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**To drink or not to drink: the effect of fluid replacement on post-exercise cardiovascular haemodynamics**

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Exercise training can induce unique physiological adaptations in the human body. While untrained populations experience post-exertional hypotension attributable to peripheral vasodilatation, this phenomenon may be secondary to reduced cardiac output in endurance-trained populations. Both fluid replacement and environmental factors may modify this acute physiological response. An interesting study by Lynn *et al.* (2009) examines these potential factors and considerably challenges our current understanding of the acute physiological response to exercise in endurance-trained male athletes. Until now, fluid replacement during prolonged exercise has been continuously emphasized, and even more so, when exercise is performed in hot environments. Who would have thought that performing exercise in the heat and without fluid replacement might actually enable endurance athletes to maintain cardiac output in the recovery period?

The acute cardiovascular response to exercise involves an intensity-mediated increase in cardiac output due to increased heart rate and stroke volume to supply metabolically active muscles with oxygenated blood. During the exercise recovery period, endurance-trained males experience post-exercise hypotension secondary to declines in cardiac output. This physiological adaptation is unique to endurance-trained athletes and is not completely understood, but may be due to adaptations to thermoregulatory mechanisms, which exacerbate reductions in preload. More specifically, thermoregulation is a complex system involving physiological processes that allow the maintenance of body temperatures under conditions of varying heat loads within a restricted physiological range

(Kaciuba-Uscilko & Gruzca, 2001). Heat stress produces a substantial acute physiological response to reduce heat load through sweating, plasma volume shifts and vasodilatation. Heat stress may also lead to increases in cardiac output secondary to an increase in heart rate and myocardial contractility (Lynn *et al.* 2009). Fluid replacement assists in maintaining plasma volume and countering the effects of heat stress.

In a recent issue of *The Journal of Physiology*, Lynn *et al.* (2009) assessed the effect of fluid replacement and heat stress during moderate-intensity exercise on post-exercise cardiovascular haemodynamics. The authors hypothesized that adequate fluid replacement during exercise would prevent the decrease in cardiac output in endurance-trained males, whereas exercise in a warm environment would further decrease cardiac output due to a greater loss of plasma volume and greater peripheral vasodilatation due to a greater thermal load. In a cross-over study design, trained males performed three cycle ergometer exercise sessions at a fixed workload (60% maximal work for 60 min), complemented both with and without fluid replacement (matched to sweat loss) in a thermoneutral environment, and without fluid replacement in a warm environment. Notably, a fourth exercise session with fluid replacement in a warm environment was not performed. Higher post-exercise cardiac output ( $\Delta 0.41 \pm 0.16 \text{ l min}^{-1}$ ;  $P = 0.027$ ), systemic vascular conductance ( $\Delta 6.0 \pm 2.2 \text{ ml min}^{-1} \text{ mmHg}^{-1}$ ;  $P = 0.001$ ) and stroke volume ( $\Delta 9.1 \pm 2.1 \text{ ml beat}^{-1}$ ;  $P < 0.001$ ) were seen in fluid replacement condition compared to the thermoneutral condition without fluid replacement. Surprisingly, when trained males exercised in the warm condition, there was no difference in cardioregulatory response between fluid replacement in the thermoneutral condition and the warm environment without fluid condition (all  $P > 0.05$ ). The authors hypothesized that this novel and unanticipated finding may be attributable to an elevated core temperature which increases heart rate and myocardial contractility.

This novel study by Lynn *et al.* (2009) leads us to consider many practical research questions to which future research is warranted. In particular, the Lynn and

colleagues study reveals several relevant research gaps, including the effects of (i) fluid replacement with carbohydrates and electrolytes, (ii) exercise performed in the heat with fluid replacement, (iii) exercise intensity and duration, (iv) training-induced cardiac structural adaptations on post-exercise cardiovascular haemodynamics, and (v) sex.

