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# **‘Exercise-Eating Linkage’ Mediated by Neuro-Endocrine Axis and the Relevance in Regulation of Appetite and Energy Balance for Prevention of Obesity**

Takahiro Yoshikawa

*Department of Sports Medicine, Osaka City University Graduate School of Medicine,  
Osaka,  
Japan*

## **1. Introduction**

A significant number of modern people of all ages are affected by obesity epidemic in developed and parts of developing world, resulting in immense health and financial burden (Field, 2002; Ogden et al., 2006; Orsi et al., 2011). Obesity is thought to be an independent risk factor for development of various medical conditions, such as type 2 diabetes, hypertension, dyslipidemia, and cardiovascular disease (Daniels, 2009; Fontaine, 2003; Ginsberg, 2000; Kahn & Flier, 2000; National Institutes of health [NIH], 1998), necessitating weight management for prevention and treatment of these serious diseases. Obesity epidemic reflects modern growing trends to promote excess energy intake (EI) and to discourage energy expenditure (EE) (Egger et al., 2001; Food and Agriculture Organization of the United Nations [FAO], 2003; Hill et al., 2003). Thus, it is well recognized that lifestyle intervention, such as changes in behavior by combination of reductions in EI and increases in physical activity, can achieve ideal weight control (Bray, 2008). Nevertheless, it seems impossible for most of people to fight down consciously their impulses of overeating in modern societies where they have easy access to unlimited supply of highly palatable and energy-dense food (Cohen, 2008).

In such circumstance, appetite is receiving extensive attention for one of key factors to adjust or disrupt energy balance (EB). In particular, an important group of the intricate factors for the appetite control are gut hormone family, including ghrelin, peptide YY (PYY), glucagon-like peptide-1 (GLP-1), oxyntomodulin (OXM), and cholecystokinin (CCK) (Huda et al., 2006; Näslund & Hellström, 2007). Recent advances have been made in understanding the structures, sources, releasers, target organs and receptors, and how these gut hormones influence brain systems for the control of appetite and EI. In brief, these gut hormones are secreted from gastrointestinal organs in response to nutrient conditions and give signals to hypothalamic and brainstem nuclei both of which are in close anatomical proximity to a circumventricular organ with an incomplete blood-brain barrier (BBB), such as median eminence and area postrema. Ghrelin is only an orexigenic hormone and secreted shortly before meals, whereas other gut hormones are all anorectic and released into circulation postprandially in proportion to calorie intake.

While these gut hormones have been well-studied for over a decade, numerous studies have so far investigated a possible association of lifestyle habit, such as exercise, with appetite and EI (King, 1999). Interestingly, recent studies, including ours, have revealed inhibitory effects of exercise on the sensation of hunger and satiety and amount of EI associated with the gut hormone release in various population (Broom et al., 2009; Martins et al., 2007; Ueda et al., 2009a, 2009b), suggesting the presence of an 'exercise-eating linkage' mediated by neuro-endocrine axis in human body. The evidence also suggest an intriguing possibility that physical activity has an impact not simply on EE but also on variations in appetite, leading to negative EB.

The present review provides an overview of 1) changes in sensation of hunger and satiety and amount of EI by various types of exercise and next highlights 2) association of exercise with blood kinetics of gut hormones and its relevance in regulation of appetite and EB. Lastly, 3) future perspective of this research field will be discussed.

