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Exercise, appetite and weight management: understanding the compensatory responses in eating behaviour and how they contribute to variability in exercise-induced weight loss

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Running Head: Exercise, appetite and obesity

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## Abstract

Does exercise promote weight loss? One of the key problems with studies assessing the efficacy of exercise as a method of weight management and obesity is that mean data are presented, and the individual variability in response is overlooked. Recent data have highlighted the need to demonstrate and characterise the individual variability in response to exercise.

Do people who exercise compensate for the increase in energy expenditure via compensatory increases in hunger and food intake? We address the physiological, psychological and behavioural factors potentially involved in the relationship between exercise and appetite, and identify the research questions which remain unanswered.

A negative consequence of the phenomena of individual variability and compensatory responses has been the focus on those who lose little weight in response to exercise; this has been used unreasonably as evidence to suggest that exercise is a futile method of weight control and managing obesity.

Most of the evidence suggests that exercise is useful for improving body composition and health. For example, when exercise-induced mean weight loss is <1.0kg, significant improvements in aerobic capacity (+6.3 ml·kg<sup>-1</sup>·min<sup>-1</sup>), systolic (-6.00 mmHg) and diastolic (-3.9 mmHg) blood pressure, waist circumference (-3.7cm) and positive mood still occur. However, people will vary in their responses to exercise; understanding and characterising this variability will help tailor weight loss strategies to suit individuals.

## **I) Introduction**

Exercise is frequently used as a method of weight control through an increase in energy expenditure with the aim of creating a sustained energy deficit. Of course, compliance to the exercise itself is an important issue, however, of more interest is the issue of compensatory responses which undermine the ability of exercise to promote the theoretical weight loss.[1]

There have been a multitude of studies examining the acute effects of exercise on compensatory responses in energy intake. The acute effects of exercise appear to be consistent and relatively well understood. The majority of research demonstrates that acute exercise does not increase hunger, desire to eat or energy intake.[2-6] Even when there is a marked increment of 4.6 MJ/d in acute exercise-induced energy expenditure (EE), and energy intake (EI) is monitored for 2 days, there is no automatic increase in EI.[7] Although the acute effects of exercise on EI are generally consistent, there are a few rare examples of a coupling between EE and EI.[8-9] More recently partial compensation to an exercise-induced energy deficit has been reported.[10-12] Exercise has also been demonstrated to improve the sensitivity of appetite control, and that regular exercisers are better at detecting the difference in energy content between low- and high-energy preloads compared with their sedentary counterparts.[13-14]

Overall, the evidence suggests that no or only slight partial compensation occurs in response to an acute exercise-induced energy deficit. Therefore, exercise should provide a successful method of weight control. The effects of chronic exercise on EI and appetite are less clear. Long term studies that have monitored EI for between 16 weeks and 18 months in overweight men and women also demonstrated no significant change in EI across the intervention.[15-17] Weight loss is typically low and variable in most of these long term studies. It is important to note that there is a tendency for the exercise sessions to be unsupervised, the absolute increase in EE is low, and the methods used to measure food intake are not reliable. Therefore, it is possible that the increase in exercise-induced EE is insufficient to up-regulate EI to a detectable level. Even when the exercise was supervised and induced an energy deficit of approximately 8.2 MJ/wk for 16 months, there was no increase in EI.[18] These data suggest that there tends to be a lack of an increase in EI to compensate

for an exercise-induced energy deficit in overweight and obese individuals. However, increases in EI in response to long term exercise have been reported in lean participants.[19-21] Therefore, one hypothesis is that lean individuals demonstrate a compensatory increase in EI to defend their relatively lower body fat reserves. It is important to recognise that for a majority of published research, mean data are reported which may disguise other trends in the data – for example individual variability. A series of systematic studies has recently provided data to identify and characterise individuals who experience different changes in body weight after 12 weeks of supervised exercise.[22-24] These data have revived interest in characterising and explaining the variability by focusing on compensatory responses.

## **II) Individual variability: do all people respond similarly?**

Whilst there is some substance in the claim that some or all of weight loss is regained, or weight loss eventually reaches a plateau (e.g.,[25-28]) the overriding assumption is that people should experience similar weight loss in response to exercise. Evidence indicates that even when the exercise is supervised and closely monitored, there is variability in weight change - both in the direction and magnitude.[24] Figure 1 shows individual changes in body weight after a 12-week supervised exercise intervention in overweight males and females. The intervention consisted of 5 high-intensity (70%  $\text{VO}_2\text{max}$ ) exercise sessions per week. The intensity and duration was fixed for all individuals and the energy expenditure of each exercise session was ~2MJ. These demonstrate that despite the same imposed increase in EE individuals will vary in their body weight response.

