PROLONGED EXERCISE IN THE HEAT

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EXERCISE IN THE HEAT

It is well established that prolonged exercise performance is impaired when undertaken in hot climatic conditions. As exercise becomes protracted, body core temperature increases and is accompanied by an elevation in cardiovascular strain (e.g. a rise in heart rate) and perceived exertion. Performance consequently deteriorates as the ability to maintain a given power output or running velocity progressively declines.

Typically, resting internal body temperature is regulated around 37°C by thermoregulatory reflexes. The initiation of these thermolytic (cutaneous vasoconstriction and shivering) and thermogenetic (cutaneous vasodilation and sweating) reflexes allows for heat to be conserved or dissipated to the environment by way of conduction, convection, radiation and evaporation. The onset threshold and intensity of the reflexes will depend on the severity of the thermal stimulus and the thermal gradient between the skin and the environment. When exercise is performed in cool conditions the gradient is negative as the temperature of the skin is lower than that of the environment. This allows for the efficient dissipation of metabolically generated heat. As a result, body temperature rises to a safe steady state. However, heat dissipation pathways are less effective in hot environments due to a reduction in the thermal gradient, whereby skin temperature approaches or surpasses environmental temperature. The rise in body core temperature is thus exacerbated, especially if relative humidity is high and sweat evaporation is limited by a low vapour pressure gradient between the air and the skin.

Interestingly, the rise in body core temperature during exercise is directly proportional to workload and relative intensity. Given that mechanical efficiency varies from 20 to 25%, most of the metabolic energy converted to produce movement is released as heat. For example, exercise performed at 80 to 90% of maximal aerobic capacity (VO_2 max) could increase core temperature by 1°C every 5 to 8 minutes if heat was not dissipated. As such, the rate of rise in body temperature during exercise is related to both the human body's ability to release heat to the environment and the intensity of exercise.

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DATE	VENUE (COMPETITION)	NO. OF STARTERS	NO. OF DNFs	% DNF	START TIME	WINNING TIME	LAST TIME	T_{amb} (°C)	RH (%)
14 Aug 1983	*Helsinki (WCh I)*	*63*	*19*	*23*	*14:00*	*2:10:03*	*3:03:10*	*18.0*	*61*
12 Aug 1984	*Los Angeles (OG)*	*78*	*29*	*27*	*17:00*	*2:09:21*	*2:52:19*	*23.2*	*35*
6 Sep 1987	*Rome (WCh II)*	*47*	*18*	*28*	*16:30*	*2:11:48*	*3:12:33*	*22.0*	*74*
2 Oct 1988	*Seoul (OG)*	*98*	*20*	*17*	*14:35*	*2:10:32*	*3:1402*	*24.5*	*65*
1 Sep 1991	*Tokyo (WCh III)*	*60*	*24*	*40*	*06:00*	*2:14:57*	*2:56:36*	*26.0*	*73*
9 Aug 1992	*Barcelona (OG)*	*87*	*25*	*22*	*18:30*	*2:13:23*	*4:00:44*	*26.6*	*65*
14 Aug 1993	*Stuttgart (WCh IV)*	*43*	*26*	*38*	*17:40*	*2:13:57*	*2:55:17*	*25.0*	*63*
12 Aug 1995	*Gothenburg (WCh V)*	*53*	*25*	*32*	*14:00*	*2:11:41*	*2:38:37*	*25.0*	*41*
4 Aug 1996	*Atlanta (OG)*	*111*	*13*	*10*	*07:05*	*2:12:36*	*4:24:17*	*23.9*	*87*
10 Aug 1997	*Athens (WCh VI)*	*70*	*38*	*35*	*08:05*	*2:13:16*	*3:14:30*	*32.7*	*43*
26 Aug 1999	*Seville (WCh VII)*	*65*	*15*	*19*	*18:45*	*2:13:36*	*2:59:20*	*33.2*	*36*
1 Oct 2000	*Sydney (OG)*	*81*	*19*	*19*	*16:00*	*2:10:11*	*3:09:14*	*19.5*	*42*
3 Aug 2001	*Edmonton (WCh VIII)*	*73*	*23*	*24*	*18:45*	*2:12:42*	*2:45:10*	*19.0*	*58*
30 Aug 2003	*Paris (WCh XI)*	*69*	*20*	*22*	*14:20*	*2:08:31*	*2:33:31*	*15.0*	*72*
29 Aug 2004	*Athens (OG)*	*81*	*21*	*21*	*18:00*	*2:10:55*	*2:50:26*	*30.0*	*39*
13 Aug 2005	*Helsinki (WCH X)*	*61*	*34*	*36*	*14:20*	*2:10:10*	*2:36:31*	*17.0*	*88*
25 Aug 2007	*Osaka (WCh XI)*	*85*	*28*	*33*	*07:00*	*2:15:59*	*3:03:47*	*30.0*	*64*
24 Aug 2008	*Beijing (OG)*	*95*	*19*	*20*	*07:30*	*2:06:32*	*2:41:08*	*26.5*	*62*
22 Aug 2009	*Berlin (WCh XII)*	*91*	*21*	*23*	*11:45*	*2:06:54*	*2:47:55*	*22.0*	*69*
4 Sep 2011	*Daegu (WCh XIII)*	*67*	*16*	*24*	*09:00*	*2:07:38*	*2:38:33*	*25.0*	*61*
12 Aug 2012	*London (OG)*	*105*	*20*	*19*	*11:00*	*2:08:01*	*2:55:54*	*21.5*	*68*

