Effect of an Acute Incremental Exercise on Plasma Peptide YY, Neuropeptide Y and IGF-1 Concentrations in Young Athletes

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ABSTRACT

This study aims to determine the effect of a single exhausting exercise on plasma PYY, NPY and IGF-1 in young athletes. Thirty-one young athletes (aged 19.52±2.75 years, body mass 79.24±16.13 kg, height 173±6.49 cm, and body fat 16.37±5.92%) volunteered to participate in this study. The participations, in randomized crossover manner, performed a single exercise bout on a cycle ergometer at 50 watts (W) for five-minute, and the power output was increased by 30 W every 3 minute until voluntary exhaustion. Venous blood samples were collected before (Pre), immediately after (Post) and 1h after (1h Post) exercise to assess plasma PYY, NPY and IGF-1 concentrations. Plasma PYY (p=0.042) and IGF-1 (p=0.001) significantly increased at Post exercise. Also, NPY was increased Post (p=0.001) and for 1h Post (p=0.021) exercise. The exhausting exercise increase plasma PYY concentration and this increase may relate to IGF-1 signals in young athletes. However, increased plasma NPY may not be related to stimulation food intake because it does not penetrate the blood-brain barrier.

Key Words: Appetite, Food Intake, Athletes, Anorexia.
INTRODUCTION

Over-weight is not desirable and has implications for health and in the case of athletes affects performance. This later implication of over-weight makes necessary weight-control among athletes. Exercise is a strategy used to counteract overweight owing to create a negative energy balance by increasing energy expenditure and influencing appetite regulating hormones (1, 2).

Interestingly, recent studies have revealed inhibitory effects of exercise on the hunger associated with these hormones in healthy subjects (3), suggesting the intriguing possibility that exercise may promote a favorable appetite hormones profile yielding sustained appetite control and weight loss (2, 4). Therefore, it’s suggested that exercise has an impact not only on energy expenditure but also on variations in appetite and post-exercise energy intake, leading to negative energy balance (5).

Previous studies have reported that intense (but not necessarily moderate or light) exercise may induce a transient suppression of hunger that has been reported in both humans (2, 6) and experimental animals (7). This temporary repression of appetite has been termed ‘exercise-induced anorexia’ and it leads to a short-term negative energy balance (2). The specific mechanisms behind regulation of exercise-associated changes in appetite are still under investigation. An important group of the intricate factors for appetite control involved in the brain-gut axis of eating behavior are ghrelin, cholecystokinin, pancreatic polypeptide, peptide YY (PYY), neuropeptide Y (NPY) and glucagon-like peptide-1. Some of these hormones suppress eating behavior, while others stimulate it. Recently, most attention has focused on PYY since this hormone is known to have strong appetite-suppressing effects (8). The peptide YY is a 36 amino acid polypeptide which is secreted predominantly from L-cells of the distal gastrointestinal tract. PYY have two main circulating form PYY1-36 and PYY3-36 (8). PYY3-36 is the major circulatory form of the hormone in the fed and fasted state (9). PYY bind with high affinity to all Y receptors, PYY3-36 shows high affinity for Y2 and some affinity for Y1 and Y5 receptors. It seems that PYY3-36 have peripheral action on appetite which may be mediated by the arcuate Y2 receptor resulting inhibition of NPY neurons and also central actions which is mediated by both Y1 and Y5 receptors results in stimulating food intake (8). Circulating PYY levels are influenced by food intake, type of food (9), gastric acid, cholecystokinin (CCK) and luminal bile salts, insulin like growth factor-1 (IGF-1), and bombesin (9). In addition, NPY is a 36 amino acid neuropeptide which is one of the most potent orexigenic agents known (9, 10). NPY acts to stimulate feeding predominantly through activation of Y1 and Y5 receptors in the hypothalamus. Circulating NPY derive from the peripheral sympaticho-adrenomedullary nervous system, platelets, lymphocytes, endothelial cells and smooth muscle (10). It seems that circulating NPY does not penetrate the blood-brain barrier (11). Therefore, NPY is produced and secreted from cell bodies of the arcuate nucleus in the hypothalamus into the paraventricular nucleus resulting increased appetite and food intake, suppressed activity of the sympathetic nervous system, decreased energy expenditure and increased activity of the parasympathetic nervous system (12).

