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## **Reply to Parkes: Effect of hypocapnia on the sensitivity of hyperthermic hyperventilation and the cerebrovascular response in resting heated humans**

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<https://doi.org/10.1152/japplphysiol.00089.2018>

PUBLISHER

© American Physiological Society

VERSION

AM (Accepted Manuscript)

PUBLISHER STATEMENT

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Tsuji, Bun, Davide Filingeri, Yasushi Honda, Tsubasa Eguchi, Naoto Fujii, N. Kondo, and Takeshi Nishiyasu. 2019. "Reply to Parkes: Effect of Hypocapnia on the Sensitivity of Hyperthermic Hyperventilation and the Cerebrovascular Response in Resting Heated Humans". figshare. <https://hdl.handle.net/2134/33838>.

## REPLY

*To the Editor:* We thank Dr Parkes for the comment on our recent paper. Dr Parkes points out that our statement “*we are the first to demonstrate that hyperthermia-induced hyperventilation is not suppressed by the resultant hypocapnia*” is incorrect as many previous studies have already reported that hyperthermia stimulates ventilation in the presence of concomitant hypocapnia.

It is true that several previous studies, including our work (1), have demonstrated that hyperventilation occurs during hyperthermia with concomitant hypocapnia. However, given that arterial CO<sub>2</sub> pressure is tightly and positively associated with ventilatory drive, it is possible that the resultant hypocapnia partly suppresses hyperthermia-induced hyperventilation. That is, the reported hyperventilation during hyperthermia previously observed may be the result of combined influences of increased respiratory drive elicited by hyperthermia, and reduced ventilatory drive due to hypocapnia.

To isolate the influence of hypocapnia on ventilatory responses during hyperthermia, we must compare ventilatory responses between hypocapnia and normocapnia, and the latter can be achieved by having individuals inhaling hypercapnic air during heating. In contrast with our recent study, none of the previous studies cited by Dr Parkes directly restored the reduced arterial CO<sub>2</sub> pressure to normocapnic levels throughout heating procedure. We are therefore the first to elucidate the effect of concomitant hypocapnia on ventilatory response during passive hyperthermia in resting humans, having used hypercapnic air breathing that kept arterial CO<sub>2</sub> pressure at normocapnic level throughout our heating protocol.

Our results clearly indicate that the concomitant hypocapnia does not influence hyperthermic hyperventilation as assessed by core temperature threshold for increases in ventilation, as well as ventilatory sensitivity to rising core temperature. With this direct evidence, we can conclude

that hyperthermic hyperventilation is not suppressed by the resultant hypocapnia. Further evidence is warranted to evaluate the role of more severe hypocapnia (e.g., arterial CO<sub>2</sub> pressure of <30 mmHg) on hyperthermic hyperventilation, as observed in the previous studies cited by Dr Parkes.

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## References

1. Tsuji B, Honda Y, Fujii N, Kondo N, Nishiyasu T. Comparison of hyperthermic hyperventilation during passive heating and prolonged light and moderate exercise in the heat. *J Appl Physiol* 113: 1388-1397, 2012.