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Review

Distance running in hot environments: a thermal challenge to the elite runner

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Endurance performance is impaired in the heat, and a combination of high temperature and high humidity presents a major challenge to the elite marathon runner, who must sustain a high metabolic rate throughout the race. The optimum temperature for marathon performance is generally about 10–12 °C. The optimum temperature may be lower for faster runners than for slower runners. Sweat evaporation limits the rise in core temperature, but dehydration will impair cardiovascular function, leading to a fall in blood flow to muscle, skin and other tissues. There is growing evidence that the effects of high ambient temperature and dehydration on performance of exercise may be mediated by effects on the central nervous system. This seems to involve serotonergic and dopaminergic functions.

For sedentary individuals in moderate ambient conditions, body temperature is typically maintained at a relatively constant level. Even allowing for the circadian rhythm, where the lowest temperature occurs at around 4 a.m. and the highest in the late afternoon, between 4 p.m. and 6 p.m., the variation is typically less than about 0.5 °C above or below the normal value of 36.6 °C. The resting metabolic rate is low, and dissipation of the heat produced by metabolic activity is achieved through a number of routes of heat loss (primarily radiation, conduction, convection and evaporation). As ambient temperature rises, the effectiveness of heat loss by physical transfer becomes compromised, and may even be reversed: heat loss becomes increasingly dependent upon the evaporation of water from the respiratory tract and of sweat from the skin surface. Sweat evaporation is a very effective means of heat dissipation, as evaporation of 1 L of sweat removes about 580 kcal of heat energy, but high humidity limits the evaporation of sweat, so water and electrolytes continue to be lost without the loss of heat from the body. The water that forms sweat is derived primarily from the blood plasma, with fluid mobilized from intracellular fluid compartments in an attempt to maintain the circulating blood volume. Sweating can therefore prevent or at least limit the development of hyperthermia, but hydration and electrolyte balance are compromised in the process: cardiovascular and thermoregulatory function will also be impaired if large fluid deficits are incurred. Athletes can drink during endurance events, but the fluid intake is generally less than the sweat loss, leading to some degree of fluid deficit, especially among the faster runners (Noakes, 1993).

During exercise, about 75–80% of the metabolic energy turnover appears as heat, and the rate of heat production in runners is determined by a number of factors. It is roughly proportional to running speed and body mass, but depends also on the individual running economy (Foster & Lucia, 2007). Faster runners perform at a higher fraction of their aerobic capacity (Maughan & Leiper, 1983), and the high rates of heat production in these runners, combined with reduced opportunities for losses by physical exchange in hot weather, mean that sweat production becomes the primary, perhaps even the only, avenue of heat loss in these conditions. Without replacement of the sweat losses, the high rates of sweat loss typically encountered when exercising in hot and humid conditions will result in a progressive reduction in blood volume and stroke volume, eventually limiting muscle blood flow (Gonzalez-Alonso, 2007). This situation is compounded by the need to supply an increased skin blood flow, which is required to transport heat away from the deep tissues to the periphery. Body water losses have been demonstrated to result in elevated exercise heart rate and the impairment of thermoregulation during exercise, leading to an elevation in core temperature at the same absolute exercise intensity (Montain & Coyle, 1992). Hypohydration during exercise in the heat is

sometimes also associated with a reduction in stroke volume, cardiac output and blood pressure, as well as a marked decline in blood flow to the working muscles (Gonzalez-Alonso, 2007).

Undertaking prolonged exercise in the heat also appears to increase the reliance upon carbohydrate (CHO) oxidation, but unlike exercise in temperate conditions (Bergstrom et al., 1967), the muscle glycogen content remains high at the point of fatigue during exercise in the heat (Parkin et al., 1999). While substrate supply may limit endurance performance in cool environments, the primary problem for the runner in the heat appears to be one of thermoregulation. If CHO depletion does not limit performance in the heat, it would be expected that increasing muscle glycogen stores would have no effect on endurance performance in these conditions. However, Pitsiladis and Maughan (1999) showed that a pre-exercise CHO loading regimen increased cycling endurance time in the heat (30 °C) from 44.0 min after a low-CHO diet to 53.2 min after a high-CHO diet. At the same power output, endurance time in the cold (10 °C) was 89.2 min after the low-CHO diet and 158.2 min after the high-CHO diet.

