Heat Acclimation

8

Sébastien Racinais, Michael Sawka, Hein Daanen, and Julien D. Périard

8.1 Introduction

Acute heat exposure impairs aerobic exercise capacity via thermoregulatory mediated cardiovascular adjustments, hyperthermia-induced skeletal muscle metabolism alterations, and central nervous system perturbations [1]. However, repeated exposures to heat allow for a physiological conditioning known as heat acclimation when exposed to hot rooms, saunas, or baths, and acclimatisation when exposed to naturally hot areas/environments [2]. These heat exposures induce numerous integrated physiological adaptations that improve performance in the heat and reduce the risk of serious heat illness [3]. This chapter covers the adaptations related to heat acclimation, including the increase in sweat rate and plasma volume, which contribute to decrease in heart rate and core temperature during exercise at a given intensity. It also covers the resulting benefit for exercise capacity in the heat, along with the potential implication for exercising in cooler environments. Lastly, the chapter covers the methods and kinetics of induction, as well as the kinetics of decay. The term heat acclimation will be used as a generic term for both acclimation and acclimatisation.

S. Racinais (✉)
Athlete Health and Performance Research Centre, Aspetar, Doha, Qatar
e-mail: sebastien.racinais@aspetar.com

M. Sawka
School of Applied Physiology, Georgia Institute of Technology, Atlanta, GA, USA

H. Daanen
Department of Human Movement Sciences, Faculty of Behavioural and Movement Sciences, Vrije Universiteit, Amsterdam, The Netherlands

J. D. Périard
University of Canberra Research Institute for Sport and Exercise (UCRISE), Bruce, ACT, Australia

© Springer Nature Switzerland AG 2019

J. D. Périard, S. Racinais (eds.), *Heat Stress in Sport and Exercise*,
https://doi.org/10.1007/978-3-319-93515-7_8

8.1.1 Historical Perspectives

In one of the first reports on human heat adaptations in 1768, Lind indicated that when relocating to East and West Indian climates, Europeans were at first affected by hot environments but, by length of time, habituated and lived comfortably [4]. In his book 'An Essay on Diseases Incidental to Europeans in Hot Climates', Lind did not yet employ the terminology 'acclimation', but recommended 'a plan for all newly arrived Europeans'. This included behavioural strategies such a reducing workload, rescheduling work to avoid the heat of the day, and using shelters. Lind also mentioned that there were probably blood adaptations allowing Europeans to 'enjoy a pretty good state of health' once adapted.

Despite the early report from Lind on adaptations to hot environments [4], research pertaining to heat acclimation only gained importance in the early twentieth century, likely in response to requirements related to the industrial revolution. Within this context, the mining industry published some of the early discoveries relating to heat acclimation. For example, it was described in 1922 that trained miners sweated more than new workers in a hot mine (4.5–8.5 L versus 2.5 L per 5 h work shift) [5]. Some mines therefore initiated acclimation programs for new workers as early as 1926 [6]. In the quest to optimise work efficiency and safety in the mining industry, Dreosti [7] performed one of the first scientific acclimation studies. He demonstrated that within 14 days of heat acclimation, heart rate was lowered and sweat rate increased, enhancing work performance during a rock-shovelling test, with the progressive beneficial effects being maintained for ~1 month [8]. Dreosti [7] also reported that resting oral temperature was lower in acclimatised than un-acclimatised workers and that even a single previous exposure was sufficient to attenuate the rise in oral temperature in hot environments. Subsequent observations confirmed that acclimatised mine workers had a lower temperature and heart rate (after work) and an increased sweat loss compared with their un-acclimatised counterparts; a finding that, in association with an improved ability to work, was considered characteristic of heat acclimatisation [9]. It was also noted that the rate of heat acclimatisation seemed relatively rapid, as clear improvements in working capacity occurred in only 3 days [9]. Interestingly, miners originating from hot regions did not appear to have a usually high heat tolerance [10].

8.2 Physiological Adaptations

8.2.1 Sweat Rate

An improved sweat rate response is considered the hallmark indicator of heat acclimation. The increase in sweat rate allows for increased evaporative cooling in environmental conditions with low vapour pressure gradients [11]. Most of the adaptive increase in sweat rate occurs in 3–4 days [11, 12] (Fig. 8.1). In essence, sweating

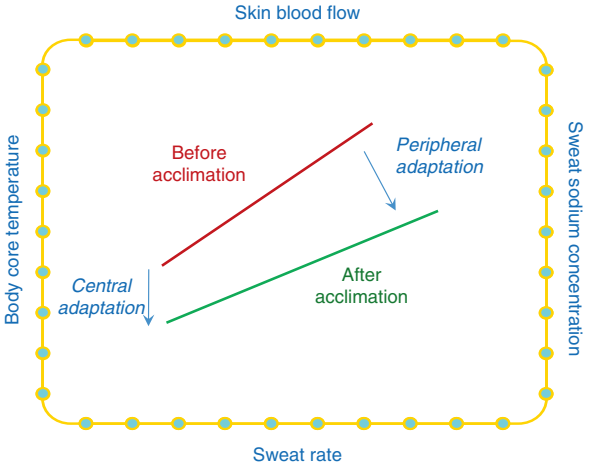

Fig. 8.1 Heat acclimation lowers the core temperate threshold for sweating and increases the rate and sensitivity of response. Heat acclimation decreases also sweat sodium concentration. Based on Periard et al. [3]

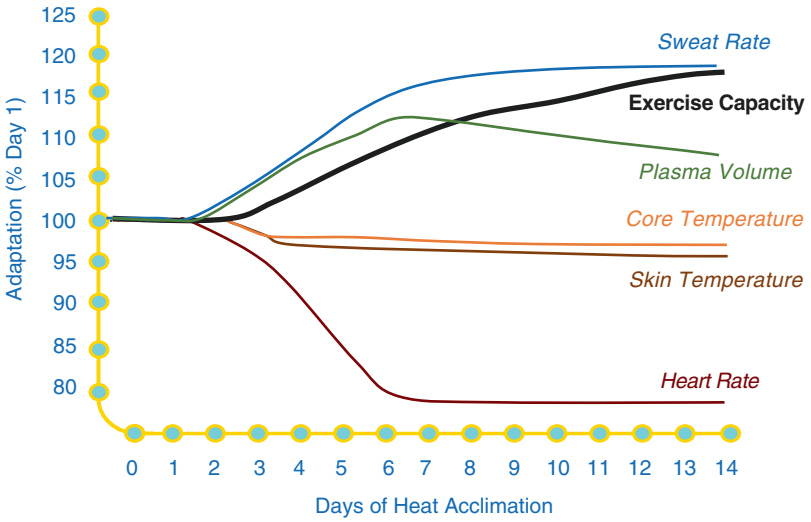

Fig. 8.2 Time course of induction in human adaptations to heat stress. Based on Periard et al. [3]

begins at a lower body temperature and increases to a greater amount for a given body temperature elevation after heat acclimation [13] (Fig. 8.2). In tropical climates, sweat rate is lower and as such less wasteful, allowing for secreted sweat to evaporate and cool the skin [14]. Sweat sodium concentration also decreases with heat acclimation for a given sweating rate [15]. The mechanisms behind the increase in sweat rate and the decrease in sweat sodium concentration with acclimation are reasonably well understood and include neurological and endocrine adaptations [16]. The decrease in sweat sodium concentration can start occurring in 2 days only and is thereafter linear during the first week of heat acclimation [17].

These sweating adaptations are supported by changes in cutaneous vasodilation with an earlier and greater increase in skin blood flow for a given body temperature after heat acclimation [12] (Fig. 8.2).

8.2.2 Blood Volume

Another commonly reported adaptation with heat acclimation is an increase in blood volume, and more specifically plasma volume. Large alterations in blood volume in response to climatic changes have been reported as early as 1922 [18]. In 1940, Bazett et al. [19] published a comprehensive description of the haematological adaptations related to acclimation based on 4 studies. Using various tracers to quantify blood volume, their work showed that heat acclimation induces an increase in both plasma volume and total circulating haemoglobin. However, the plasma volume expansion occurred more rapidly than the increase in haemoglobin, triggering a temporary decrease in haemoglobin concentration and haematocrit [19]. The increase in plasma volume became a standard observation reported in most heat acclimation studies from the 1950s [20], and typically occurs after 3–4 days of heat exposure [21, 22] (Fig. 8.1). Plasma volume expansion is particularly variable between individual with values ranging from 3% to 27% [23–27], whereas erythrocyte volume is considered to remain unchanged [28].

