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Review

Adaptations and mechanisms of human heat acclimation: Applications for competitive athletes and sports

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Exercise heat acclimation induces physiological adaptations that improve thermoregulation, attenuate physiological strain, reduce the risk of serious heat illness, and improve aerobic performance in warm-hot environments and potentially in temperate environments. The adaptations include improved sweating, improved skin blood flow, lowered body temperatures, reduced cardiovascular strain, improved fluid balance, altered metabolism, and enhanced cellular protection. The magnitudes of adaptations are determined by the intensity, duration, frequency, and number of heat exposures, as well as the environmental conditions (i.e., dry or humid heat). Evidence is emerging that controlled hyperthermia regimens where a target core temperature is maintained, enable more rapid and complete adaptations relative to the traditional constant work rate exercise heat acclimation regimens. Furthermore, inducing heat acclimation outdoors in a natural field setting may provide more specific adaptations based on direct exposure to the exact environmental and exercise conditions to be encountered during competition. This review initially examines the physiological adaptations associated with heat acclimation induction regimens, and subsequently emphasizes their application to competitive athletes and sports.

Humans have a remarkable ability to adapt to heat stress and given adequate water and protection from the sun, a healthy heat-acclimated individual can tolerate extended exposure to virtually any natural weather-related heat stress (Wenger, 1988; Hori, 1995; Sawka et al., 1996). Heat stress results from the interaction of environmental conditions (i.e., temperature, humidity, solar radiation), physical work rate (i.e., metabolic heat production), and wearing of heavy clothing that impedes heat loss (Sawka et al., 1996). Environmental heat stress and exercise interact to increase physiological strain (Nadel, 1977), which is manifested by high core, skin and brain temperatures, increased cardiovascular strain, a greater reliance on carbohydrate metabolism, and results in reduced aerobic performance (Rowell, 1974; Febbraio et al., 1994a,b; Galloway & Maughan, 1997; Périard et al., 2011b; Nybo et al., 2014). Heat acclimation results in adaptations that reduce the deleterious effects of heat stress (Table 1). Typically, adaptation occurs through morphological, chemical, functional, and genetic adjustments that decrease physiological strain under stress (Adolph, 1964; Bligh, 1973). During heat acclimation, adaptations develop following repeated heat exposures to artificial/laboratory settings that are sufficiently stressful to elicit profuse sweating and elevate skin and core temperatures. Similarly, heat acclimatization designates exposure to natural environments that elicit analogous responses (Wenger, 1988). Hence, the terms heat acclimation and heat acclimatization will be used interchangeably throughout the review.

In 1768, James Lind published the first report on the ability of humans to adapt to environmental heat (Lind, 1768). He reported that when relocating to East and West Indian climates, Europeans were at first adversely affected by the environment, but over a period of time, habituated and eventually lived comfortably (e.g., Fig. 1). This acclimatization phase included behavioral adaptations that led to reducing workload, rescheduling work periods, and utilizing shelters. Lind (1768) also highlighted that there were probably blood adaptations allowing Europeans to "enjoy a pretty good state of health" once heat acclimatized. Subsequent studies of heat acclimation have mostly been directed to military or occupational tasks (Dresoti, 1935; Horvath & Shelley, 1946; Hellon et al., 1956; Strydom et al., 1966; Wyndham, 1967; Pandolf et al., 1977), which are performed at relatively low-moderate exercise intensities (Henschel et al., 1943; Robinson et al., 1943; Weiner, 1950; Lind & Bass, 1963; Shvartz et al., 1973) with moderately fit subjects (Pandolf et al., 1977; Davies, 1981). Relatively few studies have attempted to modify and adapt heat acclimation strategies developed for occupational and military settings to the competitive athletes (Garrett et al., 2009; Lorenzo et al., 2010;

Table 1. Physiological adaptations and functional consequences associated with the heat acclimation phenotype that lead to improved thermal comfort and submaximal aerobic performance, and increased maximal aerobic capacity

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

Adapted with permission from Sawka et al. (2000, 2011).

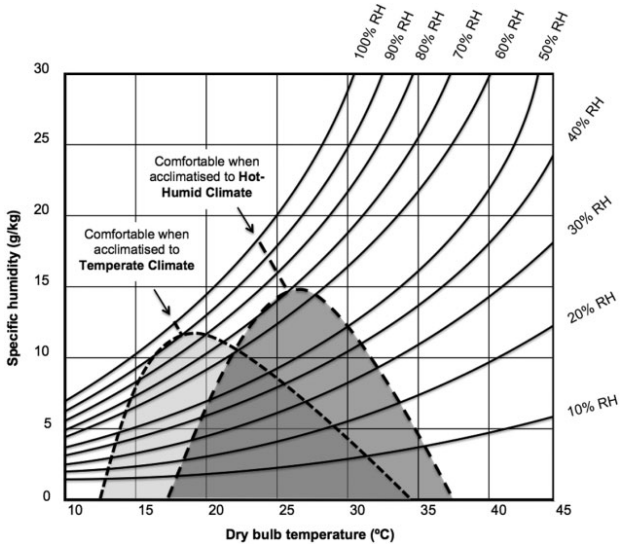

Fig. 1. Adjustment in thermal comfort with acclimatization to a tropical (hot and humid) climate in resting subjects. Comfort level is altered in relation to specific humidity (i.e., grams of water per kilogram of air), dry bulb temperature, and relative humidity. Adapted with permission from Folk (1974).

Buchheit et al., 2011; Racinais et al., 2014, 2015). Competitive athletes are often highly fit and participate in events requiring high metabolic intensities. In addition, most prior heat acclimation protocols were conducted over many days eliciting a "slow" adaptation, whereas athletes may rapidly travel from a temperate environment to a warm-hot one and may need more rapid induction of heat acclimation to optimize their performance.

This review initially examines the physiological adaptations associated with heat acclimation induction regimens, and subsequently emphasizes their application to competitive athletes and sports. The phenotypic adaptations described in the review relate to characteristics that are influenced by both an individual's genotype and the environment. Processes of physiological accommodation, which refers to the acute responses that support homeostatic regulation following heat exposure (Brooks, 1969), as well as habituation (i.e., tolerance), which relates to phenotypic adaptations that reduce physiological strain in well-adapted individuals under stress (Brück & Zeisberger, 1990) are also highlighted. It is hoped that this review will provide the scientific foundation to develop new exercise heat procedures for rapid induction of heat acclimation in athletes competing in high-intensity sporting events.

Human temperature regulation

Body core temperature is typically regulated about a mean value of ~37 °C, remaining within a narrow functional range (35 to 41 °C). Fluctuations in temperature occur naturally as a result of sleeping patterns, food intake, physical work, arousal, environmental conditions, hydration state, exercise, and fever. The regulation of body temperature is accomplished through two parallel processes: behavioral and physiological temperature regulation. Behavioral temperature regulation operates largely through conscious behavioral adjustments and may employ any means available, including standing in the shade and wearing light colored clothing. Alterations in work rate during self-paced exercise in the heat also constitute behavioral adjustments that contribute to regulate body temperature. Physiological or autonomic temperature regulation operates through thermogenic and theromolytic responses that are independent of conscious voluntary behavior. These responses include the control of metabolic heat production (i.e., shivering), vasomotor function (i.e., heat flow via blood redistribution from the core to the skin), and sudomotor function (i.e., sweating).

Human temperature regulation is mediated by physiological control systems that produce graded responses according to disturbances in a regulated variable, in this case, core temperature. Usually, the magnitude of change in response (e.g., sweating) is proportional to displacement of the regulated variable from a given basal level. Such control systems are called proportional-control systems. Both peripheral (i.e., skin) and central (i.e., brain, spinal column, and large vessels) thermal receptors provide afferent input to thermoregulatory centers located in the hypothalamus. When integrated in the thermoregulatory centers, thermal receptor input is juxtaposed to a reference temperature referred to as the "set point" (Hammel et al., 1963; Hensel, 1973). The "set point" is purely a mathematical concept used for describing the thermal control of effector responses. The concept does not imply a particular neural model of thermoregulation or set temperature, but describes different recruitment stages within the magnitude of a load error, which is the difference between the input and set point (Gisolfi & Wenger, 1984). Accordingly, thermal receptors in the core and skin send information to a central integrator, located in the preoptic anterior hypothalamus (Boulant, 1996). This integrator generates a thermal command signal that participates in the control of sweating, skin vasodilation, and vasoconstriction. Of note, core temperature changes have a greater effect (~9-fold) on heat loss responses than skin temperature changes (Sawka et al., 1996). However, ambient temperature – and thus skin temperature – changes are much greater than core temperature changes, so the total effect of skin temperature on thermoregulation should not be underestimated.

