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COMPREHENSIVE REVIEW

Sports and environmental temperature: From warming-up to heating-up

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ABSTRACT

Most professional and recreational athletes perform pre-conditioning exercises, often collectively termed a 'warm-up' to prepare for a competitive task. The main objective of warming-up is to induce both temperature and non-temperature related responses to optimize performance. These responses include increasing muscle temperature, initiating metabolic and circulatory adjustments, and preparing psychologically for the upcoming task. However, warming-up in hot and/or humid ambient conditions increases thermal and circulatory strain. As a result, this may precipitate neuromuscular and cardiovascular impairments limiting endurance capacity. Preparations for competing in the heat should include an acclimatization regimen. Athletes should also consider cooling interventions to curtail heat gain during the warm-up and minimize dehydration. Indeed, although it forms an important part of the pre-competition preparation in all environmental conditions, the rise in whole-body temperature should be limited in hot environments. This review provides recommendations on how to build an effective warm-up following a 3 stage RAMP model (Raise, Activate and Mobilize, Potentiate), including general and context specific exercises, along with dynamic flexibility work. In addition, this review provides suggestion to manipulate the warm-up to suit the demands of competition in hot environments, along with other strategies to avoid heating-up.

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Introduction

Body temperature has always been considered an indicator of health status.[1] However, only during the 20th century has thermoregulation of the healthy and active human become a major area of research. This interest was mainly driven by the requirements of the mining industry and the military. Lavoisier (1743–1794) showed that humans generated heat by a combustion process resulting in the production of carbon dioxide. Claude Bernard (1813–1878) then showed that the blood entering the lungs was warmer than that exiting, and that venous blood was warmer than arterial blood in several other organs, suggesting that these tissues were the site of heat production. Muscle heat production and dissipation during exercise and their role in increasing core temperature (T_{core}) was described in detail during the 1960s.[2] Since then, several researchers and practitioners have discussed the potential benefit for performance of increasing muscle temperature (T_m) through warming-up,[3,4] or the potential impairment in performance and increased risk to athlete health of increasing T_{core}.[5] Interestingly however, this dichotomy is generally considered independently.

From the minimal heat production associated with basal metabolic rate, heat production dramatically increases at the onset of a muscle contraction, doubling over the first minutes of intense dynamic exercise.[6] During the first 45 s, heat production mainly induces a large increase in T_m,[6] thereafter driving an increase in T_{core}.[7] Although exact absolute values of T_m are dependent of the depth of measurement, muscle measured, environmental conditions, and intensity of contraction, T_m is ~35°C at rest and will exceed T_{core} within 3–5 min of exercise, remaining 0.65–0.95°C higher than T_{core}.[7] As detailed in the first section of this manuscript, the initial increase in T_m has several benefits for athletic performance. Although the benefits of increasing T_m *in-vivo* might not be as important as originally estimated *in-vitro*, warming-up also offers non temperature-dependent effects.

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Yet, depending on both exercise intensity and climatic conditions, the compensability of the environment will vary.[8] Maintaining thermal allostasis requires transferring metabolic heat from the core to the skin and then on to the environment. This involves an increase in cutaneous circulation[9] and sweating.[10] However, the increase in sweat rate necessary for heat dissipation can lead to progressive dehydration if fluid losses are not offset by adequate fluid consumption.[11-13] Progressive dehydration precipitates a cascade of events including a decrease in plasma volume and an increase in plasma osmolality,[5] a decrease in sweat rate and evaporative heat loss[14] and a decrease in cardiac filling.[15] As detailed in the second section of this manuscript, the blood flow redistribution and other thermoregulatory demands of exercising in hot and/or humid environments represents a significant stress to the cardiovascular system[16] limiting performance,[17] as maintaining a similar relative intensity requires the reduction of absolute intensity (i.e. work load).[18] Moreover, as detailed below, hyperthermia may also affect various levels of the neural system.

Lastly, the final sections of this manuscript will present practical sporting applications for (pre)competition in the heat. Despite a long history of research on thermoregulation, the incidence of heat stroke has increased over the past decades and heat is responsible for more deaths than all other natural disasters combined.[19] This manuscript will present the current state of the literature to inform creation of a warm-up specific to the requirements of competition. This will be achieved by extracting the principles of a 3-stage systematic approach (raise, activate and mobilise, potentiate; RAMP). Also, it will be discussed how to amend the athlete's warm-up to account for environmental conditions and reduce the risk of encountering heat illness. While the most important countermeasure to protect the health and performance of athletes in hot and/or humid environments is to heat acclimatize,[20] there are other countermeasures that can be implemented during the warm-up and throughout exercise that contribute to minimize the increase in T_{core}, all the while maintaining optimal locomotor T_m and promoting non-thermal warm-up benefits.

The physiology of warming-up

An increase in body temperatures is not necessarily detrimental to the health and performance of an athlete. For example, most professional and recreational athletes perform a pre-conditioning exercise called a 'warm-up' to increase their T_m and prepare for a competitive task. The term warm-up is an appropriate representation of Asmussen & Bøje's[21] seminal work investigating the effects of body temperature on performance. They found that both active (muscular work) and passive (hot water immersion or radio-diathermy) heating improved high-intensity performance, ranging from <15 s to approx. 4–5 min.[21] Most of the ergogenic aspects of warming-up seem temperature-dependent, with both increased T_m and T_{core} potentially resulting in improved muscle force and power production,[22] improved muscle blood flow,[23,24] augmented muscle glycogen and carbohydrate (CHO) utilization,[25] accelerated oxyhaemoglobin dissociation,[26] increased metabolic rate and enzymatic reactions,[27] and changes in both mechanical efficiency and muscle fiber conduction velocities.[27,28] However, some authors have also highlighted the metabolic importance of warming-up and suggested that it should be called 'acid-up'.[29] Others have highlighted the limited scientific evidence for such a standard pre-activity.[3] Notwithstanding, most if not all athletes perform a warm-up to enhance performance and reduce the risk of injury. The current section will present the physiological adjustments associated with warming-up, both temperature or non-temperature related. The practical recommendations to construct a warm up are presented in a later section of this review (see *Application to sports – warming up for performance*). These recommendations include various stages (raise, activate and mobilise, potentiate; RAMP) and account for the environmental conditions.

Muscle adjustments

As mentioned above, muscle contractions produce heat. This heat production will increase T_m within seconds, before any visible changes in T_{core}. While muscle contractions produce heat, muscle contractility itself is also affected by temperature and thus warming-up.

Effect of increasing temperature through warming-up on muscle function

Muscle temperature effects contraction velocity.[30] While a decrease in T_m can slow-down chemical reactions,[31] delay the cross-bridge cycle[32] and decrease actomyosin sensibility to calcium[33]; an increase in T_m increases the rate of force development of a muscle twitch,[34,35] probably in relation to an increase in

myosin adenosinetriphosphatase (ATPase) activity[36] and calcium sequestration by the sarcoplasmic reticulum.[37] In addition, maximum tetanic force can also be improved by increasing T_m,[34,38,39] possibly by improving contractile protein binding.[39] Of note, slow muscles (eg. soleus) seem to be more sensitive to temperature than fast muscles (e.g. extensor digitorum longus), especially at lower temperatures (i.e., 20°C to 10°C).[35,40] However, despite the inverse relationship between T_m and time to peak twitch tension and half relaxation time *in vitro*,[38] the effect of temperature is less marked toward physiological temperatures *in vivo*.[41] Indeed, an increase in T_m across the standard range experienced *in vivo* (i.e., from 37 to 43°C) has not been observed to modify the absolute force of the muscle fiber.[42] As a consequence, an increase in T_m does not necessary modify peak twitch amplitude in human skeletal muscle (Fig. 1).[43-45] Consequently, increasing T_m through warming-up might have less benefit for muscle contractility *in vivo* than suggested by *in vitro* studies, especially in hot environments.

However, *in situ* muscle responses cannot be limited to the muscle fiber as they also involve non-contractile tissue (e.g., ligaments). Indeed, increasing temperature decreases the viscous resistance of muscle and joints.[46,47] In addition, increasing temperature will affect the muscle environment by increasing local vasodilatation[48] and increasing both nerve and sarcolemmal action potentials.[49] Lastly, as detailed in the next paragraph, warming-up also has non-thermal benefits on the muscle.

