Exercise in the Heat: Thermoregulatory Limitations to Performance in Humans and Horses

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Abstract/Résumé

This paper reviews the limits to exercise imposed by increases in ambient, hypothalamic, and contracting skeletal muscle temperature in humans and horses. Like humans, horses frequently compete in hot environments, yet their high mass-specific rate of heat production and low mass-specific surface area for heat dissipation places them at a great disadvantage compared to humans. Exercise in hot conditions increases the rate of body heat storage and reduces the time required to reach a critical hypothalamic temperature that results in voluntary fatigue. This critical temperature appears to be associated with dysfunction of the brain’s motor control centres. The ensuing voluntary cessation of exercise appears to coincide with temperature-induced alterations in skeletal muscle function with increased requirement for anaerobic ATP provision. The duration of exercise that can be performed before this critical temperature is reached can be increased by ingesting fluids, of a volume at least equal to that lost in sweat, within 60 min prior to and during exercise. Emerging research in the area of skeletal muscle heat dissipative mechanisms involves heat-induced increases in muscle sympathetic nerve activity, producing stimulation of CIII and CIV afferent nerve stimulation, and heat-induced release of nitric oxide within skeletal muscle and skin, producing muscle and skin vasodilation.

Cet article analyse les effets de l’augmentation des températures ambiante, hypothalamique et intramusculaire chez les humains et les chevaux. Tout comme les humains, les chevaux sont fréquemment en compétition par temps chaud. Comparativement aux humains, les

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chevaux ont un désavantage important: leur forte de production de chaleur relativement à la surface cutanée de déperdition. L'exercice par temps chaud accroît le taux de stockage de la chaleur corporelle et, de ce fait, réduit la période pour atteindre la température hypothalamique critique associée à la manifestation de la fatigue. Cette température critique semble reliée à une dysfonction des centres moteurs encéphaliques. L'arrêt volontaire de l'exercice semble coïncider avec une modification d'origine thermorégulatrice des fonctions musculaires avec augmentation des besoins anaérobies d'énergie. La durée de l'effort possible avant l'atteinte de cette température critique est prolongée par l'ingestion, 60 min avant ou durant l'exercice, d'un volume liquidien équivalent ou supérieur à celui perdu dans la sueur. Des nouveaux secteurs de recherche sur les mécanismes de dissipation de la chaleur musculaire sont: l'accroissement par la chaleur de l'activité nerveuse sympathique dans le muscle avec stimulation des fibres afférentes CIII et CIV; la libération par la chaleur d'oxyde nitrique dans le muscle et la peau avec vasodilatation des vaisseaux de ces régions.

Introduction

It has been well known for centuries, with a solid scientific literature dating from the 1930s, that heat impairs exercise performance (reviewed by Barr, this issue). For athletes, coaches, and physiologists, it is important to understand the mechanisms by which the thermoregulatory limitations to performance occur. For the competing athlete, exercise in the heat imposes an additional challenge to maintaining high levels of physical and mental function due to demands placed on mechanisms to regulate thermal and fluid balance. This paper presents an overview, focusing on the recent literature, of the main mechanisms leading to increases in core, hypothalamic, and muscle temperatures that may result in performance limitations.

With the onset of exercise, heat is produced by the metabolic conversion of chemical energy to the mechanical energy required for muscular contraction and limb movement. At best these processes operate at 25% efficiency, such that 75 to 80% of the chemical energy is transformed to heat within the contracting muscles. Heat moves from contracting skeletal muscle to surrounding tissues by conduction and convective flow of lymph and blood. If the intensity of muscular contraction is very high and/or the rate of heat loss from contracting muscles is insufficient, there may occur an excessive increase in muscle temperature that results in cellular injury (Brinnel et al., 1987) with ensuing impairment of muscular performance. Another mechanism by which a thermoregulatory limitation to performance may be reached is through an increase in brain temperature sufficient to result in fatigue (Nielsen et al., 1993). In general, these thermal limits to exercise performance may also be reached in cool environments when the rate of metabolic heat production is high, despite the presence of a large thermal gradient for dissipating heat to the surrounding air. When the air is hot, however, the thermal gradient for heat loss is reduced so that the rate of heat accumulation by the body is increased and fatigue, or an inability to exercise voluntarily, occurs sooner than when exercising in cool conditions (Werner, 1993).