Owing to the limitations on invasive monitoring in the field, most of the available data come from laboratory studies, and there is more evidence from cycling than from running models. It is well recognized from observations of athletes in the field, however, that endurance running performance is affected by environmental conditions. Even before statistical analysis of performance was carried out, it was apparent that athletes were more likely to encounter problems in hot environments than in temperate or cool environments. Dorando Pietri at the 1908 Olympic Games in London, where the weather was an unusually warm 26 °C, and Jim Peters at the 1954 Empire Games in Vancouver, where temperature during the race was in excess of 28 °C, both of whom collapsed on the track within sight of the finish line while leading the race, are only two of many examples.

Frederick (1983) made an attempt to examine the effects of environmental temperature on marathon running performance (Fig. 1). From this, it appears that there is an optimum temperature range for performance and that this lies somewhere between about 10 °C and 15 °C. Performance falls off progressively as the temperature either decreases of increases from this optimum. There are, however, a number of limitations to this analysis, as was well recognized by the author. There is likely to be some selection bias in the data as the best runners often choose not to compete at extremes of environmental conditions. The temperature data are inevitably limited: over the course of a marathon, there can be substantial changes in environmental temperature

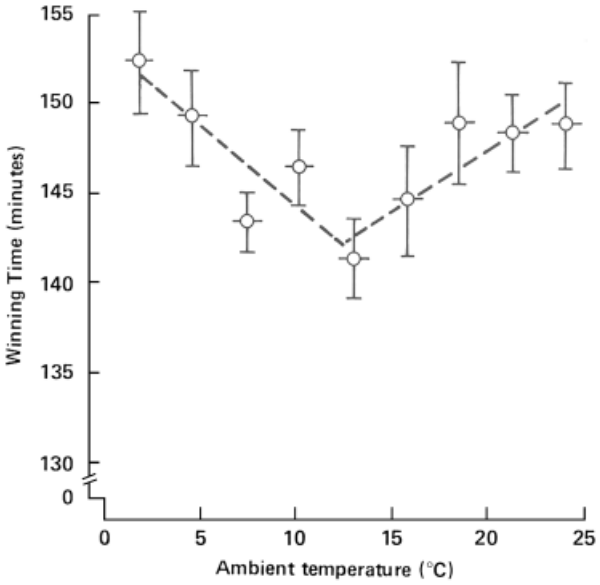

Fig. 1. Winning times at a series of marathon races in relation to ambient temperature. Redrawn from Frederick (1983).

that are not reflected in a single mean value. A falling temperature is likely to result in a more favorable outcome than a rising temperature. Other aspects of the environment are also important: humidity may be as important as air temperature as it will have a strong influence on the evaporative capacity; wind speed and direction will have a strong influence on heat exchange by convection; altitude will also affect performance. No women were included in the data analysis and the female athlete may respond differently. Finally, the analysis included no performance faster than 2 h 19 min, so may be of limited relevance to the elite athlete.

In spite of these limitations, however, the main conclusion of this data analysis was confirmed by later analyses. Buoncristiani and Martin (1983) showed that an ambient temperature (Ta) of 10–12 °C is optimal. In a much larger data set involving records from seven marathon races for periods of 6–36 years, Ely et al. (2007b) showed from a cross-sectional analysis of performance data that there is a progressive slowing of marathon performance as the wet bulb globe temperature (WBGT) increases from 5 to 25 °C. This seems true for both men and women, but increasing ambient temperature had a greater negative effect on performance among the slower populations of runners. Further analysis (Ely et al., 2008) of female runners from three women's marathons for which intermediate times were available at 5 km intervals showed that the faster runners ran at an even pace throughout the race while the slower runners ran progressively more slowly throughout the race: increasing Ta slowed the faster runners more than it did the slower runners, but the faster

runners still maintained a constant pace, i.e. they ran more slowly from the start of the race. The same authors have also shown (Ely et al., 2007a) that the solar load does not have a major influence on the relationship between temperature and performance. These responses are very similar to observations from laboratory studies of endurance cycling which showed that mean exercise time to fatigue during cycling exercise at about 70% of VO_{2max} was higher at an ambient temperature of 11 °C (93.5 min) than at 4 °C (81.4 min), 21 °C (81.2 min) or 31 °C (51.6 min; Galloway & Maughan, 1997).