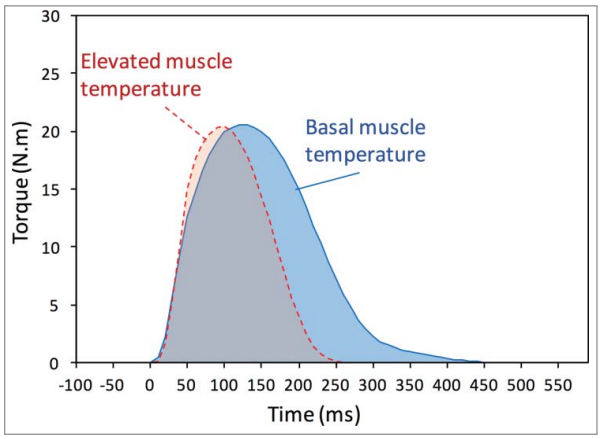

Figure 1. Muscle twitch at resting basal (plain line, blue) and elevated (dashed line, red) muscle temperature. An increase in muscle temperature generally increases the rate of force development and relaxation without modifying the peak tension. Data extracted with permission from Racinais et al.[45]

Non-thermal effects of warming-up on skeletal muscle

Independently of the increase in T_m, a warm-up can potentially increase performance by 'pre-conditioning' the muscle. This phenomena called post-activation potentiation (PAP) is generally obtain by performing a maximal or near maximal contraction,[50,51] and has been suggested to have additional benefits relative to a traditional warm-up (i.e., without such contraction) to improve performance in explosive activities.[52] The first purported mechanism explaining PAP is a phosphorylation of the myosin regulatory light chains.[50,53] However, this phosphorylation is not consistently observed in humans.[54] The second mechanism proposed is an increase in spinal synaptic transmission that could last for several minutes following a contraction.[52] Synaptic transmission can be indirectly assessed using H-reflex, a monosynaptic spinal reflex representing an electrically evoked variant of the stretch reflex. While homosynaptic post-activation depression initially decreases the H-reflex during the first minute following a contraction, independently of T_m,[55] the H-reflex can thereafter be potentiated for up to 10 min following maximal contractions.[52,56] However, the efficacy of PAP on real-world athletic performance may still remain inconclusive, when considering the trade-off between, or even coexistence of both fatigue and potentiation actions on the muscle.[51,57] Indeed, neural drive following a maximal contraction is a balance between PAP and fatigue,[51] with the latter likely being the dominant factor.[58] On a separate note, muscles develop actin-myosin cross-bridges at rest to maintain muscle tone and posture.[59] These cross-bridges increase muscle rigidity, but muscle contractions during warm-up breakdown those cross-bridges,[60] potentially increasing the rate of force development and power. However, muscle rigidity re-increases rapidly once the warm-up is completed.[61]

Neural drive adjustments

In addition to influencing muscle function, temperature and warming-up can affect the neural drive reaching the muscle.

Effect of increasing temperature through warming-up on neural drive transmission

It has consistently been demonstrated that cold exposure reduces nerve[62] and muscle[63] conduction velocity. In addition, cold exposure can increase antagonist

muscle co-contractions, likely as a protective mechanism for cold muscles and joints.[64] Shivering can also affect agonist and antagonist (co-)activation.[65] A warm-up increasing body temperatures will therefore counteract these effects and increase performance in cold environments, especially during fast movements.[30]

However, the benefits of increasing temperature on neural drive decrease as temperature increases, and are overlaid by other negative factors in hot environmental conditions. Indeed, the amplitude of electrically evoked M-waves and H-reflexes decreases at high temperatures, suggesting an alteration in peripheral neural drive transmission.[66] This decrease is likely due to a reduction in the opening time of the voltage-gated sodium channels, leading in turn to a decrease in the amplitude, duration and area of the axon potential.[67] This is in line with a negative correlation between skin temperature (T_{skin}) and the amplitude, duration, area and latency of a compound action potential,[68] with a Q_{10} effect on nerve conduction velocity between 1.1 and 2.4.[69] As a consequence, there is likely no benefit for neural drive transmission of increasing T_m to elevated levels. Even if defining an optimal temperature is contentious, as it depends on several intrinsic and extrinsic factors, it is generally accepted that a T_m slightly above resting should be aimed for when warming-up in temperate environments.

Non-thermal effects of warming-up on neural drive generation

Warming-up has psychological effects potentially improving mental preparedness. For example, warming-up provides the athlete a dedicated time to focus on the event[70] and practice mental imagery,[71] thus offering important psychological effects, beside peripheral thermal and metabolic changes.

Cardiovascular and metabolic adjustments

Effect of increasing temperature through warming-up on substrate provision and utilization

Cold exposure can increase muscle glycolysis and lactate accumulation suggesting a lower muscle efficiency and/or an effect of a lower perfusion in cold muscle.[72] Conversely, the increase in local vasodilation due to a temperature increase might benefit substrate delivery and metabolite removal.[48] This suggests that warming-up may have metabolic advantages, at least in cold conditions. Moreover, increasing temperature improves oxygen release from hemoglobin[73] and myoglobin.[74] However, increasing T_m increases ATP utilization with an increase in creatine phosphate degradation and anaerobic glycolysis.[75] This might be viewed as a positive pre-conditioning adjustment for short explosive activities, but might have negative consequences for prolonged exercise as this represents an increase in energy demand.

Non-thermal cardiovascular effects of warming-up

Abnormal electrocardiographic (ECG) responses suggestive of cardiac ischemia have been reported in 70% of participants running at a high intensity for 10–15 s without warming-up.[76,77] This very high prevalence was observed in asymptomatic males (age 21 to 52 y old) having otherwise normal ECG at heart rates of 170 bpm or higher when the load was progressively increased.[76,77] This phenomenon is likely due to an incapacity of the coronary blood flow to adapt fast enough. However, this abnormal ECG response is minimized or even suppressed by warming-up.

Injury prevention

Warming-up is commonly considered as one of the basic tools to reduce injury risk.[3] For example, several studies have reported a decrease in injury prevalence in professional athletes after incorporating preventive physiotherapy programs including supervised warm-ups.[78,79] However, the specific role of the warm-up in these studies cannot be discerned as it was accompanied by other interventions such as taping, recovery, rehabilitation or core stability exercises.[78,79]

Effect of increasing temperature through warming-up on injury prevention

In cold environments, there is an increase in antagonist/agonist muscle co-activation slowing movement velocity.[64] This slowing impairs performance but could also act as a protective mechanism for muscle injury.[31,64] A warm-up increasing body temperatures will counteract these effects and therefore increase performance in cold environments, especially during fast movements.[30] In addition, warming-up has also been reported to increase flexibility in winter sports performed in cold environments such as downhill skiing.[80] Indeed, increasing temperature increases extensibility in tendon[81] and other connective tissues.[82] As

such, increasing peripheral tissue temperature with a warm-up could reduce injury risk.[3,81,82]

Other injury-prevention effects of warming-up

Animal studies have shown that preconditioning the muscle by electrically evoked contractions increased its stretch length and the force required to tear the fibers.[83] While a part of this effect might be related to the minor (i.e., 1°C) rise in T_m reported by the authors, this study showed that preconditioning contractions reduced injury-risk.[83] An active warm-up also often includes stretching activities.[84] Stretching during warm-up was traditionally passive but it has shifted toward more dynamic exercises in the last decades.[84] While there is not enough data available to conclude on the effect of including stretching in the warm-up routine on injury prevention, some authors have suggested that this practice might reduce injury risk.[3,84] Indeed, both passively stretching or electrically contracting a muscle decreases the passive tension of the muscle-tendon unit.[85] Moreover, warming-up has been reported to reduce the magnitude of delayed onset muscle soreness (DOMS) 48 h after exercise involving a high eccentric component.[86]

The physiology of heating-up

A rise in whole-body temperature and in particular T_m enhances explosive skeletal muscle performance (e.g., sprinting and jumping) by improving metabolic and contractile function, nerve conduction, and conformational changes associated with muscle contraction.[30,87,88] Conversely, the development of thermal strain, an elevation in T_{core}, T_{skin} and T_m, is associated with increased fatigue development during sustained maximal voluntary isometric contractions (MVCs)[66,89-91] and impairs aerobic performance.[92,93-96] Moreover, while warming-up has health benefit to prevent cardiac ischemia,[76,77] and reduce the risk of injury,[79] heating-up carries a health risk. Indeed, the development of hyperthermia increases the risk of exertional heat illness (EHI), which is a serious health hazard.

Neuromuscular function

In contrast to the beneficial effects of warming-up, the development of whole-body hyperthermia impairs neuromuscular function with alterations occurring at both the central and peripheral level. From a central perspective, elevated heat stress can lead to a reduction in voluntary muscle activation and the loss of force production capacity.[66,89,97] At the skeletal muscle level, a rise in temperature (e.g., 2–3°C) increases contractile speed (i.e., twitch contraction and relaxation time decrease) and alters the force/frequency relationship.[44,98]

Central activation failure

Brück & Olschewski[99] were among the first to postulate that heat stress might affect brain function and influence exercise performance. The authors identified 3 factors of discomfort that might counteract motivation and progressively reduce the drive to exercise during hyperthermia. They suggested that the interaction of circulatory, thermal, and muscular discomfort determined endurance time and work rate. Nielsen et al.[100,101] subsequently proposed that hyperthermia *per se*, rather than circulatory failure, was the critical factor causing exhaustion during exercise under heat stress. It was further purported that the attainment of a high T_{core} (39.2–39.7°C) might influence the CNS by reducing mental drive (i.e., motivation) for motor performance.[100,101] Interestingly however, the capacity to generate force during a brief (3–5 s) MVC of the knee extensors and elbow flexors was unaltered after exhaustive cycling under heat stress.[101] This capacity to maintain force production was also preserved during 40 repeated MVCs.[89] Notwithstanding, force production was progressively impaired during a sustained (120 s) MVC relative to a contraction performed after exercise in cool conditions. The impairment was attributed to a hyperthermia-induced reduction in voluntary activation.[89] Others have also reported that the progressive increase in T_{core} via passive heating is paralleled by a decrease in voluntary activation and force production during 3–5 s MVCs.[97,102] The authors further suggested that T_{core} was the primary thermal input mediating hyperthermia-induced fatigue, as rapid cooling of the skin and muscle failed to improve voluntary drive, despite large reductions in cardiovascular and psychophysical strain. Taken together, these observations in isolated muscle contractions led to the suggestion that reaching a core temperature of ~40°C may not only influence performance in an MVC due to the development of central fatigue, by may also account for the hyperthermia-induced fatigue that develops during prolonged dynamic exercise in the heat.[89,103] However, the development of fatigue during exercise in the heat is multi-

factorial and associated with the interaction of several physiologic and psychological processes, and not caused by one single factor.[43,93,104] As such, use of the term "critical core temperature" in a reductionist manner is misleading when characterizing the influence of hyperthermia on the development of fatigue during exercise under heat stress.[105]