Figure 1 here

The phenomenon of individual variability in response to energy balance perturbations is certainly not new.[29-31] However, exercise-induced individual variability has yet to be exploited. In particular the characterisation of the variability - the behavioural and physiological compensatory processes need to be determined. More recently, individual variability has attracted new interest.[1, 22, 24, 32-36] Unfortunately, reports of the variability in exercise-induced weight change results in

the media using the evidence unreasonably ([37-38]; <http://www.telegraph.co.uk/science/6083234/Health-warning-exercise-makes-you-fat.html>; <http://www.time.com/time/health/article/0,8599,1914857,00.html>), to argue that exercise is a futile method of weight loss, by unjustly focusing on those who experience no or little weight loss. We acknowledge that some individuals do experience minimal exercise-induced weight loss and that these individuals need further examination. However, exercise should be portrayed more positively. Overall the evidence demonstrates that exercise does lead to improvements in body composition and fat mass loss[39], and more importantly, the beneficial health effects of exercise are not restricted by the absence of, or a low weight loss. Based on changes in body weight after 12 weeks of supervised exercise, two groups were identified: responders and non-responders. Despite the non-responders losing significantly less weight than the responders (0.9 v 5.2kg), they still experienced marked improvements in blood pressure, waist circumference and VO<sub>2</sub>max (see Table 1). Rather than using individual variability as an opportunity to deter those who experience poor weight loss from exercising, there is a need to understand what accounts for the variability and how that information can be used to develop weight management strategies to facilitate weight loss (and maintenance) in those people. Further, there is a need to educate people that improvements in health occur even in the absence of lower than expected weight loss. The concept of susceptibility to weight gain, and resistance to weight loss, has been discussed in detail elsewhere.[40-41] There is an urgent need to determine why some people lose less weight than is theoretically expected in response to energy balance interventions; in particular, exercise interventions.

Table 1

Variable	Group	Absolute change	% Change
Body Mass (kg)	Responders	-5.2	-5.7
	Non-Responders	-0.9	-1.0
Fat Mass (kg)	Responders	-4.9	-15.3
	Non-responders	-1.2	-4.7
Lean Mass (kg)	Responders	-0.3	-0.6
	Non-Responders	+0.3	+0.4
Waist Circumference (cm)	Responders	-6.0	-5.8
	Non-Responders	-3.7	-3.7
VO <sub>2</sub> max (ml/kg/min)	Responders	+9.1	+32.5
	Non-Responders	+6.3	+23.0
Diastolic blood pressure (mmHg)	Responders	-3.4	-3.7
	Non-Responders	-3.9	-4.6
Systolic blood pressure (mmHg)	Responders	-2.9	-1.9
	Non-Responders	-6.0	-4.3

### **III) Exercise-induced physiological processes of appetite control**

#### **i) Gastric emptying and gut physiology**

Given the central role that the gut and its related hormonal activity play in appetite control, it is possible that they also moderate the responses to acute or chronic exercise. Gastric distension, in combination with the presence of nutrients in the small intestine contributes to satiety.[42-43] Gastric emptying inevitably affects food intake and appetite control through the delivery of nutrients to the small intestine.[44] The classical studies reported an inverse relationship between the rate of gastric emptying and satiety.[45-47] More recently the importance of considering the biphasic role of gastric emptying in satiation (meal size) and satiety (inter-meal interval) has been highlighted, with the suggestion that a decrease in the initial emptying rate could result in a delay in the release of potent signals involved in meal termination.[48] As satiation and satiety are important processes in appetite regulation, inter-individual differences in gut physiology and the strength of these signals could contribute to variability in exercise-induced weight loss.

Surprisingly, very few studies have directly examined the effects of exercise on gastric emptying and appetite. A majority of studies conducted on exercise and gastric emptying have focused on the efficacy of providing optimal rates of carbohydrate and fluid replenishment as ergogenic aids in sport. Overall, the evidence points to a delay in gastric emptying with strenuous exercise (>70%  $\text{VO}_2\text{max}$ ).[49-57], whilst mild to moderate exercise stimulates (i.e., accelerates) gastric emptying.[54-55, 58-60] There have also been reports of no change in gastric emptying with moderate intensity exercise.[52, 61] Differences in study methodology such as test meal properties and exercise parameters may account for this inconsistency.

Mechanisms proposed which could contribute to exercise-induced alterations in gastric emptying include changes in contraction frequencies, antral area[62] and gastric myoelectrical activity[63-64], hormonal[56, 59, 65] and neural factors (mainly vagal in origin)[58, 66], gut blood flow[54] and the mechanical effects of “jostling of the gut”[54, 56, 59, 67] during exercise.