The decision to hold the 1996 Olympic Games in the hot and humid environment of Atlanta stimulated much interest in the problems of faced by athletes competing in endurance events in the heat. A consideration of thermal physiology led Nielsen (1996) to calculate that it would not be possible for a runner to complete a marathon in the conditions expected at the Atlanta Olympics in <3 h. This was based on a series of assumptions and the insertion of standard values into the heat balance equation:

$$S = M \pm R \pm K \pm C - E \pm \mathrm{Wk}$$

where the rate of body heat storage (S) is equal to the metabolic heat production (M) corrected for the net heat exchange by radiation (R), conduction (K), convection (C) and evaporation (E). It is a reasonable assumption in running that no external work (Wk) is done. It is also reasonable to ignore any heat exchange by conduction as this will be negligible in running.

The following equations can be applied to estimate heat exchange:

Convective loss	$C = 8.3(\mathrm{Tsk} - \mathrm{Ta})\sqrt{v}$	$W/°C/m^2$
Radiant loss	$R = 5.2(\mathrm{Tsk} - \mathrm{Tmrt})$	$W/°C/m^2$
Evap. loss	$E = 124(\mathrm{Psk} - \mathrm{Pa})\sqrt{v}$	$W/kPa/m^2$

where Tsk = Mean skin temperature (°C), Ta = Ambient temperature (°C), Tmrt = Mean radiant temperature (°C), Psk = Mean skin water vapor pressure (kPa), Pa = Ambient water vapor pressure (kPa), v = Mean air velocity (m/s)

Nielsen's calculations were based upon a fictional runner with the following characteristics: body mass 67 kg; body surface area (BSA) 1.85 m^2; running speed 19.4 km h^{-1} (5.4 m/s) equivalent to a finishing time of 2 h 10 min; ambient temperature 35 °C; relative humidity 80%; mean skin temperature 37 °C; oxygen cost 4.3 L/min; metabolic heat production 1440 W; mechanical efficiency 25%. In addition to this long list must be added the assumption that mean air velocity over the entire BSA is uniform and equals the running speed.

For the sake of simplification, it can also be assumed that heat exchange by convection and radiation are negligible and can be ignored, leaving only evaporative heat loss to balance the rate of metabolic heat production. The maximum possible rate of evaporative heat loss is given by

$$\begin{aligned} E &= 124(\mathrm{Psk} - \mathrm{Pa})\sqrt{v} \times \mathrm{BSA} \\ &= 124 \times (6.27 - 4.42) \times \sqrt{5.4} \times 1.85\,\mathrm{W} \\ &= 995\,\mathrm{W} \end{aligned}$$

This is possible with evaporation of 1475 g sweat/h. Based on the estimated rate of metabolic heat production as indicated above, heat gain will exceed the evaporative capacity of the environment by 445 W. Heat capacity of human tissue is 3.47 kJ/ °C, so body temperature would rise by 1 °C every 8.7 min. Applying the same assumptions and assuming that core temperature cannot be allowed to rise beyond 40 °C, Nielsen calculated that the fastest time possible in these environmental conditions would be about 3 h 20 min.

With the benefit of hindsight, we can see that the winning time for the men's race in Atlanta was far superior (2 h, 12 min, 36 s) and the women's winning time was also faster than this (2 h, 26 min, 05 s). The performances are real, so the assumptions must be flawed, even taking into account the fact that environmental conditions were less taxing than those used in the calculations. An examination of the possible sources of error requires a careful consideration of the factors that allow the elite endurance athlete to perform so well in these conditions.

