations that apply during competition. Precooling and per-cooling solutions appear equally effective, but their combination does not lead to further synergy. The benefits of cooling are also different across activities and sports. Endurance and intermitting athletes benefit most, whereas power athletes and sprinters do not benefit at all. The benefits of cooling for skill-based sports are less clear and should be further investigated. There is also a need to evaluate the benefits of cooling in elite athletes, as current evidence is entirely based on observations in moderate to highly trained athletes.

6.3. *Hyperhydration*

Hydration status can be severely affected during exercise in the heat, so it is recommended that athletes undertake exercise in a euhydrated state to optimize performance (181, 1020). To reduce net fluid loss and offset the deleterious effects of dehydration on performance in the heat, preloading or preexercise hyperhydration has been explored as a mitigation strategy. In this section the effects of four hyperhydration strategies will be evaluated: water, glycerol, sodium, and creatine.

6.3.1. Water loading.

The first study to examine the effects of hyperhydration using water dates back to 1965. Soldiers drinking 2 liters of 24°C water before 90 min of walking in the heat (49°C, 15% RH) demonstrated a lower end-exercise rectal temperature (~0.3°C) and 2.5% increase in sweat rate compared with those not hyperhydrating (1021). The authors concluded that "overhydration is beneficial to men working in the heat." However, there are two main limitations in this study. First, hyperhydration was not compared with euhydration, as the soldiers in the control condition were ~1% hypohydrated. It has been suggested that the higher rectal temperature in the control condition compared with the hyperhydration condition may have been the result of the slight hypohydration, rather than the positive effects of hyperhydration (601). Second, the low temperature of the drinking water may have provided a precooling effect, such that the beneficial effects were not linked to hyperhydration per se, but to precooling (see sect. 6.2). To exclude the role of precooling, water of ~37°C should be consumed to achieve hyperhydration. Two studies did so in temperate conditions (23°C, 50% RH). In one study participants drank 2.1 liters of 37°C water 60 min before 45 min of constant rate exercise (1022) and in the other study 2.5– 3.0 liters of water before 70 min of exercise (600). Both studies observed a lower end-exercise rectal temperature of ~0.2°C with hyperhydration compared with euhydration but no differences in sweat rate. These data indicate that hyperhydration using water at a temperature similar to body temperature in amounts of ~2 liters leads to a slightly lower body core temperature during exercise. However, it is difficult to consume such large volumes of fluid in the hours preceding exercise; therefore, it is recommended that protocols be practiced to determine their effectiveness and potential side effects including gastrointestinal discomfort and headaches (304).

The observations in the previous paragraph raise the question regarding how body core temperature can be lower following hyperhydration when sweat rate is not

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increased. The answer may lie with a study in which blood volume was manipulated before exercise in the heat (602). In this study, participants received an infusion of isotonic serum albumin to increase blood volume by 7.9%. During 30 min of cycling in the heat (40°C, 30% RH) at 65–70% $\dot{V}O_{2max}$, mean esophageal temperature was ~0.11°C lower following infusion and sweat rate was unaffected, which is in line with previous observations in temperate conditions (600, 1022). During hypervolemic exercise, blood volume decreased to a larger extent than in the control condition (541 vs. 421 mL) (602). The authors suggested that the expansion of blood volume was linked to a reduction in vasomotor tone (i.e., reduced vasoconstrictor activity), leading to a smaller net reabsorption of fluid from inactive tissues and greater filtration in active muscles. The reduction in vasomotor tone was associated with greater skin blood flow when hyperhydrated in the heat and thus lower body core temperature due to enhanced dry heat loss (602). Skin temperature recordings during hyperhydration failed to demonstrate a difference with those of control conditions (600, 1022), indicating that additional research is required to confirm this hypothesis. If wet and dry heat loss is not changing due to hyperhydration and core temperature shows a slower increase, the extra body volume may act as a heat sink. However, assuming a specific heat for the human body of 3.47 $kJ \cdot kg^{-1} \cdot °C^{-1}$ and a body mass of 70 kg, calculations indicate a similar heat gain of 2°C without predrinking and a gain of 1.95°C after hyperhydrating with 2 liters. Therefore, the increased heat sink only partially explains the reduction in body core temperature observed with hyperhydration. Along with the fluid shifts occurring within the central circulation during hyperhydrated exercise, fluid regulation within the bladder occurs in response to activation of the RAAS to maintain homeostasis and excrete excess fluids (see sects. 3.1 and 3.2). However, it has been reported that after 45 min of exercise, ~65% of the ingested water is still present in the central circulation and the remaining volume in the bladder (1022).

In summary, drinking ~2 liters of water before exercise in the heat seems to slightly lower body core temperature during exercise without affecting sweat rate. Hyperhydrating with water may be ineffective, however, as a rapid decrease in aldosterone concentration will signal the kidneys to excrete excess water in the form of dilute urine. Therefore, the coingestion of an osmotically active agent like sodium, glycerol, or creatine can assist with fluid retention (304). Next to oral ingestion of osmotically active agents, intravenous infusion of colloid (1023) or crystalloid (1024) solutions is used for plasma volume expansion before exercise (1025). However, no differences are observed in thermal strain when preloaded with intravenous fluids (1023, 1026). Moreover, intravenous fluid use in Olympic sports must comply with the World Anti-Doping Code and may require a therapeutic use exemptions to be granted with appropriate clinical justification (1027). Therefore, the focus in this section is on oral hyperhydration, as most athletes are unlikely to use intravenous methods.

6.3.2. Glycerol.

Glycerol, a 3-carbon alcohol metabolite, is an endogenous substance distributed across the body at low concentrations (0.05–0.3 $mmol \cdot L^{-1}$) (1028). Glycerol enhances fluid retention so it can be used to increase total body water content. This was first shown in humans in 1987 (1029). Glycerol-induced hyperhydration was on the World Anti-Doping Agency Prohibited List until January 2018, but athletes are now free to use it for hyperhydration. Elevations of glycerol levels can be achieved using venous infusion or fluid ingestion. Venous infusion increases the blood osmolality faster than the coingestion of water and dissolved glycerol, but has obvious practical limitations such as being more invasive. The effectiveness of glycerol for retaining water depends on the timing, dose and amount of fluid ingested (1028).

There are two pathways via which glycerol is suggested to increase water retention and total body water. First, glycerol attenuates the decrement in antidiuretic hormone that is observed due to water loading by increasing plasma osmolality (1030). Thus water secretion is lower following hyperhydration. Second, glycerol may have a direct effect on the kidneys as it is reabsorbed in the proximal and distal tubules, which induces an osmotic drive for the reabsorption of water. Glycerol can maintain this gradient for a relatively long period because it has a slow metabolism and is thus slowly excreted through urine, ultimately leading to higher water reabsorption (1031).