The expansion of plasma volume in later studies was reported to be a transient phenomenon [29, 30], peaking around the fifth day. However, recent studies have suggested that plasma volume could remain expanded if the adaptation stimulus was maintained constant by clamping core temperature during acclimation [24, 25]. Plasma volume maintenance may be facilitated by the oncotic effect of an increase in intravascular protein content [21, 30, 31], potentially due to an increase in protein synthesis [32, 33], a reduction in protein loss through the cutaneous capillaries in response to an acclimation-induced decrease in skin blood flow [34], and a reduction in the permeability of cutaneous capillaries to large molecules [35, 36]. Plasma volume increase and maintenance can also be facilitated by the increase in extracellular fluid due to sodium preservation [24, 25, 37]. Notwithstanding, whether plasma volume remains expanded when the stimulus for adaptation is constant requires further investigation.

Functionally, the expansion in plasma volume may improve vascular filling pressure to support cardiovascular stability (i.e. increased stroke volume and arterial blood pressure) [21], as well as increase the specific heat of blood [38], thus improving heat transfer from the core to the skin [16]. However, an acute artificial increase in plasma volume does not appear to improve thermoregulatory control [22, 39] and, therefore, the importance of plasma volume expansion in heat acclimation is still debated. Even if it is probably not the main physiological mechanism improving exercise capacity in the heat, plasma volume changes might still be a marker of short-term acclimation as the changes in haematocrit during a heat-response test appear to correlate to the changes in physical performance in the heat [40, 41] (Fig. 8.3).

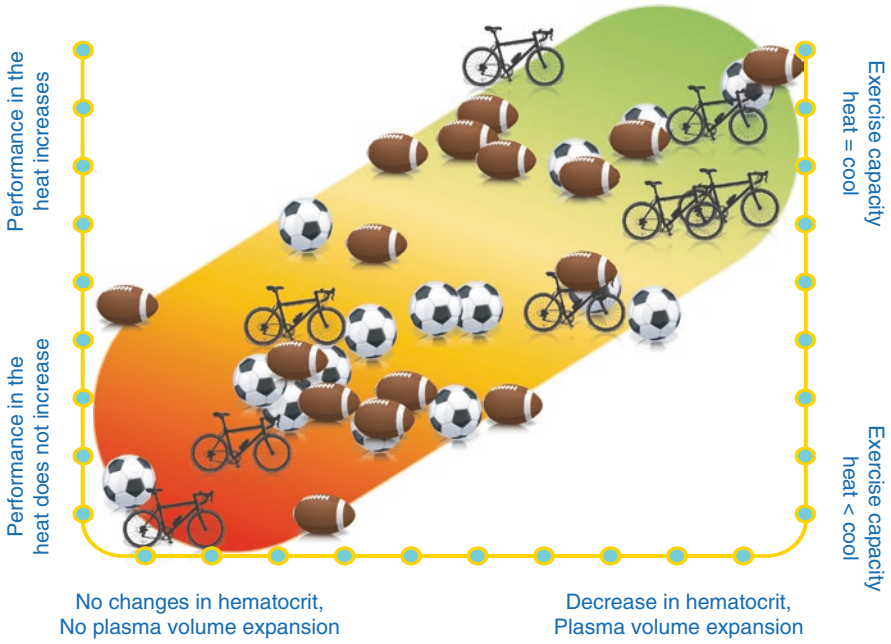

Fig. 8.3 Relationship between haematological changes and changes in exercise capacity in the heat after short-term heat acclimation. Symbols represent individual data from soccer players [40], Australian football players [41], and cyclist [27]

8.2.3 Cardiovascular Adaptations

As detailed in Chap. 3, an increase in core and skin temperatures during exercise in the heat is associated with increased cardiovascular strain [42], with a significant increase in heart rate along with decline in stroke volume, mean arterial pressure, and potentially cardiac output [43]. Some investigators believe that a high heart rate is the primary contributor to reduced stroke volume during prolonged exercise in the heat [44].

The increases in sweat rate and plasma volume support heat dissipation at the level of the skin. As such, it has been well described that heat acclimation improves evaporative cooling and lowers skin temperature more than rectal temperature, therefore increasing the internal thermal gradient and facilitating heat flow to the surface [11, 45]. This ultimately improves heat transfer via blood flow, 'sparing' blood for the rest of the circulation [11] and relieving the circulatory strain [45]. Indeed, an increased plasma volume will reduce heart rate and better sustain stroke volume during exercise in the heat [22], improving the ability to sustain blood pressure and cardiac output requirements (Fig. 8.4). Thus, a decrease in heart rate at a given work rate is a sign of a better sustained stroke volume and perhaps cardiac output during exercise-heat stress. The decrease in heart rate is probably due in-part to the increase in plasma volume supporting greater venous return, cardiac

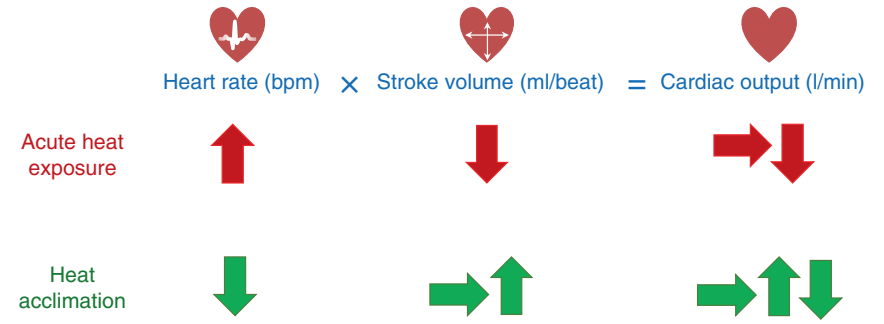

Fig. 8.4 Hyperthermia increases heart rate and decreases stroke volume. A heat acclimation-induced increase in plasma volume may allow to partly compensate for this effect

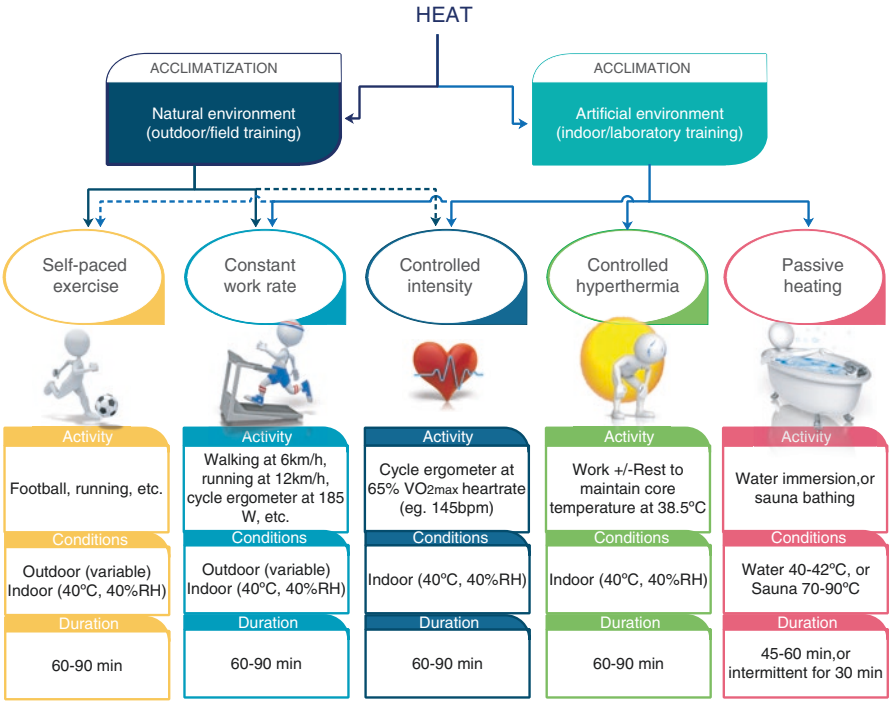

Fig. 8.5 Overview of heat acclimation methods. Based on Daanen et al. [121]

preloading, and improving ventricular filling [23, 46], but alterations in body temperature and sympathetic activity can also contribute. Accordingly, a decrease in heart rate for a given exercise intensity in the heat is considered a hallmark of heat acclimation [26, 47]. It has therefore been proposed to utilise heart rate during heat acclimation to maintain a given level of cardiovascular strain during daily exercise-heat exposures (i.e. controlled heart-rate protocol) [3] (Fig. 8.5).