Several factors may contribute to a lower than calculated energy cost; in particular, a lower body mass or a better running economy will reduce the energy cost of running at any given speed. The assumption of a body mass of 67 kg is perhaps not representative of elite marathon runners, and more especially not of the elite East African runners who have dominated distance running in recent years. The characteristics of these runners have been studied extensively in recent years and Table 1 shows some data representative of the body mass of African and Caucasian runners where comparisons have be made of factors that may influence performance.

Table 1. Body mass (kg) of male African and Caucasian runners from studies where factors related to running economy and performance have been compared

African	Caucasian	References
–	62.1	Pollock (1976)
59.5	71.1	Bosch et al. (1990)
56.0	69.9	Coetzer et al. (1993)
53.9	63.1	Lambert and Noakes (2000)
58.4	67.5	Leary and Wyndham (1965)
61.5, 56.1	66.4	Saltin et al. (1995)
61.4	64.9	Weston et al. (2000)
59.3	76.6	Marino et al. (2004)

Apart from the Caucasian runners in the study of Marino et al. (2004) all runners have a low body mass, but the body mass of the African runners is consistently much less than that of the Caucasians. The importance of a low body mass, resulting in a high surface area: volume ratio for thermal balance in the heat has been investigated in several papers. Marino et al. (2000) showed that runners with a lower body mass produced and stored heat at a slower rate when running in hot environments, delaying the time before critically high core temperatures were reached. Foster and Lucia (2007) reviewed the evidence on the role of running economy, and their general conclusion was that a superior running economy may contribute in part to the superior performance of the African runners, but that a low body mass was likely to be a more significant factor.

Based on the data in Table 1, a more reasonable estimate of the body mass of an elite runner capable of completing the distance in 2 h, 10 min would be about 60 kg. This would reduce the oxygen consumption required to meet the energy cost of running at 19.4 km/h from 4.3 to 3.9 L/min. This would reduce the rate of metabolic heat production from 1440 to 1290 W. With height remaining unchanged, there would be some reduction in body surface area with the reduced body mass, and this would decrease from 1.85 to 1.80 m^2, with a corresponding reduction in the capacity for heat exchange with the environment. A reduction in body mass will also decrease the heat storage capacity of the body.

It seems likely that an elite runner would have a higher than normal running economy, allowing any given speed to be achieved at a lower than normal energy cost. The issue of running economy has been the subject of some debate, but there is evidence of an inverse relationship between the energy cost of running and the best performance that an individual can achieve. It seems reasonable, therefore, to assume a further reduction in the energy cost: a 10% reduction in the rate of metabolic heat production would give a value for the average rate of heat generation of 1160 W. This means that the imbalance between heat production and heat loss is now reduced to 195 W, and body temperature would rise more slowly, but still too fast for the race to be completed. Assuming a higher core temperature, however, would allow a higher skin temperature and therefore a greater evaporative heat loss. Because the performance is real, and because also humans obey the laws of physics, we know that some combination of physical characteristics must exist that makes the observed performance possible.

Heat exchange in the field is influenced by many factors, and it seems unlikely that simplistic equations can adequately model the conditions that exist. Potential complicating factors include:

- Pace (and therefore energy cost) is not constant, varying with tactics, terrain and wind
- Environmental temperature and humidity vary over the duration of an event
- Skin temperature and sweat distribution are not uniform over the whole skin surface
- The whole body surface area is not available for heat exchange
- Wind velocity is never constant, and may be very high over moving limbs
- Vapor pressure depends on skin temperature and on osmolality at skin surface, and will fall as sweat evaporates, leaving a more concentrated salt solution behind

A lower skin temperature will increase the core-skin gradient, reducing the skin blood flow that is necessary to transfer heat, but will reduce the water vapor pressure at the skin surface, reducing the evaporative capacity. Raising the skin temperature will increase the vapor pressure gradient between skin and environment, increasing evaporative capacity, but at the expense of an increased requirement for skin blood flow. The limitation imposed by the available cardiac output may be especially important where the cardiovascular capacity is compromised, as may happen with hypohydration (Montain & Coyle, 1992; Gonzalez-Alonso, 2007). It should be noted also that, even though the volume of fluid ingested by elite runners is generally small, ingestion of cold fluids will act as a heat sink and slow the rate of rise of core temperature, thus potentially improving performance (Lee et al., 2008a, b).