## 2. 'Exercise-eating linkage', is it real?

So far, there has been considerable research on changes in subjective appetite parameters and amount of EI by various types of exercise. Exercise is generally assumed to induce a transient energy deficit and subsequent automatic drive in hunger and EI for energy compensation. However, a majority of previous studies failed to show that a single bout of exercise increase hunger or EI. Such discrepancy between EE and EI can be observed in a wide range of population, irrespective of age, gender, body weight, dietary restraint and exercise intensity. For instance, acute high-intensity exercise (about 70%  $VO_2$  max) is unlikely to cause the subsequent increase in EI in unrestrained males with normal body weight, favoring negative EB (Imbeault et al., 1997; Thompson et al., 1988). Similarly, poor compensation was observed in response to energy deficit by a single bout of high-intensity exercise (cycling 50 min, 70%  $VO_2$  max) in restrained young females with normal body weight (Lluch et al., 1998). Although unrestrained female counterparts rated a range of foods to be more palatable after similar type of exercise, it had no significant short-term effect on energy or macronutrient intake (King et al., 1996). Moderate physical activity (a single bout of 20-min brisk walking) suppressed appetite in middle-aged obese women who were not on special diet (i.e. unrestrained eaters) (Tsofliou, 2003). Similar to acute effects of exercise on appetite, a graded increase in EE by 7-day repeated exercise regimens did not show any significant increases in subjective feeling of hunger and food consumption in lean individuals, generating considerable negative EB (Stubbs et al., 2002a, 2002b). Given the evidence above, 'exercise-eating linkage' is, if any, unlikely to benefit the maintenance of EB, at least short term. Rather, 'exercise-eating linkage' might give rise to transient appetite suppression after exercise. However, it is known that various metabolic and behavioral responses i.e. decrease in EE and increase in appetite and EI after exercise, automatically or volitionally, operate in a long-term process of compensation for the exercise-induced energy deficit, minimizing the negative EB and body weight reduction, and the compensatory responses considerably vary among individuals (King et al., 2007, 2008). And it is assumed that such accurate adjustments of EI to an increase in EE seem to take matter of weeks (Whybrow et al., 2008).

In this regard, however, caution should be exercised when interpreting the findings from these studies. Unlike other animals, internal physiological signals for energy repletion and depletion were not sufficient for the control of appetite and eating behavior in humans. It is

well recognized that human eating behavior is a multifactorial process where various external stimuli, including sensory, cognitive, environmental (e.g. social and habitual) variables might play important roles as well as internal signals arising peripherally from gastric distension and energy imbalance (De Castro, 1996). Thus, it is reasonable that eating behavior after exercise can be largely affected by these external factors (British Nutrition Foundation, 1999) and the findings might depend on the background, such as each study setting and subject characteristics. In most previous studies in which association of (short- or long-term) exercise with EI were investigated, test meals were given to subjects *ad libitum*, where the subjects can recognize how much they have eaten, and the subject characteristics (gender, body type and taste) are inconsistent among studies and psychological condition (stress), habitual factors (binge eating) and cognitive factors (reward and preoccupation with food), such as 'food is a reward for exercise' (King, 1999), were often uncontrolled, thus often leading to mixed findings among studies.

### **3. Exercise and gut hormones: implication for 'exercise-eating linkage'**

#### **3.1 Ghrelin**

An increasing amount of research has investigated the possibilities that exercise stimulates various gut hormone release into circulation. And, some of recent studies focused on the impact of the exercise-induced gut hormone release on subsequent appetite and EI. In particular, evidence is accumulating for the effect of exercise on the plasma ghrelin levels, but somewhat inconsistent. A single bout of exercise at fasted condition has been shown not to induce significant changes in plasma levels of total ghrelin in normal weight subjects (Kraemer et al., 2004; Zoladz et al., 2005), whereas similar exercise at premeal condition can raise the circulating ghrelin concentrations in moderately overweight postmenopausal women (Borer et al., 2005). Total ghrelin levels were not affected by aerobic exercise intervention for 5 days without body weight reduction (Mackelvie et al., 2007), whereas the plasma levels gradually increased during the 12-weeks aerobic and resistance exercise with significant decreases in body weight and fat (Kim et al., 2008), suggesting that exercise has only a limited impact on the fasting total ghrelin levels unless the body weight of subjects is reduced. Changes in the fasting plasma levels of total ghrelin are likely to depend not on the duration but on the intensity of exercise (Erdmann et al., 2007). Total ghrelin is classified into two categories; orexigenic acylated ghrelin (AG) and anorectic desacyl ghrelin (DG). In contrast to the total ghrelin, orexigenic AG is suppressed by a single bout of exercise at fasted state, resulting in the negative energy balance (Broom et al., 2007). And the postexercise reduction in AG was observed in the lean as well as obese subjects (Marzullo et al., 2008). The effects of long-term exercise training on plasma AG / DG levels seem to be dependent on the duration of exercise. The longer the exercise is performed, the more decrease in AG/DG is observed in favor of body weight reduction (Kim et al., 2008).

