Antecedent exercise attenuates the glycaemia-increasing effect of a 30-second sprint

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Introduction: Recently, the glycaemia-increasing effect of a short sprint has been proposed as a potential clinical tool for the prevention of exercise-mediated hypoglycaemia in individuals with type 1 diabetes (Bussau *et al.*, 2006). However, before a sprint can be safely recommended, factors that may impair its effectiveness must be investigated. Of interest, it has been shown that an antecedent bout of moderate-intensity exercise blunts the counterregulatory hormone responses to a second bout of moderate-intensity exercise performed several hours later (Galassetti *et al.*, 2001). Whether antecedent exercise also attenuates the glycemia-increasing effect of a maximal sprint effort is not known.

Aim: To investigate whether an antecedent bout of moderate-intensity exercise attenuates the counterregulatory hormone and glycaemia-increasing responses to a 30-second sprint.

Methods: Fifteen healthy young adults (8M, 7F; aged 21.2 \pm 1.7 years; BMI of 23.5 \pm 2.1 kg·m⁻²; VO₂peak of 46.7 \pm 5.9 ml·kg⁻¹·min⁻¹; mean \pm SD) visited the research laboratory on two occasions during which they either rested for 60 minutes (CON) or performed 60-minutes of moderate-intensity exercise at 62 \pm 7.5% VO₂peak (EX) on a cycle ergometer in the morning, following a randomised counterbalanced study design. Following the morning rest and exercise conditions, each participant rested for 3 hours and 15 minutes before performing a 30-second maximal sprint. Blood samples were collected prior to and during the first hour of recovery from the sprint, and were subsequently analysed for glucose, glucose kinetics, insulin, and the counterregulatory hormones.

Results: Mean blood glucose concentrations prior to the 30-second sprint were similar between treatments ($4.47\pm0.32 \text{ mmol/L CON}$; $4.43\pm0.18 \text{ mmol/L EX}$). In response to the sprint, blood glucose levels increased to the same extent (P<0.05) in both trials, reaching similar maximal levels at 10 minutes of recovery ($5.34\pm0.39 \text{ mmol/L CON}$; $5.27\pm0.34 \text{ EX}$; P=0.18). However, there were significant differences in blood glucose levels between conditions at 30, 45, and 60 minutes of recovery, with CON exhibiting significantly higher levels at all three time points (P=0.013, P=0.003, and P=0.022 respectively). In response to the sprint, both the rate of appearance (Ra) and rate of disappearance (Rd) of glucose increased significantly in both groups by 5 minutes post-sprint. Plasma insulin levels increased transiently after sprinting, with significantly higher levels of circulating insulin in CON compared to EX at 45 minutes of recovery (P=0.016). Epinephrine levels increased rapidly in response to the sprint, peaking and returning to baseline early during recovery. Growth hormone levels peaked at 30 minutes post-sprint and were blunted by antecedent exercise, with significantly lower levels at 30 (P=0.010), 45 (P=0.002), and 60 minutes (P=0.010) of recovery in EX. Cortisol levels increased in response to the sprint with no significant difference between trials.

Conclusion: These results suggest that a prior bout of moderate-intensity exercise reduces the glycaemiaincreasing effect of a 30-second sprint in non-diabetic individuals. This highlights the need for further investigations in individuals with type 1 diabetes before sprinting can be safely recommended as a tool for the prevention of exercise-mediated hypoglycaemia.

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