An additional purpose of this paper is to compare the exercising horse to the exercising human. Both mammals produce heat rapidly at the onset of muscular contraction, and both primarily use evaporative cooling of sweat to thermoregulate
Table 1  Comparison of Physiological Indices of Heat Production and Heat Dissipation Between Horses and Humans

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Horse</th>
<th>Human</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass (BM, kg)</td>
<td>500</td>
<td>70</td>
</tr>
<tr>
<td>Contracting muscle mass (CMM, kg)</td>
<td>200</td>
<td>14</td>
</tr>
<tr>
<td>CMM (% BM)</td>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>Skin surface area (SA, m²)</td>
<td>5</td>
<td>1.8</td>
</tr>
<tr>
<td>BM : SA ratio</td>
<td>100</td>
<td>40</td>
</tr>
<tr>
<td>CMM : SA ratio</td>
<td>40</td>
<td>7.8</td>
</tr>
<tr>
<td>Max sweat rate (ml·m⁻²·min⁻¹)</td>
<td>50</td>
<td>30</td>
</tr>
<tr>
<td>% of total sweat used for cooling</td>
<td>25–30</td>
<td>30–50</td>
</tr>
<tr>
<td>Total sweat ion concentration (meq/L)</td>
<td>300⁺</td>
<td>50–80</td>
</tr>
</tbody>
</table>


during exercise (Geor and McCutcheon, 1998a; Werner, 1993). In many respects the similarities end here (Table 1). A running horse uses a greater proportion of its body mass for locomotion than does a human who is performing running or leg cycling exercise, so horses have a greater rate of heat production per unit of body mass. This poses a severe thermoregulatory limitation for the exercising horse because, compared to humans, it only has about 50% the skin surface area per unit body mass. Therefore, in terms relative to the running human, the running horse uses twice the muscle mass but only half the surface area for evaporative cooling. For these reasons, in hot humid conditions horses may gain heat at about four times the rate of humans. (For review, see Geor and McCutcheon, 1998a; Guthrie and Lund, 1998; Maughan and Lindinger, 1995; Sawka and Pandolf, 1990; Werner, 1993.)

**Exercise in the Heat**

ENVIRONMENTAL TEMPERATURE AND HEAT STORAGE DURING EXERCISE

The rate of body heat storage is a function of the rate of heat production and heat loss and, at a given steady-state exercise intensity, the rate of heat loss is a function of air temperature, velocity, and humidity (Werner, 1993). When humans exercise at cool temperatures (less than 15 °C) with low relative humidity (<60%), the body loses at least 50% of its heat by a combination of convection and conduction. Thermal balance may be maintained during exercise, albeit at a higher body temperature (Gisolfi and Copping, 1974). As environmental temperature rises, however, the thermal gradient for heat dissipation is reduced, resulting in decreased rates of convective and conductive heat loss while a larger proportion of heat is lost by evaporative cooling. In humans exercising at an ambient temperature of 30 °C (rh <60%), 70% or more of the heat loss occurs by evaporative cooling and about 20% is lost through convection and conduction (Werner, 1993).
Due to the physical limitations of surface area for heat dissipation in horses, conductive and convective heat loss is lower than in humans, comprising 9 to 13% of total heat loss with moderate intensity exercise in cool, dry conditions (Hodgson et al., 1993). As in humans, evaporative cooling in horses accounts for about 50% of total heat loss during moderate intensity exercise in cool, dry conditions. In horses, respiratory heat loss may be as high as 30% of heat produced during exercise at 40% of \( \text{VO}_2 \text{ max} \), compared to about 10% in humans (Figure 1). In non-sweating mammals the conductive transfer of heat from muscle through to the skin is an important route of heat loss (Ardevol et al., 1998).

\[\text{Figure 1. Routes of heat loss during exercise at about 40\% \text{VO}_2 \text{ max}. Human data from Werner (1993). Equine data from Hodgson et al. (1993).}\]

