Physiological limits to exercise performance in the heat

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Summary Exercise in the heat results in major alterations in cardiovascular, thermoregulatory, metabolic and neuromuscular function. Hyperthermia appears to be the key determinant of exercise performance in the heat. Thus, strategies that attenuate the rise in core temperature contribute to enhanced exercise performance. These include heat acclimatization, pre-exercise cooling and fluid ingestion which have all been shown to result in reduced physiological and psychophysical strain during exercise in the heat and improved performance.

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Introduction

The increased metabolic heat production associated with strenuous exercise, in combination with impairment of heat dissipation by elevated environmental temperature and/or humidity, creates a major physiological challenge for the exercising athlete. The eminent cardiovascular physiologist Loring Rowell stated that "Perhaps the greatest stress ever imposed on the human cardiovascular system (except for severe hemorrhage) is the combination of exercise and hyperthermia. Together these stresses can present
Physiological and metabolic factors in fatigue

There are potentially several physiological and metabolic factors contributing to the exaggerated fatigue experienced during exercise in the heat. These include alterations in energy metabolism, cardiovascular function and fluid balance, and central nervous system function and motor drive. While it is likely that they all contribute in some way, a common element in fatigue during exercise in the heat appears to be a critically high core temperature, perhaps secondary to an inability of a limited cardiac output to maintain cutaneous perfusion for heat loss.

Metabolism

Muscle glycogen depletion and hypoglycemia have long been associated with fatigue during prolonged, strenuous exercise. During exercise in the heat, the rate of muscle glycogen degradation is significantly increased (Fig. 2)\(^3\,10\,11\) with a concomitant increase in both carbohydrate oxidation and lactate accumulation. Mechanisms thought to be responsible for the enhanced muscle glycogenolysis include, but may not be limited to, elevated circulating adrenaline and increased muscle temperature.\(^12\)

There is an exaggerated hyperglycemia during exercise in the heat due to a greater liver glucose output, without any change in the exercise-induced increase in peripheral glucose uptake.\(^13\) Despite the greater mobilization and utilization of carbohydrate substrates during exercise, carbohydrate depletion is not the cause of fatigue during exercise in the heat since muscle glycogen stores remain high,\(^3\) and the total amount of carbohydrate oxi-
Cardiovascular function and fluid balance

The major mechanism for heat loss during exercise in the heat is evaporation of sweat. This requires heat transfer to the skin via cutaneous vasodilation and the loss of fluid which, if not replaced, can result in dehydration. Skin blood flow at rest is increased markedly under hot conditions, but the onset of exercise results in vasoconstriction. It has been suggested that the "circulatory conflict" between skin and active skeletal muscle during exercise in the heat results in reduced muscle blood flow; however, this does not appear to be the case if exercise intensity and the degree of dehydration are not too severe. At high exercise intensities, heat stress accelerates the decline in stroke volume, cardiac output, muscle blood flow and oxygen delivery, thereby reducing maximal oxygen uptake. Furthermore, dehydration reduces stroke volume, cardiac output, mean arterial pressure and muscle blood flow and impairs the ability of athletes to tolerate hyperthermia. The combination of dehydration and hyperthermia has even greater negative effects. Thus, impaired central cardiovascular function with heat stress limits maximal exercise performance in the heat and contributes to the exaggerated hyperthermia during prolonged exercise with dehydration. The reduced blood flow observed during the latter stages of such exercise does not impair glucose or FFA delivery to, or lactate removal from, skeletal muscle and although carbohydrate oxidation, muscle glycogen utilization and lactate production were increased, it was suggested that fatigue was due to hyperthermia rather than altered metabolism. Increases in core and skin temperatures to \( \sim 38^\circ\text{C} \) result in a leveling off in cutaneous blood flow, thereby reducing the ability for heat dissipation. In the face of ongoing skeletal muscle vasodilation, this reflects an inability of the cardiovascular system to simultaneously maintain arterial blood pressure and cutaneous vasodilation. As mentioned, with maximal exercise or dehydration during prolonged, submaximal exercise this cardiovascular limitation results in reduced skeletal muscle perfusion which may also contribute to the development of fatigue.

