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Evidence for anticipatory regulation mediated by drink temperature during fixed intensity exercise in the heat

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Letter to the Editor re:


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The very elegant study by Mündel et al. (2006) has prompted me to clarify the concepts that presently underpin the relationship between hydration, heat strain and exercise performance. The hypothesis that cold fluid ingestion provides a heat sink which could enhance heat storage, and thereby increase time to fatigue, is an interesting and practical proposition. Although we have also suggested that a heat sink through fluid ingestion would be possible under certain conditions (Kay &
Marino, 2000), the data reported by Mündel et al. (2006) indicate that rectal temperature response and subsequent time to fatigue is not explicable on the basis of the fluid ingested but possibly by an alternative anticipatory mechanism.

First, the sweat rates were identical for both drink conditions at 1.4 l.h\(^{-1}\); therefore, heat dissipation must have been similar for both conditions indicating that neither fluid condition had any effect on sweat rate per se. Second, the drink rates which were different (1.0 vs 1.3 l.h\(^{-1}\) for control and cold fluid, respectively) equate to ~ 1.26 and 1.1 litres ingested in absolute terms for cold fluid and control conditions, respectively, in turn representing only a difference of ~ 160 mL extra fluid consumed during the cold fluid condition. It would seem unlikely that this amount of extra fluid could explain any differences in temperature response and account for a 7 min increase in time to fatigue. Third, the identical sweat rates coupled with the small difference (~ 160 mL) in absolute fluid ingested between conditions does not represent what is usually claimed to be the cause of the elevated rectal temperature during exercise heat stress as the change in body mass was likely less than ~ 2% for both drink conditions; therefore, the difference in rectal temperature, if any, cannot be explained on this basis as rectal temperatures did not approach those values that are thought to limit exercise (i.e. ~ 39.5 ºC).

We have previously shown and argued that when subjects are restricted from adjusting their workload, fluid ingestion does not produce any significant differences in exercise performance (Kay & Marino, 2003; Marino et al., 2003). Sufficient cold (4.5 ºC) fluid preventing any change in body mass, or complete fluid restriction had no effect on exercise performance or thermoregulatory strain over 1 h of high intensity self-paced exercise in the heat (Kay & Marino, 2003; Marino et al., 2003).
Therefore, if cold fluid ingestion does not explain the differences in time to fatigue due to a heat sink effect in the Mündel et al. (2006) study, there must be some other mechanism which could potentially explicate the increased time to fatigue. The phenomenon of anticipatory regulation of impending thermal limits has been described in humans and rats that were either cooled or heated before exercise (for review see Marino, 2004). In each case, both humans and rats altered their rate of increase in core temperature rather than retain an identical rate of increase in each condition which would have been catastrophic. At present, there are no data that show this same phenomenon with respect to fluid ingestion when subjects exercise at a fixed intensity. However, if one re-draws the rectal temperature data provided by Mündel et al. (2006), it is evident that cold fluid ingestion changed the rate of rise in rectal temperature of the subjects as shown in Figure 1. The mechanism responsible for the termination of exercise at the critical limiting temperature is thought to be due to a reduced central nervous system (CNS) drive to the skeletal muscle (Nybo & Nielsen, 2001). However, the subjects in the Mündel et al. (2006) study stopped exercising well before reaching a critical limiting temperature of 39.5 °C, probably because they were not highly conditioned (Cheung & McLellan, 1998). We have previously argued that the critical limiting temperature hypothesis must have an advanced warning signal for impending thermal limits to invoke a reduced motor drive (Marino et al., 2004). The data provided by Mündel et al. (2006) support the model which posits that those physiological changes leading to a reduced CNS drive occur in anticipation of a critical temperature. The evidence for this is that subjects ingesting the control fluid (19 °C) fatigued ~ 7 min earlier with a higher rate of increase in rectal temperature (1.83 °C.h⁻¹ for control vs 1.45 °C.h⁻¹ for cold; Figure 1). Given that subjects finished both trials with similar
rectal temperatures (38.69 vs 38.43 °C; difference ~ 0.25 °C) can only suggest that terminal rectal temperatures do not determine the point of fatigue. The interesting result, however, is that a difference of about 160 mL of extra fluid for the cold fluid condition not only resulted in the delayed onset of fatigue in these moderately trained subjects, but it also changed the rate of increase in rectal temperature. These data indicate that a feed-forward mechanism was in operation which signalled the availability of cold fluid allowing subjects to “choose” to continue exercising. On this basis it is possible that sensory input from either receptors in the mouth and/or during faster gastric emptying of the cooler fluid may have contributed to this signal (Costill & Saltin, 1974; Sandick et al., 1984). Although it is not possible to rule out absolutely a heat sink effect, it seems just as possible that cold fluids provide a mechanism to assist in the anticipation of thermal limits allowing the organism to avoid a catastrophic outcome.

References


Figure 1. Rate of increase in rectal temperature for cold fluid ingestion (4 °C) and control fluid ingestion (19 °C). Data redrawn from Mündel et al. (2006).