Several studies have investigated the effects of glycerol induced hyperhydration during exercise in the heat. One of the first studies used a constant work rate (60% $\dot{V}O_{2max}$) protocol in hot/dry conditions (42°C, 25% RH) and observed a lower rise in rectal temperature and enhanced sweat rate after ingestion of 1 $g \cdot kg^{-1}$ body mass of glycerol with 21.4 $mL \cdot kg^{-1}$ of water, compared with no glycerol (1032). In the subsequent decade, studies investigating the effects of glycerol-induced hyperhydration (1033–1038) observed considerably different outcomes across various types of exercise in the heat. **TABLE 8** summarizes the protocols and results of these studies, all of which adopted a crossover design and provided a similar amount of water in the control trial to that of glycerol in the experimental hyperhydration trial.

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The glycerol ingestion protocols showed small differences in thermal strain and performance across studies, with most ingestion rates commensurate with recent suggestions of a glycerol dose of 1.2 g·kg^{-1} body mass with a fluid volume of 26 mL·kg^{-1} body mass per hour (1039, 1040).

Latzka et al. (1035) observed no differences in thermal strain with and without glycerol induced hyperhydration in compensable (1035) and uncompensable (1036) heat stress, although performance was enhanced with glycerol induced hyperhydration in uncompensable heat stress (1036). In their review, Latzka and Sawka (1041) concluded that there appeared to be no thermoregulatory or performance benefit from hyperhydration when euhydration was maintained during exercise and that glycerol induced hyperhydration was not better than water hyperhydration for a given hydration status at the start of exercise. Others have also reported no difference in performance between glycerol and water-induced hyperhydration during mountain bike racing (1038) and self-paced exercise in the heat (1037). These findings are in contrast with reports of improved performance during cycling time trials following glycerol-induced hyperhydration (1033, 1034) and a lower rectal temperature (0.4°C) (1033), which may have contributed to enhance performance. A meta-analysis assessing the effects of glycerol-induced hyperhydration during exercise in the heat was published by Goulet et al. (1031), but this review included only four studies (1033, 1034, 1037, 1038). The authors excluded the work of Latzka et al. (1035, 1036) since it did not meet their inclusion criteria and included a study that was not performed in the heat (685). The analysis was performed for hyperhydration studies in temperate and hot conditions and it was concluded that glycerol-induced hyperhydration improved fluid retention by 50% relative to water-induced hyperhydration. This was associated with a 6.2% improvement in endurance performance. In summary, adding glycerol to water for hyperhydration purposes results in greater water retention. However, the benefits of glycerol hyperhydration for reducing thermal strain and improving performance are equivocal and more research with sufficient statistical power is required to clarify the effect of glycerol hyperhydration.

6.3.3. Sodium.

Most of the early work showing that adding sodium to a drink increases plasma volume was performed by NASA before the turn of the century (1042). Although sodium expands plasma volume in combination with water intake due to its osmotic effect, it is also suggested that large concentrations of the sodium ions in the plasma may reduce sweat rate (621). As such, the question becomes how does sodium preloading affect thermal strain and performance in the heat. Several studies were performed to explore the effect of sodium hyperhydration on performance in the heat (**TABLE 9**), all of which were conducted following those with glycerol.

Sims et al. (1044) investigated the extent to which sodium intake (164 vs. 10 mmol·L^{-1} sodium) before exercise in the heat (32°C, 50% RH) influenced exercise capacity in eight males (1043) and 13 females. In males, plasma volume increased by 4.5% after high sodium intake and was unchanged following low sodium intake. Time to exhaustion was improved by 25% and end-exercise rectal temperature lower by 0.4°C in the high sodium group (1043). In females, all in the luteal phase, plasma volume increased by 4.4% after high sodium intake and was unchanged with low sodium intake. Time to exhaustion increased by ~26% but end-exercise rectal temperature did not differ (after recalculation of the raw data provided in the study) (1044). Sweat rate was slightly lower for the high sodium condition in females relative to low sodium intake and did not differ between conditions in males. Based on these results, the authors suggested that additional sodium intake before exercise may enhance performance. In contrast, however, drinking a sodium solution of 130 mmol·L^{-1} sodium in 26 mL·kg^{-1} water before exercise did not result in performance enhancement during an 18-km treadmill run in ~28°C, although heart rate (~5 beats·min^{-1}) and end-exercise rectal temperature (0.3°C) were lower (1046).

Two studies investigated the effect of sodium on time trial performance after 2 h of dehydrating exercise at 63% $\dot{V}O_{2max}$ (1047) or 1 h at 50% of the maximum power output (1048). The first study showed that sodium concentrations of 164 and 82 mmol·L^{-1} similarly increased plasma volume and improved time trial performance compared with no sodium during preloading, although this study showed no reduction in core temperature or increase in sweat rate (1047). The positive effects of sodium preloading on performance were also demonstrated in the second study in which participants consumed 60 mg·kg^{-1} body mass of salt (1 g of salt = 390 mg of sodium) with 2 mL·kg^{-1} of water before 60 min of submaximal exercise, followed by a cycling time trial in 30°C (1048). Participants drank ad libitum during submaximal exercise and ingested much more water following salt preloading (1,830 vs. 815 mL for placebo and 782 mL for no treatment). Water retention increased with salt intake (815 vs. 244 mL and 148 mL, respectively). Time trial performance was also improved, while sweat rate did not differ.

Sodium has also been ingested in combination with citrate instead of chloride (1045), which was found to

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Table 8. ***Summarized methods and results of studies investigating the effect of glycerol-induced hyperhydration on thermal strain and performance in the heat***

Study	Sample Size	Water, mL·kg^{-1}/ Glycerol, g·kg^{-1}	Preexercise Ingestion Time, min	Exercise Protocol	Environmental Conditions, °C, %RH	Plasma Volume, %	Core Temperature, °C (G/C)	Sweat Rate, L·h^{-1} (G/C)	Skin Temperature, °C (G/C)	Performance (G/C)
Lyons et al. (1032)	6	21.4/1.0	150	90 min at 60% $\dot{V}O_{2max}$	42/25	0.7*	37.4/38.1*	0.97/0.75*		
Latzka et al. (1035)	8	25.2/1.2 (LBM)	60	120 min at 45% $\dot{V}O_{2max}$	35/45	0.0	38.6/38.6	0.94/0.95	35.5/35.4	
Latzka et al. (1036)	8	29.1/1.2 (LBM)	60	~30 min at 55% $\dot{V}O_{2max}$	35/45	0.1	38.8/38.7	1.26/1.09	37.6/37.4	33.8/29.5 min*
Hitchins et al. (1034)	8	22.0/1.0	150	60 min at 60% $\dot{V}O_{2max}$	32/60	0.8*	38.9/39.0	1.92/1.85	33.2/33.2	472/450 kJ*
Anderson et al. (1033)	6	20.0/1.0	120	90 min at 98% LT	35/30	0.8*	38.7/39.1*			252/240 kJ*
Marino et al. (1037)	7	21.0/1.2	150	60-min time trial	35/63	0.2*	38.8/39.0	1.72/1.15*	33.7/34.0	Equal distance
Wingo et al. (1038)	12	28.0/1.0		48-km mountain bike race	WBGT 28	0.1	38.5/38.0	1.42/1.44		No time difference