#### **3.2 Satiety hormones**

Similar to ghrelin, it is only in the last few years that studies have focused on the effects of exercise-induced changes in plasma levels of satiety hormones on appetite regulation and most of these studies have been performed mainly in normal weight individuals. Majority of the early studies focused on plasma PP levels in various setting of exercise preprandially (Hilsted et al., 1980; Sullivan et al., 1984), and postprandially (Greenberg et al., 1986). PP release is likely to be exercise intensity-dependent (Holmqvist et al., 1986). Early studies

measured the circulating PP levels as the index of sympathetic activity rather than its physiological roles for appetite regulation (Holmqvist et al., 1986). Plasma GLP-1 levels have been shown to change by exercise in a small number of studies, most of which investigated the association of high intensity exercise with the fasting plasma levels in athletes (O'Connor et al., 1995, 2006). While a single bout of aerobic exercise at fasted state for 10hrs enhanced a response of plasma PYY levels to a test meal after the exercise, such changes were not shown by resistance exercise (Broom 2009).

In most of these studies, the nutrient stimuli are simply replaced by those of exercise, that is, preprandial exercise. However, because these satiety gut hormones are basically released after nutrient intake, it seems worthwhile to examine whether the postprandial plasma levels of these hormones can be affected by exercise and these hormonal changes could affect the subsequent appetite regulation. Recently, some studies, including ours, have investigated kinetics in postprandial release of gut hormones into circulation by a single bout of exercise (Martins et al., 2007; Ueda et al., 2009a, 2009b). One of these studies has demonstrated the postprandial increase in plasma levels of PYY and GLP-1 during and after 1hr exercise at the intensity of 60% of maximal HR. In particular, the time course changes in plasma PYY concentration seem to be mirror image to those in hunger ratings, suggesting that postprandial release of PYY suppress the hunger feeling during and after exercise (Martins et al., 2007). We also investigated acute effect of a single bout of 1 hr aerobic cycling exercise (50%  $\dot{V}O_2\text{max}$ ) on the postprandial plasma levels of gut hormones after standard breakfast in overweight and normal young males and determined the involvement of the hormonal changes in regulation of EB in subjects with normal body weight and overweight (Ueda et al., 2009a, 2009b). We assessed the total release of individual gut hormone during and after the exercise as the area under the curve (AUC) of the time course curve of each hormone. In both body type groups, the aerobic exercise significantly increased subsequent release of PYY and GLP-1 in plasma, while plasma levels of acylated ghrelin were not significantly altered. Of note, exercise-induced suppression of relative EI (absolute EI minus absolute EE) in overweight subjects was significantly larger than in control subjects despite lack of significant difference in PYY and GLP-1 levels between two subject groups. The discrepancy might stem from variability of cognitive and sensory factors between two body type groups. In line with this, when the data were analyzed separately in overweight and normal body type group, a significant linear correlation between the increase in AUC values of plasma PYY and GLP-1 levels by exercise and the concurrent decrease in relative EI was observed within each subject group (unpublished data). Collectively, one could argue that neuro-endocrine axis consisting of satiety gut hormones and brain appetite center could play some important roles in 'exercise-eating linkage' of exercise-induced appetite suppression for a while after a single bout of exercise.