It is clear that the environmental conditions do affect distance running performance and that performance is slowed in conditions of high heat and humidity. No clear mechanism has yet been identified for the earlier onset of fatigue in laboratory studies or for the slower pace of runners in the field. It is clear, however, from the results of championship races that elite runners can perform at very high levels even when the temperature is high and we await a clear explanation of the physiological responses and adaptations that make this possible.

Acclimatization or acclimation involving repeated exposures to exercise in the heat results in marked improvements in exercise time to fatigue, but there is little evidence in man for an improved ability to tolerate increased core temperatures in response to heat acclimation (Nielsen et al., 1993). There is also no evidence for an improved thermal tolerance in endurance-trained individuals, leading Nielsen et al. (1993) to conclude that: "the high core temperature per se, and not circulatory failure, is the critical factor for the exhaustion during heat stress." In support of this concept, Fuller et al. (1998) have

reported that there is a critical level of body temperature beyond which animals will not continue to exercise voluntarily. While this statement may prove to be true, it provides no insight into possible mechanisms by which core temperature may cause individuals to terminate exercise. It is also apparent that, if we cannot understand the factors that limit exercise tolerance in the heat, there is little chance of identifying the limitations to man's capacity to adapt.

Recent work suggests that hyperthermia may have a direct affect on the CNS, and the brain may contribute to fatigue during prolonged exercise in a warm environment. In exercising humans, Nybo et al. (2002) showed that brain temperature is higher than core (arterial) temperature: at high ambient temperature, heat loss was reduced, so brain temperature rose much faster. The same authors (Nybo & Nielsen, 2001a) also reported a $26 \pm 3\%$ reduction in middle cerebral artery mean blood velocity during prolonged exercise and a fall in global cerebral blood flow at fatigue with hyperthermia. In addition, there appears to be a reduction in the ability to voluntarily activate muscles during sustained maximal contractions when body temperature is elevated (Nybo & Nielsen, 2001b). These findings are consistent with a role for brain in the onset of fatigue in prolonged exercise in the heat, but do not identify the mechanisms responsible. There is good evidence from pharmacological interventions that drugs acting on central serotoninergic neurotransmission can influence endurance performance without affecting metabolic, cardiovascular or thermoregulatory function (Wilson & Maughan, 1992). The observation that administration of Buproprion, a dopamine receptor agonist, results in enhanced performance in the heat but not in temperate conditions (Watson et al., 2005), is consistent with a role for central dopaminergic neurotransmission in the etiology of fatigue in the heat.

Noakes and colleagues have recently made much of the role of the brain in maintaining thermal homeostasis in exercise and has stressed the importance of a "central governor" that prevents a failure of homeostasis by causing a voluntary cessation of effort – or a reduction in exercise intensity – when homeostasis is challenged (Noakes, 1998; Noakes et al., 2005). This concept of a "governor" that limits exercise performance to prevent a catastrophic failure of physiological function has helped to shift the focus of research into the causes of fatigue away from peripheral mechanisms toward an understanding of the role of the brain. The role played by the central nervous system in the fatigue that accompanies exercise was widely recognized long before this. In 1919, Bainbridge wrote that "It has long been recognized that the main seat of fatigue after muscular exercise is the central nervous system. Mosso long ago stated that 'nervous fatigue is the preponderating phenomenon and muscular fatigue is also at bottom an exhaustion of the nervous system'. There appear, however, to be two types of fatigue, one arising entirely within the central nervous system, the other in which fatigue of the muscles themselves is superadded to that of the nervous system."

