Day 2: Free Communications – Physiology and Nutrition

D2.S2.2(1). The effects of a high-intensity heavy-resistance cycling warm-up on subsequent 4-km time trial performance

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Prior exercise can improve performance in endurance cycling events. Whilst a number of different techniques have been investigated, post-activation potentiation (PAP), the phenomenon whereby the performance of a muscle is positively affected by its recent contractile history (Hodgson, Docherty, & Robbins, 2005, *Sports Medicine*, *35*, 585–595), has rarely been investigated in endurance events. Recently, such performance improvements have been found in rowing (Feros, Young, Rice, & Talpey, 2012, *Journal of Strength and Conditioning Research, 26*, 3326–3334) and running (Barnes, Hopkins, McGuigan, & Kilding, 2014, *Medicine and Science in Sports and Exercise*, *46*, 948–948). Therefore, the aim of this study was to examine the effects of a cycling based warm-up routine including high-intensity heavy-resistance contractions on short-duration endurance cycling performance and physiological measures during a 4-km time trial. With institutional ethics approval, in a repeated measures counterbalanced design, 11 well-trained male endurance cyclists ($\dot{V}O_{2max}$ 65.3 ± 5.3 mL · kg^{-1} · min^{-1}) performed two 4-km cycling time trials following a 5-minute recovery after a moderate intensity cycling warm-up (MODW) at 60% of $\dot{V}O_{2max}$ for 6.5 min, and a warm-up with PAP conditioning contractions (PAPW) consisting of 5 min at 60% of $\dot{V}O_{2max}$ then 3 × 10 s at 70% of peak power interspersed with 30-s recovery. Blood was taken to determine lactate concentrations at rest, following the warm-up and at completion of the time trial. Expired gases were analysed throughout the time trial and were assessed along with time and power output (PO) over each 500 m split. Prior to the time trial, blood lactate was elevated following PAPW compared to MODW (4.88 ± 1.36 mmol · L^{-1} vs. 1.14 m ± 0.26 mmol · L^{-1}; ES: 3.81 ± 0.73) together with small improvements in both completion time (1.7 ± 3.5 s; ES: 0.44 ± 0.49) and PO (5.1 ± 10.5 W; ES: 0.25 ± 0.26), and an oxygen uptake ($\dot{V}O_2$) elevated by 1.44 ± 1.65 mL · kg^{-1}·min^{-1} (ES: 0.28 ± 0.16). *Post-hoc* analysis revealed that the differences in time, PO and $\dot{V}O_2$ were most evident during the first 1500 m of each trial. The results suggest a warm-up with heavy-resistance efforts intended to enhance PAP leads to performance improvements, together with elevated blood lactate concentration and increased $\dot{V}O_2$ during the first 1500 m, hence would be of benefit in short endurance track cycle races such as the kilo and pursuit.

D2.S2.2(2). Determinants of W′ in the power duration relationship in normoxia and hypoxia: the effect of induced alkalosis

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Critical power (CP) demarcates the boundary between heavy and severe exercise intensities. The severe exercise intensities, between CP and VO_{2peak}, are described by W′, which represents the work capacity above CP. Determinants of W′, and associated limitations to high-intensity exercise above CP, are not fully understood. Therefore, the purpose of this study was to determine the effect of induced alkalosis on the power–duration relationship under normoxic and hypoxic conditions. Ten trained cyclists (age (mean ± SD): 32 ± 8.6 years; body mass (BM); 76.1 ± 9.2 kg; VO_{2peak}: 59.2 ± 6.8 mL · kg · min^{-1}) completed seven laboratory trials which involved the determination of individual time to peak alkalosis following sodium bicarbonate ($NaHCO_3$) ingestion, an environment-specific ramp test and 4 × 3-min "all-out" CP tests under different experimental conditions. Participants completed the following trials: (1) alkalosis and normoxia (ALN); (2) placebo and normoxia (PLN); (3) alkalosis and hypoxia (ALH) and (4) placebo and hypoxia (PLH) using a randomised crossover design. Pre-exercise alkalosis was induced via 0.3 g · kg^{-1} BM of $NaHCO_3$, with a taste-matched placebo of 0.21 g · kg^{-1} BM of sodium chloride used to match sodium molarity between conditions. Environmental conditions were set in the normobaric chamber to hypoxic (FiO_2%: 14.5%) or normoxic (FiO_2%: 20.93%). Institutional ethical approval was obtained for this investigation. Individual time to peak alkalosis was attained at 52.6 ± 14.5 min, with alkalosis significantly induced with $NaHCO_3$ compared to placebo under both normoxia (P = 0.045) and hypoxia (P = 0.05). Mean critical power was greater in normoxia compared to hypoxia (299.6 ± 39.4 W vs. 258.7 ± 32.3 W; P < 0.05; d = 1.11), whereas there was no difference between induced alkalosis within either environmental condition (ALN: 311.2 ± 51.0 W; P > 0.05; d = 0.25 and ALH: 263 ± 31.7 W; P > 0.05; d = 0.15). A significant increase in W′ was observed under both normoxia (PLN: 14.39 ± 5.94 Kj vs. ALN: 16.8 ± 4.98 Kj; P = 0.42; d = 0.44) and hypoxia (PLH: 14.93 ± 5.08 Kj vs. ALH 17.4 ± 5.16 Kj; P = 0.01; d = 0.50). Blood H^+ accumulation during exercise was greater with induced alkalosis compared to placebo in normoxic (P = 0.005) and hypoxic (P < 0.001) conditions. Induced alkalosis also increased change in blood HCO_3^- concentrations during exercise in both conditions (normoxic: P = 0.018; hypoxic: P = 0.001). In conclusion, this is the first study to show that the reinforcement of the physiochemical