8.2.4 Thermal Tolerance

In addition to the various sudomotor, vasomotor, haematological, thermoregulatory, and cardiovascular adaptations that contribute to reduce heat strain, repeated heat exposure also improves thermal tolerance. Whilst heat acclimation reduces heat strain, thermal tolerance is a cellular adaptation allowing the cells and organisms to survive a higher level of strain [48–50]. Thermal tolerance largely depends on the heat shock protein (HSP) response [51] with HSP72 being particularly responsive to heat stress and exercise [52]. Briefly, HSP binds to denatured or nascent cellular polypeptides to provide protection and support repair from various stressors including heat stress, fever, hypoxia, ischemia, viral infection, energy depletion, and acidosis [53]. Human studies have highlighted the complexity of the HSP response. For example, heat acclimation has been shown to increase basal levels of intracellular Hsp72 and HSP90, with individuals demonstrating the greatest physiological adaptations exhibiting reduced post-exercise expression (measured ex vivo via water bath incubation) [54]. In contrast, basal levels of extracellular HSP72 decreased (with an increase in the post-exercise response) after a couple of days of heat acclimation [55], but not after ~10 days [56, 57]. A potential increase in basal levels (with a decrease in the post-exercise response) after 15 days of heat acclimation has also been noted [58]. In addition to the HSP response, heat stress also up-regulates or down-regulates numerous gene expressions [59]. Globally, these adaptations allow the heat-acclimated phenotype to be more tolerant to heat as well as to other stressors [60].

Even if heat acclimation is a broader response than thermal tolerance, and it is possible to induce some level of heat acclimation without inducing significant intracellular Hsp70 responses [61], it is noteworthy that HSP responses may participate in both heat acclimation and thermal tolerance in parallel. For example, changes in HSP90 within cutaneous tissues might contribute to improved vasodilatory effects [62, 63].

8.2.5 Muscle Adaptations

Heat acclimation has been purported to act at the muscle level, possibly altering whole-body [64] and skeletal muscle metabolism [65, 66]. For example, the basal metabolic rate of young men in Asia has been reported to decrease during the warmer months of the year [67]. Heat acclimation has also been suggested to decrease oxygen uptake [68] and muscle glycogen utilisation (40–50%) [69, 70] during submaximal exercise in the heat, albeit the glycogen-sparing effect of heat acclimation may be small [65]. Heat acclimation may also reduce blood and muscle lactate accumulation during submaximal exercise [66], and increase power output at lactate threshold [71]. This could be linked to improved lactate removal due to the increase in total body water increasing splanchnic circulation [72], and to delayed lactate accumulation due to an increase in cardiac output and a decrease in metabolic rate [65, 68]. Heat acclimation may also improve muscle aerobic

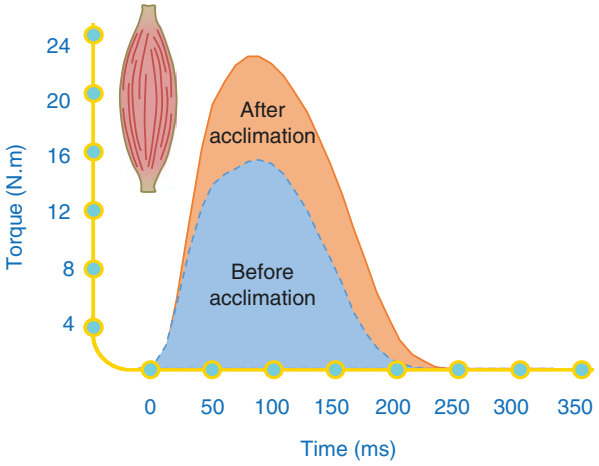

Fig. 8.6 Repeated heat exposure increases peak twitch amplitude in human plantar flexors. Based on data from Racinais et al. [124]

metabolism through mitochondrial adaptations [73] and muscle capillary growth [74]. Lastly, heat acclimation has been shown to facilitate hypertrophy in cultured [75, 76] or animals [77] muscle cells, and thus increase muscle cross-sectional area in humans [78] (Fig. 8.6).

8.2.6 Neural Adaptations

Acute hyperthermia has been shown to decrease neural drive to the muscle [79]. As detailed in Chap. 4, this impairment is due to peripheral failures in neural drive transmission and supraspinal alterations when contractions are prolonged [80]. In vitro studies have suggested that heat acclimation could reverse the peripheral failure in neural drive transmission induced by hyperthermia [81, 82]. However, in humans, the peripheral failures in neural drive transmission (estimated through M-wave and H-reflex evoked potentials) induced by hyperthermia are not reverted by heat acclimation [83]. Importantly, this suggests that the decrease in neural drive transmission in humans might not be linked to synaptic failure, but rather be a side effect of the increase in axonal conduction velocity which shortens depolarisation time [84, 85]. Conversely, heat acclimation partly restores the ability to sustain neural drive during prolonged contractions [83] (refer to the Chap. 4, Fig. 4.4). As this recovery is not accompanied by any spinal or peripheral nervous system adjustments, it suggests a supraspinal adaptation to heat acclimation [83]. The benefit of this adaptation for whole-body exercise capacity remains unclear however, as the limiting factor for prolonged exercise in the heat is more cardiovascular than neural [86, 87].

8.2.7 Perceptual and Cognitive Adaptations

Acute hyperthermia increases thermal discomfort as well as the negative/positive affects ratio [83], potentially affecting exercise capacity [88] and cognitive function [89, 90] in the heat. Heat acclimation may not affect thermal comfort per se at rest [83, 91], but it can improve the ratings of thermal sensation/comfort during training and competition in the heat for both team-sports and endurance athletes [91–93]. It is however unclear to which extend this improvement participates in the physical performance improvement associated with acclimation. Heat acclimation can also protect various cognitive tasks such as psychomotor performance [94], attention tasks [95] and planning task [96] from the deleterious effects of hyperthermia.

8.3 Exercise Capacity and Physical Performance

8.3.1 In Hot Environments

From the initial studies in the mining industry, heat acclimation demonstrated an improved work capacity under heat stress conditions [97]. This improvement has been consistently verified using either natural (acclimatisation) or artificial (acclimation) heat exposures, with a recent meta-analysis calculating an average performance improvement of 15% [91].

The decrease in VO_{2max} associated with the development of hyperthermia is likely one of the main limiting factors for prolonged exercise performance in hot ambient conditions [98]. Heat acclimation can improve VO_{2max} in temperate and hot ambient conditions; however, it cannot fully restore the initial decrease in VO_{2max} imposed by heat stress [71, 99].

The increase in VO_{2max} with acclimation is associated with a proportional improvement in exercise performance in the heat (e.g. cycling time trial) [71]. In some circumstances, heat acclimation may be sufficient to restore a similar level of performance to that observed in cooler environments [100, 101]. For example, power output during an outdoor cycling time trial (43 km) in 37 °C was ~16% lower compared to a time trial undertaken in 8 °C, with half of this decrease restored after 1 week of heat acclimation, and the decrease almost fully compensated for after 2 weeks of heat exposure [100] (Fig. 8.7). The magnitude to which heat acclimation can improve performance in the heat depends on several factors, including the severity of the environmental conditions (e.g. ambient temperature, relative humidity, wind velocity) and the type of performance (e.g. duration, intensity). Notwithstanding, heat acclimation consistently improves submaximal intensity exercise capacity in the heat.