Recently, the notion of a unified central integrator has been re-examined. Romanovsky (2007) proposed that body core temperature is regulated by independent thermoeffector loops, triggered by temperature-dependent phase transitions in thermosensory neurons, which are mediated by a particular combination of shell and core temperatures. Although this concept is interesting and persuasive, the traditional notion of central integration is supported by data indicating that the ratio of the contributions from core and skin temperature inputs to changes in sweating and skin blood flow are the same, and that the thresholds for sweating and skin vasodilation are simultaneously shifted – to a similar degree – by factors such as circadian rhythm, fever, phase of the menstrual cycle, and heat acclimation (Gisolfi & Wenger, 1984; Stephenson & Kolka, 1988). Hence, the current review will rely on the traditional model of a central integrator and "set point."

Physiological adaptations and mechanisms

Heat acclimation develops through frequent exposure to hot environmental conditions, which elicit responses that attenuate the negative effects of heat stress. Heat acclimation improves thermal comfort (Lemaire, 1960; Folk, 1974; Gonzalez & Gagge, 1976), submaximal exercise performance, and increases maximal aerobic capacity ($\dot{V}O_{2max}$) in the heat (Lorenzo et al., 2010). The benefits of heat acclimation are achieved by enhanced sweating and skin blood flow responses, plasma volume expansion, better fluid balance and cardiovascular stability, a lowered metabolic rate, and acquired thermal tolerance (Hori, 1995; Sawka et al., 1996; Horowitz, 2014). Table 1 provides a summary of the physiological adaptations and functional consequences associated with the heat acclimation phenotype.

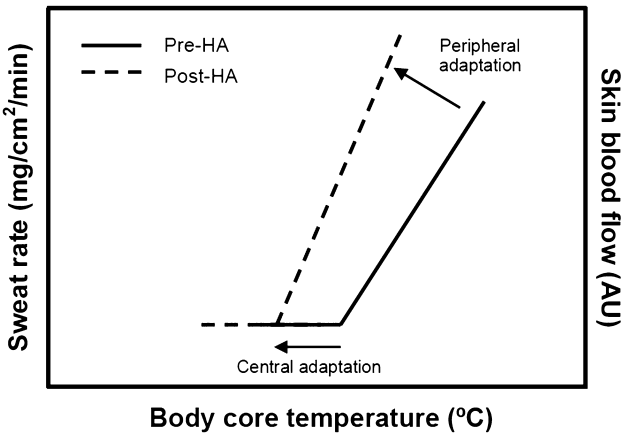

Fig. 2. Schematic representation of the central and peripheral adaptations that occur in response (Pre vs Post) to heat acclimation (HA). The body core temperature threshold for the onset of sweating is reduced, while the rate and sensitivity (i.e., slope) are increased. Concomitantly, the body core temperature threshold for the onset of cutaneous vasodilation is reduced, whereas skin blood flow sensitivity is increased. Adapted with permission from Nadel et al. (1971) and Gisolfi and Wenger (1984).

Sweating and skin blood flow

Adaptations in sweat rate and sweat composition were among the first described in response to heat acclimatization. Indeed, by the end of the 1940s, it was widely accepted that heat acclimatization increases sweat rate and decreases sweat sodium and chloride concentrations (Dill et al., 1933, 1938; Adolph & Dill, 1938; Robinson et al., 1943; Horvath & Shelley, 1946). Along with a shift in the onset threshold for sweating, which occurs earlier and at a lower core temperature (Nadel et al., 1974; Roberts et al., 1977), changes in sweat rate and composition are considered as the principal adaptive responses to heat exposure (Eichna et al., 1950; Strydom et al., 1966; Wyndham et al., 1968; Gonzalez et al., 1974; Nielsen et al., 1997). These responses are indicative of both central and peripheral adaptation (Fig. 2). At the central level, heat acclimation decreases the body temperature at which sweating is initiated. This adjustment in onset threshold is proposed to correspond to an absolute change in mean body temperature, rather than to the

attainment of a predetermined mean body temperature (Patterson et al., 2004b). Peripheral adaptations, manifested by changes in sweat rate and sensitivity (Allan & Wilson, 1971), occur at the level of the sweat gland (Fox et al., 1964; Chen & Elizondo, 1974; Inoue et al., 1999; Buono et al., 2009a,b). These adaptations include improved cholinergic sensitivity, and increased size and efficiency of eccrine glands in producing sweat per unit length of secretory coil (Sato & Sato, 1983; Sato et al., 1990). Sweat glands also become resistant to hidromeiosis so that higher sweat rates can be sustained (Fox et al., 1963a; Ogawa et al., 1982).

Along with an enhanced sweat rate, sweat composition changes with heat acclimation. Electrolytes (e.g., chloride) are reabsorbed and sodium concentration is reduced, resulting in a more dilute sweat (Dill et al., 1938; Allan & Wilson, 1971; Ogawa et al., 1982; Chinevere et al., 2008). For example, an unacclimatized person may secrete sweat with a sodium concentration of 60 mEq/L or higher and, if sweating profusely, can lose large amounts of sodium. With acclimation, the sweat glands conserve sodium by secreting sweat with a sodium concentration as low as 10 mEq/L. The preservation of sodium appears to stem from an increased sodium conservation within the reabsorptive duct of the sweat gland (Sato & Dobson, 1970; Sato et al., 1971), which is dependent on sodium depletion, as well as aldosterone, a hormone secreted in response to exercise and heat stress that facilitates the reabsorption of sodium. Of note, heat dissipation via sweat evaporation is enhanced for a given sweat rate because of the change in sweat composition, as electrolytes lower the water vapor pressure at the level of the skin for a given temperature (Taylor, 2014). Consequently, a more dilute sweat is more easily evaporated because of a widening of the water vapor gradient between the skin and ambient air.

Exercising in the heat represents a circulatory challenge for both unacclimated and acclimated athletes. However, earlier and greater sweating during acclimation improves evaporative cooling, assuming the climate allows evaporation, and reduces skin temperature and thus skin blood flow requirements. Lower skin temperature may also reduce cutaneous venous compliance so that blood volume is redistributed from the peripheral to the central circulation (Eichna et al., 1950; Rowell et al., 1967). Until recently, it was postulated that heat acclimation centrally modifies thermoregulatory responses in the skin by reducing the core temperature threshold for vasodilation, without altering the slope of the blood flow–core temperature relationship (i.e., sensitivity; Fox et al., 1963b; Roberts et al., 1977; Yamazaki & Hamasaki, 2003). However, by locally infusing an endothelium-dependent vasodilator (acetylcholine) via microdialysis, Lorenzo and Minson (2010) showed that acclimation does improve local cutaneous vascular responses (Fig. 2). The authors suggested that this peripheral response may be derived from adaptations associated with an increase in the number and sensitivity of muscarinic receptors, a decrease in cholinesterase activity leading to an improved vascular response to acetylcholine, or alterations to the pathway of vasodilation within smooth muscles or the endothelial cells. Thus, given that heat acclimation does not alter maximal skin blood flow, the modified cutaneous vascular response appears to stem from improvements in vascular function (i.e., increased sensitivity of the skin microvasculature to vasodilate), rather than structural changes that limit maximal vasodilator capacity (Lorenzo & Minson, 2010).

Blood volume and fluid balance

Most studies report that heat acclimation increases total body water by 2–3 L, or ~5–7% (Bass et al., 1955; Wyndham et al., 1968; Patterson et al., 2004a, 2014). This increase is well within the measurement resolution for total body water and thus appears to be a real physiological phenomenon. While it may be argued that such an increase in total body water may be detrimental to performance in certain sports because of the increase in body mass, the benefits (e.g., improved thermoregulation and cardiovascular stability) stemming from this increase outweigh its potential deleterious effects, especially in endurance sports. The division of the total body water increase between intracellular fluid (ICF) and extracellular fluid (ECF: plasma and interstitial fluid) is variable, as studies have reported that ECF accounts for greater, equal, and smaller than its percentage increase in total body water after heat acclimation (Sawka & Coyle, 1999). Measures of ECF have relatively high variability, and therefore, trends for such small changes are difficult to interpret. The extent to which ICF increases is unclear because it is typically calculated as the difference between total body water and ECF, and thus, measurement variability inherent in both these techniques is compounded in the calculation of ICF. The increase in total body water can be explained in part by the increased secretion of fluid conserving hormones aldosterone and arginine vasopressin, and/or renal sensitivity to a given plasma concentration. The conservation of sodium also helps to maintain the number of osmoles in the extracellular fluid, and thus, to maintain or increase ECF volume during adaptation to repeated heat exposure (Nose et al., 1988). Correspondingly, if total body water and ECF increase after heat acclimation, then expansion of plasma volume might be expected.