When humidity is increased, the water vapour gradient for evaporation of sweat is decreased, thereby reducing the rate of evaporative heat loss (Werner, 1993). Therefore the highest rate of heat storage occurs during exercise in hot, humid conditions. In thoroughbred horses (450 kg body mass) exercising on a treadmill at 50% of \( \text{VO}_2 \text{ max} \) at 20 °C and 50% rh, pulmonary artery (PA) temperature increased linearly over a 35-min period at a rate of 0.10 °C/min (Geor et al., 1995). In contrast, in hot, dry conditions (33 °C, 50% rh) the PA temperature increased at a rate of 0.15 °C/min, while in hot, humid conditions (33 °C, 83% rh) the PA temperature increased at a rate of 0.25 °C/min. Therefore the rate of heat storage when exercising in hot, humid conditions may be more than twice the rate that occurs during exercise at the same intensity in cool, dry conditions (Figure 2). It is noteworthy that the rate of increase of PA temperature was constant and, by extrapolation, after 1 hour of exercise at this intensity the PA temperature would have increased by 6 °C to just over 43 °C, much higher than the 40–41 °C core temperature reached in humans at cessation of exercise.
Figure 2. Rate of heat storage in humans during exercise at 45% VO2 max in cool dry (CD), hot dry (HD, Nielsen et al., 1993), and hot humid (HH, Nielsen et al., 1997) conditions compared to horses exercising at 50% VO2 max under similar conditions (Geor et al. 1995). In humans the increase in core temperature under cool dry conditions only occurs during first 10–15 min of exercise, after which a new steady-state core temperature is achieved.

In contrast to the horse, human athletes may exercise at relatively high intensities of 80–85% VO2 max for extended periods (e.g., marathons) under cool conditions and still maintain thermal balance, albeit at a higher body temperature than at rest (Galloway and Maughan, 1997; Gisolfi and Copping, 1974; Saltin and Hermansen, 1966; Sawka and Pandolf, 1990). In a study on male athletes exercising at 45% of VO2 max in hot, humid conditions (35 °C, 87% rh), exhaustion occurred at 45 min when core (deep esophageal) temperature reached 40 °C (Nielsen et al., 1997). The rate of increase in core body temperature was 0.066 °C/min, similar to that previously observed in hot, dry conditions (Nielsen et al., 1993).

RELATIONSHIP BETWEEN HEAT STORAGE AND VOLUNTARY FATIGUE

It appears there is a critical body temperature above which mammals will not continue to exercise voluntarily. The literature on humans consistently states that voluntary fatigue occurs at a core temperature of about 40 °C (Gisolfi and Copping, 1974; MacDougall et al., 1974; Nielsen et al., 1993, 1997; Saltin and Hermansen, 1966). Similarly, in rats exercising in various hot conditions, voluntary fatigue consistently occurred when hypothalamic temperature was 40 °C (Fuller et al., 1998). However, in horses (Hodgson et al., 1993; Marlin et al., 1996; McConaghy et al., 1995), swine (Armstrong et al., 1987), and goats (Caputa et al., 1986), voluntary exhaustion did not occur until core or rectal temperatures of 42 °C or
greater were reached. Thus there appears to be a difference in the thermal set point of the central temperature controller among mammalian species. It is of concern that in large quadruped mammals the thermal limit to exercise is similar to that associated with the induction of heat stroke.

**CONSEQUENCES OF INCREASED BRAIN TEMPERATURE**

An inability of the central temperature controller, or of the effector mechanisms responding to input from the hypothalamus, to simultaneously compensate for orthostatic, metabolic, and thermoregulatory demands may result in heat syncope, characterized by extreme peripheral vasodilation and decreased arterial pressure (Werner, 1993). This condition is not very harmful compared to heat stroke (or exhausted horse syndrome) where, during high heat stress, thermoregulation is subordinated to cardiovascular and metabolic demands (Werner, 1993).