Based on the long-term compensation of exercise-induced energy deficit described above, it seems to be necessary to examine whether sustained exercise training also have an impact on the circulating levels of satiety gut hormone and appetite control through the hormonal axis as well as a single bout exercise. The point is important for planning programs in practice for better appetite and weight control. And a few studies have only recently addressed the issue. A significant rise in fasting plasma levels of PYY was shown in overweight adolescent males and females after 32 weeks of exercise training (60-85% peak  $\dot{V}O_2$ , 45min/day, and 3days/week) (Jones et al., 2009). However, neither effects on ratings of appetite feelings nor those on EB were examined. Another study showed that, while postprandial suppression of plasma AG levels seems to be enhanced, postprandial plasma



levels of PYY and GLP-1 also tend to be higher after 12 weeks of exercise intervention (75% maximal heart rate, 500kcal energy deficit/day, and 5days/week) while the subjective feelings of hunger increased paradoxically after a test meal (Martins et al., 2010). Although these findings might suggest the possibility that long-term exercise might modify the profiles of orexigenic and satiety gut hormones in circulation, available data are still scarce, particularly as to whether these hormonal changes could suppress superfluous appetite. Further information will be needed for understanding how these gut hormones could affect the long-term regulation of appetite and EI.

#### 4. Future perspectives

As described above, findings of recent studies to date indicate the presence of 'exercise-eating linkage' via neuro-endocrinal axis. Now the following important questions remain unanswered.

Does the responsiveness vary among different intensity and style of exercise or among different body weight types? Does the responsiveness of hypothalamus to the gut hormones fluctuate during exercise? Interestingly, a single bout of 6hr exercise improves the responsiveness of hypothalamus to leptin and appetite control in male rats (Flores et al., 2006; Ropelle et al., 2008). Another study demonstrated that habitual exercise influence appetite and EI in response to covert preload energy manipulation (Long et al., 2002; van Walleghen et al., 2007). In particular, Long SJ *et al.* raised the possibility that exercise may enhance the accuracy of appetite control by the exercise-induced improvement in insulin sensitivity (Aldred et al., 1995; Haber et al., 1977; Holt et al., 1992; Poehlman et al., 2000). Insulin exerts appetite-inhibiting effects via hypothalamus activation (Schwartz 2000). It is still unknown whether the response to satiety gut hormones also can be altered by exercise. Based on previous studies in which these peptides are dosed peripherally, obesity *per se* does not seem to affect the responsiveness to some satiety gut hormones, such as PYY (Batterham et al., 2003).

Does the change in gut hormone levels with exercise have significant effects on other physiological systems as well as appetite regulation? Interestingly, PYY administration decreases respiratory quotient (RQ) and reduces adiposity in diet-induced obese mice (Adams et al., 2006). In human study, high fasting and postprandial peak PYY was observed in subjects with low resting metabolic rate (RMR) (daily EE) and low RQ (Guo et al., 2006). The findings raise an intriguing possibility that exercise might induce PYY secretion and regulate not only the appetite but also EE and fuel partitioning in favour of fat oxidation, providing the additional evidence of advantage of exercise intervention for weight and body fat reduction.

Possible association of the fluctuating gut hormone levels with subsequent appetite and EB are intriguing. However, more important question is how much effect we can expect such intrinsic signal of exercise-induced hormonal changes to modulate the overall appetite system in which other diverse extrinsic factors are largely involved. Hypothalamus is one of pivotal entry points of the peripheral internal signals into central pathways mediating brain appetite regulation along with nucleus tractus solitarius (NTS) in brainstem. There are reciprocal connections between these two entry points and these areas communicate with higher centers such as brain reward centers (e.g. ventro tegmental area, nucleus accumbens, ventral pallidum, and orbitofrontal cortex) which play a role in integration of various sensory information and hedonic signals (Wynne et al., 2005). One of recent