C, control; G, glycerol; LBM, lean body mass; LT, lactate threshold; WBGT, wet-bulb-globe temperature; RH, relative humidity. *$P < 0.05$, significant different from control.

induce a 3.6% increase in plasma volume but no reduction of thermal strain during prolonged cycling. The taste of salt drinks is often not pleasant, and salt tablets as an alternative have been investigated with regards to fluid retention capacity. Savoie et al. (1049) reported that a salt solution provided better fluid retention than tablets with equal concentrations of sodium, probably due to the time it takes the tablets to dissolve in the stomach. Sodium supplementation may be required during prolonged exercise to attenuate hyponatremia when large volumes of fluid are consumed. It must be acknowledged, however, that salt or sodium supplementation cannot prevent exercise-associated hyponatremia when persistent and excessive fluid intake produces volume overload (267) (see sect. 3.4). In summary, intake of high concentration sodium drinks (>82 mmol·L^{-1}) with a volume of at least 10 mL·kg^{-1} or of salt capsules (60 mg salt·kg^{-1} body mass) with concomitant ad libitum water intake before exercise lead to increased plasma volume. Rectal temperature at the end of exercise has been shown to be lower in some studies with sodium preloading, although none demonstrated an increase in sweat rate. Most studies observed an increased performance in the heat.

6.3.4. Creatine.

Creatine is a nonessential dietary compound synthesized in the liver and pancreas, and mainly stored in the muscles (1050). Creatine muscle storage can be increased by 10 to 40% through the use of creatine supplements, depending on initial total creatine levels (1050). The primary role of creatine is the resynthesis of ATP via the creatine kinase reaction. Thus creatine is important for short duration exercise performance and has been shown to improve sprint performance in the heat when used as a supplement (1051). As such, there has been a limited focus in the literature on the use of creatine to enhance endurance exercise performance (1052). However, creatine ingestion has been shown to have a positive effect on body water retention (1053). The increased fluid retention associated with creatine loading has been attributed to osmotic effects resulting in cell swelling and increased protein synthesis (1054). Creatine supplementation of at least 5 days is needed for increasing body mass (1–3 kg) and total body water (1050). In contrast to sodium and glycerol supplementation, creatine has no effect on renal responses (1051).

Based on these observations, a study was performed to investigate the effects of creatine supplementation on exercise capacity in the heat with the experimental group ingesting 22.8 g of creatine with a glucose polymer every day for 7 days, whereas the control group only ingested the glucose polymer (1055). Total body water increased by 1.5% in the experimental group and no changes were noted in the control group. No differences were observed in time to exhaustion between the experimental and control groups after creatine ingestion. Interestingly, there was considerable variation in the amount of creatine absorbed across participants,

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Table 9. ***Summarized methods and results of studies investigating the effect of sodium-induced hyperhydration on thermal strain and performance in the heat***

Study	Sample Size	Protocol Water/Sodium	Control Water/Sodium	Preexercise Ingestion Time, min	Exercise Protocol	Environmental Conditions, °C, %RH	Plasma Volume/Body Mass, %	Core Temperature, °C (S/C)	Sweat Rate, $L \cdot h^{-1}$ (S/C)	Skin Temperature, °C (S/C)	Performance (S/C)
Sims et al. (1043)	8	10/164	10/10	105	70% $\dot{V}O_{2max}$ to exhaustion	32, 50	4.5 PV*	38.9/39.3*	1.6/1.9		57.9/46.4 min*
Sims et al. (1044)	13	10/164	10/10	105	70% $\dot{V}O_{2max}$ to exhaustion	32, 50	4.4 PV*	39.0/39.1†	1.3/1.7*		98.8/78.7 min*
Nelson et al. (1045)	12	12/170	Gatorade	100	15% >VT for 62 min	31, 64	3.6 PV*	38.7/38.7	0.5/0.5		
Gigou (1046)	6	26/130	No sodium	110	18-km time trial run	28, 28	1.3 BM*	39.4/39.7*	1.9/1.9		85.3/85.6 min
Hamouti et al. (1047)	10	10/164	10/82	90	~171-kJ time trial	33, 30	2.1 PV	38.8/38.8	1.4/1.4	34.4/34.4	No difference
Hamouti et al. (1047)	10	10/164	No sodium	90	~171-kJ time trial	33, 30	4.5 PV*	38.8/39.1	1.4/1.4	34.4/34/6	289/269 W*
Morris et al. (1048)	7	2/60 salt	No treatment	180	200-kJ time trial	30, 18-20	0.9 BM*	37.4/37.6	Similar (no data)		773/872 s*
Morris et al. (1048)	7	2/60 salt	Aspartame	180	200-kJ time trial	30, 18-20	0.8 BM*	37.4/37.3	Similar (no data)		773/851 s*

Only studies with 6 or more participants included. BM, body mass; C, control, S, sodium; VT, ventilatory threshold; W, water. Water is in $mL \cdot kg^{-1}$, sodium is in $mmol \cdot L^{-1}$, and salt is in $mgmL \cdot kg^{-1}$, where 1 g equals 0.39 g sodium. *$P < 0.05$, significantly different from control. †After recalculation of the raw data in the study.

with those absorbing more creatine demonstrating an improvement in time to exhaustion. The experimental group also had lower sweat rates ($0.3\,L \cdot h^{-1}$) and lower end-exercise rectal temperatures (0.4°C) compared with the control group. The authors suggested that the lower increase in rectal temperature and improvement in performance may be related to the 1.5% extra body volume and concomitant increased heat storage capacity after creatine ingestion. However, as shown in sect. 6.3.1, the effect of this increased heat capacity is <0.1°C for mean body temperature and thus does not give a suitable explanation for the observations.

In a separate study, participants ingested 21.6 g of creatine monohydrate or a placebo for 7 days before a 90-min heat tolerance test that started after the participants lost 2% of their body weight due to exercise in the heat (1056). The authors reported no differences in rectal temperature and sweat rate between conditions, indicating that short-term creatine supplementation did not influence thermoregulatory responses in hypohydrated individuals. Given the conflicting outcomes of creatine ingestion in these two studies, no firm conclusions on the effects of creatine on thermoregulation during exercise in the heat can be drawn. Additional research is warranted to explore potential performance and thermoregulatory benefits and whether these may differ between constant work rate and self-paced exercise protocols.