At rest in hot environments, brain temperature may be increased by two main mechanisms: increased radiant heat load, and increased temperature of the blood due to warming of blood at the skin. With exercise in hot conditions a third mechanism becomes important: the convective flow of heated blood from contracting muscles to the core and brain contributes to the increase in brain temperature. In exercising humans (Saltin and Hermansen, 1966; Savard et al., 1988) and horses (Geor et al., 1995), skeletal muscle temperature is consistently about 1.5 °C greater than core temperature, and core temperature is about 1 °C greater than hypothalamic temperature (Brengelmann, 1993; McConaghy et al., 1995). Because of the brain’s central role in coordinating physiological responses among systems, it appears that in both humans (Brengelmann, 1993) and horses (Baptiste, 1998; McConaghy et al., 1995) the brain has mechanisms for selective cooling. But during exercise in hot/humid ambient conditions, these heat-dissipative mechanisms cannot cope with the rates of core and brain heating. There are thermal limits the brain can tolerate; beyond these limits a host of physiological reactions occur that may be aimed at reducing the rate of brain heating but often result in heat stroke. Important among these is the cessation of physical activity, which markedly reduces the rate of metabolic heat production.

Concerning the central nervous system, Nielsen and co-workers (1993, 1997) speculated that at a critical core temperature (40 °C in humans) there may be a negative effect on the brain’s motor control centres. Such effects are consistent with the loss of motor coordination and reduction in motor drive that is typical with prolonged exercise in the heat. In support of a direct effect of elevated core/hypothalamic temperature on impaired neuromuscular function are studies that show increased exercise time to fatigue when individuals exercise in cool conditions (Galloway and Maughan, 1997; Geor et al., 1995; Parkin et al., in press), when they are cooled during exercise (MacDougal et al., 1974), or when they have been cooled prior to exercise (Lee and Haymes, 1995). Associated with these central effects on motor function, a peripheral manifestation is an increase in electromechanical delay, or the time delay between myoelectric activity and muscle force generation. In a study of repeated maximal knee extension exercise in humans, a 2.1 °C increase in muscle temperature may have contributed 20–25% to the increase in electromechanical delay, from 38 ms before exercise to 56 ms postexercise (Zhou et al., 1998).
MECHANISMS BY WHICH MUSCLE TEMPERATURE IS INCREASED

The onset of muscular contraction is accompanied by a rapid increase in muscle temperature due to the inefficiency of metabolic energy conversion. In horses, during the first 15 min of exercise at 50% of VO$_2$ max in hot humid conditions, skeletal muscle (gluteus medius) temperature increases at a rate of 0.33 °C/min, and muscle temperatures approach 45 °C with continued exercise (Geor et al., 1995; Hodgson et al., 1993). This may be an upper limit for continued muscle function because some enzymes may denature, thus altering metabolism and cellular structure (Brinnel et al., 1987).

As mentioned above, the primary mechanisms for muscle cooling are the conductive transfer of heat to surrounding tissues and the convective flow of heated blood away from the contracting muscles. The exercise hyperemia associated with the onset of exercise is also beneficial, and under cool, steady-state conditions the convective heat removal from contracting tissues may keep pace with heat production. Accordingly, tissue heating as well as decreases in circulating volume would both act to reduce the rate of heat transfer from contracting muscles.

Another mechanism may also aid in heat removal from muscle during submaximal exercise lasting longer than 15 minutes. At the onset of exercise, large and rapid increases in muscle osmolality result in a net shift of fluid into contracting muscles, and this effect is primarily responsible for the rapid and pronounced decrease in plasma and blood volume (Lindinger et al., 1994; 1995). As exercise continues, much of this fluid, now heated, shifts back into the circulation as muscle phosphocreatine is resynthesized and lactate efflux increases, effectively reducing intracellular osmolality. As exercise continues, however, plasma and blood volume remain lower than at rest because about 50% of the fluid that has shifted out of the vascular compartment at the onset of exercise does not return until postexercise (Lindinger et al., 1995). Also, within 10 min of beginning exercise, fluid starts to be lost as sweat on the skin’s surface. This fluid is initially derived from the plasma compartment, but an excessive decrease in plasma (and hence blood) volume is prevented by the net shift of fluid from the intracellular compartment of many tissues (Jacobsson and Kjellmer, 1964; Lindinger et al., 1994).