Fluid replacement with carbohydrate or electrolyte solutions is commonly recommended during exercise lasting 1 h or longer in duration. In this study, although the duration of exercise was only 1 h, the addition of carbohydrates and/or electrolytes in the fluid condition may have yielded different findings for post-exercise haemodynamics. Fluids with carbohydrate and/or electrolytes (i.e. sports drinks) have been consistently found to improve exercise capacity and reduce fatigue during endurance activities compared to water (Coyle & Montain, 1992). While the composition of sports drinks are more complex than water, the rate of gastric emptying and fluid absorption of both types of beverage have been reported at  $1250 \text{ ml h}^{-1}$ , therefore causing no difference within the gastrointestinal tract. While added carbohydrates can provide working muscles with rapid energy (Coyle & Montain, 1992), electrolytes can improve fluid retention during exercise (Vrijens & Rehrer, 1999), thereby helping to maintain plasma and blood volumes. In a similar study with endurance-trained males where fluid consumption was matched to sweat loss, a group given a sports energy drink experienced higher sodium retention, which contributed to lower weight loss, an indirect measure of hydration and plasma volume, compared to the water group (Vrijens & Rehrer, 1999). Thus, the maintenance of electrolyte balance may modify post-exercise changes in cardiac output.

The inclusion of a fourth exercise condition (i.e. warm condition with fluid replacement) would have further added to our understanding of post-exercise haemodynamics. More importantly, it would have significant relevance and practical implications for endurance-trained athletes, who typically compete in warm environments over prolonged periods of time, and utilize fluids with electrolyte solutions to mitigate core

temperature and hydration status. While cardiac output was maintained during recovery without fluid replacement in a warm environment, it is possible that fluid replacement in a warm condition may actually promote an increased cardiac output during recovery such that the underlying mechanism of post-exertional hypotension is altered.

Exercise intensity and duration may also alter post-exercise cardiovascular haemodynamics due to intensity-mediated increases in heart rate and cardiovascular drift. The findings of Lynn *et al.* (2009) produce speculation that higher exercise intensities can lead to increases in core temperature which may increase heart rate and further prevent decreases in cardiac output during the exercise recovery period. However, post-exercise cardiovascular haemodynamics may also be altered due to increasing exercise duration. Prolonged exercise (i.e. lasting more than 2.5 h) research has shown that both cardiovascular drift, secondary to decreased stroke volume and a compensatory increase in heart rate, and impaired myocardial contractility independent of changes in preload, may be exacerbated (Goodman *et al.* 2009). As a result, exercise of greater intensity and duration may actually produce increases in core temperature without increased myocardial contractility, and therefore, have implications for post-exercise hypotension in this trained population.

While findings from this study are directly applicable to endurance-trained males, can these post-exercise changes be generalized

to the recreationally active and trained female populations? Echocardiography was not used to analyse cardiac function; however, chronic endurance training stimulates structural adaptations within the heart in both male and female athletes, enabling higher cardiac output during exercise. The recreationally active population may not experience the same physiological haemodynamic response, as they lack the structural adaptations seen in trained populations. Therefore, the resultant changes post-exercise may be unique to endurance-trained individuals. Sex differences may also modify the relationship between fluid replacement, heat stress and post-exercise cardiovascular haemodynamics. In particular, sex differences in acute physiological responses to exogenous and endogenous heat stress have been identified and may be attributable to the larger ratio of body surface to body mass, greater subcutaneous fat content and lower exercise capacity in women (Kaciuba-Uscilko & Gruzca, 2001). Furthermore, while women's sweat response during exercise is delayed and smaller relative to men, they exhibit similar responses to increases in core temperature due to a greater sweating evaporative efficiency (Kaciuba-Uscilko & Gruzca, 2001). Sex hormones also produce differences in resting body temperature and thermal responses to heat stress depending on menstrual cycle phase (Kaciuba-Uscilko & Gruzca, 2001), which may have implications for post-exercise hypotension.

In conclusion, the findings of Lynn *et al.* (2009) provide puzzling evidence that post-exercise hypotension is a highly regulated variable as both fluid replacement and heat stress modified cardiovascular haemodynamics during the post-exercise recovery period in trained males. Interestingly, the post-exercise hypotension occurred under both conditions even though the haemodynamics determinants were different. This relationship needs to be further substantiated with different environmental factors and populations.

## References

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