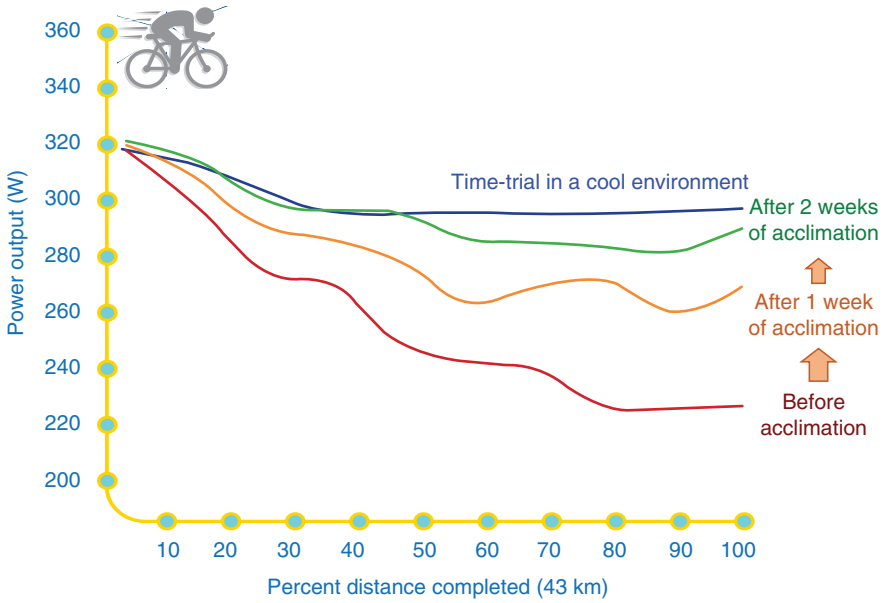

Fig. 8.7 Power output during a 43 km cycling time trial in cool ambient conditions (blue line) and in hot ambient conditions on the first (red line), the 6th (orange line), and the 14th (green line) days of heat exposure. Based on data from Racinais et al. [100]

8.3.2 In Temperate Environments

Whilst it is well accepted that heat acclimation improves both VO_{2max} and exercise capacity in the heat, there is conflicting evidence regarding its effect on exercise capacity in cooler environments. On one hand, several studies did not observe any improvement in VO_{2max} in cool environment following 5 days of isothermic heat acclimation with permissive dehydration [102], 10 days of constant intensity heat acclimation [101], or 14 days of heat acclimatisation [103]. On the other hand, other studies have reported an increase in VO_{2max} in cool conditions following heat acclimation in unfit (23%) or untrained (13%) individuals [104], but also in trained athletes (3–5%) [71, 99]. Whether heat acclimation improves VO_{2max} in cool environments therefore remains a topic of contention.

In addition to potential improvements in VO_{2max}, decreases in glycogen utilisation [65, 69, 70] and blood and muscle lactate accumulation [66] may also improve exercise capacity in cool environments following heat acclimation. The mechanisms for these adaptations are unclear, but could include an increase in total body water facilitating lactate removal through increased splanchnic circulation [72], and/or an increase in cardiac output and decrease in metabolic rate delaying lactate accumulation [65, 105]. This can translate in an increased power output at lactate threshold [71, 102], VO_{2max} [71, 99], and performance [71]. For example, heat

acclimation (via sauna bathing) improves run time to exhaustion in competitive runners with the improvement being correlated to the increase in plasma volume and total blood volume [106].

Several studies have also reported that heat acclimation increased fitness level in team-sports athlete following pre- and in-season training camps in the heat [41, 107, 108]. This is particularly interesting as obtaining rapid fitness improvements without performing specific aerobic training sessions may save time for technical training [41, 107, 108]. In summary, even if the benefits of heat acclimation in cooler environment are debated, there is no evidence to indicate that heat acclimation impairs performance in cooler environments. As such, heat acclimation should be implemented before any event with potentially hot, even if uncertain conditions.

8.4 Heat Acclimation and Decay

8.4.1 Kinetics of Adaptation

The kinetics of heat acclimation adaptation and decay were one of the primary interests of the occupational studies stemming from the mining industry and military [109, 110]. Robinson and his collaborators [111] reported in 1943 that daily walking in a hot room for 1–1.5 h decreased heart rate, skin temperature, and rectal temperature, with the decrement occurring rapidly in the first 7 days of acclimation, and then more slowly up to 23 days. As early as 1951, Ladell [12] suggested that heat acclimation occurred in two phases with an initial decrease in heart rate and temperature threshold for sweating (within 2–3 days), followed by an increase in sweat rate at a given temperature and a better resistance to fatigue. Heat acclimation is now considered as a relatively rapid process that begins from the first day of exposure, with 75–80% of the adaptations occurring in the first 4–7 days [3, 112] (Fig. 8.1).

Importantly, the kinetics of adaptation are based on daily heat exposure. Indeed, 10 days of daily heat exposure was reported to induce similar adaptations than 1 exposure every 3 day, but in the third of the time [113]. In addition, even if 1 exposure every 3 days allows for some adaptations, interspacing the exposure by 1 week does not allow for adaptations to develop [114]. Thus, if and when allowing for rest days during a pre-competition taper to avoid fatigue and hypohydration [115], it must be accounted for that intermittent heat exposure allows for lesser adaptations compared to daily exposure [116].

Of note, the constraints of the competition calendar in international sports have led recent research to focus on short-term heat acclimation [115, 117, 118]. However, even if athletes benefit from only few days of acclimation [92, 115, 119], they may require 7–10 days to achieve near complete cardiovascular and sudomotor adaptations [23, 27, 71], and two or more weeks to optimise aerobic performance in hot environments [100].

8.4.2 Heat Acclimation Induction

A classic research from 1963 originally suggested that 100 min of daily exercise in the heat was suitable to induce heat acclimation [120]. The panel of induction methods has diversified over the years and includes active, passive, or a mixture of active and passive hyperthermia [121] (Fig. 8.5). The rule of thumb to induce heat acclimation is to increase whole-body temperature, induce profuse sweating, and elevate skin blood flow [122]. This can be achieved through: (1) passive heat exposure, (2) self-paced exercise, (3) constant work rate exercise, (4) controlled hyperthermia, or (5) controlled heart-rate approaches (Fig. 8.5).

Passive exposure has been used in scientific studies to isolate the effect of repeated heat exposure without training [83, 123, 124]. However, heat acclimation before competing in the heat should be as specific as possible and as such, passive exposure is likely not as efficient as training in the heat. Notwithstanding, repeated passive heat exposures allow to partially develop a heat-acclimated phenotype [125, 126] and may be practical for athletes living in cold countries. Passive exposure may also include maintaining an elevated temperature post-training [106, 127], or even increasing temperature before active acclimation [128].

Self-paced exercise in the heat has been used for decades [129, 130], and includes working or training in natural heat. Self-paced heat acclimation has been largely replaced by more controlled methods (see below) in the recent years, but is still the primary method for team-sports athletes acclimatising through their natural outdoor training [40, 41, 107]. For endurance sports, such as cycling, choosing to naturally acclimatise through self-paced exercise, intensity blocks should be prioritised towards the beginning of the session to maintain absolute exercise intensity.

Acclimation via constant work rate exercise has been the primary model from which much of our knowledge on acclimation kinetics (see above) is based [13, 23, 111, 131]. In this model, there are fixed endogenous and exogenous thermal loads (e.g. cycling at 160 W in 35 °C, 60% RH), representing a constant forcing function. However, the relative stress imposed by of those fixed load will progressively diminish as acclimation develops [11, 132–134]. This relative decrease has been suggested as one of the reasons for the decline in plasma volume following its initial increase [24, 25]. It is important to note however, that whilst the training stimulus or forcing function may decrease as adaptations develop, a recent meta-analysis concluded that the constant work rate acclimation leads to similar adaptations than clamping core temperature to maintain the forcing function (described below) [91].

Acclimation by isothermic controlled hyperthermia (i.e. clamping core temperature at 38.5 °C) has been proposed to provide a constant forcing function and thus maintain the acclimation stimulus as adaptations develop [24, 117, 135, 136]. This concept has recently seen a regain in interest, but it is not new and was proposed half a century ago [132, 135]. In general, isothermic protocols may not allow appropriate training for athletes as the exercise intensity dramatically decreases when the target core temperature is reached, with exercise completely ceasing in some circumstances [137], thus reducing one of the main drivers for adaptation (i.e. sweating).