With prolonged exercise in the heat, the decrease in vascular volume due to thermoregulatory sweating poses important problems for thermoregulation and cardiovascular function. There is competition among tissues for fluid and available cardiac output by both contracting muscles and skin to meet metabolic and cooling demands (McConaghy et al., 1996; Nielsen et al., 1997). The situation is exacerbated when exercise is prolonged without fluid replacement of lost sweat. As vascular volume decreases, so does stroke volume, but cardiac output and blood flow to contracting muscles and the skin are maintained by increases in heart rate (Geor et al., 1995; Nielsen et al., 1997; Savard et al., 1988).

As the temperature of the blood progressively increases, the thermal gradient for heat dissipation decreases, so that blood leaving the cutaneous circulation loses less heat than during early exercise. This is manifested by increases in PA blood and core temperatures and is referred to as uncompensable exercise-heat stress (Latzka et al., 1998; Nielsen et al., 1993). Furthermore, the increase in circulating blood temperature reduces the thermal gradient for heat transfer at contracting muscles and, if muscle activity is maintained, muscle temperature continues to increase (Geor et al., 1995; Hodgson et al., 1993; Saltin and Hermansen, 1966).
Associated with these responses is the defense of the vascular volume at the expense of intracellular fluid volume (Kozlowski and Saltin, 1964).

It is highly likely that the progressive dehydration of intracellular fluids during exercise in the heat contributes to the reported impairments of neural and skeletal muscle function, and contributes to the heat-induced fatigue response. Therefore, progressive decreases in vascular volume during exercise (exercise-induced dehydration) are associated with increasing skeletal muscle and body temperatures in both horses (Geor and McCutcheon, 1998b) and humans (Armstrong et al., 1997).

EFFECTS OF MUSCLE TEMPERATURE ON SKELETAL MUSCLE FUNCTION

In humans at rest in thermoneutral conditions, limb skeletal muscle temperature is about 36 °C (Saltin and Hermansen, 1966), while deep muscle temperature, e.g., in the middle gluteus of horses, is around 37.8 °C (Geor et al., 1995; Hodgson et al., 1993). With high intensity exercise, or with prolonged moderate intensity exercise in the heat, the temperature of contracting skeletal muscle increases and, in horses, may approach 45 °C. Thus the excursion in skeletal muscle temperature when going from rest to heat-induced fatigue may exceed 5 °C. A number of studies have shown that skeletal muscle function is affected over this range of temperature.

In vitro studies on rat skeletal muscle have shown that increasing muscle temperature from 35 to 40 °C resulted in a decrease in maximum isometric tetanic force, a 20% decrease in time to peak tension (attributed to increased myosin ATPase activity), a 20% decrease in 1/2 relaxation time (attributed to increased sarcoplasmic reticulum Ca ATPase activity), a 25% decrease in time to fatigue, an increase in stimulation frequency required to reach 70% of maximum tetanic force, and a 25% decrease in the number of stimuli required to fatigue the muscle (Segal et al., 1986). These changes would be associated with accelerated kinetics of action potentials (Ward and Thesleff, 1974), and the reduced relaxation time also points to a requirement for increased Na,K ATPase activity. These heat-induced increases in ATP utilization require a markedly increased metabolic rate to supply ATP.

Young and co-workers (1985) have shown that when humans perform leg exercise in the heat (49 °C, 20% rh), compared to cool conditions (21 °C, 30% rh), there is a decrease in the aerobic metabolic rate of contracting skeletal muscle and an increase in anaerobic metabolic rate, as demonstrated by increased muscle glycogen utilization and muscle lactate accumulation. It is now known, in humans at least, that these changes occur with minimal to no change in muscle blood flow or leg VO$_2$ (Savard et al., 1988). The increase in VO$_2$ reported during exercise in hot conditions (Gisolfi and Copping, 1974; Galloway and Maughan, 1997) must therefore be due to increased metabolism by heart and skin, as suggested by MacDougall et al. (1974). It is also known that increases in circulating epinephrine are greater when exercising in the heat, and that increased epinephrine may partly account for the increased rates of skeletal muscle glycogenolysis and lactate production (Febbraio et al., 1994; 1996). Exercise in the heat may also be associated with a reduction in muscle free fatty oxidation, as there is an increased rate of carbohydrate oxidation (Hargreaves et al., 1996a).