6.3.5. Combinations of glycerol, sodium, and creatine.

Several studies have examined the use of a combination of different hyperhydrating agents. For example, Savoie et al. (1049) reported that sodium and glycerol hyperhydration have similar beneficial thermoregulatory effects during exercise. However, the combination of glycerol ($1.4\,g \cdot kg^{-1}$ fat free mass) and sodium ($7.5\,g \cdot L^{-1}$ of table salt) dissolved in water ($\sim 30\,mL \cdot kg^{-1}$ fat free mass) led to significantly greater fluid retention (1.4 liters) compared with sodium (1.1 liters) or glycerol (0.7 liters) alone (1039). These findings suggest that the combination of sodium and glycerol is more effective at retaining fluid than each component separately.

The combined effects of creatine and glycerol supplementation were investigated to determine their effects on physiological variables during 40 min of constant-load exercise at 63% of maximum work rate and performance during a subsequent 16.1-km time trial in the heat (30°C) using four experimental conditions: placebo, creatine, glycerol and the combination of creatine and glycerol (1057). Two daily doses of glucose (i.e.,

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placebo: 11.4 g), creatine (11.4 g), glycerol (1 g.kg^{-1} body mass), and creatine and glycerol combined were taken for 7 days. Glycerol increased total body water by 0.50 liters, creatine by 0.63 liters and the combination of the two by 0.87 liters compared with placebo. Creatine and the combination of creatine and glycerol significantly attenuated heart rate, rectal temperature, and perceived exertion during constant rate exercise, but no regimen influenced time trial performance. It has also been shown that the beneficial effects of creatine and glycerol combined on attenuating the increases in heart rate and rectal temperature are confined to exercise performed in hot (35°C) but not cold (10°C) conditions (1058). The high amounts of water retained by the combined ingestion of creatine and glycerol and the slower increase in body core temperature are linked to the creatine-induced increase in intracellular water and simultaneous increase of extracellular water due to glycerol, but the exact mechanisms remain to be elucidated (1057).

6.3.6. Summary.

Hyperhydration using water alone or coingested with sodium, glycerol or creatine increases body water content and thus the potential for preserving fluid homeostasis for longer during exercise. The increase in plasma volume secondary to volume loading is particularly important when exercise is performed in the heat as it may aid with heat dissipation. Drinking ~2 liters of water at or below body temperature in the hours before exercise in the heat may slightly attenuate the rise in body core temperature, without affecting sweat rate. The ingestion of glycerol with water before exercise in the heat appears to increase fluid retention and has been shown to increase sweat rate and lower end-exercise body core temperature and seems to enhance exercise capacity (i.e., constant work rate), but not exercise performance (i.e., self-paced). Most studies in which sodium was ingested before exercise showed enhanced exercise capacity but mixed results regarding exercise performance. Some studies showed an attenuated rise in body core temperature, but no study showed any change in sweat rate. Only two studies were identified in which creatine was used to hyperhydrate before exercise in the heat and reported conflicting findings. Since the effects of hyperhydration on thermal strain and exercise capacity and performance in the heat depend on many factors like the amount of fluid ingested, the exercise task, ambient conditions, and concentration of coingested sodium, glycerol, and creatine, the available studies currently only provide a first step toward a better understanding of the complex interactions.

7. CONCLUSIONS

The last decade has been the warmest on record and part of a persistent long-term trend. Along with an increase in the incidence and severity of heat waves, this trend places both elite and recreational athletes of all ages at greater risk of exertional heat illness and provides sporting organizations with increasing logistical constraints when preparing for an event. Although humans are capable of regulating body core temperature within a narrow range in a variety of scenarios, exercise under heat stress provides a unique and integrative challenge to physiological function. The factors that constitute the thermal environment determine the compensability of a setting and whether thermoregulatory responses can maintain heat balance. Under heat stress, endurance exercise capacity and performance are impaired as heat gain often exceeds heat loss. This impairment is primarily mediated by hyperthermia-induced adjustments in cardiovascular, central nervous system, and skeletal muscle function. Failure or inability to replenish excessive body water loss leads to a reduction in blood (plasma) volume and increase in osmolality that compromises thermoregulatory capacity and further exacerbates the rise in thermal strain, precipitating the development of fatigue. The decision to discontinue exercise at a constant work rate or reduce self-paced exercise work rate in the heat, with or without hypohydration, occurs across a spectrum of physiological and perceptual responses. These responses are specific to the exercise being undertaken, intensity of effort, prevailing ambient conditions, contextual parameters, and individual expectations. Given the controversy regarding how best to hydrate during exercise, similar factors should be considered when deciding on which hydration approach to utilize. Factors to consider include the intensity and duration of the exercise task, ambient conditions, availability of fluids, and fluid needs.

Several strategies may be adopted to mitigate the deleterious influence of heat stress. For example, heat acclimation elicits physiological adaptations that enhance thermoregulation, fluid balance, cardiovascular function, and metabolism, all of which contribute to enhance performance in the heat. A variety of active and/or passive approaches can be employed to induce adaptations, with regimens of 10–14 days proposed to optimize responses. Pre- and per-cooling techniques may also enhance endurance performance in the heat. These techniques are based on either internal (e.g., ice-slurry ingestion) or external (e.g., cold water immersion) cooling, with the selection of an appropriate technique determined by its effectiveness, feasibility, and event-

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specific compatibility. Hyperhydration using water or coingested with sodium, glycerol, or creatine increases body water content and has been suggested to preserve fluid homeostasis for longer during exercise in the heat. While some of these approaches have been reported to provide benefits (e.g., attenuated rise in core temperature and enhanced exercise capacity), there appears to be a requirement for further performance and mechanistic studies.

7.1. Contemporary Controversies and Avenues of Research

The impact of heat stress on exercise performance and physiological responses has been extensively investigated, as have strategies to mitigate the deleterious effects of hyperthermia and dehydration. Nevertheless, several aspects of these areas of research remain under debate. This section highlights the main areas of contention and controversy, as well as avenues of research that warrant further investigation and elucidation. Traditionally, thermoregulation at rest and during exercise in the heat is described using the model of a central integrator of peripheral and central thermal inputs that activates thermoeffector responses (e.g., vasoconstriction and sweating) when core temperature is displaced beyond a certain point. This simple model has been challenged, however, and more complex models involving several integrators and other afferent signals (e.g., heat flux) should be considered.

Exercise in the heat is associated with an increased risk of EHI, particularly in uncompensable conditions. Exertional heat stroke is a life-threatening condition and often affects athletes that previously performed exercise at a similar intensity and duration under comparable environmental conditions, without experiencing any problems or complaints. Although several risk factors have been identified, there remains a need for the development and validation of algorithms to estimate person-specific risk calculations of EHI during exercise in the heat. Novel approaches such as (big) data science and deep learning may be adopted in addition to traditional studies utilizing thermophysiological and epidemiological assessments. Technological developments to allow affordable measurement of real-time insight into core temperature may also aid in assessing the risk for heat stroke using wearables, as such measurements are currently largely restricted to the science domain. Furthermore, the involvement of genetics should be further explored as an increasing number of studies suggest a role for inherited factors (1059–1061).