Adjustments in blood volume in response to climatic changes were first reported by Barcroft et al. (1922). Thereafter, Bazett et al. (1940) published a comprehensive description of the hematological adaptations associated with heat exposure, indicating that changes in plasma volume develop quite rapidly, causing a temporary decrease in haemoglobin concentration and

hematocrit, and occasionally in plasma protein concentration. Plasma volume expansion is usually present after 3 to 4 days of repeated heat exposure (Sawka & Coyle, 1999), but is also influenced by seasonal changes. A ~5% expansion in resting plasma volume occurs in the hottest months and a ~3% contraction in the coldest months (Doupe et al., 1957; Sawka & Coyle, 1999). This occurs with considerable variability with some individuals not experiencing an expansion in plasma volume. During acute heat acclimation, plasma volume expansion is generally 4–15%, but can range from 3% to 27% (Bass et al., 1955; Senay et al., 1976; Nielsen et al., 1993; Patterson et al., 2004a, 2014), while erythrocyte volume remains unaltered by heat acclimation or season (Sawka & Young, 2000). The magnitude of increase in plasma volume is dependent on the heat acclimation day, the hydration state when measured, skin temperature and whether the individual is at rest or performing exercise (Sawka et al., 1983b; Harrison, 1985; Kenefick et al., 2014). It may also relate to fitness level, with endurance-trained athletes already exhibiting an expanded plasma volume. Notwithstanding, plasma volume expansion seems to be greatest during upright exercise on about the fifth day of heat acclimation in fully hydrated individuals. Conversely, during water-based sports (e.g., long distance swimming), the hydrostatic pressure conferred by the water and the prone position adopted during exercise may act to minimize the decreases in renal blood flow and central venous pressure, resulting in reduced fluid regulation and plasma volume expansion (Harrison, 1985; Convertino, 1991). Ultimately, plasma volume expansion has two obvious physiological advantages: (a) increasing vascular filling to support cardiovascular stability; and (b) increasing the specific heat of blood to slightly lower skin blood flow responses (Sawka et al., 2011).

Although the rapid increase in plasma volume expansion was previously described as being a transient phenomenon (Wyndham et al., 1968; Senay, 1979; Shapiro et al., 1981), it appears that this may have been an experimental artifact related to the traditional constant work rate model of heat acclimation. Recent findings suggest that by using the controlled hyperthermia technique, which maintains a constant adaptation stimulus by clamping core temperature (e.g., 38.5 °C) throughout acclimation, plasma volume remains similarly expanded (~14%) after 8 and 22 days of heat exposure (Patterson et al., 2004a, 2014). The mechanism(s) responsible for this hypervolemia are unclear, but may include an increase in ECF mediated by retention of crystalloids, primarily sodium chloride, and perhaps an increase in plasma volume modulated by the oncotic effect of intravascular protein (Mack & Nadel, 1996; Patterson et al., 2004a, 2014).

A reduction in total body water (i.e., dehydration) will adversely affect thermoregulation and increase cardiovascular strain (Morimoto, 1990; Sawka & Coyle, 1999), which can counteract the benefits conferred by heat acclimation and high aerobic fitness (Buskirk et al., 1958; Sawka et al., 1983b). Dehydration increases core temperature during exercise in temperate and hot environments, and the greater the water deficit, the greater the elevation in core temperature. Dehydration also impairs dry and evaporative heat loss via the development of plasma hyperosmolality and hypovolemia (Sawka & Coyle, 1999). However, recent evidence suggests that heat acclimation might attenuate the adverse effects of hyperosmolality on impairing sweating and skin blood flow responses (Takamata et al., 2001). As such, total body water expansion stemming from heat acclimation may confer a protective benefit against dehydration. Moreover, moderate permissive dehydration during exercise heat acclimation may facilitate adaptation (Taylor & Cotter, 2006) by increasing fluid-electrolyte retention, plasma volume expansion, and cardiovascular responses to heat stress, particularly during short-term heat acclimation (Garrett et al., 2011, 2014). Thus, although dehydration has clear detrimental effects on performance, heat acclimation regimens that allow for permissive dehydration may contribute to accelerate the acclimation process.

Fluid balance improvements from heat acclimation include better matching of thirst to body water needs, reduced sweat sodium losses, increased total body water, and increased blood volume (Mack & Nadel, 1996; Sawka & Coyle, 1999). Thirst is often not a good index of body water requirements as *ad libitum* water intake can result in incomplete fluid replacement or voluntary dehydration during exercise heat stress (Adolph & Dill, 1938; Bean & Eichna, 1943; Eichna et al., 1945; Adolph, 1947; Greenleaf & Sargeant, 1965; Greenleaf et al., 1983; Armstrong et al., 1985; Greenleaf, 1992). Heat acclimation improves the relationship of thirst to body water needs so that voluntary dehydration is markedly reduced (~30%; Bean & Eichna, 1943; Eichna et al., 1945, 1950). Consequently, heat-acclimated individuals are better able to maintain hydration during exercise in the heat, and thus minimize body water deficits and voluntary dehydration, provided that access to fluids is not restricted (Fig. 3). This is an important adaptation as heat acclimation increases sweating rate and if fluid replacement is not proportionately increased, greater dehydration will occur, especially in humid environments. Along with the expansion of plasma volume induced during heat acclimation, the ability to better maintain fluid balance through thirst represents an adaptive response that contributes to reduce cardiovascular strain during subsequent heat stress.

Cardiovascular stability

Improved cardiovascular stability can be best illustrated by a reduced likelihood of syncope during repeated days of exercise heat exposure. Figure 4 provides redrawn

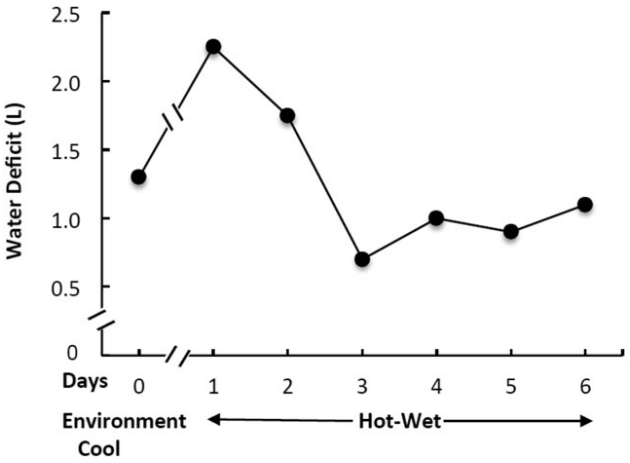

Fig. 3. Difference between *ad libitum* water intake and water loss in 15 men during work in humid heat over a 6-day period, compared with work in a cool environment (Eichna et al., 1945). Reproduced with permission from Sawka et al. (1984).

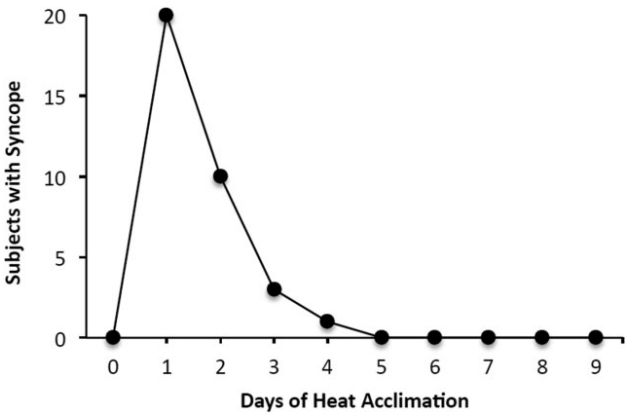

Fig. 4. Incidence of syncope in 45 men walking with a 9 kg load for 20 km in 49 °C and 20% relative humidity. Redrawn with permission from Bean and Eichna (1943).

data from Bean and Eichna (1943). On the first day of exercise in the heat, heart rate is much higher than in temperate conditions, and stroke volume is lower; thereafter, heart rate begins to decrease when exercising at a given work rate. These changes are rapid at first, but continue more slowly for about a week. Various mechanisms participate in reducing cardiovascular strain and their relative contributions vary over the course of heat acclimation and also among individuals (Wenger, 1988; Sawka et al., 1996). These mechanisms include (a) improved skin cooling and redistribution of blood volume; (b) plasma volume expansion; (c) increased venous tone from cutaneous and noncutaneous vascular beds; and (d) reduced skin and core temperature. It has also been suggested that a decrease in sympathetic nervous activity (i.e., plasma norepinephrine concentration) may contribute to reduce heart rate during heat acclimation (Berlyne et al., 1974; Hodge et al., 2013), while in rats, myocardial adaptations could increase compliance and changes in the distribution of myosin isoenzymes, reducing myocardial energy cost (Horowitz et al., 1986).