Training in the heat at a given heart rate has therefore been suggested as a more suitable method for athletes [3]. Heart rate represents a global index of the cardiovascular stress imposed by the thermal stress and exercise (see above), and decreases rapidly with acclimation (Fig. 8.1). Thus training at a clamped heart rate accounts for the acute effect of heat stress via power output adjustments, but then allows for maintaining a given level of cardiovascular strain as acclimation develops and power output increases at the pre-determined heart rate [3].

8.4.3 Individual Responses

There are large inter-individual differences in heat acclimation responses [40, 138], with some athletes acclimating in 1 week and others requiring 2 weeks or longer [121]. Although heat acclimation improves exercise capacity in the heat in a variety of populations including mine workers, military personnel, and athletes, an individualised strategy may be required in elite athletes to truly optimise performance. For example, when analysing a soccer game in dry heat after 1 week of heat acclimation, some football players can have the same physical activity than they had 1 week before in temperate environment, whereas other players from the same team have to dramatically reduce their physical activity [40]. Anecdotally, although the changes in performance following short-term acclimation may correlate to the changes in haematocrit during a heat-response test (Fig. 8.3), there is currently no test in temperate environment or resting blood measures allowing to quantify heat acclimation [27, 40, 41].

8.4.4 Decay and Re-acclimation

Early studies on miner usually working in hot ambient conditions [110, 139] or on soldier undergoing 12 days of acclimation [140] suggest that most adaptations are lost within 6–7 days without heat exposure. However, Robinson et al. [111] reported that 3 of 5 participants could still benefit from the adaptations induced by 23 days of heat acclimation 2–3 weeks after the end of the procedure, whereas two other participants lost most of their adaptations. This support the early study from Dreosti [7] suggesting that some heat acclimation benefits could be maintained for up to 1 month after 14 days of heat acclimation. Although those early reports are informative, there is limited research available on the kinetics of heat acclimation decay. A recent systematic review and meta-analyses reported that despite athletes maintaining some adaptations for several weeks after acclimation, the magnitude of those adaptations decreased daily by ~2.5% for heart rate and body core temperature during exercise [121]. Sweat rate showed a fast decay, but too little data was available to draw firm conclusions. Noteworthy is that the rate of decay may likely be slowed by training and regular heat exposure post-acclimation. Importantly, re-acclimation during this period appears to be faster than the initial rate of acclimation

[121]. Thus, 4–5 days of re-acclimation could be enough to regain complete heat acclimation if undertaken within 1 month [141].

8.5 Conclusion

Repeated exercise-heat exposures induce heat acclimation, characterised by a series of physiological adaptations improving thermoregulation and reducing physiological strain. These adaptations include an increase in sweat rate, a decrease in heart rate, and a decrease in core temperatures at a given exercise intensity. To obtain these adaptions, it is necessary to increase both whole-body temperature (i.e. core, skin, muscle, and tissue temperature), as well as stimulate profuse sweating. The adaptations improve exercise capacity in warm-hot environments and their transfer to temperate environments remains debated. Most adaptations can be obtained within a few days of daily heat exposure, but exercise capacity in the heat may improve optimally after ~2 weeks.

References

1. Nybo L, Rasmussen P, Sawka MN. Performance in the heat-physiological factors of importance for hyperthermia-induced fatigue. Compr Physiol. 2014;4(2):657–89.
2. Armstrong L, Maresh C. The induction and decay of heat acclimatisation in trained athletes. Sports Med. 1991;12(5):302.
3. Périard JD, Racinais S, Sawka MN. Adaptations and mechanisms of human heat acclimation: applications for competitive athletes and sports. Scand J Med Sci Sports. 2015;25:20–38.
4. Lind J. An essay on diseases incidental to Europeans in hot climates, with the method of preventing their fatal consequences. Printed by William Duane; 1768.
5. Kuno Y. Human perspiration. Springfield: Thomas; 1956.
6. Pogue TE. The evolution of research collaboration in South African gold mining: 1886–1933. Doctoral thesis. Universiteit Maastricht. 2006 June 29; p. 1–219.
7. Dreosti AO. The results of some investigations into the medical aspects of deep mining on the Witwatersrand. J Chem Metal Mining Soc S Afr. 1935;6:102–29.
8. Buskirk ER. The temperature regulatory system. Exercise physiology - people and ideas. Oxford: Oxford University Press; 2003. p. 423–51.
9. Weiner JS. Observations on the working ability of Bantu mineworkers with reference to acclimatization to hot humid conditions. Br J Ind Med. 1950;7(1):17–26.
10. Ladell WS. Physiological observations on men working in supposedly limiting environments in a West African gold-mine. Br J Ind Med. 1955;12(2):111–25.
11. Eichna LW, Park CR, Nelson N, Horvath SM, Palmes ED. Thermal regulation during acclimatization in a hot, dry (desert type) environment. Am J Physiol. 1950;163(3):585–97.
12. Ladell WS. Assessment of group acclimatization to heat and humidity. J Physiol. 1951;115(3):296–312.
13. Nadel ER, Pandolf KB, Roberts MF, Stolwijk JA. Mechanisms of thermal acclimation to exercise and heat. J Appl Physiol. 1974;37(4):515–20.
14. Gonzalez RR, Pandolf KB, Gagge AP. Heat acclimation and decline in sweating during humidity transients. J Appl Physiol. 1974;36(4):419–25.
15. Allan JR, Wilson CG. Influence of acclimatization on sweat sodium concentration. J Appl Physiol. 1971;30(5):708–12.

16. Sawka MN, Leon LR, Montain SJ, Sonna LA. Integrated physiological mechanisms of exercise performance, adaptation, and maladaptation to heat stress. Compr Physiol. 2011;1(4):1883–928.
17. Buono MJ, Kolding M, Leslie E, Moreno D, Norwood S, Ordille A, et al. Heat acclimation causes a linear decrease in sweat sodium ion concentration. J Therm Biol. 2018;71:237–40.
18. Barcroft J, Meakins JC, Davies HW, Duncan Scott JM, Fetter WJ. Observations upon the effect of high altitude on the physiological processes of the human body. On the relation of external temperature to blood volume. Phil Tr Roy Soc. 1922;211:455–64.
19. Bazett HC, Sunderman FW, Doupe J, Scott JC. Climatic effects on the volume and composition of blood in man. Am J Physiol-Leg Content. 1940;129(1):69–83.
20. Glaser EM. Acclimatization to heat and cold. J Physiol. 1950;110(3–4):330–7.
21. Senay LC, Mitchell D, Wyndham CH. Acclimatization in a hot, humid environment: body fluid adjustments. J Appl Physiol. 1976;40(5):786–96.
22. Sawka MN, Coyle EF. Influence of body water and blood volume on thermoregulation and exercise performance in the heat. Exerc Sport Sci Rev. 1999;27:167–218.
23. Nielsen B, Hales JR, Strange S, Christensen NJ, Warberg J, Saltin B. Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. J Physiol. 1993;460:467–85.
24. Patterson MJ, Stocks JM, Taylor NAS. Sustained and generalized extracellular fluid expansion following heat acclimation. J Physiol. 2004;559(Pt 1):327–34.
25. Patterson MJ, Stocks JM, Taylor NAS. Whole-body fluid distribution in humans during dehydration and recovery, before and after humid-heat acclimation induced using controlled hyperthermia. Acta Physiol (Oxford). 2014;210(4):899–912.
26. Périard JD, Travers GJS, Racinais S, Sawka MN. Cardiovascular adaptations supporting human exercise-heat acclimation. Auton Neurosci. 2016;196:52–62.
27. Karlsen A, Nybo L, Norgaard SJ, Jensen MV, Bonne T, Racinais S. Time course of natural heat acclimatization in well-trained cyclists during a 2-week training camp in the heat. Scand J Med Sci Sports. 2015;25:240–9.
28. Sawka MN, Convertino VA, Eichner ER, Schnieder SM, Young AJ. Blood volume: importance and adaptations to exercise training, environmental stresses, and trauma/sickness. Med Sci Sports Exerc. 2000;32(2):332–48.
29. Wyndham CH, Williams CG, Morrison JF, Heyns AJ, Siebert J. Tolerance of very hot humid environments by highly acclimatized Bantu at rest. Br J Ind Med. 1968;25(1):22–39.
30. Senay LC. Effects of exercise in the heat on body fluid distribution. Med Sci Sports. 1979;11(1):42–8.
31. Harrison MH, Edwards RJ, Graveney MJ, Cochrane LA, Davies JA. Blood volume and plasma protein responses to heat acclimatization in humans. J Appl Physiol. 1981;50(3):597–604.
32. Horowitz M, Adler JH. Plasma volume regulation during heat stress: albumin synthesis vs capillary permeability. A comparison between desert and non-desert species. Comp Biochem Physiol A Comp Physiol. 1983;75(1):105–10.
33. Yang RC, Mack GW, Wolfe RR, Nadel ER. Albumin synthesis after intense intermittent exercise in human subjects. J Appl Physiol. 1998;84(2):584–92.
34. Harrison MH. Effects on thermal stress and exercise on blood volume in humans. Physiol Rev. 1985;65(1):149–209.
35. Senay LC. Movement of water, protein and crystalloids between vascular and extra-vascular compartments in heat-exposed men during dehydration and following limited relief of dehydration. J Physiol. 1970;210(3):617–35.
36. Senay LC. Changes in plasma volume and protein content during exposures of working men to various temperatures before and after acclimatization to heat: separation of the roles of cutaneous and skeletal muscle circulation. J Physiol. 1972;224(1):61–81.
37. Mack GW, Nadel ER. Body fluid balance during heat stress in humans. In: Fregly MJ, Blatteis CM, editors. Handbook of physiology environmental physiology. New York: Oxford University Press; 1996. p. 187–214.