Hyperthermia with and without dehydration reduces exercise capacity and performance through several integrative pathways involving alterations in cardiovascular function (i.e., skeletal muscle blood flow and metabolism), adjustments in central neural drive (i.e., voluntary muscle activation), and intensification of perceptual strain (i.e., exertion, thermal discomfort, thirst). The increase in thermal strain and compromise in hydration status that occur during prolonged exercise in the heat without adequate fluid replacement provide a significant hemodynamic challenge that progressively reduces $\dot{V}O_{2max}$. Although the reduction in maximal aerobic power represents a primary determinant in fatigue development, the decision to volitionally terminate exercise (i.e., $\dot{V}O_{2max}$ test or constant work rate exercise) or reduce work rate (i.e., time trial exercise) remains task specific and occurs across a continuum of physiological and perceptual endpoints. As such, recognizing the nuances associated with impairments in aerobic exercise under heat stress and appropriately contextualizing the impact of hyperthermia and dehydration are required to further advance our understanding of fatigue development.

A compromise in hydration status is well recognized as a factor exacerbating fatigue development during aerobic exercise in the heat. However, the precise body water deficit associated with impaired endurance exercise remains contentious, as it relates to several factors, including the nature of the exercise task (i.e., constant work rate vs. self-paced) and whether initiated in a state of hypohydration, or if dehydration is induced. A further point of contention relates to the optimal approach used to hydrate during exercise: drink ad libitum or plan to drink. This debate is nuanced and influenced by the intensity and duration of exercise, environmental conditions, and context (e.g., indoors or outdoors) in which exercise is undertaken, along with a host of personal factors (e.g., experience, fitness, and acclimation status). Thus, to further elucidate the impact of a loss in body water on performance and the approach utilized to hydrate, the factors described above should be well controlled and research conclusions appropriately contextualized.

Heat acclimation adaptations improve thermoregulatory capacity and fluid balance and enhance exercise capacity and performance in the heat. However, contention remains regarding the enhancement of performance in cool conditions following heat acclimation and the precise mechanisms mediating this potential improvement. Further research is therefore required to elucidate if heat adaptations are beneficial to exercise performed in cooler conditions and the pathways via which this occurs. Additional research is also needed to clarify whether blood/plasma volume

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expansion is maintained during the adaptive process (i.e., time course of expansion and retention) by ensuring a constant thermal impulse. The potential for permissive dehydration, or restricted fluid consumption, to enhance the adaptive response (i.e., plasma volume expansion) by further challenging fluid regulatory responses also warrants additional research. There is also a need for research that bridges the gap between long-term (30 to 60 days) animal-based mechanistic (i.e., genomic responses, molecular signaling, and epigenetics) research and classic shorter term (10–14 days) human physiological heat acclimation undertaken in sport, occupational, and military settings. Investigating the molecular and cellular responses associated with the heat acclimation phenotype will lead to a better understanding of what drives the adaptive process.

Pre- and per-cooling improve exercise performance in the heat. However, a large variability in the effect of different strategies is observed across studies, highlighting the need for individual person data meta-analyses to elucidate the influence of personal factors (i.e., age, sex, and training status), ambient conditions (i.e., air temperature, wind speed, and humidity) and exercise characteristics (i.e., intensity, duration, and protocol) on the magnitude of cooling benefits. Such an approach has sufficient statistical power to answer these relevant questions, as it is often not feasible to conduct subanalyses in a single study due to the limited sample size. To facilitate such initiatives, it is recommended to present individual data beyond aggregated outcome measures (e.g., mean, median, and effect size) in future publications. This applies not only for determining the influence of cooling strategies but also for elucidating the acute and chronic effects of heat stress and hypohydration.

Hyperhydration before exercise in the heat has been investigated as a compensatory mechanism to offset the detrimental effects of dehydration. Although hyperhydration using intravenous fluids has been reported in some sports, strong evidence of its beneficial effects on thermal strain and performance is lacking. Water hyperhydration has been shown to lower the rate of increase in body core temperature during exercise in the heat, but the temperature of the ingested water plays a role in this that requires clarification. Adding glycerol, sodium, and/or creatine to ingested water increases water retention, but does not necessarily reduce thermal strain and increase sweat loss. The direct effect of sodium on the sweat gland may play a role that requires elucidation. The extent to which intracellular or extracellular fluids are affected by the osmotically active agents should also be further examined. Hyperhydration seems to be more beneficial for time to exhaustion than self-paced exercise, although more work is required to delineate the specific effects of hyperhydration on various exercise protocols.

APPENDIX A

TABLES A1, A2, A3, and A4 provide protocol details of the studies in TABLES 2–5.

Table A1. ***Constant work rate studies with dehydration***

Study	Sample Size	Exercise Protocol	Environmental Conditions, °C, %RH, m·s^{-1}	Dehydration, %body mass	Performance Outcome
de Melo-Marins et al. (673)	11	Cycling: 70% $\dot{V}O_{2max}$ to exhaustion	34.0, 40.0, –	EU: −0.2 ± 0.4 DE1: −1.0 ± 0.5 DE2: −1.3 ± 0.6	EU: 38.0 ± 9.0 min DE1: 37.0 ± 8.0 min DE2: 37.0 ± 9.0 min
Fallowfield et al. (672)	8	Running: 70% $\dot{V}O_{2max}$ to exhaustion	20.0, –, –	EU: −0.8 DE: −2.0	EU: 103.0 ± 35.1 min DE: 77.7 ± 21.8 min*
Marino et al. (674)	8	Cycling: 70% peak power output to exhaustion	31.3, 63.3, –	EU: −0.2 ± 0.1 DE1: −1.0 ± 0.4 DE2: −1.7 ± 0.5	EU: 41.2 ± 17.1 min DE1: 40.6 ± 14.0 min DE2: 32.5 ± 16.3 min*
Maughan et al. (671)	6	Cycling: 70% $\dot{V}O_{2max}$ to exhaustion	Temperate	EU: −0.7 DE: −1.8	EU: 76.2 ± 22.3 min DE: 70.2 ± 20.3 min
McConell et al. (670)	7	Cycling: 69% $\dot{V}O_{2peak}$ for 2 h + 90% $\dot{V}O_{2peak}$ to exhaustion	21.3, 43.0, –	EU: −0.1 ± 0.1 DE1: −1.8 ± 0.1 DE2: −3.2 ± 0.1	EU: 328.0 ± 246.1 s DE1: 248.0 ± 283.1 s DE2: 171.0 ± 198.4 s*

Protocol details are from TABLE 2. RH, relative humidity; DE, dehydration; EU, euhydration. *$P < 0.05$, significantly different from EU.