Table 1: World Championships and Olympic Games marathons for men. WCh=World Championships, OG=Olympic Games, DNF=did not finish, T_{amb}=ambient temperature, RH=relative humidity. Adapted with permission from Martin[1].

The marathon example

One of the ultimate challenges of human endurance is that of the marathon (42 km). During a 2 hour 10 minute marathon the calculated rate of heat production is approximately 1400 $kcal.h^{-1}$. Depending on the climatic conditions (i.e. temperature and humidity), performance may be severely impaired if the rate of heat loss does not offset the rate of heat gain. Table 1 illustrates the influence of hot and humid climatic conditions on Olympic and International Association of Athletics Federation World Championship marathon performances from 1983 to 2012. Greater decrements in performance, represented by slower winning times and greater percentages of athletes not finishing the race, occur when temperature and humidity are high at the start of the race and progressively increase (e.g. Tokyo 1991, Athens 1997, Osaka 2007). Conversely, the top 10 marathons of all time, ranging from 2:03:38 to 2:04:50, have all been run on occasions when the temperature at the start of the race was below 13°C. In fact, optimum temperature for competitive endurance performance is around 11 to 12°C. A notable exception is the performance of Kenyan runner Sammy Wanjiru at the 2008 Beijing Olympics. Despite a starting ambient temperature of 26.5°C and a relative humidity of 62%, he ran a blistering 2:06:32. This undoubtedly reflected his tremendous fitness, acclimatisation state and mental strength.

PERFORMANCE LIMITATIONS

Although field and laboratory-based studies have both demonstrated that aerobic exercise is impaired in the heat, contention remains as to the cause of the impairment. Therefore, a question within the field of environmental exercise physiology is whether performance is impaired via cardiovascular or central nervous system limitations. This question has led to the development of various experimental models attempting to reveal the mechanism(s) limiting prolonged exercise performance under heat stress. Currently, two mechanisms are proposed to explain the potential pathway by which fatigue develops. These hypotheses include:

- Cardiovascular limitations impairing systemic oxygen delivery and local oxygen uptake in exercising muscles and
- The attainment of a specific hyperthermic state, reducing central neural drive to exercising muscles.

Cardiovascular strain

It is well known that a rise in thermal strain (i.e. core and skin temperature) during prolonged moderate intensity exercise in the heat leads to a reflex increase in skin blood flow (Figure 1). This thermoregulatory mediated redistribution of blood towards cutaneous vascular beds increases skin temperature and narrows the core-to-skin temperature gradient.

In turn, this increases the skin blood flow requirements for heat dissipation, especially via convection. Consequently, competition for blood flow develops between thermoregulatory (skin blood flow) and

metabolic (muscle blood flow) processes. During exercise at a fixed workload, this competition is manifested as a decrease in central blood volume, which is accompanied by a reduction in mean arterial pressure and a progressive increase in heart rate towards maximum (Figure 2).

