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Total PYY and GLP-1 responses to submaximal continuous and supramaximal sprint interval cycling in men

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ABSTRACT

Exercise-induced changes in appetite-regulating hormones may be intensity-dependent, however a clear dose-response relationship has not been established. The purpose of this study was to examine changes in anorexigenic markers (total PYY and GLP-1) in response to rest or exercise at submaximal and supramaximal intensities. Ten active males completed four experimental sessions in randomized order: 1) Moderate intensity continuous training (MICT; 30 min cycling at 65% VO_{2max}); 2) High intensity continuous training (HICT; 30 min cycling at 85% VO_{2max}); 3) Sprint interval training (SIT; 6 × 30 s "all-out" cycling bouts with 4 min recovery periods); 4) Control (CTRL; no exercise). Blood samples were obtained immediately pre- and post-exercise, as well as 90-min post-exercise for the measurement of total PYY and GLP-1. Subjective hunger was assessed using a visual analog scale pre-breakfast and at the three blood sampling time-points. Total PYY concentrations increased immediately post-exercise following both HICT ($P = 0.006$) and SIT ($P < 0.001$) versus CTRL, while SIT was also greater ($P = 0.005$) compared to MICT. Total GLP-1 concentrations changed similarly across time-points ($P < 0.001$), with no differences between sessions ($P = 0.280$). Perceptions of hunger also changed similarly across time-points ($P < 0.001$) with no differences between trials ($P = 0.085$). These findings suggest that total PYY increases only after high-intensity exercise and exhibits a greater responsiveness to SIT compared to moderate-intensity exercise. Compensatory increases in hunger do not seem to occur at any exercise intensity. These findings support a dose-response relationship between exercise intensity and total PYY, though the effects on total GLP-1 and hunger perceptions seem unclear.

1. Introduction

Recent obesogenic trends can be attributed to reductions in energy expenditure (EE) coupled with increases in energy intake (EI), highlighting the need for improved weight loss therapies (Hussain & Bloom, 2013). While exercise is widely utilized as a weight loss tool, its effectiveness in the absence of dietary restriction is suboptimal (Jakicic et al., 2001). This may be due to compensatory increases in post-exercise EI, which prevent the energy deficit required for weight loss (King, Hopkins, Caudwell, Stubbs, & Blundell, 2008; King et al., 2012). Thus, exercise protocols that increase EE without promoting subsequent increases in EI are highly desirable.

High-intensity interval training (HIIT) involves brief near maximal exercise bouts separated by short recovery periods and promotes similar physiological adaptations as moderate-intensity (50–75% VO_{2max}) continuous training (MICT) of much longer duration (Gibala, Gillen, & Percival, 2014). Sprint interval training (SIT) is a more intense but equally effective form of intermittent exercise that consists of four to six 30-sec supramaximal efforts (>100% VO_{2max}) interspersed with 4 min recovery periods (Gibala et al., 2014). In addition to numerous health and performance benefits (Gibala, Little, MacDonald, & Hawley, 2012), studies involving HIIT/SIT have demonstrated fat loss despite significantly lower training volume and time-commitment than MICT (Boutcher, 2011; Hazell, Hamilton, Olver, & Lemon, 2014; MacPherson, Hazell, Olver, Paterson, & Lemon, 2011). This may be attributable to elevations in post-exercise metabolism (Beaulieu, Olver, Abbott, & Lemon, 2015; Chan & Burns, 2013; Hazell, Olver, Hamilton, & Lemon, 2012; Skelly et al., 2014; Townsend, Couture, & Hazell, 2014) and/or a substrate shift that favors fat utilization in

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recovery (Beaulieu et al., 2015; Chan & Burns, 2013). However, some studies have suggested that the magnitude of these effects is likely inadequate for explaining the fat loss observed with HIIT/SIT (Kelly, King, Goerlach, & Nimmo, 2013; Williams et al., 2013). Therefore, the improvements in body composition may also be facilitated by other aspects of energy balance, potentially via changes in appetite and/or EI (Schubert, Desbrow, Sabapathy, & Leveritt, 2013).

Physiologically, appetite is regulated by neuroendocrine interactions involving peripheral hormones and key brain regions involved in energy homeostasis (Hussain & Bloom, 2013). A recent meta-analysis indicates that exercise does not lead to compensatory increases in EI (Schubert et al., 2013) and may even influence hypothesized appetite-regulating hormones in a manner that reduces EI (Schubert, Sabapathy, Leveritt, & Desbrow, 2014). The gastrointestinal hormones glucagon-like peptide-1 (GLP-1) and peptide YY (PYY) are expressed and secreted by enteroendocrine cells and their major/bioactive forms ($GLP_{7\text{-}36}$, $GLP\text{-}1_{7\text{-}37}$, $PYY_{3\text{-}36}$) act as episodic signals that increase satiety in response to food intake (Cummings & Overduin, 2007; Hussain & Bloom, 2013; Psichas, Reimann, & Gribble, 2015). The transient suppression of appetite that is observed following strenuous endurance exercise (Broom, Stensel, Bishop, Burns, & Miyashita, 2007; King, Burley, & Blundell, 1994) may be partly mediated by changes in PYY and GLP-1 (Broom, Batterham, King, & Stensel, 2009; King et al., 2011; Martins, Morgan, Bloom, & Robertson, 2007; Ueda, Yoshikawa, Katsura, Usui, & Fujimoto, 2009). Furthermore, there is evidence to suggest that HIIT/SIT may lead to a greater suppression of appetite (Deighton, Barry, Connon, & Stensel, 2013; Deighton, Karra, Batterham, & Stensel, 2013; Williams et al., 2013) and subsequent EI (Alkahtani, Byrne, Hills, & King, 2014; Crisp, Fournier, Licari, Braham, & Guelfi, 2012; Sim, Wallman, Fairchild, & Guelfi, 2014) compared to lower-intensity exercise protocols. Given that several mechanisms proposed to influence appetite-regulating hormones may be intensity-dependent (Hazell, Islam, Townsend, Schmale, & Copeland, 2016), a direct comparison between exercise protocols at different intensities is warranted. Though several studies have compared HIIT/SIT with MICT, few to date have included high-intensity (75–85% VO_{2max}) continuous training (HICT), making it difficult to establish a dose-response relationship between exercise intensity and appetite regulation.