Indeed, the development of exercise-induced hyperthermia has been shown to impact on performance at both the central and peripheral levels. In a recent study, Lloyd et al.[106] examined the impact of a range of T_m (22 to 38.5°C) on voluntary drive during brief (3 s) and sustained (120 s) MVCs. The authors reported that with T_{core} remaining relatively stable, voluntary activation and hence force production, was inversely related to T_m during sustained contractions, but unaffected during brief MVCs. The observed reduction in voluntary drive was assigned to altered peripheral fatigue rates and/or sensitivity to peripheral fatigue occurring during the sustained contraction. It was estimated that a ratio of 5.5 to 1 represented the impact of T_{core} and T_m on voluntary muscle activation, respectively.[106] Périard et al.[107] have also demonstrated that voluntary activation and force production are similarly reduced during a 20 s MVC following self-paced exercise in hot and cool conditions. The authors reported that the post-exercise decline in voluntary activation accounted for ~20% of the decrease in total force production. It was thus suggested that the 0.8°C higher T_{core} (39.0 vs. 39.8°C) at time trial completion in the heat did not exacerbate central fatigue and that the loss of force was mainly of peripheral origin and a consequence of the prolonged contractile activity associated with exercise.[107] It has also been shown that a similar increase in T_{core} (39.5–39.8°C) either via active or passive heating elicits a similar reduction in voluntary activation during a sustained (45 s) MVC, but that a faster rate of decline in force production capacity occurs following exercise.[90] Together, these studies indicate that the loss of force production capacity originates from both central and peripheral fatigue factors, with the combination of heat stress and prior contractile activity (i.e., exercise) exacerbating the rate of decline.

To separate the effect of hyperthermia from the effect of exercise, several studies have used a passive hyperthermia approach. In the absence of exercise, passive hyperthermia has been shown to affect the peripheral nervous system, but also to induce a

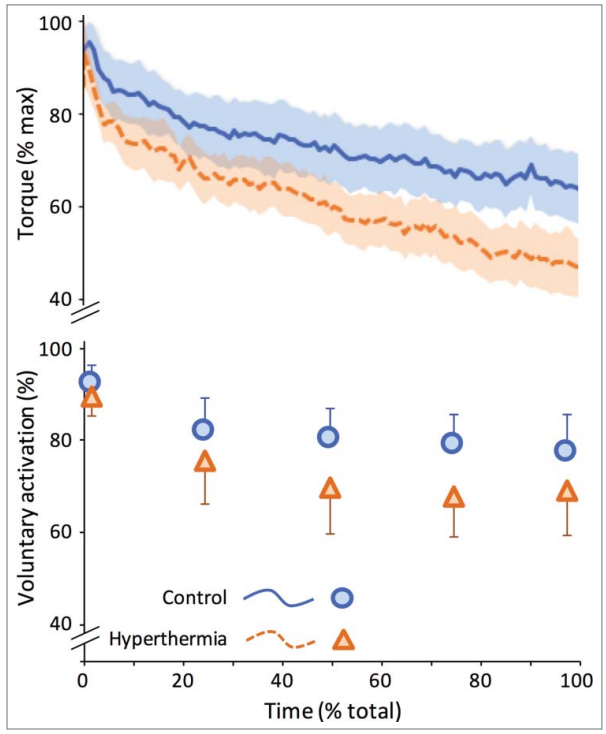

Figure 2. Hyperthermia reduces the ability to sustain force/torque production (upper panel) and neural drive to the muscle (lower panel) during a 120-s maximal voluntary contraction. Data represent mean and 95% confidence-intervals from 14 participants in control or hyperthermic (rectal temperature 39°C) state. Reproduced with permission from Racinais et al.[111]

supraspinal failure when a contraction is prolonged.[66] Consequently, hyperthermia reduces voluntary activation[66,97,102] (Fig. 2). To identify the mechanism(s) and localize the site of failure in voluntary activation during hyperthermia, Todd et al.[108] induced passive hyperthermia (38.5°C) and evaluated brief (2–3 s) and sustained (2 min) MVC performance in the elbow flexors with transcranial magnetic stimulation (TMS). They observed that hyperthermia induced decrements in voluntary torque and cortical activation during both contractions, with decreases being greater in the sustained MVC. Interestingly, the authors noted that peak muscle relaxation rate (measured during the silent period following TMS) during the sustained contraction was ~20% faster in hyperthermia. This observation of an increase in contractile speed within heated muscle corroborated previous data,[98,109,110] and led to the suggestion that the greater central fatigue observed during longer contractions may be indicative of a failure in voluntary drive to account for temperature-related adjustments in muscle contractile speed. Hence, while high motor unit firing rates may be transiently attained during brief MVCs, these may not be

sustained during prolonged contractions.[108] To further investigate the potential influence of a temperature-induced increase in motor unit firing rate in mediating the additional central fatigue observed during sustained contractions, Périard et al[91] actively and passively heated participants from 37.1 to 38.5, and on to 39.4°C. The authors demonstrated that both active and passive hyperthermia increased peak muscle relaxation rate when performing brief (5 s) and sustained (30 s) MVCs. Moreover, an increase from moderate (38.5°C) to severe (39.5°C) passive hyperthermia further increased relaxation rate, but without exacerbating force loss or voluntary muscle and cortical activation. It was thus concluded that centrally mediated rates of activation (i.e., motor unit firing) are sufficient to overcome active and passive hyperthermia-induced increases in peak muscle relaxation within physiologically relevant ranges (i.e., 10–30 Hz).[91] As such, the reduction in voluntary muscle activation noted during hyperthermia, whether induced actively or passively, does not appear to originate from a failure in voluntary drive to account for temperature-related adjustments in muscle contractile speed. Rather, voluntary activation failure is partly link to alterations in the peripheral transmission of the neural drive and the supraspinal generation of neural drive when contractions are prolonged.[66] Of note, peripheral alterations are not protected by heat acclimation[111] and voluntary activation remains depressed.[45] However, the supraspinal failure is reverted by acclimation, thus protecting from the additional hyperthermia-induced decrease in force when contractions are prolonged in acclimated humans.[111]

Peripheral perturbations

High T_{core} and T_m have been shown to influence muscle function and cellular metabolism in humans. Indeed, exercise in the heat leads to a greater reliance on muscle glycogen and anaerobic metabolism,[112,113] and causes a greater post-exercise accumulation of ammonia as well as muscle and blood lactate.[75,112,114] This elevated muscle lactate production is linked with muscle fatigue, and the decline in force observed during work at high glycolytic rates.[87] It is also highly correlated with the release of force-depressing free hydrogen ions (H^+).[115,116] Temperature-induced impairments in sarcoplasmic reticulum function or structural damage compromising sarcoplasmic reticulum calcium ion regulatory capacity may also influence skeletal muscle force production.[87,117]

Moreover, it is well established that isometric exercise involves partial to complete occlusion of blood flow,[118,119] further increasing T_m and stimulating chemoreflexes and mechanoreflexes.[120,121] The afferent limb of these reflexes (i.e., group III and IV polymodal fibers) responds to chemical, mechanical and thermal stimulation,[122,123] which increases muscle sympathetic nervous activity.[124] Depending on the intensity of contraction, the rise in muscle sympathetic nervous activity can alter motor unit excitability, modifying the relationship between central neural drive, motor unit recruitment, and firing rate coding.[125,126] Impairment in neuromuscular function may thus relate to failure in the peripheral transmission of neural drive at any level from cortical activity to sarcolemma depolarization.[66] Although hyperthermia influences neuromuscular performance, it has been demonstrated recently that chronic exposure to heat stress (i.e., heat acclimation) improves skeletal muscle contractile function.[45] This occurs via increases in evoked peak twitch amplitude, torque production at a given voluntary activation as well as improvement in the relative torque/EMG linear relationship (Fig. 3).

Perceptual and mental perturbations

The reduction in voluntary activation observed under heat stress (i.e., hyperthermia-induced central fatigue) may well represent a psychophysiological phenomenon whereby central neural drive is reduced via alterations in neuromuscular function and motivation. Indeed, considerable effort is required to sustain a maximal contraction, along with a willingness to withstand discomfort and pain. Mild sensations of discomfort are generally sensed at the onset of a contraction, which eventually develop into severe pain that alters the perception of sensations in the contracting musculature.[127] As a result, mental fatigue, which involves tiredness, limited attention span and an aversion or decreased commitment to continuing a task or activity,[128] may contribute to decrements in voluntary activation. Conscious signals originating from both central and peripheral afferent pathways could mediate behavior and reduce motivation in order to minimize discomfort[129] and lead to the abandonment of a task in which the energetic demands (i.e., effort) outweigh the perceived benefits of continued performance.[130] Essentially, a lack of motivation can cause central neural drive and motoneurone firing to decline, leading to a loss of force production.[131]