The effects of heat acclimation on stroke volume and cardiac output during exercise heat stress include responses dependent upon the exercise intensity and heat stress type. Two studies reported increases in stroke volume with little change in cardiac output as heart rate decreased with acclimation (Rowell et al., 1967; Wyndham et al., 1968). Another study reported a decrease in cardiac output, associated with a reduction in "surface blood flow," but little change in stroke volume as heart rate decreased (Wyndham, 1951). Still, another study reported a mixed pattern, with two subjects showing a steady increase in stroke volume, one a transient increase reversing after the sixth day, and one showing no increase with heat acclimation (Wyndham et al., 1976). The reason for these differences is unclear, although one possibility might be that Rowell et al. (1967) described dry heat acclimation, while Wyndham (1951) and Wyndham et al. (1968, 1976) described humid heat acclimation. Nielsen et al. (1993, 1997) examined stroke volume responses during exercise before and after heat acclimation. One study acclimated subjects for 9–12 days in hot-dry (40 °C, 10% relative humidity; RH) conditions while cycling at 50% $\dot{V}O_{2max}$ (Nielsen et al., 1993). They reported that during exercise, heat acclimation increased stroke volume (~21 mL/beat) and cardiac output (~1.8 L/min). In the other study, subjects acclimated for 8–13 days in hot-humid (35 °C, 87% RH) conditions while cycling at 45% $\dot{V}O_{2max}$ (Nielsen et al., 1997). Heat acclimation in these conditions did not alter stroke volume or cardiac output. Notably, both studies reported a plasma volume expansion of 9–13%.

It has also been shown that 10 days of heat acclimation improves maximal stroke volume and cardiac output during a $\dot{V}O_{2max}$ test in cool (13 °C), but not hot (38 °C) conditions, without influencing maximum heart rate (Lorenzo et al., 2010). Furthermore, short-term (5 days) moderate-intensity (70% $\dot{V}O_{2max}$, 30 min/day) exercise heat acclimation has been shown to increase plasma volume and stroke volume, and decrease heart rate (Goto et al., 2010). Ultimately, these observations indicate that heat acclimation improves central hemodynamics, but that the magnitude of improvement may depend on the environmental condition (i.e., dry vs humid heat), acclimation regimen (i.e., stimulus impulse), exercise intensity, and subject population.

Whole-body and skeletal muscle metabolism

Heat acclimation has been shown to alter whole-body (Sawka et al., 1996) and skeletal muscle metabolism (Young et al., 1985; Febbraio et al., 1994a). Consistent with this is the frequent but not universal observation that basal metabolic rate is decreased during warmer

months (Hori, 1995). The oxygen uptake response to submaximal exercise is also reduced by heat acclimation (Sawka et al., 1983a), as is muscle glycogen utilization (40–50%; King et al., 1985; Kirwan et al., 1987). However, the glycogen-sparing effect of heat acclimation has also been shown to be quite small and apparent only during exercise in cool conditions (Young et al., 1985). A further effect of heat acclimation is the reduction of blood and muscle lactate accumulation during submaximal exercise (Febbraio et al., 1994a) and the increase in power output at lactate threshold (Lorenzo et al., 2010). The mechanism(s) mediating these adaptations remains unclear, but could stem from the increase in total body water enhancing lactate removal through increased splanchnic circulation (Rowell et al., 1968), or through increased cardiac output and decreased metabolic rate, delaying lactate accumulation (Sawka et al., 1983a; Young et al., 1985).

Thermal tolerance

Thermal tolerance refers to cellular adaptations from a severe nonlethal heat exposure that allows the organism to survive a subsequent and otherwise lethal heat exposure (Moseley, 1997; Horowitz, 1998). It has been suggested that thermal tolerance and heat acclimation share a common basis, as both may potentially be governed by the heat shock response (Kuennen et al., 2011). As such, thermal tolerance and heat acclimation are complimentary, as acclimation reduces heat strain and tolerance increases survivability to a given heat strain. For example, rodents with fully developed thermal tolerance can survive 60% more heat strain than what would have been initially lethal (Maloyan et al., 1999). Thermal tolerance is associated with heat shock proteins (Hsp) binding to denatured or nascent cellular polypeptides and providing protection and accelerating repair from heat stress, as well as fever, hypoxia, ischemia, viral infection, energy depletion, and acidosis (Kregel, 2002).

Hsp are grouped into families based upon their molecular mass (8 to 110 kDa), with Hsp72 being particularly responsive to heat stress and exercise (Locke, 1997). Hsp families have different cellular locations and functions. At the intracellular (iHsp) level, Hsp process stress-denatured proteins, manage protein fragments, maintain structural proteins, and chaperone other proteins across cell membranes. In the extracellular (eHsp) milieu, it is purported that Hsp act as a signal, triggering an immuno-stimulatory response (Pockley, 2003; Asea, 2007; Noble et al., 2008). The export of Hsp outside the cell occurs via translocation across the plasma membrane, release via lipid vesicles and through passive release following necrosis (De Maio & Vazquez, 2013).

Hsp expression increases during and following exposure to heat stress with differing responses across various tissues (e.g., brain and liver exhibit a greater response than skeletal muscle). After the initial exposure, mRNA levels peak within an hour and subsequent Hsp synthesis depends upon both the severity and cumulative heat stress (Maloyan et al., 1999). Passive heat exposure and physical exercise both elicit Hsp synthesis (Febbraio & Koukoulas, 2000); however, the combination of exercise and heat exposure elicits a greater Hsp response than either stressor independently (Skidmore et al., 1995). During exercise in the heat, the expression of eHsp has been shown to be both duration and intensity dependent, relating to the level of hyperthermia attained and rate of rise in core temperature (Périard et al., 2012b).

Horowitz and Robinson (2007) noted that heat acclimation increases Hsp70 reserves and accelerates the heat shock response in animals. In addition, Hsp expression with heat acclimation has been associated with increased physical performance and aerobic power in military working dogs (Bruchim et al., 2014). In humans, the heat shock response during acclimation and the concomitant expression of iHsp and eHsp remain somewhat unclear. For example, it has been shown that 2 days of exercise heat exposure reduces basal levels of eHsp72 and that an increase occurs immediately post-exercise (Marshall et al., 2006). It has also been shown that iHsp72 is unaffected over the same time course, although a slight nonsignificant increase in both basal and post-exercise expression was observed (Marshall et al., 2007). Yamada et al. (2007) demonstrated that when acclimation was extended to 10 days, basal iHsp72 expression increased within 6 days, which blunted the post-exercise induction response. In contrast, eHsp72 remained unchanged. During a 15-day heat acclimation regimen, basal eHsp72 was shown to progressively increase and post-exercise expression decrease in a 29-year-old male ultra marathon runner preparing for the Marathon des Sables, suggesting that a longer heat acclimation period may induce greater cellular adaptations (Sandstrom et al., 2008). McClung et al. (2008) conducted a further study in which iHsp72 and iHsp90 responses to 10 days of exercise heat acclimation were correlated with physiological adaptations. The authors noted that acclimation increased basal levels of both proteins and that individuals demonstrating the greatest physiological adaptations exhibited a reduced post-exercise expression (measured *ex vivo* via water bath incubation). More recently, an 11-day controlled hyperthermia (1 °C core temperature elevation) acclimation regimen resulted in an increase in basal iHsp72 levels, while eHsp72 expression remained unchanged (Magalhaes et al., 2010). As with the previous study, the expression following exercise heat exposure was blunted in both iHsp72 and eHsp72. Thus, it appears that iHsp may be more sensitive to heat stress than eHsp and that an increase in basal level during acclimation results in a blunting of the acute response to exercise as acclimation develops. Moreover, Hsp expression may depend on the heat acclimation technique utilized, the level of heat strain attained, as well as sustained. This is consistent with observations that 11

days of moderate exercise (50% $\dot{V}O_{2max}$) and mild heat exposure (33 °C and 40% RH) for 90 min induced adaptations associated the heat acclimation phenotype, without inducing significant iHsp70 responses (Hom et al., 2012).