The purpose of this study was to examine the effects of MICT, HICT, and SIT on plasma levels of total PYY and GLP-1 as well as hunger sensations, in order to establish a dose-response relationship between exercise intensity and appetite regulation. We hypothesized that exercise would elicit intensity-dependent effects on appetite regulation, where both SIT and HICT would result in higher PYY and GLP-1 concentrations than MICT, with consequently lower hunger sensations.

2. Methods

2.1. Participants

Ten active young males were recruited. All participated regularly (at least 3 times per week) in vigorous exercise and were comfortable in completing high-intensity and exhaustive exercise bouts. Participants were apparently healthy, non-smokers, and not taking any medications. All were instructed to perform no physical activity or ingest any caffeine for 48 h prior to any laboratory visit. Prior to the initiation of the study all participants provided written informed consent, passed the Physical Activity Readiness Questionnaire (PAR-Q) health survey, and participated in a familiarization visit (one week prior to the 1st experimental session). The University of Lethbridge Human Subject Research Committee approved all experimental procedures in accordance with the ethical standards of the 1964 Declaration of Helsinki.

2.2. Pre-experimental procedures

During the familiarization session, participants were acclimatized to the exercise equipment as well as the types and degrees of effort required during the different exercise protocols. Height was measured to the nearest 0.5 cm and body weight to the nearest 0.1 kg by a physician beam scale (Health-o-meter Professional, Sunbeam Products, Inc. Florida, USA). Participants then completed a cycling VO_{2max} test using an incremental test to exhaustion on a mechanically braked cycle ergometer (model 874-E, Monark Exercise, Stockholm, Sweden) using an online breath-by-breath gas collection system (Quark CPET, Cosmed, California, USA). Heart rate (HR) was recorded throughout the test using an integrated HR monitor (Polar Electro, New York, USA). Before testing, gas analyzers were calibrated using gases of known concentration and a 3-L syringe for flow. After a 5-min warm-up at a resistance of 1.5 kg and 70 rpm (105 W), an additional 0.5 kg resistance was added every 2 min until 70 rpm could no longer be maintained. Verbal encouragement was provided throughout the test. VO_{2max} was defined as the greatest 30-s average over the course of the test and established by the presence of a plateau in the VO_2 values (<1.35 mL kg^{-1} VO_2 increase) despite an increase in resistance or two of the following criteria: 1) a respiratory exchange ratio (RER) value $\geq$ 1.10; 2) the achievement of a maximal HR within 10 bpm of the predicted maximal HR (220-age) and/or; 3) visible exhaustion.

2.3. Experimental sessions

At least one week after completing the preliminary exercise test, participants completed a randomized four-way crossover study with all experimental sessions (Fig. 1) performed 1 week apart and on the same day of the week. The four experimental sessions consisted of: 1) moderate-intensity continuous training (MICT; 65% VO_{2max}); 2) high-intensity continuous training (HICT; 85% VO_{2max}); 3) sprint interval training (SIT) and; 4) no exercise control CTRL). For 24 h before the first experimental session, participants recorded their weighed food intake using a food record diary. Participants were instructed to consume the same food for the 24 h prior to each subsequent session. On the day of the experimental session participants arrived at the laboratory at 0800 h following a 12 h overnight fast and remained in the laboratory for the next ~3 h (Fig. 1). Upon arrival participants were given ~15 min to consume a standardized breakfast (16.8 kJ kg^{-1} body mass) consisting of a Clif bar (up to 1050 kJ; Clif Bar & Company, California, USA), plain Quaker rice cake (up to an additional 147 kJ; PepsiCo, Ontario, Canada), and natural peanut butter (up to all remaining allotted kJ; Costco Wholesale, Washington, USA). Water was provided ad libitum throughout experimental sessions. After allowing sufficient time for digestion (~45 min), the exercise session began at 0900 h and included a 5 min standardized warm-up, a 30 min exercise protocol (27 min for SIT with an additional 3 min rest prior to warm-up to match), and a 5 min cool-down. Upon completion of exercise (0940 h), participants rested in the laboratory for an additional 90 min while resting quietly (i.e. reading or using a laptop). Venous blood samples were obtained (detailed below) at 0900 h (pre-exercise), 0940 h (immediately post-exercise), and 1110 h (90 min post-exercise). Subjective hunger (i.e. “How hungry do you feel?”) was assessed at pre-breakfast and each blood sampling time-point hereafter using a visual analog scale anchored at each end with contrasting statements (i.e. “I am not hungry at all” and “I have never been more hungry”) (Flint, Raben, Blundell, & Astrup, 2000). Identical procedures were followed during the