Passive Heating as an Option for Improving Glucose Control: Take A Bath!

Steve H. Faulkner, Sarah Jackson, Kate McDaid, George Havenith FASCM, Christof A. Leicht

Loughborough University, Loughborough, Leicestershire, LE11 3TU, United Kingdom

Obesity and type 2 diabetes are increasing in prevalence and can be treated by lifestyle interventions such as diet and exercise. Acute exercise has an insulin sensitising effect, leading to improved glucose control. This may in part be due to the attenuated synthesis of heat shock protein 70 (HSP70) in obesity and diabetes. HSP70 may be elevated by exercise and passive heating (PH). Furthermore, animal studies suggest PH may be beneficial to glucose control.

PURPOSE: To determine the effect of PH versus exercise on the HSP70 response and glucose control in overweight and lean humans.

METHODS: In a crossover design, ten physically inactive males (5 lean (L) BMI = 23.5 kg.m⁻², fat mass = 9.5 ± 4.8 kg; 5 overweight (OW) BMI = 28.0 kg.m⁻², fat mass = 20.9 ± 5.3 kg) underwent 60 minutes of exercise at a fixed rate of metabolic heat production (EX, 7.5W.kg⁻¹). In a second trial, participants underwent PH (2.0 W.kg⁻¹) matched to EX for duration, core temperature (Tc) increase and dietary intake. A venous blood sample was obtained before and immediately following heating for analysis of HSP70. Continuous glucose monitoring was used to measure interstitial glucose for the subsequent 24 hours.

RESULTS: There were no differences in ∆Tc between groups (L = 0.87 ± 0.23°C; OW = 0.85 ± 1.7°C, p = 0.784) or conditions (EX = 0.8 ± 0.2°C, PH = 0.9 ± 0.2°C, p = 0.913) following heating. Resting HSP70 was negatively correlated with fat mass (r=-0.528, p<0.05). There was a trend between ∆HSP70 and fat mass (r=-0.303, p = 0.194). HSP70 increased 6.8% after heating (p<0.05) with a greater increase in L (0.40 ± 0.44 ng.mL⁻¹) compared to OW (-0.26 ± 0.27 ng.mL⁻¹, p<0.05) after PH but no difference in ∆HSP70 between L and OW following EX. There was no difference in glucose area under the curve between EX and PH, but peak glucose in the first meal following heating was lower for PH (6.7 ± 1.4 mmol.L⁻¹ vs. 7.4 ± 1.2 mmol.L⁻¹, p<0.05).

CONCLUSION: The HSP70 response after PH is impaired in OW compared to L and appears linked to fat mass. As PH resulted in no increase in HSP70 in OW, this suggests a need for greater inflammatory stimulus from muscular contraction to elicit rises in HSP70 in OW. PH reduces peak glucose excursions in the first meal after heating and may contribute to improved insulin sensitivity following repeated acute PH exposures which warrants future investigation.