Moreover, it seems likely that other cellular systems, besides Hsp, contribute to improved thermal tolerance and heat acclimation. Research has identified ~130 genes that are up-regulated and ~89 genes down-regulated during heat stress (Sonna et al., 2001). By comparing the transcript profiles of passive heat exposure and aerobic training with that of exercise heat acclimation, Kodesh et al. (2011) were able to describe the influence of each individual stressor on the reprogramming of gene expression in the development of the exercise heat acclimation phenotype. The authors showed that genes affected by chronic heat exposure were associated with the excitation-contraction coupling cascade, whereas aerobic training up-regulated genes involved with calcium turnover. As such, it was purported that the interaction between heat exposure and exercise is not merely additive, but should be viewed as an independent stress, which leads to a specific transcriptional program (Kodesh et al., 2011). For a thorough review on the role of heat acclimation on cytoprotection and epigenetics, the reader is referred to a review by Horowitz (2014).

Performance in hot environments

The effect of heat acclimation on submaximal exercise performance can be quite dramatic, such that acclimated individuals can easily complete tasks in the heat that earlier were difficult or impossible. Pandolf and Young (1992) reported that of 24 subjects, none were able to complete a 100-min walk in 49 °C and 20% RH on the first day of exposure. However, 40% were successful by day 3, 80% by day 5, and all but one were successful by the seventh acclimation day. Racinais et al. (2015) also showed that cycling time trials (43 km) undertaken in hot outdoor (i.e., field setting) conditions (~37 °C) were initiated at a similar power output to that of a time trial conducted in cold conditions (~8 °C). Subsequently, however, a marked decrease in power output occurred in the heat, which was partly recovered after 1 week of heat acclimatization and almost fully restored after 2 weeks. Accordingly, heat acclimation/acclimatization mediates improved submaximal exercise performance by reducing physiological strain and abating a variety of other potential fatigue mechanisms (Nybo et al., 2014).

Interestingly, relative to values recorded in temperate conditions, heat stress mediates a reduction in $\dot{V}O_{2max}$ in trained individuals that cannot be abated by heat acclimation. Indeed, Sawka et al. (1985) demonstrated that heat acclimation improved $\dot{V}O_{2max}$ by 3.5% in moderate (21 °C) conditions and by 4.2% in the heat (49 °C). However, the improvement noted in the hot environment could not compensate for the ~8% reduction in $\dot{V}O_{2max}$ conferred by heat stress both before and after acclimation, when compared with moderate conditions. Recently, Lorenzo et al. (2010) observed improvements of 8% and 5% in the $\dot{V}O_{2max}$ of trained individuals in hot (38 °C) and cool (13 °C) conditions, respectively, following a traditional 10-day heat acclimation regimen (40 °C for 100 min at ~50% $\dot{V}O_{2max}$). As with previous observations, the larger improvement in $\dot{V}O_{2max}$ noted in the hot condition after acclimation failed to compensate for the ~20% reduction conferred by heat stress, both before and after acclimation. Notwithstanding, the authors also observed increases in cycling time trial performance of 8% (hot) and 6% (cool) (Lorenzo et al., 2010). The improvements in self-paced exercise performance were proportional to the increase in $\dot{V}O_{2max}$ in each condition, which reinforces the notion that relative exercise intensity strongly influences performance in the heat (Périard et al., 2011b; Périard, 2013). More specifically, these improvements in aerobic performance were associated with increases in maximal cardiac output and lactate threshold, plasma volume expansion, lower skin temperatures, and a larger core-to-skin gradient after heat acclimation. In contrast, no differences were observed for the control group (Lorenzo et al., 2010).

Following two studies from Nielsen et al. (1993, 1997) in which subjects heat acclimated by exercising to exhaustion in hot-dry and hot-humid conditions, it was proposed that heat acclimation does not alter the maximal core temperature an individual can tolerate during exercise in the heat. This was proposed because exhaustion coincided with a core temperature of ~39.8 °C on each day of acclimation, despite exercise duration increasing throughout the acclimation period (Nielsen et al., 1993; Nielsen et al., 1997). Interestingly, exhaustion occurred at relatively low heart rates both prior to (~158 beats/min) and after (~150 beats/min) acclimation. Moreover, no reductions in cardiac output and muscle blood flow were observed at exhaustion, or changes in substrate utilization and blood lactate accumulation (Nielsen et al., 1993). This led the authors to suggest that exercise may have been terminated prematurely because of a reduction in motivation. Accordingly, several studies have shown that motivated individuals reach exhaustion at a heart rate above 95% of maximum during constant rate exercise to exhaustion and that decrements in cardiac output occur (Rowell et al., 1966; Gonzalez-Alonso et al., 1999; Périard et al., 2011a, 2012a).

Moreover, there is evidence that individuals living and training over many weeks in the heat might tolerate higher maximal core temperatures than those heat acclimated over 1 or 2 weeks (Sawka et al., 2001), and that trained individuals can tolerate higher core temperatures (Pugh et al., 1967; Cheung & McLellan, 1998; Gonzalez-Alonso et al., 1999; Mora-Rodriguez et al., 2010; Périard et al., 2012a). For example, Robinson (1963) showed that highly trained and likely

acclimatized runners can reach core temperatures of 41.1 °C during a 3-mile run (14:15 min) in 30.6 °C conditions. It has also been shown that trained runners performing an 8-km running time trial in warm conditions (27 °C wet-bulb-globe temperature; WBGT) are able to sustain running velocity, despite a core temperature exceeding 40 °C (Ely et al., 2009). More recently, it was shown that trained cyclists reach core temperatures of 40.1–40.2 °C at the end of a 43.3-km time trial in hot (37 °C) conditions (Racinais et al., 2015). Therefore, it does appear that aerobic fitness confers an increased capacity to tolerate higher core temperatures. However, whether heat acclimation provides a similar benefit remains to be determined.

Performance in cool environment

The observation of enhanced aerobic performance in cool conditions by Lorenzo et al. (2010) supports previous findings that heat acclimation improves $\dot{V}O_{2max}$ in untrained (13%) and unfit (23%) individuals in cool conditions (Shvartz et al., 1977). It also reinforces the 32% increase in run time to exhaustion noted in fit individuals by Scoon et al. (2007) after acclimation via post-exercise sauna bathing, and the observation that swimmers training in a tropical climate (30 °C, 80% RH) in 30 °C water had greater improvements (10%) in performance when returning to a temperate environment (27.1 °C pool water) than swimmers who kept training in a temperate environment (Hue et al., 2007). Other studies have also observed that team-sport athletes participating in preseason (Racinais et al., 2014), in-season (Buchheit et al., 2011), and off-season (heat and altitude; Buchheit et al., 2013) training camps in the heat (~34 °C) improve performance by 7% and 44% (Yo-Yo Intermittent Recovery test level 1 and 2, respectively) in temperate conditions (~22 °C). The mechanisms modulating the transfer between hot and cool conditions could be linked to a variety of ergogenic responses, with cardiovascular/thermoregulatory (Lorenzo et al., 2010) and cellular (Bruchim et al., 2014) adaptations. As recently highlighted by Corbett et al. (2014), heat acclimation may provide a stimulus for improving performance in nonthermally challenging environments via improvements in $\dot{V}O_{2max}$, lactate threshold, and economy. Interestingly, it has also been suggested that heat acclimation may serve to preserve or enhance performance at altitude (White et al., 2014). The purported pathways for such a response include plasma volume expansion, improved cardiac efficiency, and involve the up-regulation of hypoxia-inducible factor-1 in boosting oxygen delivery. Currently, however, additional research with well-designed protocols (e.g., sufficient participants and control groups) is required to elucidate the mechanisms associated with improvements in performance at altitude and in cool conditions, as well as fully substantiate the ergogenic benefits of heat acclimation on performance in these environments.