The rise in heart rate decreases ventricular filling time and end-diastolic volume, ultimately reducing stroke volume and compromising the maintenance of cardiac output. When exercise is protracted and performed to exhaustion, these circulatory adjustments result in the attainment of maximum heart rate at submaximal workloads. This leads to a reduction in maximum cardiac output, and concomitantly VO_2 max, which has been shown to decrease in proportion to the rise in thermal strain[2]. Thus, the large displacement of blood flow toward the skin during constant load aerobic exercise in the heat appears to play a key role in the development of cardiovascular strain and fatigue.

It was recently demonstrated that a progressive rise in cardiovascular strain was similarly associated with reductions in performance during self-paced exercise in the heat[3]. During a 40 km cycling time trial in hot and cool conditions, it was noted that the development of thermal strain during the hot trial resulted in a sustained elevation in heart rate. It was also noted that mean arterial pressure, stroke volume and cardiac output decreased throughout the hot exercise time trial. This occurred despite the maintenance of a lower power output.

During the final maximal effort from 39 to 40 km it was found that peak oxygen uptake declined significantly more in the hot (18%) than in the cool condition when compared with baseline VO_2 max measurements. The reduction was associated with an inability to increase mean arterial pressure and cardiac output, despite the attainment of maximum heart rate. These alterations in cardiovascular function contribute to the premise that exercise in the heat, whether performed at a constant rate or self-paced, is significantly influenced by circulatory adjustments.

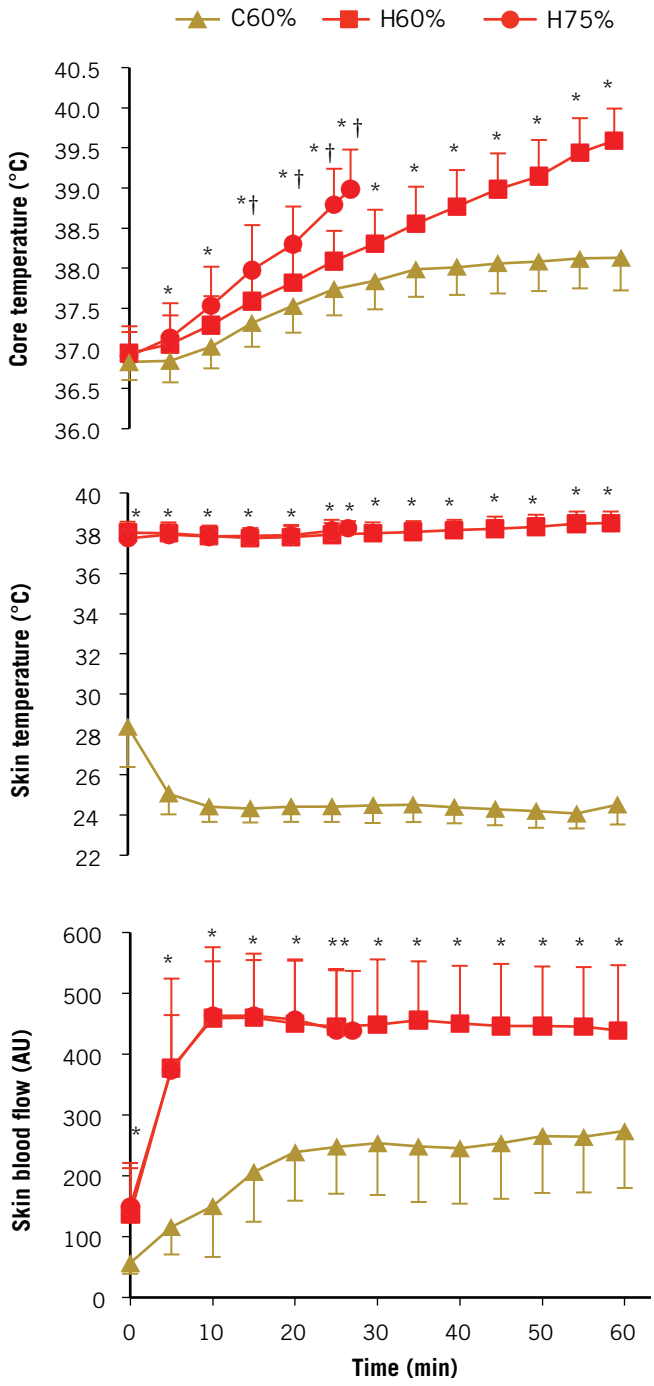

Figure 1: Core temperature, mean skin temperature and forearm skin blood flow during 60 minutes of steady state exercise in a control condition (C60%) and at 60% (H60%) and 75% (H75%) VO_2 max to exhaustion in the heat. * Significant difference between both exercise conditions in the heat vs C60%, $P<0.05$. † Significant difference between H60% and H75%, $P<0.05$. Values are means ± SD for 10 subjects. Reprinted with permission from Périard et al[10].