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Table A2. ***Constant work rate studies with hypohydration***

Study	Sample Size	Hypohydration Protocol	Exercise Protocol	Environmental Conditions, °C, %RH, m·s^{-1}	Hypohydration, % body mass	Performance Outcome
Baker et al. (668)	8	Running: 2-h intermittent 70% $\dot{V}O_{2max}$	Running: 85% $\dot{V}O_{2max}$ exhaustion run	30.0, 40.0, –	EU: 0.0 HY1: −2.0 HY2: −3.3	EU: ∼21.0 min HY1: ∼14.0 min* HY2: ∼8.0 min*
Cheung and McLellan (677)	15	Walking: 4.5-6.0 km·h^{-1}, 3–7% gradient until 2.5% body mass loss, 15 h before exercise protocol	Walking: 3.5 km·h^{-1}, 0% gradient in protective clothing to exhaustion	40.0, 30.0, –	Moderate Fitness-Pre EU: 0.0 ± 0.0 HY: −2.8 ± 0.9 Moderate Fitness-Post EU: −0.1 ± 1.2 HY: −2.6 ± 0.6 High Fitness-Pre EU: 0.0 ± 0.0 HY: −2.0 ± 0.5 High Fitness-Post EU: 0.0 ± 0.7 HY: −1.9 ± 0.8	Moderate fitness-Pre EU: 98.6 ± 19.6 min HY: 78.3 ± 16.9 min* Moderate Fitness-Post EU: 101.4 ± 11.4 min HY: 80.6 ± 18.0 min* High Fitness-Pre EU: 114.5 ± 27.4 min HY: 100.9 ± 20.4 min* High Fitness-Post EU: 115.6 ± 18.4 min HY: 110.5 ± 29.7 min*
Cheung and McLellan (678)	8	Walking: 5 km·h^{-1} 5–7% gradient until 2.5% body mass loss, 15 h before exercise protocol	Light walking: 3.5 km·h^{-1}, 0% gradient in protective clothingHeavy walking: 4.8 km·h^{-1}, 4% gradient in protective clothing to exhaustion	40.0, 30.0, –	Light Exercise EU: 0.0 ± 0.0 HY: −2.2 ± 1.0 Heavy Exercise EU: 0.0 ± 0.0 HY: −2.2 ± 0.9	Light Exercise EU: 106.5 ± 22.1 min HY: 87.1 ± 14.2 min* Heavy Exercise EU: 59.7 ± 9.5 min HY: 53.3 ± 8.9 min*
Cheung and McLellan (368)	15	Walking: 4.5–5.5 km·h^{-1}, 3–6% gradient until 2.5% body mass loss, 15 h before exercise protocol	Walking on motorized (speed not mentioned) treadmill in protective clothing to exhaustion	40.0, 30.0, –	Training Group-Pre EU: 0.0 ± 0.0 HY: −2.0 ± 0.4 Training Group-Post EU: −0.2 ± 0.5 HY: −2.1 ± 0.5 Control Group-Pre EU: 0.0 ± 0.0 HY: −1.9 ± 0.6 Control Group-Post EU: +0.6 ± 0.9 HY: −2.2 ± 0.8	Training Group-Pre EU: 93.1 ± 18.9 min HY: 75.8 ± 14.4 min* Training Group-Post EU: 94.0 ± 16.2 min HY: 80.3 ± 11.7 min* Control Group-Pre EU: 85.3 ± 10.2 min HY: 74.6 ± 10.1 min* Control Group-Post EU: 90.9 ± 11.9 min HY: 79.6 ± 10.3 min*
Ebert et al. (675)	8	Cycling: 53% maximal aerobic power for 2 h	Cycling: simulated hill climb (8% gradient) at 88% maximal aerobic power to exhaustion	29.3, 36.7, 4.2	EU: +0.3 ± 0.4 HY: −2.5 ± 0.5	EU: 19.5 ± 6.0 min, 313 ± 28 W HY: 13.9 ± 5.5 min*, 308 ± 28 W*
Walsh et al. (676)	6	Cycling: 70% $\dot{V}O_{2peak}$ for 1 h	Cycling: 90% $\dot{V}O_{2peak}$ to exhaustion	32.0, 60.0, 0.8	EU: −0.2 HY: −1.8	EU: 9.8 ± 3.9 min HY: 6.8 ± 3.0 min*

Protocol details are from TABLE 3. RH, relative humidity; EU, euhydration; HY, hypohydration. *$P < 0.05$, significantly different from EU.

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Table A3. ***Self-paced exercise studies with dehydration***

