Thermoregulation, pacing and fluid balance during mass participation distance running in a warm and humid environment

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Reference research paper by Lee et al, published in Eur J Appl Physiol. 2010 Jul;109(5):887-98

INTRODUCTION

Elevated deep body temperature (Tc) is implicated as a causative factor of accelerated fatigue in the heat but the nature of its involvement is controversial.

Exercise trials observe that tasks are completed with a reduction in work rate prior to marked hyperthermia. Thus, anticipatory regulation hypothesis suggests that exercise intensity is regulated before attainment of a critical Tc.

Fluid ingestion is not a significant predictor of Tc responses despite laboratory evidence that fluid replacement attenuates rate of rise of Tc.

Objectives:

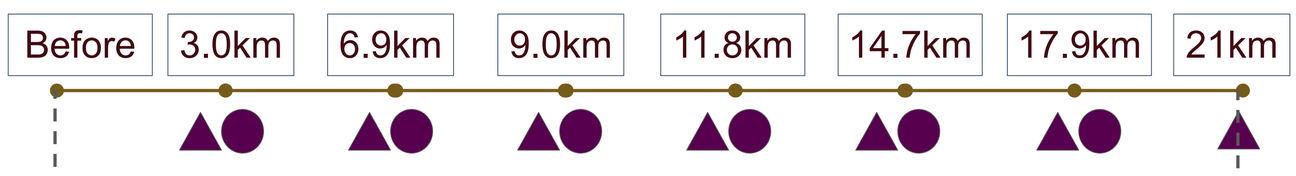
- Investigate existence of a critical Tc threshold for fatigue
- Investigate anticipatory regulation hypothesis
- Quantify fluid balance with precision and investigate contribution of body water variables to Tc response

METHODS

Participants

- 29 volunteers recruited from Singapore Armed Forces and 2 from local running clubs

Participants characteristics	
Age (years)	25.3 ± 3.2
VO_{2max} (ml kg^{-1} min^{-1})	59.1 ± 4.2
Running economy (ml kg^{-1} km^{-1})	211 ± 12
vVO_{2max} (m min^{-1})	280 ± 21



- Telemetric temperature sensor ingested
- Urine sample and venous blood collected; body mass measured
- Heart rate telemetry system and ambulatory body core temperature recorder fitted

- Post-race venous blood, urine sample collected
- Nude body mass measured

- *Triangle*: split time recorded; *Circle*: fluid stations
- Deep body temperature and heart rate recorded at 15s intervals

RESULTS

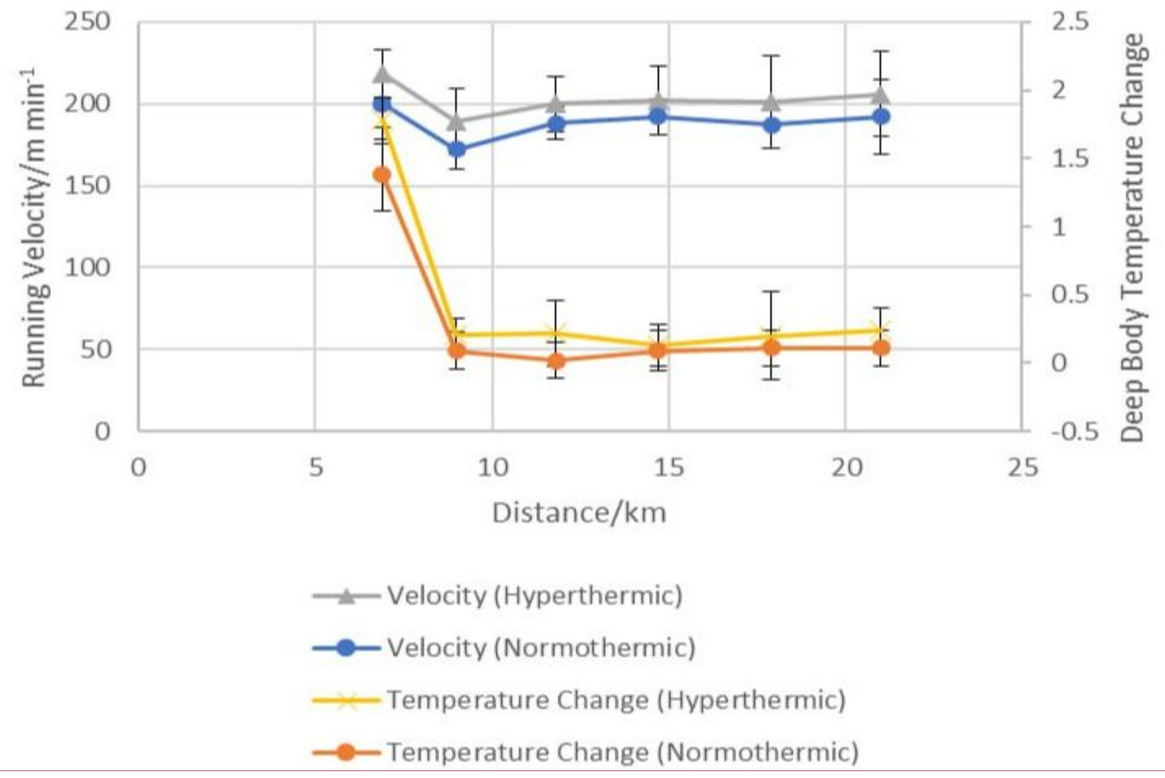


Figure 1 illustrates that the mean running velocity was significantly faster for hyperthermic runners from 0-6.9km (218 ± 15 cf. 200 ± 14, P<0.01), 6.9-9.0 km (189 ± 20 cf. 172 ± 120,P<0.01) and 9.0-11.8km (200 ±17 cf. 188±10, P<0.01). There was no significant difference at race distance >11.8km.

The figure also illustrates change in deep body temperature was significantly greater for hyperthermic runners from 0-6.9km (1.78 ± 0.17 c.f 1.38 ±0.26, P<0.05), 6.9-9.0km (0.21±0.12 c.f 0.09 ± 0.14, P<0.05) and 9.0-11.8km (0.22±0.24 c.f 0.02 ± 0.13, P<0.05). There was no significant difference at race distance >11.8km.

Fluid balance	
Mean water body loss/kg	2.47 ± 0.63
Sweat loss/l	2.58 ± 0.59
Volume of sports drink consumed/ml	341 ± 259
Volume of water consumed/ml	132 ± 107

Figure 2 illustrates that race fluid intake replaced 15 ± 11% of sweat loss. When pre-race fluid intake was included, this increased to 21± 12%. Serum sodium concentration increased significantly from 140 ± 2 pre-race to 146 ± 2 mmol/l (P<0.01)

DISCUSSION

Hyperthermia (Tc >39.5°C) was not found to be associated with fatigue, exhaustion, heat illness, or an acute reduction in exercise intensity.

Running velocity was the main significant predictor variable of ΔTc at 21 km, and the main discriminating variable between hyperthermic and normothermic runners up to 11.8 km.

Our data support the anticipatory regulatory hypothesis in that exercise intensity is reduced prior to marked hyperthermia; however, Tc rate of change was not found to influence subsequent exercise intensity.

Further evaluation of the aforementioned hypothesis was untenable as only 10 runners achieved a Tc >40°C; this hindered the elucidation of a Tc threshold for an acute reduction in exercise intensity.

The **physiological benefit derived from water consumption** in actual field settings is **likely to be small** - fluid ingestion during competitive races have no detectable effect on variables related to Tc or performance.