neurophysiological studies revealed that the sensory inputs produced by sight, smell, taste and texture of food converge at specific brain regions, such as orbitofrontal cortex, and interact with peripheral hunger/satiety signals at hypothalamus, including autonomic nerve activation and gut hormones, and finally determine eating behavior (Rolls, 2005, 2006). In addition, taste information is also sent to the reward system, which mediates the motivation of eating of palatable food, and finally transfers the signals to the hypothalamus (Yamamoto, 2008). While human intrinsic appetite control systems remain unchanged in recent few decades, the excess food stimuli override appetite suppressive responses, such as humoral and neural satiety signals arising from gastrointestinal organs, resulting in overeating and subsequent obesity. Exercise might partly correct the disparity between the primitive appetite control systems and the current expanding food consumption by upregulating the peripheral satiety signals. Interestingly, a recent human study using brain functional MRI demonstrated that, after intravenous infusion of PYY at physiological plasma concentrations, mimicking the fed state, hypothalamic neural activity was not correlated with caloric intake, while the changes in neural signals within higher center, the orbitofrontal cortex, correlated with caloric intake, suggesting that brain activity predicting caloric intake appeared to switch from hypothalamus to higher center, orbitofrontal cortex in the presence of PYY (Batterham et al., 2007). Based on this observation, the exercise-induced increase in the plasma PYY levels might cause some possible effects on the neural