Time course of adaptation

Induction

Heat acclimation is a relatively rapid process that begins on the first day of exposure (Fig. 5). In fact, 75–80% of the adaptations occur in the first 4–7 days (Pandolf, 1998; Shapiro et al., 1998). The development of these adaptations can be divided in three broad periods (Taylor, 2014), namely, physiological accommodation, short-term, and long-term adaptation (Sundstroem, 1927; Candas, 1987). The timeline can further be categorized as short-term acclimation (< 7 days), medium-term acclimation (8–14 days), and long-term acclimation (> 15 days; Garrett et al., 2011). The exercise heat acclimation phenotype is generally achieved through one of three induction pathways: (a) constant work rate exercise (Robinson et al., 1943; Nadel et al., 1974; Nielsen et al., 1993, 1997); (b) self-paced exercise (Nelms & Turk, 1972; Armstrong et al., 1986); and (c) controlled hyperthermia, or isothermic heat acclimation (Fox et al., 1967; Regan et al., 1996; Patterson et al., 2004a; Garrett et al., 2009). Although exercise in the heat is the most effective method for developing heat acclimation, passive heat exposure also results in some adaptation (Takamata et al., 2001; Beaudin et al., 2009; Brazaitis & Skurvydas, 2010). However, the specificity of exercise environmental adaptation lies with the stimulus representing the conditions in which the athlete will eventually perform. Thus, to achieve optimal adaptation, work rate and environmental conditions should closely replicate those of the competition setting.

The foundation of our understanding regarding the induction of heat acclimation stems from studies conducted in the early to mid-20th century (Dresoti, 1935; Dill et al., 1938; Bean & Eichna, 1943; Robinson et al., 1943; Eichna et al., 1945, 1950; Ladell, 1951; Wyndham, 1951; Buskirk et al., 1958). These studies demonstrated that during the initial heat exposure, physiological strain is high, as manifested by elevated core temperature and heart rate. However, the physiological strain induced by heat stress progressively decreases each day of acclimation. Through daily exercise in a hot climate (dry or humid), most of the improvements in heart rate, skin and core temperature, and sweat rate are achieved during the first week of exposure. The heart rate reduction develops most rapidly in 4–5 days. After 7 days, the reduction in heart rate is virtually complete. Most of the improvements in skin and core temperature have also occurred by 7 days. The thermoregulatory benefits of heat acclimation are generally thought to be complete after 10–14 days of exposure; however, improvements in physiological tolerance may take longer.

Following on from these initial investigations, it was suggested that continuous daily exposure to dry heat for

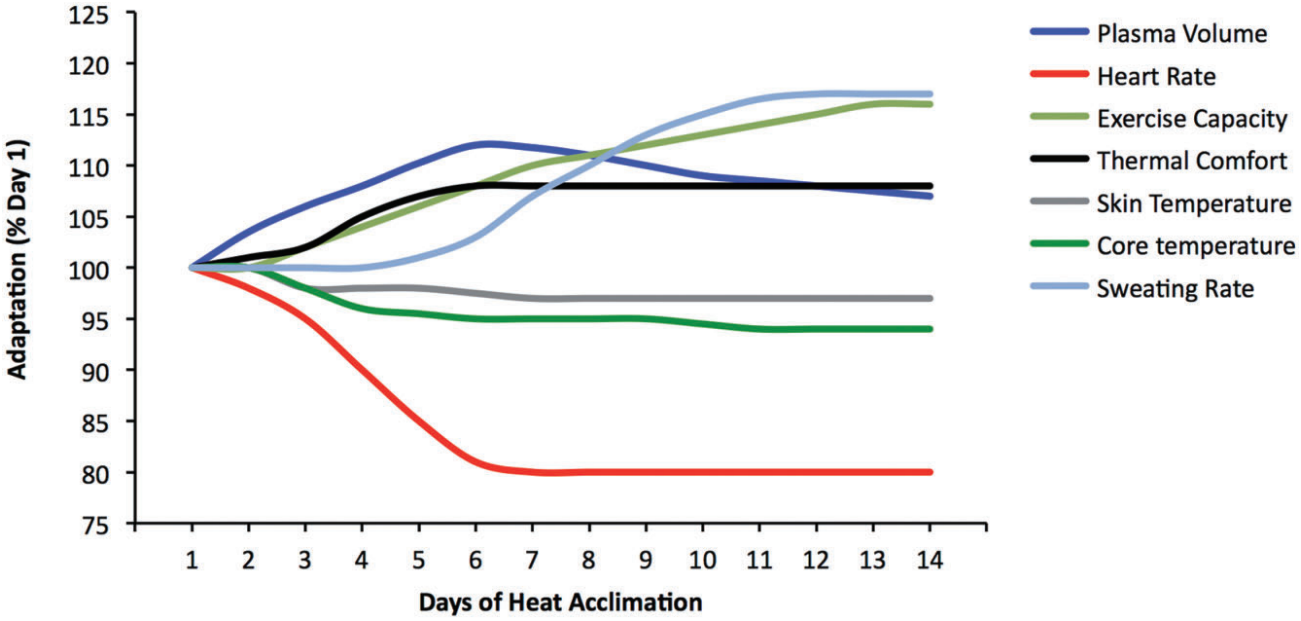

Fig. 5. Time course of induction in human adaptations to heat stress. Within this first week of exercise heat acclimation, plasma volume expands and heart rate decreases during exercise at a given work rate. Perceptually, the rating of thermal comfort improves. From a thermoregulatory perspective, core and skin temperature are reduced during exercise at a given work rate, whereas sweat rate increases. Consequently, aerobic exercise capacity is increased. Of note, the magnitude of these adaptations is dependent on the initial level of acclimation, the environmental conditions (i.e., dry or humid), exercise intensity, and acclimation regimen.

100 min produced optimal heat acclimation responses (Lind & Bass, 1963). Interestingly, while studies investigating heat acclimation have generally used daily heat exposure, Fein et al. (1975) examined the time course of physiological adaptations to 10 days of heat exposure, with subjects exposed to heat daily, or every third day. Using this paradigm, one group completed the acclimation regimen in 10 days and the other in 27 days. Both regimens were equally effective in inducing heat acclimation, but with daily heat exposure, it took approximately one-third of the total time. More recently, it was shown with a similar regimen that minimal adaptation occurs with intermittent heat exposure relative to daily exposure, which was clearly a more effective strategy for inducing heat acclimation (Gill & Sleivert, 2001). Although some adaptations occur when intermittently exposed to heat (72-h intervals), it appears that 1-week intervals do not induce heat acclimation (Barnett & Maughan, 1993). Notwithstanding, intermittent heat exposures may be beneficial in a high-performance context when fatigue and dehydration should be avoided (e.g., taper period; Chalmers et al., 2014). Correspondingly, in highly training athletes, heat acclimation sessions should be viewed and utilized as specific interventions, separate from those of daily training. Conversely, a heat acclimation regimen may supplement regular training in less fit individuals.

Most recently, considerable focus has been placed on short-term heat acclimation in benefiting highly trained endurance and team-sport athletes (Garrett et al., 2009, 2011; Chalmers et al., 2014). While it is expected that cardiovascular adaptations develop over this time course (5–7 days), it does not provide a strong-enough stimulus for sudomotor adaptation (Cotter et al., 1997). Nevertheless, the expansion of plasma volume, along with the emergence of thermoregulatory and metabolic adaptations, improves the perception of effort and fatigue. Hence, short-term heat acclimation appears to be sufficient at inducing performance-enhancing adaptations, which may be more pronounced after fluid regulatory strain from a permissive dehydration acclimation regimen (Taylor & Cotter, 2006; Garrett et al., 2011, 2014). Although short-term acclimation regimens do not induce adaptations as complete as long-term acclimation, these regimens may be particularly beneficial as a preseason training camp, for tapering before competition, and as a mid-season performance-enhancing tool.

Magnitude

The magnitude of physiological adaptations induced by heat acclimation depends largely on the intensity, duration, frequency, and number of heat exposures (Sawka et al., 1996). For example, it has been shown that low-intensity long-duration (60 min at 50% $\dot{V}O_{2max}$) exercise elicits similar heat acclimation benefits (i.e., reduced exercising heart rate, core temperature, and metabolism) to that of moderate-intensity short-duration (30–35 min 75% $\dot{V}O_{2max}$) exercise (Houmard et al., 1990). It has also been shown that certain adaptations (i.e., shorter sweating latency and lower core temperature threshold for sweating) are more pronounced during the specific daily time period in which previous heat exposure has occurred (Shido et al., 1999).