Central nervous system function and motor drive

Observations that fatigue during exercise in the heat is associated with attainment of a so-called "critical" level of body core temperature have led to suggestions that hyperthermia may be acting via effects on the central nervous system and motor activation. Such an hypothesis is teleologically appealing since a reduction in motor drive (and therefore exercise intensity) slows the rate of metabolic heat production. It has been demonstrated that both exercise-induced and passive hyperthermia reduce force during sustained maximal voluntary contractions (MVC), effects that appear to be mediated by both "central fatigue" and temperature-related changes in muscle contractile properties. The reduction in force production was not observed during brief MVCs, suggesting that hyperthermia may affect the ability to maintain voluntary activation. There is evidence of neuromuscular fatigue during prolonged cycling exercise in the heat and the power output during self-paced exercise is reduced when environmental temperature is increased (Fig. 1). Interestingly, body core temperatures during exercise were similar in the cool and hot trials, suggesting complex regulation and integration of the thermoregulatory and motor control systems. Indeed, a recent study has proposed that the rate of heat storage, possibly with input from skin and blood thermoreceptors, contributes to such regulation, thereby ensuring that exercise intensity is adjusted so as to prevent excessive heat production and accumulation under hot conditions. Understanding the complex neural mechanisms underlying such regulation is a significant challenge for the future. There are a number
of changes in cerebral perfusion and metabolism and brain EEG activity that accompany fatigue during exercise-induced hyperthermia, although causal relationships are not well defined. Hyperprolactinemia during exercise in the heat provides indirect evidence of the potential involvement of central serotoninergic activity in the aetiology of fatigue under these conditions. Furthermore, administration of a dopamine/noradrenaline reuptake inhibitor enhanced exercise performance in warm conditions. Collectively, these studies implicate altered central nervous system function in hyperthermia-induced fatigue. Agents that alter the perception of fatigue may exert ergogenic effects via these mechanisms; however, they may also oppose inhibitory signals arising from hyperthermia and increase the risk of heat injury. Was the death of British cyclist Tom Simpson on the ascent of Mt. Ventoux in the 1967 Tour de France perhaps the terrible consequence of amphetamine-mediated inhibition of signals that might have otherwise prevented the development of fatal hyperthermia?

Strategies to enhance exercise performance in the heat

Since hyperthermia appears to be the major factor in fatigue during strenuous exercise in the heat, the primary goal of strategies to enhance exercise performance should be to attenuate the rise in body core temperature. Effective strategies include acclimatization, pre-cooling and fluid ingestion.

Acclimatization

Repeated bouts of exercise in a hot environment result in a number of physiological and metabolic adaptations that are associated with improved exercise performance. These include an expanded plasma volume, increased stroke volume and cardiac output and enhanced sweat rate and sensitivity during exercise in the heat. Muscle glycogen use during exercise in the heat is reduced following heat acclimatization, as a consequence of lower core temperature and plasma adrenaline levels. Interestingly, the rate of rise in core temperature during exercise in the heat was similar before and after acclimatization in trained subjects and the increased exercise time to fatigue, which corresponded with attainment of a similar core temperature, was due to a reduction in resting body temperature.

Fluid ingestion

The loss of body fluid due to sweating results in dehydration which has negative effects on cardiovascular, thermoregulatory and metabolic function, increasing the development of fatigue during exercise in the heat (see above). Ingestion of fluids is an effective strategy to attenuate many of these negative consequences of dehydration. The inclusion of carbohydrate in rehydration beverages may provide additional benefits. The physiological benefits of fluid intake appear to be proportional to the volume ingested and athletes are encouraged to replace fluids up to, but not exceeding, their sweating rate. In practical terms, this may be difficult due to large sweat losses, variations in gastrointestinal motility and fluid bioavailability and

Figure 3 Esophageal temperature relative to cycling exercise time at 60% VO_{2} max in trained men at 40 °C following no intervention (control), precooling or preheating. Values are means ± S.E. (n = 7). Data from González-Alonso et al. (1999a) and reproduced from Coyle (1999).
access to appropriate fluids. There is debate as to whether "full" fluid replacement is necessary or even desirable. 37 Most studies examining the benefits of fluid ingestion on physiological and metabolic function have been laboratory-based and while they provide valuable insight, direct extrapolation to all athletic conditions in the field may be difficult. In a recent study there was no relationship between fluid intake and core temperature during outdoor running 38 and it has been observed that successful athletes are often significantly dehydrated at the end of endurance events. 37 That said, there is compelling evidence that fluid ingestion "ad libitum" enhances endurance exercise performance; whether ingestion of larger volumes is required in outdoor settings is contested. 39 Excessive over drinking resulting in an increase in body mass during exercise should be avoided since this can result in hyponatremia, a potentially fatal condition. Given these considerations, it is essential for athletes to develop drinking strategies that maximise physiological benefits, but prevent overhydration.

References