Central nervous system alterations

The premise that central nervous system function impairs prolonged exercise performance in the heat originates from observations that fatigue consistently occurs at a specifically elevated core temperature. The attainment of this 'critical' core temperature during exercise-induced hyperthermia has been suggested to elicit a reduction in central neural drive to exercising muscles (central fatigue). It is proposed that alterations in the cerebral motor cortex act to reduce voluntary muscle activation in order to limit the accumulation of heat and prevent thermal injury.

To explain this mechanism, researchers have evaluated voluntary activation levels using electrical stimulation during sustained maximal voluntary isometric contractions. Such contractions were performed after cycling to exhaustion in the heat, as well as following submaximal steady state exercise in cool conditions for a comparable time period[4]. It was shown that greater losses of force production occurred after exercise in the heat because

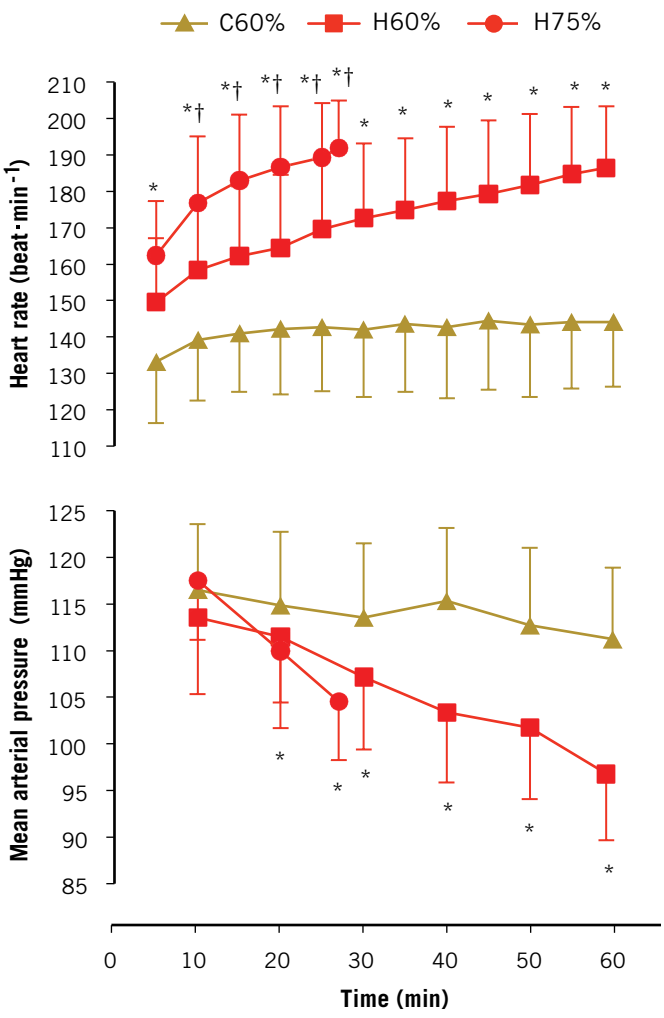

Figure 2: Heart rate and mean arterial pressure during 60 minutes of steady state exercise in a control condition (C60%) and at 60% (H60%) and 75% (H75%) VO_2 max to exhaustion in the heat. * Significant difference between both exercise conditions in the heat vs C60%, P <0.05. † Significant difference between H60% and H75%, P <0.01. Values are means ± SD for 10 subjects. Reprinted with permission from Périard et al[10].