Previous training studies have examined changes in fasting hormone responses before and after an exercise training intervention (13), but there are limited and controversial data regarding impact of acute exercise on plasma PYY (4, 14, 15), with studies have reported the absence of an effect on PYY post-exercise (15, 16), and an increase in

plasma PYY concentration (14, 17, 18). Regarding plasma NPY response to acute exercise, there are only three studies with reporting significant increase in plasma NPY after 45 min cycle ergometer (19) or significant decrease in plasma NPY after short-term treadmill exercise in obese rats (20) and incremental cycle ergometer test to exhaustion in athletes (21). Therefore, the aim of the present study was to investigate the effects of a single exhausting exercise bout on plasma PYY, NPY and IGF-1 (because PYY concentrations are influenced by IGF-1 signal) in young athletes.

MATERIALS AND METHODS

Participants. Thirty-one young men athletes were recruited to participate in this study (Table 1). All subjects had at least 6 years of experience in wrestling and were among the top 10 in national championships. The study was limited to males to reduce variation in hormonal response to the exercise. Subjects were fully informed of the study details and give their written consent prior to participation. The Institutional Review Board of the University approved the research protocol. All subjects had at least 4 years experience in wrestling. Exclusion criteria were history of gastrointestinal, endocrine, cardiovascular or psychological disease, and consuming any supplementation, alcohol and tobacco products.

| Table 1. Participants' Characteristics (M±SD) |
|-------------------|-------------|-------------|-------------|
| Age (year)        | Height (cm) | Weight (kg) | BF (%)     |
| 19.52±            | 173±        | 79.24±      | 16.37±     |
| 2.75              | 6.49        | 16.13       | 5.92       |

Protocol. Subjects undertook a randomized crossover design with an interval of 7 days between two study tests. To avoid diurnal rhythm effects, testing sessions began on the same time of the day. Subjects came to the laboratory at 0800 h and, a standard breakfast (biscuits, yogurt, and jelly: 560 kcal, 16.5% protein, 19.3% fat, and 64.2% carbohydrates) was then served at 0830 h and participants remained seated quietly. At 1030 h, a venous blood sample (6 ml) pre-exercise (pre) was taken then the subject exercised on the cycle ergometer. Then blood samples were collected immediately after (post), and 1h after (1 h post) exhaustive exercise into tubes containing heparin. Each subjects performed the incremental exercise test to exhaustion began at 50 watts (W) for five-minute, and the power output was increased by 30 W every 3 minute until voluntary exhaustion or the subject could no longer maintain a pedal cadence of 60 rpm despite strong verbal encouragement. After the end of the exercise (post), and 1 h after (1 h post) blood samples were collected into tubes containing heparin. Subjects were instructed to refrain from exercise 72 h prior experimental session. All participants were instructed to follow their habitual diet throughout the study period and to fill out a food recall form for 3 days before the testing session (Table 2). The food recall forms were analyzed using the Nutritionist IV software (Diet analysis model 3.5.2, the Hearst Corporation, San Bruno, CA 94066) to determine total calories intake, carbohydrates, proteins and lipids.

Table 2. Dietary Intake Assessed during 3 Days prior to Exercise Session

<table>
<thead>
<tr>
<th>Dietary intake</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy intake (kcal)</td>
<td>2695.8±63.99</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>87.95±23.54</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>303.40±100.64</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>130.52±35.98</td>
</tr>
</tbody>
</table>

Blood Sampling and Hormonal Measurement. The taken blood samples were centrifuged at 1500 × g for 15 min then plasma was stored at -20 °C until analysis. Plasma total PYY was analyzed in a single
run and in duplicate using Enzyme-Linked immunosorbent Assay Kit (Cat. No: E91067Hu, USCN Life Science Inc), IGF-1 and NPY was measured using Enzyme-Linked immunosorbent Assay Kit (Boldon, Tyne & Wear, United Kingdom) according to the manufacturer’s protocol. To eliminate inter-assay variance, all samples for a particular assay thawed once and analyzed in the same assay run in duplicate. The coefficients of variations (CV) were 7.1% for PYY, 6.9% for IGF-1 and 7.3% for NPY. The changes of plasma volume were calculated based on hemoglobin and hematocrit estimation. Results were adjusted in order to prevent pre-analytic error in the hormonal values (22).