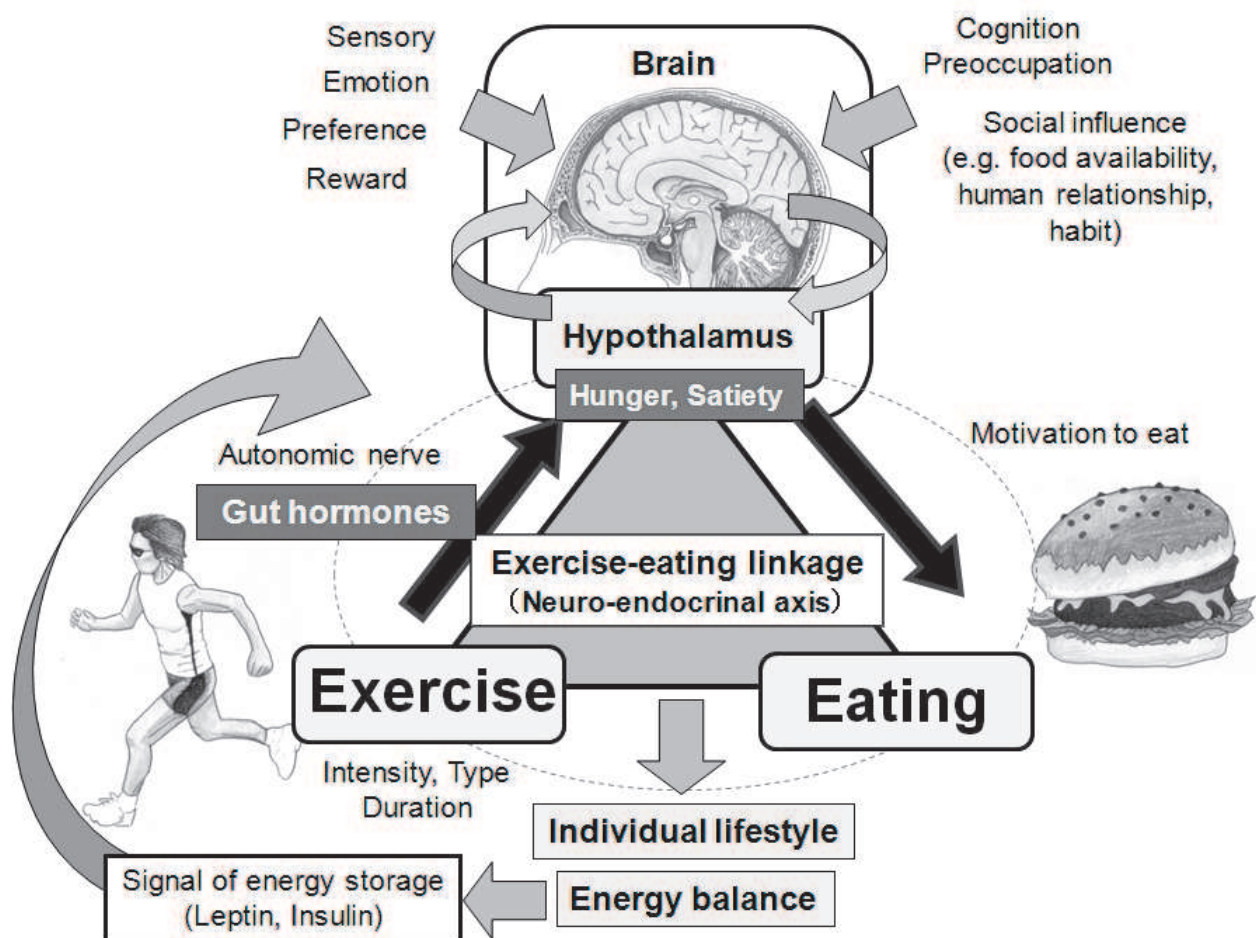


Fig. 1. Overview of exercise-eating linkage via neuro-endocrinal axis

activity in higher centers for appetite control. In addition to the changes in gut hormones, exercise also influences brain dopaminergic, GABAergic, noradrenergic and serotonergic systems (Meeusen & De Meirleir, 1995). In rat models, changes in synthesis and metabolism of these neurotransmitters and receptors during exercise were observed in distinct brain regions including hypothalamus (Kramer et al., 2000). Although it is beyond the scope of this review to include these neurophysiological findings, we should keep in mind the large general picture of these control systems before we consider the implication for the association of exercise with appetite, EB and gut hormones. Future studies will be needed to investigate the effects of (acute or long-term) exercise on the overall appetite regulatory systems (exercise-eating linkage) based on the crosstalk interactions between peripheral endocrine system and central appetite centers (neuro-endocrinal axis) (Figure). Furthermore, the understanding might give us clues to reveal the reasons why there is inter-individual variability in ratings of hunger and amount of food ingested after exercise (King et al., 2007), leading to tailored strategies of preventive programs and treatment for overeating and obesity.

In conclusion, The evidence of exercise-induced changes in satiety gut hormones with suppression of hunger feeling and subsequent relative EI provides considerable support for the value of exercise not only in a measure of expenditure but also in preventing from overeating and obesity. Understanding of this area in endocrinology combined with neurophysiology of eating behaviour in higher brain centers might facilitate the development of promising approach to maintain better dietary life with appropriate exercise and healthy body weight.

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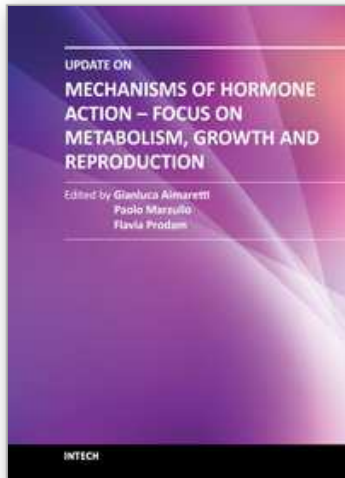
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The purpose of the present volume is to focus on more recent aspects of the complex regulation of hormonal action, in particular in 3 different hot fields: metabolism, growth and reproduction. Modern approaches to the physiology and pathology of endocrine glands are based on cellular and molecular investigation of genes, peptide, hormones, protein cascade at different levels. In all of the chapters in the book all, or at least some, of these aspects are described in order to increase the endocrine knowledge.

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Phone: +385 (51) 770 447  
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Unit 405, Office Block, Hotel Equatorial Shanghai  
No.65, Yan An Road (West), Shanghai, 200040, China  
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元  
Phone: +86-21-62489820  
Fax: +86-21-62489821



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