The "central governor" has been described at length in recent publications, but in the absence of any underlying mechanisms, it remains a "black box". The key role of the central nervous system (CNS) in setting the limits to exercise performance and in anticipating demands is obvious from the pacing strategies adopted by runners, cyclists and other athletes. The relationship between speed and distance in athletic events was well described by AV Hill (1925), and by many others before and since. Given that a fairly uniform pace is maintained in most athletic events, the pace adopted at the outset is based on experience of what the maximum tolerable pace is likely to be. This is then subject to modulation by signals arising in the peripheral tissues as described by Bainbridge (1919). Every elementary coaching manual published in the last century or more has emphasized the need for the inexperienced athlete to be cautious in the early stages of a race and set off at a more modest pace. Every athlete also knows that there are "good days" and "bad days" when performance is above or below what is expected.

Tucker et al. (2006) suggested that the brain is able to calculate the rate of heat storage allowable under the prevailing environmental conditions and that this information, along with knowledge of the exercise duration, will determine running pace at different times during the exercise. This system is proposed to limit the rate of heat production during exercise, thus allowing a task to be completed before catastrophe (fatigue). Others, however, have argued against such a mechanism (Nybo, 2008; Jay & Kenny, 2009). There has been some debate as to whether this is a subconscious or a conscious control mechanism, but the fact that it is a learned response perhaps argues in favor of a conscious process.

Unless one subscribes to the concept of intelligent design whereby some higher being controls the destiny of the human race, there must be a physical basis for the CNS limitations to performance that so manifestly exist. The physical mechanisms involved in the "central governor" are not well understood, but there are several possibilities. One is the temperature of the brain itself, which can be sensed and will reflect in part the body core temperature, but also responds to the metabolic heat production of the brain itself. Although brain temperature is commonly considered to be closely regulated, it has long been known that brain temperature responds

to brain neuronal activity. James (1890) described an experiment reported by Schiff some 20 years previously in which he measured the brain temperature in conscious dogs that had been deprived of food for a period, and found an increase in temperature of $\sim 1\ ^{\circ}C$ on food presentation. More recent investigations have confirmed that there is some link between activity and energy metabolism in the conscious human brain (Raichle & Gusnard, 2002). Normal fluctuations of ± 2 to $3\ ^{\circ}C$ in brain temperature are reported to occur in response to various behaviors, and while there is some correlation with body core temp, brain temperature is also responsive to neural metabolic activity (Kiyatkin 2005). Although the brain represents only about 2% of the body mass, it accounts for about 20% of the resting metabolic rate (Clark & Sokoloff, 1999).

If there is a neurological basis for the CNS limitation to performance, then there must be a neurochemical mechanism, or more likely a number of mechanisms acting in concert. Various pharmacological interventions have been applied to test this hypothesis, and the outcomes are generally consistent with a role for some key central neurotransmitters, specifically dopamine, serotonin and noradrenaline, in the fatigue process (Meeusen & Roelands, 2010).

Perhaps the most convincing evidence for a role of the brain in the fatigue process comes from pharmacological interventions. There is a marked increase in exercise capacity following the administration of amphetamines to both rodents (Gerald, 1978) and humans (Borg et al., 1972; Chandler & Blair, 1980). Amphetamines are thought to enhance exercise performance through the maintenance of DA release late in exercise, as an elevation in catecholaminergic neurotransmission is typically linked to arousal, motivation and reward. Many different psychotropic drugs that act through changes to central dopaminergic and noradrenergic neurotransmission with varying degrees of receptor specificity to treat/manage psychiatric disorders are now available. Bupropion acts on central dopamine and noradrenaline receptors, and has recently been shown to enhance endurance performance in the heat, but this effect was not seen when exercise was undertaken in temperate conditions (Watson et al., 2005). A higher power output was maintained during exercise in the heat after administration of this drug, but the subjects' perception of effort and thermal discomfort was the same on both the treatment and placebo trials. Core (rectal) temperature at the end of the trial was, however, higher on the treatment trials than on the placebo trials, which is consistent with the higher mean power output. It is possible that dopaminergic drugs may dampen or override inhibitory signals arising from the CNS to cease exercise due to hyperthermia, and enable an individual to maintain a high power output in spite of an elevated core temperature. To further emphasize the potential involvement of dopaminergic neurones in the fatigue process, there is some recent evidence that rats selectively bred for either high or low running capacity show differences in the expression of genes related to dopaminergic function in some regions of the brain (Foley et al., 2006). Attempts to find a genetic basis for the success of specific athletic populations, such as the East African distance runners, have generally been unsuccessful (Scott & Pitsiladis, 2007), perhaps because they have looked in the wrong place. Other neurotransmitter systems, including especially the involvement of central serotoninergic neurones, are likely to be involved, as reviewed by Meeusen and Roelands (2010).

