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Appetite-regulatory hormone responses on the day following a prolonged bout of moderate-intensity exercise

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HIGHLIGHTS

- Circulating acylated ghrelin concentrations were reduced on the day after exercise.
- Circulating leptin concentrations were reduced on the day after exercise.
- Exercise did not affect circulating insulin or total PYY on the day after exercise.
- Appetite perceptions were unaltered on the day after exercise.

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ABSTRACT

Exercise increases energy expenditure however acutely this does not cause compensatory changes in appetite or food intake. This unresponsiveness contrasts the rapid counter-regulatory changes seen after food restriction. The present investigation examined whether corrective changes in appetite-regulatory parameters occur after a time delay, namely, on the day after a single bout of exercise. Nine healthy males completed two, two-day trials (exercise & control) in a random order. On the exercise trial participants completed 90 min of moderate-intensity treadmill running on day one (10:30–12:00 h). On day two appetite-regulatory hormones and subjective appetite perceptions were assessed frequently in response to two test meals provided at 08:00 and 12:00 h. Identical procedures occurred in the control trial except no exercise was performed on day one. Circulating levels of leptin were reduced on the day after exercise (AUC 5841 ± 3335 vs. 7266 ± 3949 $ng^{-1} \cdot mL^{-1} \cdot 7$ h, $P = 0.012$). Conversely, no compensatory changes were seen for circulating acylated ghrelin, total PYY, insulin or appetite perceptions. Unexpectedly, levels of acylated ghrelin were reduced on the exercise trial following the second test meal on day two (AUC 279 ± 136 vs. 326 ± 136 $pg^{-1} \cdot mL^{-1} \cdot 3$ h, $P = 0.021$). These findings indicate that short-term energy deficits induced by exercise initially prompt a compensatory response by chronic but not acute hormonal regulators of appetite and energy balance. Within this 24 h time-frame however there is no conscious recognition of the perturbation to energy balance.

1. Introduction

The relationship between exercise and appetite regulation has important implications regarding the role of exercise in weight management [1]. In recent years, advancements in scientific understanding regarding the psycho-biological regulation of appetite and food intake have ignited research interest around the interaction between exercise, appetite regulation and energy balance [2]. Within this sphere, one particular issue that has received significant attention is the impact of exercise on hormonal mediators of appetite which are central components of the body's homeostatic system governing energy balance and weight control [3,4].

The body's appetite-regulatory system includes several peptides of gastro-intestinal, pancreatic and adipose tissue origin, which communicate acute nutrient status and chronic energy availability to the central nervous system [4]. Leptin and insulin act as chronic mediators of energy balance, with circulating concentrations being present in proportion to stored energy within adipose tissue [5]. Additionally, on a meal-to-meal basis, food intake is regulated by a selection of gastrointestinal peptides, most notably acylated ghrelin, peptide-YY (PYY), glucagon-like peptide-1 (GLP-1), cholecystokinin (CCK) and oxyntomodulin [6].

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Ghrelin is secreted from the stomach and remains unique as the only circulating appetite stimulating hormone. Furthermore, the prandial profile of ghrelin is suggestive of an important role in meal initiation [7,8]. Conversely, each of the other short-acting peptides has an inhibitory effect on appetite. Highly prominent is PYY which is secreted chiefly from the distal intestine and colon in direct proportion to the energy content of an ingested meal [9,10]. Within key appetite-regulatory brain centres these afferent signals are integrated and the summed response initiated which impacts directly up on appetite and eating, as well as thermogenesis and substrate metabolism [11].

Research has demonstrated that single bouts of exercise have a marked impact on the circulating levels of appetite-regulatory hormones with changes occurring rapidly after the initiation of exercise [2]. Notably however, these alterations appear to be transient. For example, circulating levels of acylated ghrelin are distinctly suppressed during exercise of moderate-intensity or higher [12–14]. This perturbation however is absent within 30 min after exercise. Similarly, circulating concentrations of PYY increase during moderate- to high-intensity exercise however customary levels are re-established shortly thereafter [2,15,16]. Each of these responses is consistent with an appetite-inhibitory profile which may in part contribute to a well-characterised inhibition of appetite at moderate-high exercise intensities, a phenomenon which has been termed 'exercise-induced anorexia' [17].