38. Blake AS, Petley GW, Deakin CD. Effects of changes in packed cell volume on the specific heat capacity of blood: implications for studies measuring heat exchange in extracorporeal circuits. Br J Anaesth. 2000;84(1):28–32.
39. Watt MJ, Garnham AP, Febbraio MA, Hargreaves M. Effect of acute plasma volume expansion on thermoregulation and exercise performance in the heat. Med Sci Sports Exerc. 2000;32(5):958–62.
40. Racinais S, Mohr M, Buchheit M, Voss SC, Gaoua N, Grantham J, et al. Individual responses to short-term heat acclimatisation as predictors of football performance in a hot, dry environment. Br J Sports Med. 2012;46(11):810–5.
41. Racinais S, Buchheit M, Bilsborough J, Bourdon PC, Cordy J, Coutts AJ. Physiological and performance responses to a training camp in the heat in professional Australian football players. Int J Sports Physiol Perform. 2014;9(4):598–603.
42. González-Alonso J, Crandall CG, Johnson JM. The cardiovascular challenge of exercising in the heat. J Physiol. 2008;586(1):45–53.
43. Périard JD, Cramer MN, Chapman PG, Caillaud C, Thompson MW. Cardiovascular strain impairs prolonged self-paced exercise in the heat. Exp Physiol. 2011;96(2):134–44. Available from: http://ep.physoc.org/content/96/2/134.long.
44. Coyle EF, González-Alonso J. Cardiovascular drift during prolonged exercise: new perspectives. Exerc Sport Sci Rev. 2001;29(2):88–92.
45. MacDonald DKC, Wyndham CH. Heat transfer in man. J Appl Physiol. 1950;3(6):342–64.
46. Senay LC. An inquiry into the role of cardiac filling pressure in acclimatization to heat. Yale J Biol Med. 1986;59(3):247–56.
47. Sundstroem ES. The physiological effects of tropical climate. Physiol Rev. 1927;7:320–62.
48. Moseley PL. Heat shock proteins and heat adaptation of the whole organism. J Appl Physiol. 1997;83(5):1413–7.
49. Horowitz M. Do cellular heat acclimation responses modulate central thermoregulatory activity? News Physiol Sci. 1998;13:218–25.
50. Maloyan A, Palmon A, Horowitz M. Heat acclimation increases the basal HSP72 level and alters its production dynamics during heat stress. Am J Phys Regul Integr Comp Phys. 1999;276(5 Pt 2):R1506–15.
51. Kuennen MR, Gillum TL, Dokladny K, Bedrick EJ, Schneider SM, Moseley PL. Thermotolerance and heat acclimation may share a common mechanism in humans. Am J Phys Regul Integr Comp Phys. 2011;301(2):R524–33.
52. Locke M. The cellular stress response to exercise: role of stress proteins. Exerc Sport Sci Rev. 1997;25:105–36.
53. Kregel KC. Heat shock proteins: modifying factors in physiological stress responses and acquired thermotolerance. J Appl Physiol. 2002;92(5):2177–86.
54. Mcclung JP, Hasday JD, He J-R, Montain SJ, Cheuvront SN, Sawka MN, et al. Exercise-heat acclimation in humans alters baseline levels and ex vivo heat inducibility of HSP72 and HSP90 in peripheral blood mononuclear cells. Am J Phys Regul Integr Comp Phys. 2008;294(1):R185–91.
55. Marshall HC, Ferguson RA, Nimmo MA. Human resting extracellular heat shock protein 72 concentration decreases during the initial adaptation to exercise in a hot, humid environment. Cell Stress Chaperones. 2006;11(2):129–34.
56. Yamada PM, Amorim FT, Moseley P, Robergs R, Schneider SM. Effect of heat acclimation on heat shock protein 72 and interleukin-10 in humans. J Appl Physiol. 2007;103(4):1196–204.
57. de Castro Magalhães F, Amorim FT, Passos RLF, Fonseca MA, Oliveira KPM, Lima MRM, et al. Heat and exercise acclimation increases intracellular levels of Hsp72 and inhibits exercise-induced increase in intracellular and plasma Hsp72 in humans. Cell Stress Chaperones. 2010;15(6):885–95.
58. Sandström ME, Siegler JC, Lovell RJ, Madden LA, Mcnaughton L. The effect of 15 consecutive days of heat-exercise acclimation on heat shock protein 70. Cell Stress Chaperones. 2008;13(2):169–75.

