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ORIGINAL ARTICLE

Effects of resistance exercise volume on appetite regulation and lipid profile in overweight young men

Effets d'exercices de renforcement musculaire (RE) à différents volumes sur l'appétit, ses régulateurs hormonaux et les lipoprotéines plasmatiques chez les hommes en surpoids

F. Rahmani-Nia[a], **B. Mirzaei**[a], **R. Rahimi**[b,*]

[a] *Department of exercise physiology, faculty of physical education and sport science, university of Guilan, P.O. Box 41635-1438, Rasht, Iran*
[b] *Department of physical education and sport sciences, university of Kurdistan, P.O. Box 416, 66177-15175 Sanandaj, Iran*

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Summary
Objectives. — This study investigated the effect of resistance exercises (RE) with different volumes on appetite, its hormonal regulators (PYY and insulin), and plasma lipoprotein in overweight men.
Methods. — Nine healthy overweight male students (age: 20.88 ± 2.52 years, weight: 99.53 ± 14.46 kg, body mass index: 29.7 ± 2.74 kg/m^2) selected randomly and performed two RE protocols with high volume (5 sets × 12 repetitions at 75% of 12RM) and low volume (3 sets × 12 repetitions at 75% of 12RM load), in two randomized separate sessions. Blood samples were collected pre-exercise, immediately after, 1, 3, and 6 h after resistance exercise sessions.
Results. — Plasma PYY concentrations significantly increased after both RE protocols. Also, plasma insulin concentrations were significantly increased in both protocols at 1 h after exercise compared to pre-exercise, 3 and 6 h post-exercise. Appetite suppression at the end of RE with high volume was higher than RE with low volume. Appetite measure was significantly lower immediately post-RE protocols at 1, 3 and 6 h post-exercise. Plasma HDL-C level increased significantly after resistance exercise.

* Corresponding author.
E-mail addresses: rahman.rahimi@yahoo.com, r.rahimi@uok.ac.ir (R. Rahimi).

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Conclusion. — Appetite suppression after resistance exercise that was observed in the present study may be due to increases in anorexigenic hormones such as PYY and insulin. Also, resistance exercise increased HDL-C levels that have protective effect against plaques generation.

MOTS CLÉS
Peptide YY (PYY) ;
Insuline ;
Renforcement musculaire ;
Profil lipidique

Résumé
Objectifs. — Cette étude a permis d'évaluer les effets d'exercices de renforcement musculaire (RE) à différents volumes, sur l'appétit, ses régulateurs hormonaux (PYY et insuline), et les lipoprotéines plasmatiques chez les hommes en surpoids.
Méthodes. — Neuf étudiants du sexe masculin en surpoids, sans pathologies connues (âge : 20,88 ± 2,52 ans, poids : 99,53 ± 14,46 kg, indice de masse corporelle : 29,7 ± 2,74 kg/m^2) ont été choisis au hasard, et deux protocoles de RE leur ont été proposés, l'un avec un volume élevé (5 séries de 12 répétitions à 75 % de 12RM) et l'autre à faible volume (3 séries de 12 répétitions à 75 % de 12RM), en deux sessions séparées, et réalisées de manière randomisée. Un prélèvement sanguin a été réalisé avant l'exercice, immédiatement après, 1, 3, et 6 h après les séances de RE.
Résultats. — Les concentrations plasmatiques de PYY ont considérablement augmenté après les deux protocoles de RE. La concentration d'insuline dans le plasma a augmenté dans les deux protocoles, 1 h après l'exercice (par rapport aux mesures réalisées avant l'exercice), et 3 et 6 h après l'exercice. L'appétit était diminué 1, 3 et 6 h après les exercices de RE. L'effet suppresseur de l'appétit des exercices était plus important après le RE à volume élevé qu'après le RE à plus faible volume. Les concentrations plasmatiques de HDL-C ont augmenté de façon significative après les exercices de résistance.
Conclusion. — La baisse de l'appétit après les exercices de renforcement musculaire pourrait être due à l'augmentation des hormones anorexigènes telles que le PYY et l'insuline. En outre, les exercices de résistance abaissent les concentrations plasmatiques de HDL-C, ce qui représente un facteur de protection contre la formation de plaques d'athérome.

1. Introduction

Overweight and obesity are major problems in the 21st century. Obesity is a contributor to cardiovascular disease, diabetic, skeletal malformation, respiratory and metabolic problems and also is incidence agent in some cancers and psycho-social disorders [1]. Obesity is primarily due to increased energy intake (EI) along with decreased energy expenditure (EE), and this imbalance contributes to increased body fat mass. Body weight is regulated by a balance between both EI and EE. A potent and complex physiological system exists to balance EI and EE including hypothalamus, brainstem and reward centers that regulate energy homeostasis via neuropeptides.

From previous studies in humans, it is thought that exercise is an effective method to create a negative energy balance [2,3], which relies not only directly on its impact on increasing EE, but also indirectly on its potential to a short-term hunger suppression [4]. The effect of acute exercise on appetite regulation is controversial, with studies demonstrating no change in hunger and energy intake during or after exercise [3], as well as increase [5,6] and decrease [2,7]. The mechanisms behind exercise-induced appetite regulation are three categories, which included long-term signals such as leptin and insulin, intermediate signals including post-absorptive signals associated with macronutrient oxidation and short-term mechanisms including post-ingestive signals such as the gut peptides ghrelin, cholecytokinin, glucagon-like peptide-1 and peptide YY (PYY) [4,8].

Gastrointestinal (GI) peptides seem to play an important role in the regulation of feeding [9]. Among these peptides, PYY is an anorexigenic 36-amino acids peptide which was first isolated in 1980 from porcine intestinal mucosa [10]. This peptide is synthesized and released by specialized endocrine cells called L-cells found predominantly within the distal GI tract [11] in response to intraluminal nutrients [12]. Its production level increases after food intake and mediates its effects through the G-protein coupled receptors: Y1-Y6. PYY promotes satiety by binding to Y2 receptors of neuropeptide Y (NPY) within the hypothalamus and inhibiting NPY secretion [13,14]. NPY is one of the most potent orexigenic agents that act to stimulate feeding predominantly through activation of Y1 and Y5 receptors in the hypothalamus [15]. Also, among long-term signals, insulin is a major metabolic hormone produced by the pancreas. It is likely that insulin crosses the blood—brain barrier by a saturable mechanism that involves both transporter and receptor specific proteins [16]. Once insulin enters the brain, it acts as an anorexigenic signal, decreasing intake and body weight. In addition, injections of insulin directly into the hypothalamic paraventricular nucleus decrease food intake and rate of weight gain in rats [17].

Little is known about the PYY and appetite response to resistance exercise (RE). Only Broom, et al. [18] has investigated PYY response to an acute bout of RE and did not observe a rise in post-exercise plasma PYY concentration,