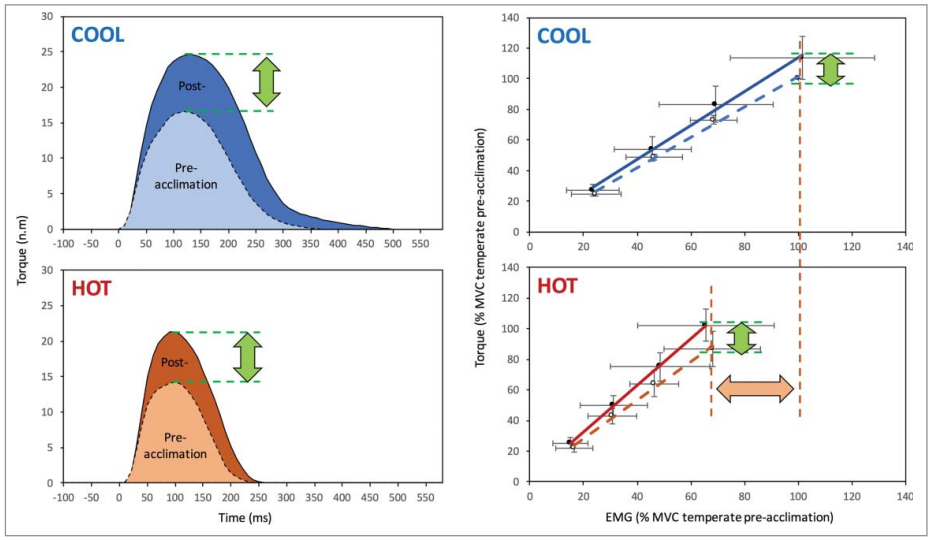

Figure 3. Heat acclimation increases peak twitch amplitude in both normothermic (COOL) and hyperthermic (HOT) state (left panel). Heat acclimation also improves the torque/EMG relationship (right panel). Reproduced with permission from Racinais et al.[45]

During dynamic exercise, it has recently been demonstrated that endurance exercise capacity at 80% VO_{2max} in a hot environment (30°C) is impaired by both a passive increase in T_{core} prior to exercise, and a 90 min mentally fatiguing task.[132] It was further demonstrated that the performance impairment was exacerbated by the combination of these interventions, which acted synergistically to influence the exercise task. In contrast, a mentally fatiguing task undertaken prior to 45 min of moderate cycling and a ~15 min self-paced cycling effort in 30°C did not influence time trial performance.[133] This was likely due to mental faculties already being strained by the combination of high intensity exercise and hot environmental conditions, and the mentally fatiguing task failing to increase negative valence and perceived exertion. A similar mechanism (i.e., failure in down-regulating perceived exertion) was purported to modulate the inability of carbohydrate mouth rinsing to improve 60-min cycling time trial performance in hot-humid conditions, which appears to have been influenced more significantly by the elevated levels of thermal and cardiovascular strain.[134] Notwithstanding, Flouris and Schlader[135] have suggested that thermal perception influences the rating of perceived exertion and concomitantly work rate at the onset of exercise when only T_{skin} is elevated. However, as thermal strain increases, factors associated with cardiovascular strain[94,95,136] more likely mediate the rating of perceived exertion and the voluntary reduction in work rate.

Cognitive function

A moderate increase in core temperature can initially improve cognitive function.[137] Conversely, the development of hyperthermia can impair cognitive function,[138,139] with the impairments being task and complexity dependent.[140-144] It has been suggested that the performance of cognitive tasks under heat stress deteriorates when the total cognitive resources are insufficient to support both the adequate completion of the task and processing of the thermal stress.[139] As such complex tasks are more sensitive to hyperthermia than simple tasks.[140,141,143,144] Importantly, variations in temperature have been shown to induce pleasure or displeasure if they favor or perturb homeostasis, respectively.[145] Thus, a model was developed linking the decrement in complex task performance to the alliesthesial change accompanying compensatory physiological responses to hot environmental conditions.[144] More specifically, increases in temperature during heat exposure generated unpleasant stimuli, as measured by the Positive and Negative Affect Schedule (PANAS), which could be considered as a 'cognitive load'. It was proposed that this load might reduce the available resources for concurrent cognitive tasks. Interestingly, this model could explain why reducing thermal discomfort, by cooling the head for example, can restore some complex cognitive functions in a hot environment.[66,142] Lastly, based on recent data showing that passive hyperthermia increased the rate of false alarms during a sustained attention task,[142] and led to faster but false responses

during a complex planning task,[143] it has been suggested that hyperthermia may increase impulsivity.[144]

Thermal and circulatory strain

Skin blood flow

The circulatory requirements associated with aerobic exercise under heat stress include an increase in skin blood flow and the maintenance of cerebral and exercising muscle perfusion. Although a transient vasoconstrictor-mediated reduction in skin blood flow occurs at the onset of exercise, cutaneous blood vessels eventually dilate to aid in the dissipation of accumulating metabolic heat. The T_{core} threshold at which skin blood flow begins to rise is directly related to exercise intensity, and a delay in active vasodilation shifts this threshold to the right relative to rest.[146–149] During prolonged exercise in the heat, the rate of rise in skin blood flow markedly decreases after 20–30 min when T_{core} nears 38°C, reaching a virtual plateau at ~50% of maximum flow capacity.[150,151] At this point, the perfusion requirements of exercising muscles take precedence over thermoregulatory control. Despite this attenuation and the potential for splanchnic and renal vasoconstriction to redistribute 600–800 $ml.min^{-1}$ of blood to the periphery, the large displacement of blood (6–8 $l.min^{-1}$) to cutaneous vascular beds mediates a progressive decline in arterial pressure, central venous pressure and stroke volume that is accompanied by an increase in heart rate.[152,153] Interestingly, the relationship between T_{skin} and skin blood flow is minimally influenced by T_{core}.[151,154] However, a strong link exists between the core-to-skin temperature gradient and skin blood flow. For example, a 10°C increment in ambient temperature is associated with a 4.5°C decrease in the core-to-skin temperature gradient.[155] This narrowing of the gradient leads to a reflexive rise in skin blood flow,[154,156,157] enhancing non-evaporative heat loss to the environment in compensable conditions.

Muscle blood flow and oxygen delivery

In exercising muscles, blood flow requirements relate to a rise in relative exercise intensity whereby the increase in oxygen demand is matched by an increase in systemic and muscle oxygen delivery, as well as perfusion pressure.[158] Interestingly, recent studies show that elevated tissue/blood temperatures also induce an increase in skeletal muscle blood flow at rest and during exercise.[24,159,160] The mechanism(s) mediating this increase may include an interaction of metabolic and thermal stimuli inducing the release of erythrocyte-derived ATP, a potent vasodilator.[160] In absolute terms, muscle blood flow can increase from ~0.3 $l{\cdot}min^{-1}$ at rest, to 10 $l{\cdot}min^{-1}$ during maximal exercise in less than 10 s.[161] If work rate is stable, a steady-state is reached within 30–90 s and blood flow rises only slightly when exercise intensity increases.[162] As such, muscle blood flow and oxygen uptake are quite stable during prolonged exercise at fixed intensities in thermoneutral conditions.[163] Under conditions of heat stress however, a rise in thermal strain exacerbates the cardiovascular response as metabolic and thermoregulatory processes compete for cardiac output.[152,164,165] The concept of competition or conflict between regulatory systems has recently been suggested to rather represent commensalism, which is an integrated balance of regulatory control where one circulation benefits without substantially affecting the other.[166]

The classic hypothesis of circulatory/cardiovascular limitations to exercise in the heat proposed by Rowell[152] lies with the redistribution and peripheral pooling of blood, which reduces central blood volume and concomitantly ventricular filling pressure, end-diastolic volume, and stroke volume. A more contemporary hypothesis suggests that the reduction in stroke volume is primarily due to an increase in intrinsic heart rate,[167-169] mediated by the direct effects of temperature on the sinoatrial node, and/or baroreflex modulation of sympathetic and parasympathetic activity.[170] In effect, the decline in stroke volume during moderate intensity prolonged exercise in the heat likely originates from both a reduction in central blood volume reducing cardiac filling pressure, and a shorter diastolic filling time reducing ventricular end-diastolic volume. Indeed, when exercise is sustained in highly motivated individuals, the attainment of maximum or near maximum heart rate is a well-documented response.[92] Concurrently, maximum cardiac output is decreased,[171172] and the cardiovascular system is forced toward a functional limit at submaximal workloads and oxygen uptake.[173-175] It is suggested that this reduction in cardiovascular reserve is the primary factor limiting constant rate aerobic exercise, and manifested as an increase in relative exercise intensity and perceived exertion for a given workload.[43,129,171,172,176]

Cheuvront et al.[93] have previously highlighted that the T_{core} tolerated at exhaustion is inversely related to whole-body skin blood flow requirements, suggesting

that fatigue during exercise in the heat may correspond with adjustments in muscle and cerebral perfusion related to increases in cardiovascular strain. During short but intense cycling at 80% of peak power output, Gonzalez-Alonso[177] noted that to exhaustion was associated with a reduction in systemic and exercising muscle blood flow, oxygen delivery and uptake. These reductions were exacerbated by heat stress, which accelerated the decline in mean arterial pressure and cardiac output, reducing VO_{2max}. It has also been shown that during maximal incremental and constant rate exercise, systemic oxygen delivery is blunted by a plateau or decrease in cardiac output at intensities below VO_{2max}.[178] From 50–90% VO_{2max}, systemic and exercising muscle blood flow, along with oxygen delivery, match the rise in VO_2. However, beyond 90% VO_{2max} a levelling off occurs that attenuates the rate of rise in VO_2, despite maximum increases in arteriovenous oxygen difference and heart rate. This impairment indicates an inability of the cardiovascular system to sustain a linear increase in oxygen delivery to exercising muscles. Aerobic power and capacity are therefore impaired by reductions in cardiac output and oxygen delivery to the exercising musculature, partly due to an enhanced muscle sympathetic nervous activity which attenuates leg blood flow.[178-180] These findings have been corroborated during maximal and supramaximal intensity cycling[181] and although metabolic energy requirements are greater in supramaximal exercise, a plateau in cardiac output and exercising muscle vascular conductance was noted at similar levels of oxygen delivery at both intensities. Taken together, these observations challenge the postulate that muscle oxygen delivery increases linearly from rest to VO_{2max}.[158,182,183] During prolonged submaximal exercise in the heat, a similar compromise in oxygen delivery appears to occur, especially as heart rate nears maximum.

