Effect of heavy and light training periods on blood glucose response during rowing

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From randomly performed blood test, Reid et al. (2004: British Journal of Sports Medicine, 38, 42 – 45) observed non-fasting hypoglycaemia in 28% of 36 competitive athletes undertaking repeated bouts of intensive training. The aim of the present study was to investigate whether rowers demonstrated altered blood glucose responses at rest and during exercise when they were at the end of heavy and light training periods, and whether this might coincide with changes in other metabolic variables and exercise performance. Ethical approval for this study was granted through the University Ethics Committee. Twelve highly trained male and female university rowers (mean ± s: age 21 ± 1 yrs, stature 1.87 ± 0.07m, body mass 83.2 ± 11.0 kg) completed an incremental rowing protocol consisting of five, four minute stages with one minute recovery in between, followed by a final stage where participants tried to achieve as high a power output as possible over four minutes. The intensity for the fifth stage was determined from the rower’s two km time, from which the time per 500-m was found and then 4-s were added. Finally, this time was converted into a power output. Power outputs for other stages (the 4th stage to the 1st stage) were then calculated by taking 30-W off each stage in turn. At the end of each stage, mean power output, heart rate (Polar Sportstester, Polar Electro, Finland), RPE (Borg: 1986 In The perception of exertion in physical work, edited by G. Borg and D. Ottson. London, Macmillan), capillary blood lactate (Lactate ProLT-1710, Akray, Japan) and glucose (β-glucose 201 Photometer, Haemocue Ltd, England) were measured. Following the final stage, heart rate was recorded immediately after exercise cessation and at one, four and seven minutes post-exercise, along with blood lactate and glucose. Immediately prior to each test, participant’s body mass (SECA 770, Germany), urine osmolality (Advanced Micro Osmometer, Model 3300, Advanced instruments mc, USA), blood pressure (Omron MS-i, England), lactate and glucose were measured. Prior to this, participants had undertaken an “orthostatic test” (Nummela and Rusko, 2000: Journal of Sports Sciences, 18, 411 – 419) where they lay in a supine position for five minutes and then stood up for three minutes. Mean heart rate was recorded during the final minute in the supine position and between 90-s and 120-s after standing up. Peak heart rate within 30-s of standing was also noted. Participants completed the tests following a heavy, six week period of training and then again following a light, three week period of training. They consumed only water in the seven hours preceding the tests. Having recorded their dietary intake in the 48-hours prior to the first test participants replicated their intake before the second test. As data were normally distributed a dependent T-test was used to establish if a systematic bias was present between trials. A significance level of P<0.05 was adopted throughout. Differences between trials were found only in the blood glucose responses, following incremental protocol stages one (T=2.657, P=0.033, ω²=0.39), two (T=2.370, P=0.050, ω²=0.28), three (T=7.659, P=0.000, ω²=0.81), four (T= 4.5 13, P=0.030, ω²=0.60) and six 1 min to 7 min (T=3.456-
5.053, \( P=0.010-0.001, \omega^2=0.46-0.65 \). This suggests that a greater liver glycogenolysis occurred following the heavy period of training. This could have been due to low pre-exercise muscle glycogen or an elevated stress response to the exercise. However, parasympathetic/sympathetic nervous system modulation at rest was unchanged. Whether an elevated blood glucose response to exercise is a symptom of an “overreached” condition is unknown.