The contractile alterations in skeletal muscle performance with increased muscle temperature (Segal et al., 1986) also appear to be associated with
impairments in cellular calcium handling (Byrd et al., 1989), mitochondrial function (Brooks et al., 1971; Willis and Jackman, 1994), membrane phospholipid-protein interactions, and cell signaling pathways. Very few studies have been done in these areas that can be applied to the in vivo situation, and there is a need for continued research in these areas.

MECHANISMS TO INCREASE HEAT FLOW

There are three important ways in which dissipation of metabolic heat may be improved. The first is by continuous cooling of the skin to provide a large gradient between heat production sites and the skin for heat dissipation (Geor and McCutcheon, 1998a; MacDougall et al., 1974). The second is by heat acclimation or acclimatization, a topic well reviewed by Geor and McCutcheon (1998a) and Werner (1993). The third is by providing fluids during exercise, as discussed in detail by both Maughan and Galloway in this issue.

The importance of trying to maintain vascular and body fluid volume to improve thermoregulation and exercise performance during exercise in the heat is well known (Gisolfi et al., 1974) and continues to receive attention. The premise driving this concept is that maintenance of fluid volume helps to ensure increased rates of fluid flow to the heart and brain, the skin (heat loss site), contracting skeletal muscle (heat production site) and, lastly, other tissues. In humans, blood flow to contracting skeletal muscle does not appear to be compromised (see above), so the main benefit can be attributed primarily to increased skin blood flow. Leg blood flow in quadruped mammals such as the rat (Laughlin and Armstrong, 1993) and pigs (Armstrong et al., 1987) is also not compromised, but actually progressively increases (up to 49% increase) with time during prolonged treadmill exercise. It appears there are no studies that have examined the changes in skeletal muscle blood flow in horses during prolonged exercise, and increases in limb blood flow in exercising horses cannot be ruled out at this time.

Hargreaves et al. (1996b) found that fluid ingestion sufficient to replace sweat losses (compared to no fluids) during 2 hrs of cycling exercise at 67% VO\textsubscript{2} max at about 21 °C resulted in lower heart rate and lower rectal and contracting muscle temperatures, while muscle glycogen was greater and muscle lactate was lower at the end of exercise. Similar results were obtained in horses exercising on a treadmill at 50% VO\textsubscript{2} max in the heat (34.5 °C, 48% rh), and it was further demonstrated that provision of fluid 2 hrs prior to exercise provided no thermoregulatory advantage (Geor and McCutcheon, 1998b). Provision of fluids during the 1-hr period preceding exercise resulted in an increased endurance time in both horses (Ecker and Lindinger, 1996) and humans (Latzka et al., 1998) that can be attributed to an increase in the time required to reach a core temperature at which exhaustion from heat strain occurs (Latzka et al., 1998).

Rapidly emerging areas of study have identified other ways in which muscle temperature may be regulated. Muscle heating results in an increase in sympathetic nerve activity with stimulation of Group III and IV muscle afferents (Ray and Gracey, 1997). This may contribute to the increases in skeletal muscle vasodilation and blood flow associated with increased heart rate and cardiac output. Lastly, nitric oxide release in skeletal muscle also appears to be under thermosensitive control and may play an important role in modulating the blood supply to the vascular bed during exercise in the heat (Simon, 1998).
Conclusion

In conclusion, thermoregulatory mechanisms are in place to provide for effective transfer of heat from contracting skeletal muscle. However, when exercise intensity is too high, or ambient temperature too hot, the rate of heat dissipation is inadequate, resulting in progressive increases in skeletal muscle, core, and hypothalamic temperatures. These increased temperatures set into motion a series of responses that ultimately appear to result in both central and muscle (peripheral) fatigue, necessitating a reduction in exercise performance.

References


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