The impairment in prolonged self-paced exercise occurring in the heat (Fig. 4) has also been attributed to this detrimental increase in circulatory/cardiovascular strain.[93-95,136,184,185] It has been shown that a

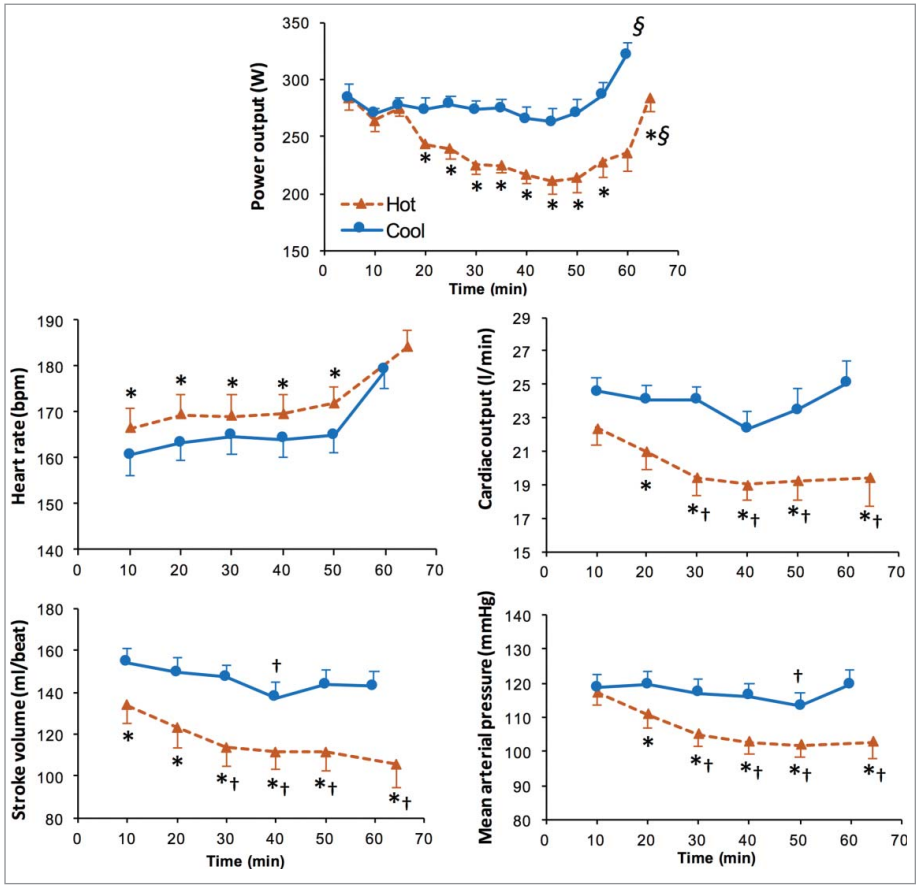

Figure 4. Power output and cardiovascular response during a 40-km cycling time trial in Hot (35°C) and Cool (20°C) conditions. Values are means ± SD for 8 subjects. ∗Significantly different from Cool ($P < 0.05$). §Significantly higher than previous ($P<0.01$). †Significantly lower than 10 min ($P < 0.05$). Reproduced with permission from Périard et al.[17]

thermoregulatory-mediated rise in cardiovascular strain is associated with reductions in VO_{2max} and power output during prolonged (60 min) intense self-paced cycling in the heat (Fig. 4).[94] Although exercise in both hot and cool conditions leads to a progressive decrease in VO_{2max}, the extent of the decrement is greater in the heat.[95] Despite the larger decrease in aerobic capacity and concomitantly work rate (e.g., power output), relative exercise intensity (i.e., $\%VO_{2max}$) in the heat is maintained within a fairly narrow range (i.e., 2–5%), similar to that of cooler conditions. This range widens under heat stress however, as exercise becomes protracted and a disassociation develops between relative exercise intensity and heart rate and RPE.[95]

Cerebral blood flow

The development of hyperthermia is associated with a progressive reduction in cerebral blood flow during self-paced exercise (Fig. 5), attributed in part to an increase in cutaneous blood flow, decreases in cardiac output and arterial blood pressure, and hyperventilation-induced hypocapnia.[184,186-188] Interestingly, reductions in cerebral blood flow and concomitantly oxygen delivery to the brain, have been suggested to compromise central neural drive to exercising muscles during strenuous exercise in the heat.[186,189,190] However, the development of fatigue during such exercise is associated with an enhanced cerebral metabolism,[191] manifested by a compensatory increase in oxygen extraction in the brain.[192] Moreover, comparable

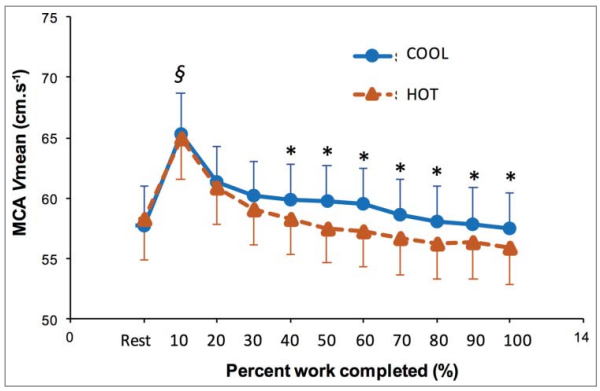

Figure 5. Middle cerebral artery mean blood velocity (MCA Vmean) during a self-paced time trial (750 kJ) in HOT and COOL conditions. Values are means ± SEM. *Significant difference between HOT and COOL ($P < 0.05$). §Significantly higher than all other values ($P < 0.05$). Reproduced with permission from Périard and Racinais.[184]

impairments in force production capacity have been observed following self-paced cycling (40 km) in hot and cool conditions,[107] and exhaustive incremental exercise has been shown to decrease voluntary activation by the same extent in hot and cool conditions.[43] It therefore does not appear that an exacerbated reduction in cerebral blood flow under heat stress mediates performance decrements via central inhibition. Notwithstanding, the development of hyperthermia does influence the ability to generate muscle force.

Exertional heat illness

Environmental heat stress and physical activity interact to increase the risk of EHI. This risk represents a continuum of medical conditions with potentially severe consequences that can affect physically active individuals in both hot and temperate environments. The severity of EHI can escalate from relatively benign symptoms such as muscle cramping, to heat exhaustion, heat injury and heat stroke.[5,19,193] Heat exhaustion is considered a mild to moderate EHI associated with a T_{core} of 38.5 to 40°C and an inability to maintain cardiac output. Heat injury is defined as a moderate to severe illness characterized by organ (e.g., liver and renal) and tissue (e.g., gut and muscle) damage with a high body temperature, typically >40°C. The most severe EHI, heat stroke, is characterized by high body temperatures, profound central nervous system dysfunction (e.g., combativeness, delirium, seizures and coma), as well as organ and tissue damage, and can lead to death. Unlike classic heat stroke, which is primarily observed in vulnerable and immunocompromised populations during seasonal heat waves, exertional heat stroke occurs in healthy young individuals performing strenuous physical activity (e.g., exercise or occupational tasks). Notwithstanding, heat stroke and other EHIs often occur in the presence of certain risk factors, such as extreme environmental conditions, medication and drug use, compromised health status, and underlying genetic conditions.[19] Heat stroke often occurs in individuals considered low risk and performing routine physical activities under heat stress.[5] Accordingly, it is difficult to identify individuals that may be at risk of EHI. Although the prevalence of EHI increases during exercise performed in the heat, athletes can adopt various strategies to mitigate the influence of heat

stress on performance, as well as the risk of heat illness.[20] As detailed below, these involve following an individualized hydration regimen, utilizing cooling strategies and most importantly, acclimatizing to the heat.[5,194,195]

Application to sports – warming-up for performance

Types of warm-up

There are many preconditioning or priming techniques at an individual's disposal prior to competition. These involve preparing the body physically and psychologically. Psychological warm-up strategies (e.g. mental imagery, mental rehearsal) are catered towards enhancing cognitive function, a function that is specifically key in team sports where decision making is one of the primary factors contributing toward successful performance outcome.

A common physical warm-up may consist of progressive intensity building with a range of explosive movements utilizing the stretch shortening cycle, while also utilizing stretching interventions aimed at relieving muscle tension and improving range of motion. While certain strategies are beneficial for maximal strength and force production, others may well be detrimental, which will be discussed in sections below.

Structuring a warm-up

An effective warm-up can last for more than 30-min[196] and should include a main component of general exercise (usually sport specific), as well as context specific exercises and flexibility work.[80] Such a protocol is commonly used in the field and involves targeting 4 physiological pillars and in 3 specific stages[197]: Raise, Activate and Mobilize, Potentiate (RAMP; Fig. 6). This model, incorporated with appropriate exercise selections that replicate the biomechanical and psychological demands of the chosen competition, should provide the necessary tools for effective warm-up design. Under heat stress, the duration of each stage may differ due to the detrimental effects of heating up. Accordingly, certain adjustments in the warm-up protocol are advised (Fig. 6).