A possible mechanism that may link fluid balance, thermoregulatory function and central serotoninergic or dopaminergic function involves changes in the permeability of the blood brain barrier (BBB). It has recently been shown that performance of prolonged exercise in the heat may be associated with an increased permeability of the BBB (Watson et al., 2005). This conclusion was based on an increased concentration of a brain-specific protein (S-100β) in the circulation after exercise in the heat, but not after a similar exercise bout performed in cool conditions. The function of the BBB is to protect the brain by preventing access by pathogens and by small molecules that may disrupt CNS function. It also acts to prevent escape from the brain of valuable nutrients. The BBB is normally impermeable to S-100β, though this protein can escape from the brain in various stress situations that disrupt barrier function (Marchi et al., 2003). If S-100β can escape from the central nervous system during exercise, it has to be assumed that other compounds can also escape from or enter the brain. The fact that increased permeability is observed when core temperature is elevated by exercise in the heat, may be of no significance, but may be a factor in the early fatigue that occurs in this situation. Further circumstantial evidence in support of this suggestion comes from the observation that the rise in serum S-100β with exercise in the heat is at least partially abolished by the ingestion of water, which helps maintain plasma osmolality (Watson et al., 2006). This may be associated with the better control of osmotic equilibrium across the BBB, limiting the structural changes that would otherwise take place. While there is evidence that relatively mild levels of dehydration can negatively influence cognitive performance (Gopinathan et al., 1988), little is known about the mechanisms behind the effects of hypohydration on the brain. These data suggest that changes in whole body fluid balance may directly influence the CNS, and this may play a role in the deterioration in mental

and physical performance seen with dehydration and hyperthermia.

The high cerebral temperature may lead to alterations in motor drive that affect the ability to recruit sufficient muscle fibers to meet the demands of an exercise task (Nybo & Nielsen, 2001b). This effect may be mediated, at least in part, by changes in cerebral blood flow occurring in response to redistribution of cardiac output in response to the combined stress of exercise and heat. During exercise in temperate conditions, cerebral blood flow is markedly increased during exercise (Ide & Secher, 2000), but a decline in blood flow to the brain has been reported during exercise with hyperthermia (Nybo & Nielsen, 2001a). It is clear that exercise coupled with heat stress results in metabolic and circulatory perturbations within the brain. At present it is impossible to single out any single factor that may be responsible for development of central fatigue and reduction in exercise performance in the heat, but it is likely to involve an interplay between these responses, with all contributing to varying degrees.

In summary, performance of both physical and mental tasks is significantly reduced by heat and by dehydration, though elite marathon runners perform relatively well even under unfavorable conditions. The cardiovascular and thermoregulatory systems are particularly stressed under these conditions and provision of fluids can prevent dehydration and can greatly reduce the adverse effects of heat stress. There is growing evidence that the effects of high ambient temperature and dehydration on performance of exercise may be mediated by effects on the central nervous system. Hyperthermia results in changes in the electrical activity of the brain, a marked reduction in the ability to maintain voluntary contractions and an increase in perceived exertion. While the precise role of the CNS in the development of fatigue is yet to be determined, evidence supports a role of neurotransmission in the fatigue process. A number of circulatory perturbations, including a reduction in cerebral blood flow and an opening of the BBB, may also influence performance when exercise is undertaken in high ambient temperatures, particularly in the presence of significant levels of dehydration.

Key words: marathon running, hyperthermia, dehydration, fatigue.

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