Study	Sample Size	Exercise Protocol	Environmental Conditions, °C, %RH, m·s^{-1}	Dehydration, % body mass	Performance Outcome
Adams et al. (690)	7	Cycling: 2 h at 55% $\dot{V}O_{2peak}$ + 5-km time trial	35.0, 30.0, 4.5	EU: −0.2 ± 0.6 DE: −2.2 ± 0.4	EU: 295 ± 29 W, 12.9 ± 0.8 min DE: 276 ± 29 W*, 13.5 ± 1.0 min*
Bachle et al. (679)	10	Cycling: 1-h time trial	20.6, 72.1, –	EU: +0.8 DE: −1.0	EU: ∼127 W DE: ∼123 W
Backx et al. (681)	8	Cycling: 1-h time trial	20.0, 70.0, 0.3	EU: −0.7 DE1: −1.3 DE2: −1.7	EU: 43.1 ± 2.1 km, 291 ± 35 W DE1: 43.0 ± 2.3 km, 290 ± 39 W DE2: 43.2 ± 2.5 km, 295 ± 42 W
Bardis et al. (683)	10	Cycling: 1 h at 70-75% HR_{max} + 5-km outdoor hill climb	29.0, –, 1.0	EU: −1.4 ± 0.3 DE: −2.2 ± 0.2	EU: 16.6 ± 2.3 min DE: 17.6 ± 2.9 min*
Bardis et al. (687)	10	Cycling: 3 × (5 km at 50% peak power output + 5-km time trial)	31.6, –, –	EU: −0.5 ± 0.3 DE: −1.8 ± 0.7	EU: 30.2 ± 2.4 km.h^{-1} DE: 28.8 ± 2.6 km.h^{-1}*
Below et al. (669)	8	Cycling: 50 min at 80% $\dot{V}O_{2max}$ + ∼10-min time trial	31.2, 54.0, 3.5	EU: −0.5 DE: −1.9	EU: 10.2 ± 0.8 min, 276 ± 17 W DE: 10.9 ± 0.9 min*, 258 ± 17 W*
Daries et al. (685)	8	Running: 90 min at 65% $\dot{V}O_{2max}$ + 30-min time trial	25.0, 55.0, 3.6–4.2	EU: −1.3 DE1: −2.6 DE2: −3.2	EU: 15.4 ± 1.4 km.h^{-1} DE1: 15.6 ± 1.1 km.h^{-1} DE2: 15.8 ± 0.9 km.h^{-1}
Dion et al. (691)	10	Running: 21.1-km time trial	30.2, 42.0, 3.9	EU: −1.3 DE: −3.1	EU: 89.6 ± 7.7 min, 14.2 ± 1.2 km.h^{-1} DE: 89.8 ± 7.7 min, 14.2 ± 1.2 km.h^{-1}
Dugas et al. (688)	6	Cycling: 80-km time trial	33.0, 50.0, 9.0–11.0	EU: −0.5 DE1: −1.9 DE2: −2.1 DE3: −2.9 DE4: −3.9 DE5: −4.3	EU: 125.4 ± 5.8 min, 207 ± 25 W DE1: 126.1 ± 4.8 min, 205 ± 18 W DE2: 124.2 ± 5.8 min, 214 ± 24 W DE3: 129.9 ± 6.1 min, 190 ± 20 W DE4: 129.4 ± 8.1 min, 194 ± 26 W DE4: 128.3 ± 6.3 min, 196 ± 25 W
Hillman et al. (686)	7	Cycling: 90 min at 95% of lactate threshold + 5-km time trial	23.0, –, –33.9, –, –	EU1: −0.1 ± 0.5 DE1: −3.0 ± 0.8 EU2: −0.2 ± 0.5 DE2: −3.8 ± 0.8	EU1: 282 ± 37 W DE1: 268 ± 32 W EU2: 262 ± 42 WD E2: 229 ± 32 W*
Kay and Marino (682)	7	Cycling: 1 h time trial	19.8, 63.3, –33.2, 63.3, –	EU1: −0.1 DE1: −1.8 EU2: 0.0 DE2: −2.2	EU1: 30.8 ± 5.7 km, 217 ± 40 W DE1: 32.6 ± 6.4 km, 235 ± 49 W EU2: 30.1 ± 5.0 km, 225 ± 34 W DE2: 30.5 ± 4.8 km, 225 ± 45 W
McConell et al. (680)	8	Cycling: 45 min at ∼80% $\dot{V}O_{2peak}$ + 15-min time trial	20.9, 41.0, –	EU: 0.0 ± 0.1 DE1: −1.0 ± 0.1 DE2: −1.9 ± 0.0	EU: 299 ± 28 W DE1: 297 ± 25 W DE2: 304 ± 25 W
Perreault-Briere et al. (689)	9	Cycling: 1-h time trial	30.0, 50.0, 7.0–8.0	EU: −0.6 ± 0.2 DE1: −2.2 ± 0.3 DE2: −2.9 ± 0.4	EU: 35.7 ± 2.0 km, 240 ± 34 W DE1: 35.8 ± 2.0 km, 241 ± 33 W DE2: 35.6 ± 1.9 km, 237 ± 31 W
Robinson et al. (684)	8	Cycling: 1-h time trial	20.0, 60.0, 3.0	EU: +0.9 DE: −2.3	EU: 42.32 ± 1.6 km, 303 ± 8 W DE: 43.05 ± 1.9 km*, 293 ± 7 W*

Protocol details are from TABLE 4. RH, relative humidity; DE, dehydration; EU, euhydration. *$P < 0.05$, significantly different from EU.

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Table A4. ***Self-paced exercise studies with hypohydration***

Study	Sample Size	Hypohydration Protocol	Exercise Protocol	Environmental Conditions, °C, % RH, m·s^{-1}	Hypohydration, % body mass	Performance Outcome
Armstrong et al. (683)	8	Diuretic 5 h before exercise	Running: 1,500-m time trialRunning: 5,000-m time trialRunning: 10,000-m time trial	15.7, 31.8, 3.6	1,500 m EU: 0.0 HY: −1.9 5,000 m EU: 0.0 HY: −1.6 10,000 m EU: 0.0 HY: −2.1	1,500 m EU: 4.7 min HY: 4.9 min5,000 m EU: 18.2 min HY: 19.5 min*10,000 m EU: 38.9 min HY: 41.5 min*
Bardis et al. (703)	10	Cycling: 2 × (25 min at 70−75% maximum heart rate with 5-min rest)	Cycling: 3 × 5-km time trial	EU: 32.9, –, 3.2HY: 33.0, –, 3.2	EU: 0.0 ± 0.0 HY: −1.0 ± 0.1	EU: 268 W HY: 254 W*
Berkulo et al. (704)	12	Cycling: 45 min at ∼50% peak power output, 30-min rest	Cycling: 40-km time trial	35.2, 51.0, 7.0	EU: 0.0 ± 0.2 HY1: −1.1 ± 0.2 HY2: −1.3 ± 0.3	EU: 70.1 ± 4.1 min, 223 ± 32 W HY1: 71.2 ± 4.0 min, 217 ± 39 W HY2: 69.9 ± 5.6 min, 224 ± 35 W
Burge et al. (667)	8	24-h fluid/food restriction with 1.5 liters of fluid 2 h before exercise	Rowing: 4,200 flywheel revolutions time trial	–, –, –	EU: −0.0 HY: −3.1	EU: 7.0 ± 0.5 min, 294 ± 20 W HY: 7.4 ± 0.6 min*, 279 ± 23 W*
Casa et al. (696)	17	22 h fluid restriction	Running: 12-km time trial	EU: 26.3, –, –HY: 28.0, –, –	EU: 0.8 HY: −2.3	EU: 53.2 ± 6.1 min HY: 55.7 ± 7.5 min*
Castellani et al. (701)	7	Walking: 2.5–3.0 h in 50°C without fluid replacement	Cycling: 30 min at ∼55% $\dot{V}O_{2peak}$ + 30 min time trial	27.5, 27.0, 0.8–1.4	EU: −0.6 ± 0.5 HY: −4.0 ± 0.4	EU: 193 ± 57 W HY: 168 ± 65 W*
Cheung et al. (705)	11	Cycling: 90 min at 50%	With mouth rinse: 20-km cycling time trialWithout mouth rinse: 20-km cycling time trial	35.0, 10.0, 3.0	With mouth rinse EU: −0.5 HY: −2.0 Without mouth rinse EU: −0.5HY: −2.1	With mouth rinse EU: 2172.0 ± 155.0 s HY: 2185.0 ± 131.0 s Without mouth rinse EU: 2180.0 ± 150.0 s HY: 2133.0 ± 142.0 s
Cheuvront et al. (698)	8	3 h of passive heat stress in 45 °C	Cycling: 30 min at 50% $\dot{V}O_{2peak}$+ 30 min time trial	2.0, 50.0, 2.220.0, 50.0, 1.0	EU1: −0.3 ± 0.6 HY1: −2.9 ± 0.7 EU2: −0.4 ± 0.7 HY2: −3.0 ± 0.8	EU1: 154 ± 36 W HY1: 150 ± 35 W EU2: 152 ± 30 W HY2: 140 ± 30 W*
Fleming and James (697)	10	24-h fluid restriction + 45 min run at 75% $\dot{V}O_{2peak}$	Prehabituation to hypohydration: 5-km running time trialPosthabituation to hypohydration:5 km running time	22.0, –, –	Prehabituation EU: 0.2 ± 0.3 HY: −2.4 ± 0.3 Posthabituation EU: −0.1 ± 0.1 HY: −2.4 ± 0.1	Prehabituation EU: 1381.0 ± 237.0 s HY: 1459.0 ± 250.0 s* Posthabituation EU: 1366.0 ± 211.0 s HY: 1381.0 ± 200.0 s
Funnell et al. (708)	14	Cycling: 120 min at 50% of maximal power	Blinded hydration:∼15-min cycling time trialUnblinded hydration:∼15-min cycling time trial	31.1, 47.6, 5.9	Blinded hydration EU: −0.6 ± 0.5 HY: −3.0 ± 0.5 Unblinded hydration EU: −0.5 ± 0.3 HY: −3.0 ± 0.3	Blinded hydration EU: 903.0 ± 89.0 s HY: 1008.0 ± 121.0 s* Unblinded hydration EU: 874.0 ± 108.0 s HY: 967.0 ± 170.0 s*