59. Sonna LA, Sawka MN, Lilly CM. Exertional heat illness and human gene expression. Prog Brain Res. 2007;162:321–46.
60. Horowitz M. Genomics and proteomics of heat acclimation. Front Biosci (Schol Ed). 2010;2:1068–80.
61. Hom LL, Lee EC-H, Apicella JM, Wallace SD, Emmanuel H, Klau JF, et al. Eleven days of moderate exercise and heat exposure induces acclimation without significant HSP70 and apoptosis responses of lymphocytes in college-aged males. Cell Stress Chaperones. 2012;17(1):29–39.
62. Fujii N, Zhang SY, McNeely BD, Nishiyasu T, Kenny GP. Heat shock protein 90 contributes to cutaneous vasodilation through activating nitric oxide synthase in young male adults exercising in the heat. J Appl Physiol. 2017;123:844–50.
63. Carter HH, Green DJ. HSP90: an unappreciated mediator of cutaneous vascular adaptation? J Appl Physiol. 2018;124:521.
64. Sawka MN, Wenger CB, Pandolf KB. Thermoregulatory responses to acute exercise-heat stress and heat acclimation. In: Fregly MJ, Blatteis CM, editors. Handbook of Physiology Environmental Physiology. New York: Oxford University Press; 1996. p. 157–85.
65. Young AJ, Sawka MN, Levine L, Cadarette BS, Pandolf KB. Skeletal muscle metabolism during exercise is influenced by heat acclimation. J Appl Physiol. 1985;59(6):1929–35.
66. Febbraio MA, Snow RJ, Hargreaves M, Stathis CG, Martin IK, Carey MF. Muscle metabolism during exercise and heat stress in trained men: effect of acclimation. J Appl Physiol. 1994;76(2):589–97.
67. Hori S. Adaptation to heat. Jpn J Physiol. 1995;45(6):921–46.
68. Sawka MN, Pandolf KB, Avellini BA, Shapiro Y. Does heat acclimation lower the rate of metabolism elicited by muscular exercise? Aviat Space Environ Med. 1983;54(1):27–31.
69. King DS, Costill DL, Fink WJ, Hargreaves M, Fielding RA. Muscle metabolism during exercise in the heat in unacclimatized and acclimatized humans. J Appl Physiol. 1985;59(5):1350–4.
70. Kirwan JP, Costill DL, Kuipers H, Burrell MJ, Fink WJ, Kovaleski JE, et al. Substrate utilization in leg muscle of men after heat acclimation. J Appl Physiol. 1987;63(1):31–5.
71. Lorenzo S, Halliwill JR, Sawka MN, Minson CT. Heat acclimation improves exercise performance. J Appl Physiol. 2010;109(4):1140–7.
72. Rowell LB, Brengelmann GL, Blackmon JR, Twiss RD, Kusumi F. Splanchnic blood flow and metabolism in heat-stressed man. J Appl Physiol. 1968;24(4):475–84.
73. Tamura Y, Matsunaga Y, Masuda H, Takahashi Y, Takahashi Y, Terada S, et al. Postexercise whole body heat stress additively enhances endurance training-induced mitochondrial adaptations in mouse skeletal muscle. Am J Physiol Regul Integr Comp Physiol. 2014;307(7):R931–43. Available from: http://eutils.ncbi.nlm.nih.gov/entrez/eutils/elink.fcgi?dbfrom=pubmed&id=25080501&retmode=ref&cmd=prlinks.
74. Kuhlenhoelter AM, Kim K, Neff D, Nie Y, Blaize AN, Wong BJ, et al. Heat therapy promotes the expression of angiogenic regulators in human skeletal muscle. Am J Physiol Regul Integr Comp Physiol. 2016;311(2):R377–91. Available from: http://eutils.ncbi.nlm.nih.gov/entrez/eutils/elink.fcgi?dbfrom=pubmed&id=27357800&retmode=ref&cmd=prlinks.
75. Yamashita-Goto K, Ohira Y, Okuyama R, Sugiyama H, Honda M, Sugiura T, et al. Heat stress facilitates stretch-induced hypertrophy of cultured muscle cells. J Gravit Physiol [Internet]. 2002;9(1):P145–6. Available from: http://eutils.ncbi.nlm.nih.gov/entrez/eutils/elink.fcgi?dbfrom=pubmed&id=15002522&retmode=ref&cmd=prlinks.
76. Goto K, Okuyama R, Sugiyama H, Honda M, Kobayashi T, Uehara K, et al. Effects of heat stress and mechanical stretch on protein expression in cultured skeletal muscle cells. Pflugers Arch. 2003;447:247–53.
77. Uehara K, Goto K, Kobayashi T, Kojima A, Akema T, Sugiura T, et al. Heat-stress enhances proliferative potential in rat soleus muscle. Jpn J Physiol [Internet]. 2004;54(3):263–71. Available from: http://eutils.ncbi.nlm.nih.gov/entrez/eutils/elink.fcgi?dbfrom=pubmed&id=15541204&retmode=ref&cmd=prlinks.

78. Goto K, Oda H, Kondo H, Igaki M, Suzuki A, Tsuchiya S, et al. Responses of muscle mass, strength and gene transcripts to long-term heat stress in healthy human subjects. Eur J Appl Physiol. 2011;111:17–27.
79. Nybo L, Nielsen B. Hyperthermia and central fatigue during prolonged exercise in humans. J Appl Physiol. 2001;91(3):1055–60.
80. Racinais S, Gaoua N, Grantham J. Hyperthermia impairs short-term memory and peripheral motor drive transmission. J Physiol. 2008;586(Pt 19):4751–62.
81. Kelty JD, Noseworthy PA, Feder ME, Robertson RM, Ramirez J-M. Thermal preconditioning and heat-shock protein 72 preserve synaptic transmission during thermal stress. J Neurosci. 2002;22(1):RC193.
82. Karunanithi S, Barclay JW, Brown IR, Robertson RM, Atwood HL. Enhancement of presynaptic performance in transgenic Drosophila overexpressing heat shock protein Hsp70. Synapse. 2002;44(1):8–14.
83. Racinais S, Wilson MG, Gaoua N, Périard JD. Heat acclimation has a protective effect on the central but not peripheral nervous system. J Appl Physiol. 2017;123(4):816–24.
84. Bolton CF, Sawa GM, Carter K. The effects of temperature on human compound action potentials. J Neurol Neurosurg Psychiatry. 1981;44(5):407–13.
85. Rutkove SB, Kothari MJ, Shefner JM. Nerve, muscle, and neuromuscular junction electrophysiology at high temperature. Muscle Nerve. 1997;20(4):431–6.
86. Racinais S, Girard O. Neuromuscular failure is unlikely to explain the early exercise cessation in hot ambient conditions. Psychophysiology. 2012;49(6):853–65.
87. Périard JD, Caillaud C, Thompson MW. Central and peripheral fatigue during passive and exercise-induced hyperthermia. Med Sci Sports Exerc. 2011;43(9):1657–65.
88. Stevens CJ, Mauger AR, Hassmèn P, Taylor L. Endurance performance is influenced by perceptions of pain and temperature: theory, applications and safety considerations. Sports Med. 2018;48(3):525–37.
89. Marks LE, Gonzalez RR. Skin temperature modifies the pleasantness of thermal stimuli. Nature. 1974;247(5441):473–5.
90. Gaoua N, Grantham J, Racinais S, Massioui El F. Sensory displeasure reduces complex cognitive performance in the heat. J Environ Psychol. 2012;32:158–63.
91. Tyler CJ, Reeve T, Hodges GJ, Cheung SS. The effects of heat adaptation on physiology, perception and exercise performance in the heat: a meta-analysis. Sports Med. 2016;46(11):1699–724.
92. Sunderland C, Morris JG, Nevill ME. A heat acclimation protocol for team sports. Br J Sports Med. 2008;42(5):327–33.
93. Kelly M, Gastin PB, Dwyer DB, Sostaric S, Snow RJ. Short duration heat acclimation in Australian football players. J Sports Sci Med. 2016;15:118–25.
94. Walker SM, Dawson B, Ackland TR. Performance enhancement in rally car drivers via heat acclimation and race simulation. Comp Biochem Physiol Part A Mol Integr Physiol. 2001;128(4):701–7.
95. Radakovic SS, Maric J, Surbatovic M, Radjen S, Stefanova E, Stankovic N, et al. Effects of acclimation on cognitive performance in soldiers during exertional heat stress. Mil Med. 2007;172(2):133–6.
96. Racinais S, Wilson MG, Gaoua N, Periard J. Heat acclimation has a protective effect on the central but not peripheral nervous system. J Appl Physiol. 2017;123(4):816–24.
97. Dreosti AO. The results of some investigations into the medical aspect of deep mining on the Witwatersrand. J Chem Metal Mining Soc S Afr. 1935;36:102–29.
98. Périard JD, Racinais S. Self-paced exercise in hot and cool conditions is associated with the maintenance of %VO2peak within a narrow range. J Appl Physiol [Internet]. 2015;118(10):1258–65. Available from: http://eutils.ncbi.nlm.nih.gov/entrez/eutils/elink.fcgi?dbfrom=pubmed&id=25814635&retmode=ref&cmd=prlinks.
99. Sawka MN, Young AJ, Cadarette BS, Levine L, Pandolf KB. Influence of heat stress and acclimation on maximal aerobic power. Eur J Appl Physiol Occup Physiol. 1985;53(4):294–8.