Raise

The first stage (Raise) specifically targets the thermoregulatory homeostatic systems of the body by increasing muscle and by extension core temperature. This stage also impacts upon the heart rate response, blood flow viscosity and ventilation. At the onset of exercise T_m rapidly increases, usually within 3–5 min. It is after 10–20 min of moderate intensity steady-state exercise (80–100% lactate threshold) that T_m plateaus[198] with elevated temperatures (i.e., 2–4°C)

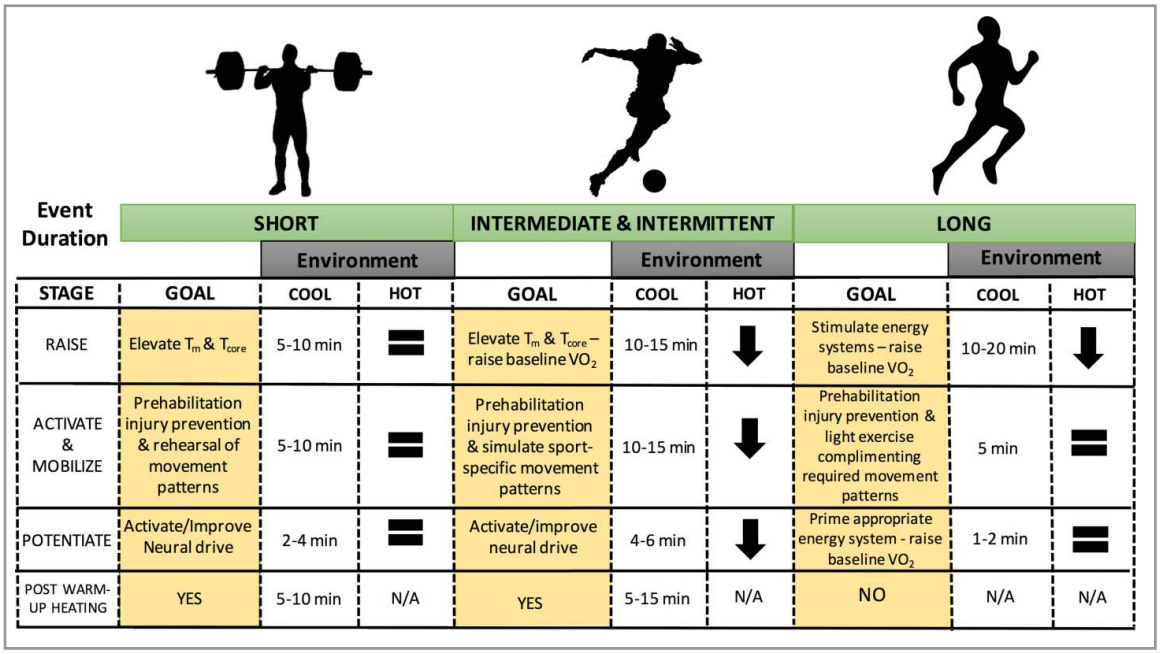

Figure 6. Warming-up for short-, intermediate- and long-duration events and the impact of environmental heat stress on the duration of 'RAMP' based warm-up protocols. Physiological underpinning of the "Raise," "Activate and Mobilize," and "Potentiate" stages are highlighted for each sporting duration, in addition to demonstration of the potential impact of post-warm up passive heating strategies.

shown to significantly enhance performance.[22,198] The increased maximum shortening velocities and development of tension,[35] alongside viscosity changes[46] in warmer muscles likely prepares the athlete for the next stages of the warm-up. When undertaken in hot climatic conditions, the Raise stage of the warm-up may be shortened for intermediate to prolonged events (Fig. 6), so as to avoid and excessive increase in whole-body temperature.

Activate and mobilize

The second warm-up stage (Activate and Mobilize) can be broken down into 2 parts. Activation is highly specific to not only the working muscle groups, but also the specific function and movement patterns the working muscles carry out during competition. Typically, "activation" can be achieved using now termed "prehabilitation" exercises, defined as *"preparing an individual to withstand a stressful event through enhancement of functional capacity"*.[199] This stage often utilizes light loads, aiming to stimulate the neuromuscular and proprioceptive responses to competitive exercise through simulated movement patterns – a key determinant for chronic-exercise induced neural adaptation.[200] Duration of this phase will depend on the individual and competition demands. "Mobilization" usually involves dynamic movements, aiming to activate key muscle groups involved in successful performance outcome. Early research suggested static stretching and proprioceptive neuromuscular facilitation techniques may contribute to improvements in both injury prevention and exercise performance.[3,84] More recent observations suggest that while long-term muscle function and performance may be improved from regular stretching interventions,[201] there may be little effect of either static stretching or proprioceptive neuromuscular facilitation on various performance outcomes, across a range of intensities.[202-204] Recent data suggest high baseline athlete flexibility (e.g. gymnasts), and the specific stretching protocol employed (intermittent vs continuous) may improve the impact of static stretching on performance.[205] Dynamic-based stretching and loading likely remains a more effective approach when aiming to maximize athletic readiness during the mobilization stage.[206] Given the nature of the activates undertaken in the Activate and Mobilize phases, the types of events most likely to impose a modification of this stage are those of intermediate duration (e.g., team-sports; Fig. 6).

Post-activation potentiation

Prior to commencing PAP, a warm-up induced functional change to both temperature- and load-dependent actions on muscle and tendinous tissues should be present. This yields a protective effect as greater forces and length of stretch are required to tear isometrically preconditioned (warm) animal muscle[83] when compared to cold muscle. PAP can then involve high rates of force production and muscle-length changes at high velocity, thereby involving muscles containing an abundance of fast-twitch fibers. These muscle fiber types in particular may be more susceptible to injury due to their differing functional capacities.[207] While increases in T_m lead to improvements in muscle fiber conduction velocity,[27,28] these findings do not necessarily relate to muscular activity.[208] Unlike the inhibitory neural conduction rate responses that occur when T_m is reduced,[23] findings of increased neural conduction rates are not necessarily present when T_m is elevated.[208] PAP therefore usually involves performing intense sports-specific movements in order to fully simulate the demands of performance, in turn stimulating the necessary neural pathways.

The PAP phenomenon can be usefully applied prior to events that require production of high forces and power outputs, especially in conditions of elevated T_m[209]. PAP for the power based athlete may be utilized through relatively high-resistance movements,[210] with 2 recent studies demonstrating significantly beneficial effects on countermovement jump[211] and vertical jump[212] performances when 1–3 repetitions of resistance conditioning activities ranging from 80–93% 1RM were performed 2–6 min prior to testing. It is recommended that individualized intensities and exercise modalities be employed to suit the capability (maximal strength) of the athlete.

Depending on the demands of the event, the duration and intensity of PAP will vary dramatically. The potentiating benefit in endurance athletes would aim to preserve fatigue at submaximal intensities, through altering low frequency firing rates and force development at the level of the motor unit.[210] This would theoretically allow the athlete to achieve higher force for lower calcium activation.[213] While potentiation is capable of compensating for low firing frequency at submaximal workloads, potentiation-induced alteration of high frequency forces is not necessarily observed.[57] This implies any PAP-induced improvement in sustained workload during fatiguing

endurance exercise would be resultant from increased low frequency force, in-turn compensating for the gradual decline in high frequency force that occurs in fatiguing tasks. Indeed, in states of elevated T_m, a rightward shift in the force-frequency relationship occurs[44] and therefore the benefit of PAP on endurance performance would be counteracted by elevated T_m.[210] For the strength- or power-based athlete however, PAP induced under conditions of elevated T_m would be additive (see *protocol specificity* for more).

Protocol specificity

How long is too long?

The "optimal" warm-up strategy likely varies between sports.[4] It also depends on environmental conditions (Fig. 6, Fig. 7). Indeed, warm-up duration might be reduced before intermediate or prolonged exercise in hot environments (Fig. 6). Warm-ups should be designed to activate the energy systems of interest, while at the same time challenging sport-specific movement patterns the athlete is likely to experience throughout the competition period. This follows recommendations that neural activation is one of the most important mechanisms of warming-up for an athlete.[214]

While Raise and Activate and Mobilize may be necessary for most competing athletes, the importance of neural activation is larger for strength- or power-based athletes compared to endurance athletes.[210] Equally, longer durations of prior aerobic exercise can have deleterious effects on maximal power production, especially at warm-up intensities >60% VO_{2max}.[215] This followed further observations that maximal cycling peak power output was achieved after warm-up sets performed at between 33 and 48% VO_{2max}.[215] It may therefore be advisable for strength- and power-athletes to focus on performing light-intensity (< 50% VO_{2max}) exercise during the Raise stage. Once the warm-up is complete, heated garments may be utilized with the aim of maintaining elevated T_m and T_{core} prior to strength- or power-based events (see '*Time between warm-up and competition*'). Such post warm-up heating is however not necessary in hot ambient condition and could even be replaced by some cooling strategies in particular circumstances (Fig. 7).

