Time Trial Performance and Pacing in Heat is Determined by Rate of Heat Gain

Dear Editor-in-Chief

The very elegant study by Racinais et al. (6) shows for the first time that at least one outcome of heat acclimatization is altered pacing strategy. A key finding from their Figure 1 was the attenuated reduction in power output over the percent distance completed from the first (TTH-1) to the third time trial (TTH-3) in the heat. Interestingly, the rectal temperature (T_{re}) in both trials was not different over the trial distance as shown in their Figure 2 (*bottom panel*) so that subjects terminated performance with identical values (40.1°C–40.2°C). The authors correctly concluded that a critical temperature threshold of 40°C is unlikely to determine performance. Thus, this study should now close the chapter on the presumption that humans cannot continue exercising beyond a critical core temperature of 39.5°C–40°C.

Nevertheless, it still remains to be elucidated as to what thermoregulatory factors either determine or regulate pacing with and without heat acclimatization. Fortunately, retrospective interpretation of their data can shed further light on this issue. According to their Figure 1, regardless of power output over the duration of both trials in the heat, T_{re} response was almost identical. The time to complete the two trials was different with trial 1 completed in about 77.28 min, whereas trial 3 was completed in 65.61 min, approximately 11.66 min faster. Taking the change in T_{re} over the total time, it is apparent that the rate of increase in T_{re} was different, approximately 1.44°C · h^{-1} and 1.88°C · h^{-1} for TTH-1 and TTH-3, respectively. Coupled with the finding that terminal T_{re} were identical, these data suggest that pacing was determined by the rate of increase in T_{re} as previously posited (7,8).

Although the authors suggest that attenuated cardiovascular strain with acclimatization could also explain the improved performance, this negates the obvious observation that as cardiovascular strain increases, power output can also increase (4,5) or maintained as Racinais et al. (6) now show (as per their Figs. 1 and 2). An alternative possibility is that skeletal muscle recruitment is regulated relative to thermal strain during self-paced time trials (2,8). This is consistent with the finding that subjects ended both trials with identical T_{re} albeit with significantly different power outputs.

Their data also show that power output explains approximately 90% of the variance for T_{re} response (R^2 ~0.88–0.97). Remarkably, power output range was larger in trial 1 (~220–320 W) compared with trial 3 (~280–320 W) where it was tightly controlled. These data are consistent with the proposition that pacing in the heat, even after acclimatization, is regulated in anticipation of the terminal T_{re} so that exercise is completed without the development of hyperpyrexia (3). This phenomenon was first noted over 50 yr ago by Ladell (1) who concluded that "The wonder is, not that anyone gets hyperpyrexia, but that so few of us ever do" (p. 206). Perhaps it is now time to embrace the possibility that humans, like all other animals, are able to regulate their exercise responses in order to avoid cellular catastrophe.

Frank E. Marino, PhD
School of Human Movement Studies
Faculty of Science
Charles Sturt University
Bathurst, New South Wales, AUSTRALIA

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