The magnitude of adaptation may also relate to the induction pathway. Taylor (2000, 2014) has highlighted that repeated exposure to a constant work rate regimen (i.e., traditional heat acclimation) results in physiological habituation, whereas the progressive overload approach (e.g., controlled hyperthermia to a core temperature of 38.5 °C) induces more complete adaptation. In effect, the traditional heat acclimation model offers a constant forcing function (i.e., fixed endogenous and exogenous thermal loads), the influence of which progressively decreases as adaptations develop (Eichna et al., 1950; Fox et al., 1963a; Rowell et al., 1967). In contrast, the adaptation impulse is maintained during controlled hyperthermia as the individual adapts because the forcing function (i.e., metabolic heat production) is increased through the manipulation of endogenous and/or exogenous thermal loads (Taylor, 2000).

Alternatively, we propose that utilizing a controlled intensity regimen, whereby a given level of cardiovascular strain is maintained during daily exercise heat exposure, may also elicit a sustained forcing function. This strain level might be achieved by identifying the heart rate associated with a specific relative intensity (e.g., a percentage of $\dot{V}O_{2max}$ or lactate threshold) in cool conditions, and adjusting work rate accordingly during exercise in the heat to sustain this heart rate throughout the acclimation period. As with the controlled hyperthermia regimen, absolute work rate would increase after the initial accommodation phase. This approach appears practical given that Garrett et al. (2012) showed the maintenance of a mean heart rate around 121 beats/min throughout a short-term controlled hyperthermia acclimation regimen, despite work rate increasing from day 1 to 5. Consequently, this approach could have greater real-world application and relevance to athletes and coaches training with heart rate, rather than with body core temperature. Moreover, the controlled intensity regimen may offer a greater cardiovascular adaptation impulse based on the level of strain targeted.

Of note, it has been suggested that natural heat acclimatization may provide more complete and specific adaptations than artificial heat acclimation (Edholm, 1966). Given the complexity of competitive sports and the influence of various factors (e.g., strategy, terrain, pacing) on performance, heat acclimation in a laboratory may indeed not adequately replicate certain sport-specific responses (Bergeron et al., 2012). Furthermore, in a performance setting, each athlete is required to perform optimally and the adaptations related to heat acclimation might differ between athletes. Racinais et al. (2012) observed large interindividual variations in the responses to a heat acclimatization training camp in high-level soccer players. Some players experienced a reduction in running activity during a match played in the heat (43 °C) after acclimatization, whereas those with the "best acclimatization responses" were able to maintain their running activity, relative to a match played in temperate conditions (21 °C). The authors noted that changes in performance were correlated with changes in hematocrit from pre- to post-acclimatization during a heat-response test. It was suggested that this parameter might be a good indicator of the short-term heat acclimatization responses of team-sport athletes (Racinais et al., 2012, 2014) and potentially useful in estimating acclimation level (Bergeron et al., 2012). In endurance sports, interindividual variation in the magnitude of increase in $\dot{V}O_{2max}$ noted after acclimation may represent a strong indicator of aerobic performance improvement.

Decay

Heat acclimation is transient and gradually disappears if not maintained by continued repeated heat exposure. The heart rate improvement, which develops rapidly during acclimation, is also lost more rapidly than thermoregulatory responses (Williams et al., 1967; Pandolf et al., 1977). However, there is no agreement concerning the rate of decay for heat acclimation. The beneficial effects of the 14 days of heat acclimatization reported in the study from Dresoti (1935) appeared to be maintained for ~1 month. Lind (1964) believed that heat acclimation might be retained for 2 weeks after the last heat exposure and then be rapidly lost over the next 2 weeks. Williams et al. (1967) reported some loss of acclimation in sedentary individuals after 1 week, with the percentage loss being greater with increasing time. By 3 weeks, losses of nearly 100% for heart rate and 50% for core temperature were observed. Conversely, Pandolf et al. (1977) observed greatly attenuated losses in physically trained individuals, indicating that aerobic fitness and regular exercise contribute to retaining the benefits of heat acclimation for a longer period.

More recently, it was shown that 2 weeks after the final exposure to heat, a 35% loss of acclimation occurred in both heart rate and core temperature in individuals exercising for 60 min at 60% $\dot{V}O_{2max}$ in temperate conditions (18 °C; Saat et al., 2005). In contrast, Weller et al. (2007) demonstrated that following a 10-day acclimation (46 °C and 18% RH) regimen, there was no decay in core temperature and minimal decay in heart rate after 12 and 26 days without heat exposure. During this period, however, regular physical activity patterns were maintained. Consequently, heat reacclimation was accomplished in 2 and 4 days, respectively (Weller et al., 2007). A particular feature of this study was the use of controlled hyperthermia (38.5 °C) to elicit heat acclimation, which may have enhanced the benefits of heat acclimation. Notwithstanding, these observations support the notion that aerobic fitness and regular exercise are critical during the decay period in providing stimulus for sustaining adaptation (Pandolf et al., 1977). It has also been suggested that 1 day of exercise in the heat is required for every 5 days spent without heat exposure (Pandolf et al., 1977;

Taylor, 2000), which challenges the notion that 1 day of acclimation is lost for every 2 days spent without exposure to heat stress (Givoni & Goldman, 1972). With short-term heat acclimation, it appears that adaptations persist for 1 week, but not 2 (Garrett et al., 2009). Future research endeavors may wish to examine the minimum intensity, duration, and frequency of exposure required to maintain the benefits of heat acclimation.

Aerobic fitness

Endurance training in temperate climates reduces physiological strain and increases exercise capacity in the heat, as endurance-trained athletes exhibit many of the characteristics of heat-acclimated individuals (Armstrong & Pandolf, 1988). However, while physical training by virtue of the thermoregulatory strain can impart some heat acclimation, the requirement of profuse sweating and warm skin is critical. Trained individuals exercising at the same relative intensity, but at a higher metabolic rate as untrained individuals, experience a higher rate of heat storage (Mora-Rodriguez et al., 2010) and fatigue at a similar (Sawka et al., 1992; Périard et al., 2012a), or higher core temperature (Cheung & McLellan, 1998; Selkirk & McLellan, 2001). Hence, adaptations related to training may allow for greater rates of body heat accumulation before a reduction in work rate occurs, be it voluntary or involuntary (Mora-Rodriguez, 2012). In addition, aerobically fit individuals develop heat acclimation more rapidly than their less fit counterparts, and high aerobic fitness might reduce the susceptibility to heat injury/illness (Gardner et al., 1996). It has been estimated that $\dot{V}O_{2max}$ accounts for approximately 44% of the variability in exercise heat tolerance, and the number of days required for complete development of heat acclimation (Pandolf et al., 1977; Shvartz et al., 1977). However, endurance training alone does not totally replace the benefits of heat acclimation produced by a program of exercise in the heat (Armstrong & Pandolf, 1988).

For endurance training to improve thermoregulatory responses during exercise in the heat, the exercise training sessions must produce a substantial elevation in sweat rate and core temperature (Henane et al., 1977). Interestingly, however, an elevated aerobic capacity does not always improve heat tolerance (Avellini et al., 1982). To achieve improved thermoregulation from endurance training in temperate climates, either strenuous interval training or continuous training at an exercise intensity greater than 50% $\dot{V}O_{2max}$ should be employed. Lesser training intensities produce questionable effects on performance during exercise heat stress (Armstrong & Pandolf, 1988). The endurance training must last at least 1 week (Nadel et al., 1974) and some authors show that the best improvements require 8–12 weeks of training (Armstrong & Pandolf, 1988).

Adaptations and mechanisms of heat acclimation

Acclimation to dry and humid environments

Although heat acclimation in a dry environment confers a substantial advantage in humid heat, the physiological and biophysical differences between dry and humid heat lead one to expect that humid heat acclimation would produce somewhat different physiological adaptations from dry heat acclimation. Early investigations supported this premise, indicating that dry heat acclimation might be better retained than humid heat acclimation (Bean & Eichna, 1943; Henschel et al., 1943; Robinson et al., 1943; Lind & Bass, 1963). Fox et al. (1964) compared the effects of acclimation to dry and to humid heat on the inhibition of sweating using controlled hyperthermia (~38.2 °C core temperature). They showed that most of the improvement in the ability to maintain higher sweat rates in hot-humid conditions following acclimation was associated with a diminution of hidromeiosis. Along with this adaptation, the ability to evaporate sweat at a rate sufficient to achieve thermal balance is imperative.