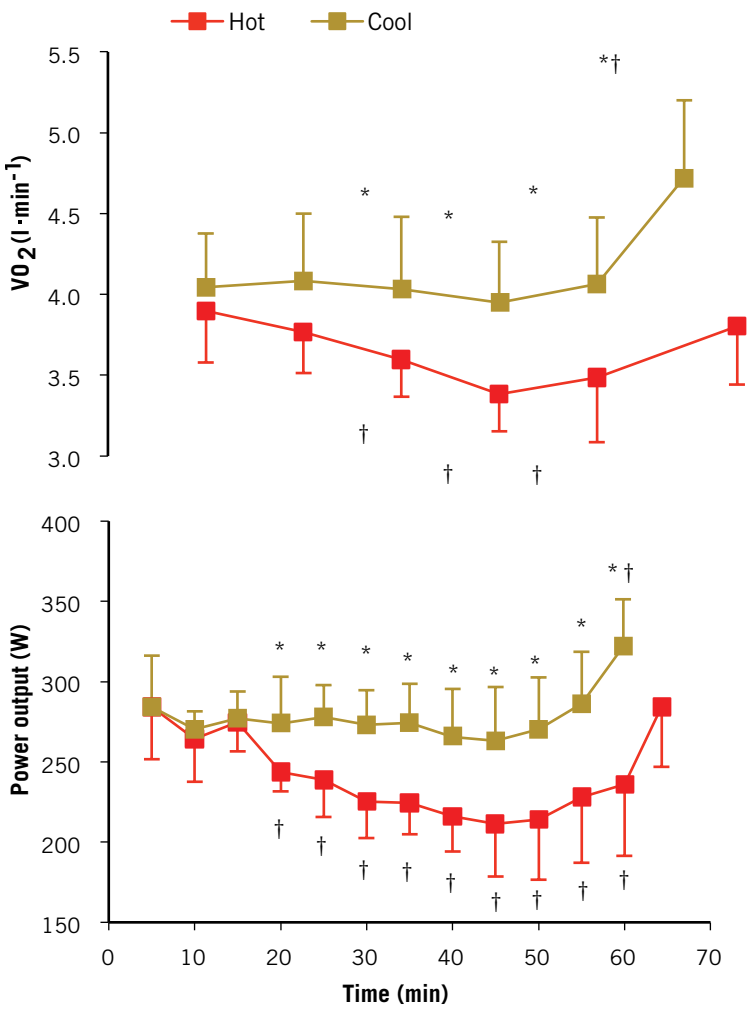

Figure 3: Oxygen uptake at 10 minute intervals and mean power output at 5 minute intervals and during the final kilometre at maximal effort during a 40 km cycling time trial in hot and cool conditions. Values are means ± SD for eight subjects. * Significantly higher than hot (P <0.05). † Significantly different from 10 minutes (P <0.05). Reprinted with permission from Périard et al[3].

of a decline in central neural drive to the active musculature. However, it has recently been demonstrated that the loss of force production capacity is not entirely attributable to central fatigue following fixed and self-paced exercise in the heat[5,6]. In fact, only 20 to 40% of the additional loss of force could be linked to central factors. The remainder was related to alterations occurring at the peripheral level in the muscle. As such, the precise contribution of hyperthermia-induced central fatigue to the loss of force production capacity requires further investigation.

Interestingly, increases in core and skin temperature are associated with reductions in cerebral blood flow and cerebral oxygen delivery during moderate-intensity exercise to exhaustion in the heat. It is suggested that these reductions, along with an increase in cerebral temperature, may mediate the development of central fatigue. However, reductions in cerebral perfusion prior to exhaustion are associated with an increase in oxygen extraction, which enhances global brain metabolism and central nervous system drive[7]. This indicates that a significant oxygen reserve is maintained by the brain at exhaustion, which may act to protect it against declines in perfusion. Furthermore, it is important to note that although exhaustion often occurs at an elevated core temperature, it also occurs in conjunction with a significant level of cardiovascular strain. A recent comparison of the thermal and cardiovascular responses of trained and untrained subjects exercising to exhaustion at moderate and high intensities in the heat demonstrated this phenomenon[8]. It was shown that both groups fatigued earlier at the higher intensity, attaining a 0.5°C lower core temperature at exhaustion. However, the level of cardiovascular strain reached at exhaustion was similar between exercise intensities and fitness groups.

COUNTERMEASURES AND STRATEGIES TO IMPROVE PERFORMANCE IN THE HEAT

Although aerobic exercise performed in the heat is impaired relative to when

it is conducted in cool conditions, various strategies can be utilised to minimise the impairment.