100. Racinais S, Périard JD, Karlsen A, Nybo L. Effect of heat and heat acclimatization on cycling time trial performance and pacing. Med Sci Sports Exerc. 2015;47(3):601–6.
101. Keiser S, Flück D, Hüppin F, Stravs A, Hilty MP, Lundby C. Heat training increases exercise capacity in hot but not in temperate conditions: a mechanistic counter-balanced cross-over study. Am J Physiol Heart Circ Physiol. 2015;309(5):H750–61.
102. Neal RA, Corbett J, Massey HC, Tipton MJ. Effect of short-term heat acclimation with permissive dehydration on thermoregulation and temperate exercise performance. Scand J Med Sci Sports. 2015;26(8):875–84.
103. Karlsen A, Racinais S, Jensen MV, Norgaard SJ, Bonne T, Nybo L. Heat acclimatization does not improve VO2max or cycling performance in a cool climate in trained cyclists. Scand J Med Sci Sports. 2015;25:269–76.
104. Shvartz E, Shapiro Y, Magazanik A, Meroz A, Birnfeld H, Mechtinger A, et al. Heat acclimation, physical fitness, and responses to exercise in temperate and hot environments. J Appl Physiol. 1977;43(4):678–83.
105. Sawka MN, Toner MM, Francesconi RP, Pandolf KB. Hypohydration and exercise: effects of heat acclimation, gender, and environment. J Appl Physiol. 1983;55(4):1147–53.
106. Scoon GSM, Hopkins WG, Mayhew S, Cotter JD. Effect of post-exercise sauna bathing on the endurance performance of competitive male runners. J Sci Med Sport. 2007;10(4):259–62.
107. Buchheit M, Voss SC, Nybo L, Mohr M, Racinais S. Physiological and performance adaptations to an in-season soccer camp in the heat: associations with heart rate and heart rate variability. Scand J Med Sci Sports. 2011;21(6):e477–85.
108. Buchheit M, Racinais S, Bilsborough J, Hocking J, Mendez-Villanueva A, Bourdon PC, et al. Adding heat to the live-high train-low altitude model: a practical insight from professional football. Br J Sports Med. 2013;47(Suppl 1):i59–69.
109. Henschel A, Taylor HL, Keys A. The persistence of heat acclimatization in man. Am J Phys. 1943;140:321–5.
110. Wyndham CH, Jacobs GE. Loss of acclimatization after six days of work in cool conditions on the surface of a mine. J Appl Physiol. 1957;11(2):197–8.
111. Robinson S, Turrell ES, Belding HS, Horvath SM. Rapid acclimatization to work in hot climates. Am J Phys. 1943;140:168–76.
112. Shapiro Y, Moran D, Epstein Y. Acclimatization strategies--preparing for exercise in the heat. Int J Sports Med. 1998;19(Suppl 2):S161–3.
113. Fein JT, Haymes EM, Buskirk ER. Effects of daily and intermittent exposures on heat acclimation of women. Int J Biometeorol. 1975;19(1):41–52.
114. Barnett A, Maughan RJ. Response of unacclimatized males to repeated weekly bouts of exercise in the heat. Br J Sports Med. 1993;27(1):39–44.
115. Chalmers S, Esterman A, Eston R, Bowering KJ, Norton K. Short-term heat acclimation training improves physical performance: a systematic review, and exploration of physiological adaptations and application for team sports. Sports Med. 2014;44(7):971–88.
116. Gill N, Sleivert G. Effect of daily versus intermittent exposure on heat acclimation. Aviat Space Environ Med. 2001;72(4):385–90.
117. Garrett AT, Goosens NG, Rehrer NJ, Rehrer NG, Patterson MJ, Cotter JD. Induction and decay of short-term heat acclimation. Eur J Appl Physiol. 2009;107(6):659–70.
118. Garrett AT, Creasy R, Rehrer NJ, Patterson MJ, Cotter JD. Effectiveness of short-term heat acclimation for highly trained athletes. Eur J Appl Physiol [Internet]. 2012;112(5):1827–37. Available from: http://eutils.ncbi.nlm.nih.gov/entrez/eutils/elink.fcgi?dbfrom=pubmed&id=21915701&retmode=ref&cmd=prlinks.
119. Garrett AT, Rehrer NJ, Patterson MJ. Induction and decay of short-term heat acclimation in moderately and highly trained athletes. Sports Med. 2011;41(9):757–71.
120. Lind AR, Bass DE. Optimal exposure time for development of acclimatization to heat. Fed Proc. 1963;22:704–8.
121. Daanen HAM, Racinais S, Périard JD. Heat acclimation decay and re-induction: a systematic review and meta-analysis. Sports Med. 2018;48(2):409–30.

122. Racinais S, Alonso JM, Coutts AJ, Flouris AD, Girard O, González-Alonso J, et al. Consensus recommendations on training and competing in the heat. Scand J Med Sci Sports. 2015;25:6–19.
123. Brazaitis M, Skurvydas A. Heat acclimation does not reduce the impact of hyperthermia on central fatigue. Eur J Appl Physiol. 2010;109(4):771–8.
124. Racinais S, Wilson MG, Periard J. Passive heat acclimation improves skeletal muscle contractility in humans. Am J Phys Regul Integr Comp Phys. 2017;312:R101–7.
125. Takamata A, Yoshida T, Nishida N, Morimoto T. Relationship of osmotic inhibition in thermoregulatory responses and sweat sodium concentration in humans. Am J Phys Regul Integr Comp Phys. 2001;280(3):R623–9.
126. Beaudin AE, Clegg ME, Walsh ML, White MD. Adaptation of exercise ventilation during an actively-induced hyperthermia following passive heat acclimation. Am J Phys Regul Integr Comp Phys. 2009;297(3):R605–14.
127. Zurawlew MJ, Walsh NP, Fortes MB, Potter C. Post-exercise hot water immersion induces heat acclimation and improves endurance exercise performance in the heat. Scand J Med Sci Sports [Internet]. 2016;26(7):745–54. Available from: http://eutils.ncbi.nlm.nih.gov/entrez/eutils/elink.fcgi?dbfrom=pubmed&id=26661992&retmode=ref&cmd=prlinks.
128. Mee JA, Peters S, Doust JH, Maxwell NS. Sauna exposure immediately prior to short-term heat acclimation accelerates phenotypic adaptation in females. J Sci Med Sport. 2018;21(2):190–5.
129. Nelms JD, Turk J. A self-regulating method for rapid acclimatization to heat. J Physiol. 1972;221(1):2P–3P.
130. Armstrong LE, Hubbard RW, DeLuca JP, Christensen EL. Self-paced heat acclimation procedures. Natick: U.S. Army Research Institute of Environmental Medicine; 1986. p. 1–36.
131. Nielsen B, Strange S, Christensen NJ, Warberg J, Saltin B. Acute and adaptive responses in humans to exercise in a warm, humid environment. Pflugers Arch. 1997;434(1):49–56.
132. Fox RH, Goldsmith R, KIDD DJ, LEWIS HE. Acclimatization to heat in man by controlled elevation of body temperature. J Physiol. 1963;166:530–47.
133. Rowell LB, Kraning KK, Kennedy JW, Evans TO. Central circulatory responses to work in dry heat before and after acclimatization. J Appl Physiol. 1967;22(3):509–18.
134. Taylor NAS. Principles and practices of heat adaptation. J Hum-Environ Syst. 2000;4(1):11–22.
135. Fox RH, Goldsmith R, Hampton IF, Hunt TJ. Heat acclimatization by controlled hyperthermia in hot-dry and hot-wet climates. J Appl Physiol. 1967;22(1):39–46.
136. Regan JM, Macfarlane DJ, Taylor NA. An evaluation of the role of skin temperature during heat adaptation. Acta Physiol Scand. 1996;158(4):365–75.
137. Gibson OR, Mee JA, Taylor L, Tuttle JA, Watt PW, Maxwell NS. Isothermic and fixed-intensity heat acclimation methods elicit equal increases in Hsp72 mRNA. Scand J Med Sci Sports. 2015;25:259–68.
138. Corbett J, Rendell RA, Massey HC, Costello JT, Tipton MJ. Inter-individual variation in the adaptive response to heat acclimation. J Therm Biol. 2018;74:1–35.
139. Williams CG, Wyndham CH, Morrison JF. Rate of loss of acclimatization in summer and winter. J Appl Physiol. 1967;22(1):21–6.
140. Adam JM, Fox RH, Grimby G. Acclimatization to heat and its rate of decay in man. J Physiol J Physiol. 1960;152:26–7.
141. Ashley CD, Ferron J, Bernard TE. loss of heat acclimation and time to re-establish acclimation. J Occup Environ Hyg. 2015;12(5):302–8.