To achieve a high evaporative cooling rate in a humid environment, it is necessary to overcome the high ambient water vapor pressure by maintaining either a higher vapor pressure at the skin (which requires a higher skin temperature), or a larger wetted skin area, as compared with what would be necessary in a dry environment. Unless core temperature is allowed to rise along with skin temperatures, the higher skin temperature must be achieved by increasing core-to-skin thermal conductance, which requires a higher skin blood flow. Therefore, one expected difference between acclimation to humid heat and acclimation to dry heat is for the former to involve greater circulatory adaptations, to support higher skin blood flow with minimal circulatory strain.

Another difference that might be expected between acclimation to humid heat and dry heat is for humid heat to enable more efficient use of the skin as an evaporating surface. Accordingly, it was proposed that humid heat acclimation allows for a greater portion of sweat production to occur at the level of the limbs (Höfler, 1968; Shvartz et al., 1979; Regan et al., 1996). However, Patterson et al. (2004b) showed that humid heat acclimation does not elicit a preferential sweat redistribution toward the limbs. Nevertheless, they demonstrated interregional variations in the capacity to increase local sweat rate, as the increase in forearm sweat rate (117%) exceeded that at the forehead (47%) and the thigh (42%), and the increase in chest sweat rate (106%) exceeded the thigh (Patterson et al., 2004b). Therefore, while humid heat acclimation does not elicit preferential sweat redistribution toward the limbs, interregional variations in the capacity to increase local sweat rate do occur.

The magnitude of cross-acclimation that exercise combined with either dry or humid heat confers during exercise in either climate has not been fully elucidated.

Studies indicate that some cross-acclimation can occur between humid heat and dry heat exposure. Passive dry heat or passive humid heat acclimation elicited similar core temperatures during exercise in both hot climates (Fox et al., 1967). Exercise dry heat acclimation conferred an advantage (over no heat acclimation) during exercise in humid heat (Bean & Eichna, 1943; Fox et al., 1967) and vice versa (Eichna et al., 1945). In addition, Shapiro et al. (1980) reported that exercise (35% $\dot{V}O_{2max}$) in dry heat elicited equal or greater core temperatures during exercise in humid heat than a matched WBGT (34 °C) dry heat climate. Unfortunately, that study did not report pre-acclimation data and employed only dry heat acclimation. Sawka et al. (1983b) found that exercise (29% $\dot{V}O_{2max}$) in matched hot-dry and hot-humid WBGT climates (32 °C) resulted in the attainment of similar core temperatures both before and after completing the heat acclimation regimen, which consisted of daily alternating dry heat and humid heat exposures.

Griefahn (1997) compared the physiological course of acclimation to humid heat, dry heat, and radiant heat at equivalent WBGT (33 °C) over a 15-day exercise heat acclimation program. An unknown number of subjects participated in one to all three of the heat acclimation regimens, which were interspersed by at least 52 days. These investigators reported that humid heat elicited a more rapid acclimation and less physiological strain (i.e., core temperature, heart rate, and sweating rate) than dry heat exposure. Unfortunately, the experimental design (i.e., lack of matched groups or crossover testing) did not allow cross-acclimation effects to be evaluated, nor did the authors speculate on their findings to that issue.

To our knowledge, no study has directly compared loss of heat acclimation for matched groups after humid heat and dry heat acclimation. Pandolf et al. (1977) acclimated soldiers to dry heat (49 °C, 20%) and studied their loss of acclimation over 3 weeks. They reported that 13% and 4% of the core temperature advantages, and 23% and 29% of the heart rate advantages were lost after 1 and 3 weeks without exposure, respectively. Williams et al. (1967) acclimated African miners to humid heat (35 °C, 80%) and studied their decay over 3 weeks. They reported that 26% and 45% of the core temperature advantages and 65% and 92% of their heart rate advantages were lost after 1 and 3 weeks without exposure to heat stress, respectively. Together, these studies suggest that heat acclimation decay might occur more rapidly for humid than dry heat.

Future areas of research

Heat acclimation research and our understanding of the adaptations related to heat exposure have greatly evolved since the first observations of Lind (1768). However, there remain several avenues of research that require elucidation. These involve the following:

- Developing more athlete-specific heat acclimation regimens based on performing exercise in the heat at high intensities.
- Investigating the differences in adaptations between natural and artificial heat exposure (i.e., acclimatization vs acclimation), as well as the possible interspacing of artificial exposure with natural competition-like conditions for optimizing performance.
- Examining the minimum intensity, duration, and frequency of exposure required to maintain the benefits of heat acclimation.
- Determining whether heat acclimation provides an ability to tolerate greater body temperatures at exhaustion and/or during prolonged intense exercise.
- Evaluating whether heat acclimation regimens that allow for permissive dehydration improve the rapidity and magnitude of heat acclimation adaptations.
- Exploring the mechanism(s) mediating the reduction in blood and muscle lactate accumulation during submaximal exercise, and the increase in power output at lactate threshold following heat acclimation.
- Substantiating the ergogenic benefits of heat acclimation on performance in cool conditions and at altitude, and developing a better understanding of the mechanisms associated with these improvements.
- Developing our understating of heat shock protein responses at the intracellular and extracellular level, as well as the role of epigenetic mechanisms in transcriptional regulation during the development of heat acclimation.
- Investigating whether a controlled intensity regimen – exercising at a given level of cardiovascular strain – elicits a sustained forcing function that allows for optimal adaptation.

Perspectives

Exercise heat acclimation induces physiological adaptations that improve thermoregulation, attenuate physiological strain, reduce the risk of serious heat illness, and improve aerobic performance in warm-hot environments. The adaptations include improved sweating, improved skin blood flow, lowered body temperatures, reduced cardiovascular strain, improved fluid balance, altered metabolism, and enhanced cellular protection. The magnitude of adaptation depends on the intensity, duration, frequency, and total number of heat exposures, as well as the acclimation regimen. Most adaptations to daily heat exposure develop during the first 4 days, with the remainder complete by 3 weeks. The adaptations vary somewhat depending on the exposure to dry or humid heat. Improved sweat secretion is the most critical factor in heat acclimation. Earlier onset and higher sweating rate improves evaporative cooling, which reduces skin temperature and heat storage. The resultant lower skin temperature decreases skin blood flow and

skin venous compliance, shifting blood volume to the central circulation.

While heat acclimation improves aerobic performance in the heat, it may also enhance performance in cool or temperate conditions, and act as a training tool during specific phases of the competitive season (e.g., pre-season, taper). The benefits of short-term heat acclimation in particular have been the focus of numerous studies to determine the effectiveness of such a regimen in the context of elite endurance and team-sports performance. Evidence has emerged that this approach is well suited for individuals and teams with limited time and resources, as it allows for performance improvements related to plasma volume expansion and increased cardiovascular stability. Research examining the role of heat acclimation in potentially improving performance at altitude (i.e., hypoxic conditions) is also emerging; however, well-controlled studies are required to fully elucidate this paradigm.

The use of a controlled hyperthermia regimen, whereby internal and/or external heat load is manipulated, provides a sustained adaptation stimulus and averts the physiological habituation associated with the traditional constant work rate exercise heat acclimation regimen. This approach may thus be better suited to investigate the mechanisms of heat acclimation and induce more complete adaptation. Similarly, utilizing a controlled intensity heat acclimation regimen in which a specific level of cardiovascular strain (% $\dot{V}O_{2max}$ or lactate threshold) is achieved based on maintaining a given heart rate, may also provide a sustained forcing function and allow for maximizing adaptation. Although this approach needs to be thoroughly investigated, it offers greater relevance and ease of implementation to athletes and coaches.

Specificity of exercise and environmental conditions during competition may need to be replicated during acclimation to optimize performance when competing. Thus, additional research is also required to investigate the difference and specificity of adaptation between extended natural and artificial heat exposure (i.e., acclimatization vs acclimation) and the possible interspacing of artificial exposure with natural competition-like conditions. Heat acclimatization may provide more specific adaptations based on direct exposure to the exercise environmental conditions to be encountered, including the exercise task, solar radiation, and terrain/geography.

Key words: Exercise performance, fluid balance, heat acclimatization, thermal tolerance, thermoregulation.

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