Continued

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Table A4.—*Continued*

Study	Sample Size	Hypohydration Protocol	Exercise Protocol	Environmental Conditions, °C, % RH, m·s^{-1}	Hypohydration, % body mass	Performance Outcome
James et al. (707)	7	Cycling: 155-min intermittent at 50% peak power output	Cycling: 15-min time trial	34.0, 50.0, 0.3–0.4	EU: −0.1 ± 0.1 HY: −2.4 ± 0.2	EU: 183 ± 24 W HY: 169 ± 27 W*
Kenefick et al. (702)	32	Walking: 3-h intermittent at 5 km·h^{-1} and 4% gradient in 50 °C	Cycling: 30 min at 50% $\dot{V}O_{2peak}$ + 15 min time trial	10, –, –20, –, –30, –, –40, –, –	EU1: within −1.0 HY1: −4.1 EU2: within −1.0 HY2: −4.2 EU3: within −1.0 HY3: −4.0 EU4: within −1.0 HY4: −4.1	EU1: 221 ± 41 W HY1: 216 ± 40 W* EU2: 220 ± 24 W HY2: 199 ± 22 W* EU3: 220 ± 23 W HY3: 193 ± 33 W* EU4: 174 ± 21 W HY4: 136 ± 41 W*
Logan-Sprenger et al. (494)	9	Cycling: 90 min at ∼65% $\dot{V}O_{2peak}$	Cycling: time trial at 6 kJ per kg of body mass	23.0, 32.5, –	EU: −0.0 ± 0.0 HY: −2.3 ± 0.4	EU: 31.8 ± 4.1 min, 266 ± 19 W HY: 36.0 ± 3.1 min*, 250 ± 19 W*
Merry et al. (694)	12, 6 trained, 6 untrained	Cycling: 90 min at ∼60% $\dot{V}O_{2peak}$ + 14-17 h fluid restriction	Cycling: 40 min at 70% $\dot{V}O_{2peak}$ + 40-min time trial	24.3, 50.0, 4.5	Trained EU: within −0.5 HY: −1.5–2.0 Untrained EU: within −0.5 HY: −1.5–2.0	Trained:EU: ∼20.7 km HY: ∼18.0 kmUntrained: EU: ∼24.9 kmHY: ∼23.4 km
Oliver et al. (669)	13	Walking: 1.5 h at 50% $\dot{V}O_{2peak}$ 24 h and 48 h before exercise protocol	Running: 30-min time trial	19.7, 58.8, 2.0	EU: −0.6 ± 0.4 HY: −3.2 ± 0.5	EU: 6295.0 m, 12.6 km.h^{-1} HY: 6107.0 m, 12.2 km.h^{-1}
Slater et al. (692)	17	24-h fluid restriction to 4% body mass loss + partial rehydration	Rowing: 2,000-m time trial	21.1, 29.0, –32.4, 60.4, –	Males EU1: 0.8 HY1: −2.0 Females EU1: 1.7 HY1: −1.2 Males EU2: 1.1 HY2: −2.0 Females EU2: 1.7 HY2: −1.1	MalesEU1: 398.2 ± 7.4 s HY1: 400.3 ± 7.4 s Females EU1: 453.7 ± 10.8 s HY1: 457.2 ± 9.3 s Males EU2: 403.0 ± 6.0 s HY2: 403.3 ± 7.8 s Females EU2: 457.9 ± 10.1 s HY2: 461.4 ± 11.2 s
Stearns et al. (695)	17	22-h fluid restriction	Running: 12 km as 3 × 4 km with 4-min rest	WBGT: ∼26.2	EU: −0.8 ± 1.0 HY: −2.1 ± 1.3	EU: 3191.0 ± 366.0 s HY: 3339.0 ± 450.0 s*
Stewart et al. (700)	7	Cycling: 2 h at 50-65% of peak power output	Cycling: 5-km time trial	18.0–25.0, 20.0–30.0, –	EU: 0.2 HY: −3.8	EU: 7.1 ± 1.3 min HY: 7.3 ± 1.5 min
Wall et al. (706)	10	Walking and cycling at 60% $\dot{V}O_{2max}$	Cycling: 25-km time trial	33.0, 40.0, ∼9.0	EU: 0.0 ± 0.1 HY1: −2.1 ± 0.1 HY2: −3.0 ± 0.0	EU: 40.6 ± 2.2 min, 249 ± 27 W HY1: 40.6 ± 2.1 min, 251 ± 28 W HY2: 406 ± 2.1 min, 253 ± 30 W

Protocol details are from TABLE 5. RH, relative humidity; EU, euhydration, HY, hypohydration, WBGT, wet-bulb-globe temperature. *$P < 0.05$, significant difference from control/euhydrated condition.

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ACKNOWLEDGMENTS

The authors thank Harry Brown and Thomas Topham for help with collating the hydration data in sect. 5.4.

DISCLOSURES

J.D.P and T.M.H.E are scientific advisory board members for Inuteq. J.D.P. received funding from the Coca Cola Company to perform consultancy work after this article was first submitted.

AUTHOR CONTRIBUTIONS

J.D.P., T.M.E., and H.A.D. analyzed data; J.D.P., T.M.E., and H.A.D. prepared figures; J.D.P., T.M.E., and H.A.D. drafted

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manuscript; J.D.P., T.M.E., and H.A.D. edited and revised manuscript; J.D.P., T.M.E., and H.A.D. approved final version of manuscript.

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