The endurance athlete, depending on the competitive ambient environment, may wish to predominate the undertaking of the Raise and Activate and Mobilize stages. The modality and movement pattern requirements, should dictate emphasis of activation and mobilization. Endurance events that utilize cyclical activities with one predominant movement pattern (i.e., cycling) may reap less benefit from Activation and Mobilization compared with the endurance cross-country runner, or team-sport athlete that constantly undertake multi-directional movements, performed across a range of joint angles and velocities. High load explosive Potentiation prior to competition may not be necessary in endurance athletes. While a prior 6-min exercise bout in endurance trained cyclists can accelerate phase II VO_2 kinetics during a subsequent heavy-domain (85% VO_{2max}) exercise task,[216] this phenomenon occurs after both moderate- (~52% VO_{2max}) and high-intensity (85% VO_{2max}) priming.[216] As previously alluded to regarding co-activation of both potentiation and

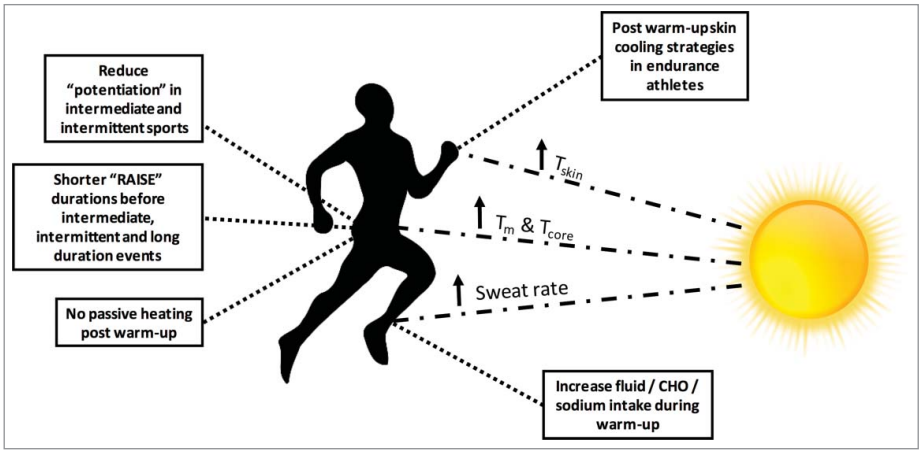

Figure 7. Altering a warm-up to accommodate for hot ambient conditions. Some applied protocol recommendations for altering the RAMP protocol; the use of post warm-up passive heating/cooling strategies and altering nutritional intake to reduce the impact of heat stress on subsequent performance. CHO = carbohydrate.

endurance-induced fatigue, PAP may naturally occur in fatigued states, even when performing submaximal contractions (i.e., endurance competition). While training status may have influence on this mechanism, the antagonistic nature of elevated T_m on the efficacy of PAP in the endurance athlete, may provide rationale for designing a warm-up that aims to target an efficient priming of aerobic energy systems, rather than focusing on high-intensity potentiating conditioning bouts, or elevating T_m to optimal temperatures to improve sprint- or power-based performance.

The trade-off between priming the body and inducing fatigue is of vital consideration. Limiting variables to performance are highly contrasting between sprint- or power-based tasks and endurance events (e.g., marathon running or cycling races). Therefore, warm-ups should be designed following a needs analysis of each individual sport (Fig. 6).

Time between warm-up and competition

T_m has been observed to decrease exponentially after warming-up,[22] and approximately 70% of the warm-up induced elevation in T_{core} can be lost during a 15-min transition period between the warm-up and performance task.[217] In temperate conditions ($\leq$ 19°C) strategies to maintain T_m and T_{core} through either localized muscle heating elements[22] or application of blizzard survival jackets[217] have been shown to be effective interventions in improving power-based exercise tasks. Indeed, applying external heating elements to the limbs can maintain T_m.[22,218] However, there is no further benefit in applying such heating elements to the working muscles during the warm-up itself[22] and their use should be restricted between the warm-up and the event. The issue of maintaining the benefits of warming-up is of particular interest to swimmers who cannot swim for ~20 min after exiting the warm-up pool prior to their event. In this setting, maintaining T_{core} and T_m via dry-land exercises may benefit performance, and using heated jackets in addition to conducting sports specific dry-land exercises can further improve performance.[219,220] Naturally, event duration plays a pivotal role in the decision to apply heating elements. It is proposed that for a 1°C rise in T_m at depths between 1–4 cm, improvements of 4–10% in peak power output (PPO) could occur.[22] Conversely, when assessing more prolonged intermittent-exercise tasks, lower body temperatures may delay the onset of fatigue.[221]

The classic study by Sargeant & Dolan[215] showed a decrease in peak power output after a 6 min warm-up at 87% VO_{2max}, with baseline values (i.e., without warm-up) being recovered following just 1 min of rest, but maximal peak power output (9.6% above baseline) being observed after 6 min of recovery.[215] This supports recommendations that power- and sprint-based athletes should plan a recovery period of 5–10 min between warm-up completion and competition, first to maintain the benefit of potentiating conditioning work[57] and second to preserve elevated T_{core} and T_m,[22] while simultaneously allowing for sufficient phosphocreatine (PCr) and anaerobic energy system resynthesis.[222,223] Endurance athletes on the other hand may wish to simply focus on commencing competition once energy system repletion occurs following appropriate priming warm-up exercises, with the goal of avoiding early onset fatigue via elevated temperature-related mechanisms.[221] Indicative durations for each warm-up stages in both cool and hot environments are suggested in Fig. 6.

Warm-up strategies and heat stress

While warm-up strategies should aim to augment performance of previously highlighted metabolic, musculoskeletal and cardiovascular systems via manipulation of intensity, duration and movement patterns; the consideration of first the demands of competition and second environmental stressors, should dictate the specific prescription of the warm-up protocol. In cool conditions, power-based and team-sport athletes may wish to focus on raising T_m and T_{core} through intense sports-specific drills that simulate the demands of competition, in turn sufficiently stimulating neural activation following performance of applicable motor recruitment patterns. With passive heating interventions also at an athlete's disposal, the use of T_m maintenance for a power-based athlete (aiming to maintain a 2–4°C T_m elevation) may also improve quality of work. When these sports are undertaken in the heat over, passive heat maintenance is likely unnecessary and may be eliminated.

Endurance-based athletes (e.g., triathletes, marathon runners, cyclists) on the other hand may wish to take a different approach to their pre-competition strategy. The goal of the warm-up in these populations is to optimize physiological readiness through activation of predominant energy systems and movement patterns,

without producing unnecessary metabolic heat that may become more performance-limiting the longer an event lasts.[224] The impact of a potentiating and temperature elevating warm-up prior to competition is likely intensity dependent, with power and sprint-type tasks showing the greatest improvements.[22,196,217]

Application to sports – warming-up or cooling-down?

The undertaking of necessary mechanical work, capable of stimulating the responses described above undoubtedly results in thermal homeostatic perturbations, leading to increases in both T_m and T_{core}.[196,225] In hot and/or humid ambient conditions, these increases can be exacerbated and bring the athlete from a 'warmed-up' to a 'heated-up' state. The section below focuses on how to minimize thermal strain by employing various cooling techniques as part of the warm-up routine. It also includes recommendations on hydration during warm-up and competition, as well as some suggestions regarding half-time in team-sports.

Cooling-down while warming-up

Skin cooling can reduce cardiovascular strain during exercise in the heat, while whole-body cooling can decrease organ and skeletal muscle temperatures. Several studies carried out in controlled laboratory environments (e.g., uncompensable heat-stress), with or without fanning during exercise, have reported that pre-cooling can improve endurance,[92,226-231] and high-intensity[232] exercise performance. However, the benefits of pre-cooling on intermittent or repeated-sprint exercise performance vary, with observations of performance enhancement[233-236] and no changes[233,237-239] in a range of studies. Given its effect of lowering T_m, whole body cooling appears to be detrimental to performance during a single sprint, or the first few repetitions of an effort involving multiple sprints.[240,241]

Several reviews conclude that prolonged exercise capacity in the heat can be improved following cooling interventions,[242-249] however its effectiveness when comparing hot laboratory and competitive environments appears to differ, with suggestions that the benefits of pre-cooling may be overestimated in outdoor settings.[250] Hence, the impact of cooling in competition settings remains equivocal and recommendations are currently limited to prolonged exercise bouts in hot ambient conditions, or conditions with limited air flow.

Cold-water immersion

A range of cold water immersion (CWI) protocols are available when aiming to optimize physiological readiness for competition (for reviews see:[247,251-253]). Notwithstanding, the most common techniques for effectively lowering T_{core} include whole-body CWI, usually for ~30 min at water temperatures between 22−30°C, or body segment (e.g., legs) immersion at lower temperatures (10−18°C).[247] More extreme external cooling protocols will lead to more rapid reductions in T_m, in turn having deleterious effects on important priming mechanisms such as nerve conduction rate and muscle contraction velocities.[30] Athletes employing these more "aggressive" CWI strategies may therefore need to plan an active re-warming strategy into their competition preparation prior to commencing the event. Aggressive cooling of locomotor muscles will undoubtedly reduce the initial development of thermal and cardiovascular strain, however may hamper performance in the early stages of exercise. As such, various new products such as cooling garments, specifically designed to cool the torso, have been designed to reduce T_{core} (or limit its increase) without affecting T_m.

Cooling garments

The original use of ice towels to promote cooling lead to the development of several ice-cooling jacket devices to cool athletes prior to or during exercise in the heat.[228,233,254,255] CWI induces greater reductions in T_{core} when compared to cooling vest application,[249] however cooling vests can effectively lower T_{skin}, in turn having advantageous effects on both cardiovascular strain and heat storage,[256] without deceasing T_m. Cooling through cooling garments carries higher ecological validity as athletes can more easily apply these garments during warm-up periods, or recovery breaks, depending on environmental and competition demands.

Mixed methods cooling strategies

It is suggested that the combined use of internal (e.g., cold beverages) and external (e.g., CWI) cooling strategies can lead to greater cooling capacity when compared to using the same techniques in isolation.[249] Mixed cooling methods have so far proven beneficial to football performance in tropical environments,[257] lacrosse players training in hot environments,[258] and laboratory-based cycling tasks.[230] The combination of mixed cooling methods is practically applicable to

athletes. Valid mixed cooling methods may integrate the use of ice-slurry beverages, cooling-vests and fanning during or after warm-up.

