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ORIGINAL ARTICLE

Oral tyrosine supplementation improves exercise capacity in the heat

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Abstract Increased brain dopamine availability improves prolonged exercise tolerance in the heat. It is unclear whether supplementing the amino-acid precursor of dopamine increases exercise capacity in the heat. Eight healthy male volunteers [mean age 32 ± 11 (SD) years; body mass 75.3 ± 8.1 kg; peak oxygen uptake ($\dot{V}O_{2peak}$) 3.5 ± 0.3 L min^{-1}] performed two exercise trials separated by at least 7 days in a randomised, crossover design. Subjects consumed 500 mL of a flavoured sugar-free drink (PLA), or the same drink with 150 mg kg body mass^{-1} tyrosine (TYR) in a double-blind manner 1 h before cycling to exhaustion at a constant exercise intensity equivalent to 68 ± 5% $\dot{V}O_{2peak}$ in 30°C and 60% relative humidity. Pre-exercise plasma tyrosine:large neutral amino acids increased 2.9-fold in TYR ($P < 0.01$), while there was no change in PLA ($P > 0.05$). Subjects cycled longer in TYR compared to PLA (80.3 ± 19.7 min vs. 69.2 ± 14.0 min; $P < 0.01$). Core temperature, mean weighted skin temperature, heart rate, ratings of perceived exertion and thermal sensation were similar in TYR and PLA during exercise and at exhaustion ($P > 0.05$) despite longer exercise time in TYR. The results show that acute tyrosine supplementation is associated with increased endurance capacity in the heat in moderately trained subjects. The results also suggest for the first time that the availability of tyrosine, a nutritional dopamine precursor, can influence the ability to subjectively tolerate prolonged submaximal constant-load exercise in the heat.

Keywords Central fatigue · Amino acid · Mild hyperthermia

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Introduction

It is clear that prolonged exercise capacity (Galloway and Maughan 1997) and performance (Tatterson et al. 2000) are impaired in the heat when compared with temperate conditions. This fatigue, defined as an inability to maintain a given work rate or power output, appears to involve changes within the central nervous system (Nybo and Nielsen 2001), secondary to an increase in body (i.e. core, muscle and brain) temperature per se (Nielsen et al. 1993). Hyperthermia may reduce the will or the "drive" to exercise (Brück and Olschewski 1987), and fatigue during prolonged exercise in the heat may therefore represent reduced arousal or motivation to continue exercise (Nielsen et al. 2001). In support of this, a slowing of frontal cortex electroencephalogram waves has been noted with increasing body (oesophageal) temperature during prolonged exercise in the heat, indicative of reduced arousal (Nielsen et al. 2001), and this correlates strongly with linear increases in subjective exertion (RPE; Nybo and Nielsen 2001). However, to date no study has been able to identify a precise neurobiological cause for this "central fatigue".

Alterations in brain neurotransmitter availability may provide a possible cause and some suggest that a high ratio of brain dopamine:5-Hydroxytryptophan (5-HT) augments prolonged exercise and a low ratio induces lethargy and reduced motivation (Davis and Bailey 1997). This may be