Studies have shown that acute energy deficits induced by food restriction lead to rapid and quite striking compensatory alterations to appetite and appetite-regulatory hormones i.e., hormones change in directions expected to stimulate appetite and eating [14,18]. Intuitively, it may be expected that energy deficits induced by exercise would lead to similar responses in appetite-regulatory parameters however several studies have failed to observe any compensatory changes in circulating appetite hormones (acylated ghrelin or PYY) on the day that exercise is performed. This unresponsiveness occurs with bouts of exercise associated with high levels of energy expenditure i.e., large perturbations to energy balance, and over several hours of observation afterwards [13,14]. This is consistent with a lack of change in energy intake [19]. It remains possible that there is a time-delay before exercise-induced energy deficits manifest as alterations in appetite, appetite regulatory parameters and food intake. This notion was postulated half a century ago [20] and in the absence of altered oro-gastric input, may reflect a greater time-span necessary for the body to detect and respond to exercise-induced energy balance perturbations. This notion is supported by previous evidence which identified latent changes in circulating leptin on the day after exercise [21,22]; and the findings of a recent study which documented an exercise-induced suppression of fasting and meal-stimulated plasma acylated ghrelin response 12 h after undertaking exercise [23].

The present study assessed the latent effects of exercise on appetite and critical mediators of appetite control and energy balance on the day after a single bout of exercise. Specifically, we sought to confirm and extend previous findings by characterising the meal-stimulated (breakfast and lunch) responses of key acute and chronic appetite-regulatory hormones (acylated ghrelin, total PYY, leptin & insulin) on the day after a single bout of exercise. We hypothesised that meal-stimulated acylated ghrelin (suppression) and PYY (elevation) responses would be attenuated on the day after exercise whilst circulating levels of leptin would be reduced. Furthermore, we thought that these changes would be associated with commensurately altered subjective appetite perceptions.

2. Materials & methods

2.1. Participants

After receiving local ethical advisory committee approval nine young, healthy male volunteers (age 22.0 ± 1.2 y; weight 72.0 ± 6.9 kg; BMI 22.6 ± 1.8 $kg \cdot m^2$; waist circumference 74.4 ± 1.8 cm; estimated basal metabolic rate 7247 ± 405 kJ; $\dot{V}O_2$ max 60.6 ± 7.6 $mL \cdot kg \cdot min^{-1}$) gave their written informed consent to participate. Participants were weight stable (<2 kg change in body mass in the last three months), non-smokers, free of cardio-metabolic disease, had a BMI within the healthy range (18.5–24.9 $kg \cdot m^2$) and were not taking any medications or supplements. Participants were active i.e., typically games players, but were not accustomed to undertaking endurance exercise regularly.

2.2. Pre-assessment and study familiarisation

Before main trials, participants attended the laboratory where they were familiarised with the study procedures and underwent necessary pre-assessments. Participants completed questionnaires assessing health status and physical activity habits after which measurements of height, weight and waist circumference were taken. Participants then completed two treadmill running tests; 1) a progressive 16 min submaximal test to determine the relationship between treadmill running speed and oxygen consumption; 2) a maximum oxygen uptake test ($\dot{V}O_2$ max). These tests have been described in depth previously [12].

2.3. Main experimental trials

In subsequent weeks participants completed two main experimental trials (exercise and control) separated by a washout period of at least seven days. Each main trial spanned across two days and was preceded by a 48 h lead-in phase where diet and physical activity (absence of) were standardised. Within this standardisation phase dietary intake was controlled by the participants i.e., on each participant's first trial they ate ad libitum however participants recorded what they ate and replicated it exactly in the lead up to their second main trial. Adherence to this procedure was confirmed verbally by the study experimenters before main trials. Each main trial was composed of an intervention phase (day one) and a data-collection phase (day two). This design permitted the assessment of appetite-regulatory responses on the day after exercise. The order of main trials was randomised with five participants completing the control trial first and four completing the exercise trial first. Fig. 1 provides a schematic illustration of the main trial protocol.

Main trials began on the morning of day one and ended at approximately 15:10 on day two. During this period participants were required to attend the laboratory between 10:00–13:30 on day one and 07:30–15:10 on day two. In the time away from the laboratory participants were instructed to remain completely inactive and this was checked repeatedly by the study experimenters via telephone. During the study, participants travelled to and from the laboratory via motorised transport unless they lived within 400 m in which case they were permitted to walk. During main trials participants were provided with all of their food which was consumed at set times that were standardised across trials. Water was permitted ad libitum on day one, however to avoid any impact on appetite and/or gastric function during the data-collection phase of trials water consumption was standardised on day two.

On day one of the exercise trial participants consumed their standardised breakfast at home at 07:30. At 10:00 participants arrived at the laboratory ahead of their treadmill run (10:30–12:00). Herein, participants ran on a motorised treadmill (Technogym Excite Med, Cesena, Italy) for 90 min at a speed predicted to elicit 70% of their maximum oxygen uptake. At 15 min intervals oxygen uptake was assessed via expired air collections into a Douglas Bag and the speed of the treadmill was adjusted if necessary to maintain the desired exercise intensity. Ratings of perceived exertion were also assessed using the Borg scale [24]. Following the run participants rested in the laboratory until lunch (13:00). After lunch participants went home where they remained (inactive) until returning to the laboratory the following morning.