Fitness

Aerobic fitness in itself improves the capacity to exercise in the heat and to tolerate greater levels of hyperthermia. This benefit stems from the physiological adaptations that occur in response to regular exposure to high core temperatures during exercise. Well-trained athletes have a larger plasma volume and may display better central venous pressure and cardiovascular stability under heat stress than their untrained counterparts. Trained individuals also exhibit larger net gains in body core temperature during heat-stress exercise, mostly due to their lower starting core temperature. Other proposed adaptations include a greater sensitivity in the thermal effector responses for cutaneous vasodilation and sweating. This corresponds to an earlier onset of heat dissipation, which attenuates the rise in body core temperature.

Acclimatisation

Heat acclimatisation is the process by which we develop a tolerance to the heat. When an individual is first exposed to unusually hot environmental conditions these may seem very stressful. However, following a few days the conditions will appear more tolerable. Initially, acclimatisation can be viewed as a behavioural adaptation whereby activity level is reduced and a change occurs in diet and the selection of clothing. The greatest adaptations, however, are those that occur physiologically. During acclimatisation, blood volume increases, resting core temperature decreases, as does the concentration of sodium in sweat and urine. From a sweating perspective, acclimatisation can be viewed as a training of the sweat glands to produce more sweat. Maximal sweat rate increases and a given submaximal rate of sweating is achieved at lower core and skin temperatures. As a result, core temperature and cardiovascular strain are reduced for given levels of exertion because evaporative heat loss capacity is enhanced. These adaptations can develop naturally when exposed to the elements in an outdoor environment (acclimatisation), or artificially when performing a laboratory-based passive or exercise heat-exposure regimen (acclimation).

Hydration and cardiovascular strain

During hyperthermia, cardiovascular function is greatly influenced by hydration status. In fact, gradual dehydration has been shown to exacerbate the rise in thermal and cardiovascular strain during heat-stress exercise in proportion to the extent of body water loss[2]. This accelerates the reductions in cardiac filling and stroke volume, impairing the maintenance of cardiac output and systemic vascular conductance. It leads to a decrease in systemic, skin and muscle blood flow. It also leads to a greater reliance on muscle glycogen as fuel, and a reduction in exercising muscle oxygen uptake at exhaustion.

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Therefore, it is important that the extent of dehydration or body water loss be minimised in order to maintain performance. Currently, it is suggested to prevent per cent body mass losses beyond 2% when exercising in the heat to maintain performance. However, these recommendations are mostly based on laboratory experiments in which exercise intensity and workload are controlled. In field settings, athletes have been shown to have reached high core temperatures and lose significant amounts of body mass via sweating, while incurring minimal reductions in performance. However, the ability to perform in the heat may relate to various factors including pre-exercise hydration status, acclimatisation state, exercise intensity and aerobic fitness. Thus, it is better to err on the side of caution and undertake exercise in a well-hydrated state and limit the loss in total body water by hydrating properly during sustained exercise.

Pre-cooling

In an effort to reduce the starting core and skin temperature of athletes competing in the heat, researchers have developed various ways in which the body can be pre-cooled. This strategy is used to provide a wider range in which core temperature can rise, thus delaying the increase in cardiovascular strain. Several strategies can be employed to pre-cool athletes prior to competition including cold-water immersion, wearing ice vests, draping with cold towels and ingesting cold slurry drinks. Pre-cooling has been reviewed in a previous edition of this journal and the reader is referred to the article by Laursen[9] for a comprehensive and practical examination of current practices.

SUMMARY

Prolonged exercise performance is impaired in hot environments. Although heat acclimatisation and strategies such as pre-cooling can improve performance and exercise-heat tolerance, the deleterious effects of hyperthermia are likely to develop when exercise is undertaken in hot and/or humid conditions. The mechanisms potentially mediating this performance decrement remain hotly debated. At present, fatigue cannot be attributed to a single causative factor as exercise in the heat is a multi-factorial endeavour influenced by alterations in circulatory, metabolic and neuromuscular function. The interplay of psychological factors further influences performance and our understanding of fatigue by modifying behavioural responses. However, it appears that in motivated individuals, a thermoregulatory-mediated rise in cardiovascular strain may play a primary role in mediating fatigue during exercise in the heat.

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