**Statistical Analysis.** Descriptive statistics including means, standard deviations, and ranges were calculated for all measures. A two-way ANOVA was used to analyze the dependent measures of PYY and NPY with repeated-measures design. Bonferroni’s post hoc test was used to determine significant findings over time, with significance accepted at p < .05. The Student’s paired t-test was used to determine the difference in IGF-1 concentration. All statistical analyses were performed using Statistical Package for Social Sciences (SPSS, Version 19.0) software.

**RESULTS**

There was a significant increase in adjusted plasma PYY concentration at post-exercise in experimental group (Figure 1). The NPY concentration increased afterwards and 1h post-exercise in experimental group (Figure 2). Also, IGF-1 concentration was significantly increased at post exercise in experimental group (Figure 3).

![Figure 1. Plasma PYY Concentration at Pre-, Post- and 1h Post-Exercise. *: Significant difference with Pre at p<0.05. §: Significant difference with control p<0.05.](attachment:image.png)

**DISCUSSION**

Appetite control plays a critical role in the competition between energy consumption and energy expenditure. Exercise-induced changes in appetite are of interest over the last decade. Recently, special attention has been paid to understand the central and peripheral mechanisms involved in the regulation of energy balance, especially in the appetite control (21).
PYY and NPY have been reported to influence on appetite and can result in suppression and stimulation of food intake, respectively (9). As mentioned previously, PYY is secreted predominantly from L-cells of the distal gastrointestinal tract and exert its effect through the arcuate Y receptors resulting in inhibition of NPY neurons and food intake (23). The finding of this study revealed that a single exhausting exercise session increased plasma PYY concentration at post-exercise. These data are consistent with the finding of Mackelvie et al. (2007) reported significant increase in PYY levels following 60 min of cycling at 65% of maximal hart rate (MHR) (1h after standardized breakfast) in normal- and overweight male adolescents (17). In addition, Ueda et al. (2009) reported significant increase in plasma PYY levels after standardized breakfast followed by constant cycling exercise at 50% of VO2 max in obese...
young males (4, 5). Also, Broom et al. (2009) investigated PYY response to an acute bout of aerobic exercise (60 min run at a speed 70% of VO2max on treadmill) (18). They observed significant elevation in post-exercise plasma PYY concentration and appetite is suppressed during and for a short time after exercise in healthy males (18). Also, Ueda et al. (2013) observed significant increase in PYY level after 60 min of cycling at 65% of max heart rate (MHR) (1 h after breakfast) in middle-aged women subjects (15). In contrast to our finding, however, Larson-Meyer et al. (2012) were not able to show significant increase in PYY level after 60 min run or walk at 70% of VO2max, in nine middle-aged women runners (16). This contrasting finding may be explained by the different in sex of the subjects examined. Larson-Meyer et al. (2012) used middle-aged women as subjects (16) whereas our subjects and/or published studies (4, 5, 17, 18) were males and some evidence showed that appetite hormones/metabolic responses may differ in females compared with males (2) although further research is required before conclusions can be drawn.

In this study, PYY returned to pre-exercise level following 1h recovery. Similarly to this result, some of the previous studies reported that PYY levels remained elevated at least up to 1 h post exercise (3-5, 17). However, in contrast to our finding, previous study reported PYY level remained elevated up to 3 and 7 h after 50 min submaximal bout of exercise on the cycle ergometer at 60% of VO2max (14) and running on treadmill (4). Collectively, these findings suggest that acute exercise affect circulating PYY level which may regulate appetite following exercise.