Hydration during warm-up

It is common for rested, well-fed humans to be sufficiently hydrated.[259] However, as fluid replacement capacity is limited while exercising in the heat, it is recommended to avoid being is a state of dehydration before competing in the heat,[20] especially during warm-up. A prescribed fluid intake of 5–6 ml of water per kg of body mass, at a frequency of every 2–3 h, as well as 2–3 h prior to either training or competing in the heat is advisable. As with other strategies, this hydration protocol should be practiced prior to its implementation during competition as it will increase urinary output.

While cold fluid ingestion prior to exercise can potentially improve performance,[260,261] it is thought that fluid-induced activation of thermoreceptors, probably located in the abdominal area,[262] can cause reductions in sweating, reducing the potential for evaporative cooling in dry heat.[263] As such, there does not seem to be advantageous performance responses when cold fluids are ingested during exercise.[264,265] This practice could however be beneficial during warm-up to support cooling and fluid preservation before the event.

Ice slurry beverages may be a more effective athlete cooling strategy, based on the theory of enthalpy. Ice requires substantially more heat energy (334 J/g) to cause a phase change from solid to liquid (at 0°C) compared with the energy required to increase the temperature of water (4.2 J/g/°C). Several reports recently supported the consumption of ice slurry beverages prior to[231,266,267] or during[268] exercise. In order to observe benefits in endurance or intermittent-sprint exercise tasks, ~1 L crushed ice at ≤ 4°C may be advisable. However, the practicality and evidence for using ice-slurry require additional study. In the meantime, as for cold drink, ice-slurry is likely more suitable during warm-up than competition.

Half-time strategies

Re-warming up

While half-time intervals may provide recovery opportunities for team-sport athletes, previous observations reveal soccer players cover less distance[269] or produce less medium intensity running (11.1 – 19 kph) efforts in the second-half of matches compared with first-half performances.[270] This observed decrement could be due to various (thermoregulatory and metabolic) factors. As previously alluded to in this review, reduced T_m is correlated with reduced sprint performance capacity during soccer matches.[196,271] Firstly, a 35-min sports specific warm-up lead to a 3°C rise in T_m compared to rest (39.4°C vs. 36°C). After the first half, a 15-min half-time interval produced a natural 2°C reduction in T_m (39.7°C to 37.7°C) in the control group. When an active re-warming (moderate intensity) strategy was introduced 7-min into the half-time break, the natural decline in T_m was reduced to just 0.5°C (39.7°C to 39.2°C), resulting in a similar starting T_m when compared to pre-game temperature (39.2°C vs 39.0°C, respectively). Subsequently, second-half sprint performance was maintained in the active re-warm group.[196] In temperate environments, athletes that perform high-intensity efforts should aim to maintain warm-up induced elevations in T_m through either passive heating interventions[22] or moderate intensity active re-warming strategies.[196]

When environmental heat-stress is added to the competitive environment (i.e., performing in 40°C ambient conditions), increased glycolysis, leading to more rapid depletion of muscle glycogen occurs when compared to exercising in the cool.[75] The risk of exercise-induced hyperthermia increases with rising environmental air temperature.[271] As such, implementation of half-time cooling techniques (discussed below), rather than interventions aiming to maintain elevated T_m, may help to prolong exercise capacity and avoid early onset of fatigue in hot ambient conditions.[221]

The first half of soccer matches may lead to significant muscle glycogen depletion by half-time[272] potentially having detrimental effects on performance capacity in temperate conditions.[273] These findings are corroborated by Winnick et al.[274] showing regular CHO feeding during rest-intervals improves both peripheral and CNS function between 45 and 60 minutes of team-sport activity when compared to placebo feeding.[274] In addition, both external heat-stress and higher internal T_m result in increased CHO oxidation rates for a given workload during exercise. As such, appropriate nutritional refeeding during this time-period may therefore contribute to enhance second-half performance (45–90 minutes), both in temperate and hot environments.

Cooling down

The influence of external cooling on reducing skin blood flow and promoting a rise in the central circulation,[275] along with improvements in perceptual responses to heat stress,[276] may ultimately provide the impetus for enhancing performance. A pre-cooling strategy utilizing CWI for between 5–12 min in 14°C water during a 15-min period between bouts can potentially improve intense exercise in the subsequent effort in hot environments.[277,278] The translation between reducing internal heat strain, though ingestion of cold water[279] or ice-slurry[280] does not necessarily translate to improved subsequent bout performance outcomes. Together, studies on body cooling (internal or external) suggest that cooling might aid recovery from intense exercise in uncompensable heat-stress in laboratories and, in some cases, might improve performance in subsequent intense exercise bouts. The effects of aggressive cooling versus simply resting in the prevailing hot ambient conditions, or in cooler conditions, remains to be validated in a competition setting (e.g., half-time in team-sports).

Other recommendations to avoid heating-up

Hydration

The development of hyperthermia during exercise in hot ambient conditions is associated with a rise in sweat rate, which can lead to progressive dehydration. Exercise-induced hypo-hydrated states are associated with reductions in plasma volume and an increase in plasma osmolality that are proportional to the reduction in total body water.[5] The increase in the T_{core} threshold for vasodilation and sweating at the onset of exercise is closely linked to the ensuing hyperosmolality and hypovolemia.[281,282] In addition, dehydration decreases cardiac filling and challenges blood pressure regulation.[15,283,284] The rate of heat storage and cardiovascular strain is therefore exacerbated and the capacity to tolerate exercise in the heat is reduced.[285-287] In competition settings, however, hydration is dependent on several factors, including fluid availability and the demands of the events. As such, hydration is easier during warm-up than competition. Of note, although it is recommended for competitive athletes to minimize their body mass losses, recreational athletes involved in prolonged exercise should be cautious not to overhydrate during the exercise.

Heavy sweaters may choose to deliberately increase sodium intake prior to either training or competing in the heat. Increasing sodium (i.e., salt) intake (e.g., 3.0 g of salt added to 0.5 L of a carbohydrate-electrolyte drink) can help maintain plasma sodium balance in heavy sweaters, as the main electrolyte lost in sweat is indeed sodium (20–70 mEq/L).[288,289] In athletes experiencing muscle cramping, it is recommended to increase the sodium supplementation to 1.5 g/L of fluid.[290] Athletes should also aim to include 30–60 g/h of carbohydrates in their hydration regimen for exercise lasting longer than 1 h,[291] and up to 90 g/h for events lasting over 2.5 h.[292] This can be achieved through a combination of fluids and solid foods.

Heat acclimatization

Appropriate preparation for exercising in the heat starts a long time before warming-up. Repeated training in the heat increasing body (T_{core} and T_{skin}) temperature, sweating and skin blood flow induces heat acclimatization.[5,293-297] Heat acclimatization can lead to various advantageous physiological responses when exercising in the heat. These include increased sweating and cutaneous skin blood flow responses, better matching of fluid-electrolyte balance, plasma volume expansion subsequently improving the ability to sustain blood pressure and cardiac output.[5,194,293] These changes enhance submaximal and maximal aerobic exercise performance in warm-hot conditions.[101,298,299] Most adaptations occur within the first week of training in the heat and continue to change at slower rates over the following 2-weeks.[300-302] If an athlete is constrained by time leading into a competition, they may well therefore benefit from only a few days of acclimatization.[303-305] In order to achieve near complete cardiovascular and sudomotor adaptations, however, as much as 2 weeks training may be needed[101,298,306] to optimize aerobic performance in hot conditions.[299] Athletes who do not have the possibility to travel to naturally hot ambient conditions (so called “acclimatization”) can train in an artificially hot indoor environment (so called “acclimation”).

In addition, it is worth mentioning that some[298,307] but not all[308,309] studies have reported an increase in exercise capacity in cool conditions. This cross benefit is currently debated,[310,311] but seems to be supported by both early,[173312] and recent[313,314] observations. For example, field studies have observed performance improvements in temperate conditions following pre-

season training camps in the heat.[315-317] While the mechanisms, magnitude and populations benefiting of such cross adaptations remain to be clarified, it should be acknowledged that there is no evidence that heat acclimation impairs cool weather performance. As such, heat acclimation should be implemented by default before a competition with uncertain environmental conditions potentially changing from temperate to hot.

Conclusion

One of the primary aims of warming-up is to increase T_m. This has several benefits for performance, to which we should add the non-temperature related benefits of warming-up (e.g., psychological preparedness). However, the concomitant increases in thermal strain (i.e., T_{skin} and T_{core}) might be detrimental to prolonged exercise capacity in hot ambient conditions via the development of cardiovascular strain. The warm-up routine should therefore be adapted to the environmental conditions. For example, cooling interventions (e.g., cooling-vest) can minimize the increase in T_{skin} and T_{core} during the warm-up. Athletes should also minimize dehydration during the warm-up. Athletes and coaches should keep in mind that warming-up induces both temperature and non-temperature related responses and is an important part of the pre-competition preparation in all environmental conditions. However, while an increase in whole-body temperature is beneficial to performance in cold environments, the warm-up should be adapted before exercising in the heat to promote muscle adaptations while minimizing the increase in T_{skin} and T_{core} based on the event.

Abbreviations

CHO	carbohydrate
EHI	exertional heat illness
MVCs	maximum isometric voluntary contractions
PAP	post-activation potentiation
RAMP model	Raise, Activate and Mobilize, Potentiate
T_{core}	core temperature
T_m	muscle temperature
T_{skin}	skin temperature
VO_2 kinetics	oxygen uptake kinetics
VO_{2max}	maximal oxygen uptake

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