The mechanisms underlying the changes in plasma PYY during exercise are unclear in detail. However, it is reported that IGF-1 stimulation expression and secretion of PYY in rodent (9). This increased IGF-1 at post-exercise in experimental group [in this study consistently with previous investigations (24-26)], and it can explain that increased PYY concentration immediately after exhausting exercise may be related to IGF-1 signals which may influence appetite in young athletes.

Even though plasma NPY levels do not reflect NPY secretion in the brain, however, plasma NPY levels may originate from peripheral sympathetic nerve secretion or the adrenal gland and/or adipose tissue during exercise in humans (27, 28). In addition our finding show that plasma NPY concentration increased significantly at post and 1h post a single exhausting exercise session in young athletes. This finding is accordance with previous study that reported significant increase in plasma NPY after incremental cycle ergometer test to exhaustion in athletes (19). As mentioned earlier, NPY secreted into both circulating and paraventricular nucleus in hypothalamus (12). Circulating NPY does not penetrate the blood-brain barrier (29). Therefore, plasma NPY level may be unrelated to appetite regulation following strenuous exercise. Increased plasma NPY level after incremental cycle ergometer test to exhaustion may stimulate angiogenesis (30), regulate blood pressure and electrolyte homeostasis (31).

Our study has some limitations. We measured total PYY rather than PYY3-36, however, previous studies reported a high positive correlation between total PYY and PYY3-36 (32) and acute exercise-induced changes blood PYY3–36 levels is similar to the time course of changes in blood total PYY level (5). Furthermore, we did not assess hunger following our single exhausting bout of exercise or obtain food intake measurements into the recovery period which could have provided additional importance.

**CONCLUSION**

In conclusion our findings showed that plasma PYY concentration increased
following a single exhausting exercise session in young athletes. The actions of this gut peptide is not yet completely understood but it seems that via central circulation, PYY cross the blood brain barrier and directly interact with the arcuate Y receptors resulting inhibition of NPY neurons and food intake. Therefore, increased plasma PYY concentration after a single exhausting exercise session in young athletes may possibly contribute to appetite suppression. However, increased plasma NPY after this type of exercise may not be related to appetite regulation because it does not cross the blood brain barrier, rather it may assumed to stimulate angiogenesis and show a vasoconstrictive and mitogenic effect on blood vessels (12, 32).

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REFERENCES
Acute Exercise and Appetite Peptides


اثر حاد فعالیت ورزشی فضایی بر غلظت پپتید YY، نورپپتید Y و IGF-1 پلاسما در ورزشکاران جوان

حسن فرجی، رحیمی، سعید دباغ

چکیده
مشخص شده است که فعالیت ورزشی یک استراتژی جهت مقابله با چاقی محسوب می‌شود. این کار توسط ایجاد تعادل انرژی منفی به وسیله افزایش مصرف انرژی و اثر بر تنظیم هورمون‌های اشتها انجام می‌شود. هدف از پژوهش حاضر بررسی اثر یک جلسه فعالیت ورزشی تا سر حد خستگی بر پپتید YY، نورپپتید Y و فاکتور رشد شبه انسولین (IGF-1) در ورزشکاران جوان بود. سی و یک مرد ورزشکار (سن: 75/2 ± 52/19، وزن: 13/16 ± 24/79، قد: 49/6 ± 73/1 و چربی بدن 92/5 ± 37/16 درصد) به طور داوطلبانه در این مطالعه شرکت کردند. آزمودنی‌ها یک جلسه فعالیت ورزشی را در خاک کارسنج با مقاومت 50 وت به مدت 5 دقیقه پیدا کردند. نتایج نشان داد که یک جلسه فعالیت ورزشی تا حد یک جلسه هورمون‌های YY و IGF-1 و نورپپتید Y، PYY پلاسما را در افزایش غلظت یافته است. این نتایج که در گروه ورزشکاران جوان از جمله کشتی‌گیران جوان اخراج نمی‌شوند، پیامد این آزمودنی‌ها بود که در تامین انرژی کافی برای زیستن برای روند رفتاری و احداث اشتهایی کمک کرده است.

واژگان کلیدی: اشتها، غذای مصرفی، ورزشکاران، پپتید YY
Short-Term Interval Training Courses on Fitness and Weight Loss