In terms of adaptation to regular exercise, very few studies have examined the impact of chronic exercise exclusively on gastric emptying at rest or during exercise. Despite not controlling for age, Shimamoto et al[68] reported that at rest, gastric emptying was slower in inactive compared to active elderly individuals, which was associated with decreased gastric electroactivity in the inactive group. In a cross sectional study a faster basal rate of gastric emptying was observed in marathon runners compared to sedentary controls.[69] Training-induced enhanced parasympathetic tone was proposed as one possible explanation.

Of course, physical activity (hence EE) might not be the only factor to vary between inactive and active people. Dietary habits, including total EI, frequency of eating and macronutrient intake could also vary. Collectively, these dietary factors could affect gastric motility, via the quantity, frequency and quality of nutrients which pass through the gut and small intestine. A high-fat/low-carbohydrate diet attenuates the effects of fat on gastric emptying[70-71] and intestinal transit.[72] Harris et al[73] reported rapid orocecal transit time (OCTT) in chronically active individuals with concomitant high energy intakes, and concluded that the high energy intake associated with chronic exercise may be associated with significant gastrointestinal adaptations (Figure 2). However, the causal nature of this association is not possible to determine from this cross sectional study. It could also be speculated that increased physical activity levels may have led to faster OCTT and thus higher caloric intakes as a result of a shorter satiety period.

Figure 2 about here

## ii) **Appetite Peptides**

Appetite related peptides play an important role in the stimulation or inhibition of eating in accordance with the principles of energy balance. For example, ghrelin is an orexigenic peptide secreted from the stomach.[74] Increased postprandial ghrelin suppression is associated with reduced appetite.[75] The majority of studies indicate that acute exercise has no influence on total ghrelin (TG) concentrations.[76-81] One study showed that TG levels are suppressed for an hour after the cessation of

exercise[82], whilst 3-h of moderate intensity exercise has been shown to increase TG levels.[83] The consensus is that acute exercise appears not to influence TG levels independent of mode, intensity or metabolic state in normal weight (NW) and overweight/obese adults.[84]

Unfortunately, few studies have examined the active component of the hormone – acylated ghrelin (AG). The first study to investigate AG has shown that running for 60 min at ~75%  $VO_2$ max caused a suppression of this orexigenic peptide, with a simultaneous suppression of appetite during and immediately post-exercise in NW trained men.[85] This evidence suggests that TG and AG respond differently to exercise. Further to this, TG and AG were measured in both NW and obese adults who cycled until exhaustion.[86] No change in TG was evident but AG decreased significantly in both groups after exercise even though the obese group reached exhaustion earlier than the NW group. The results of this study emphasise that the mechanism causing the decrease in AG during exercise is independent of adiposity status or acute exercise.[86]

Fewer studies have investigated the effect of acute exercise on anorexigenic gut peptides. Cholecystokinin (CCK), polypeptide YY (PYY) and glucagon-like peptide-1 (GLP-1) are examples of anorexigenic peptides released in response to intestinal nutrients.[74] One of the first studies in this area investigated the effects of 60min cycling (at 65% HR max) vs rest (control) on gut peptides and subsequent energy intake in twelve lean adults after consuming a fixed breakfast.[87] There was a significant exercise-induced increase in PYY, GLP-1 and pancreatic polypeptide (PP), but no change in TG. The GLP-1 continued to be elevated into the post-exercise period, whilst the increase in PYY was short-lived. A similar study (cycling at 50%  $VO_2$ max for 60min), also reported that both PYY and GLP-1 were significantly increased by exercise but TG levels remained unaffected.[88]

Of course, in acute exercise studies, weight loss is not an issue, whereas in chronic studies in which weight loss occurs, changes in peptides will also be associated with body composition. A significant increase in fasting TG plasma levels has been reported after exercise-induced weight loss in NW[89], overweight/obese adults[90] and in overweight/obese children.[91-92] However, no change in TG plasma levels was reported in overweight twins after 3 months of supervised

exercise, inducing a 5kg weight loss.[93] Regarding AG, no change in fasting plasma levels after long-term exercise was reported in overweight children[92] and adolescents.[94] However, a significant increase in fasting and postprandial AG levels was reported in NW and overweight adolescents after 5 days of supervised exercise - but no change in TG.[95] We have also recently shown a significant increase in fasting AG plasma levels, despite no change in TG, in overweight/obese adults after 12-weeks of supervised exercise, inducing an average 3.5kg weight loss, together with a significant increase in the postprandial suppression of AG – see Figure 3.[96]

Figure 3 about here

Although the increase in TG and/or AG plasma levels observed in some exercise-induced weight loss studies loss can be seen as a counter-regulatory mechanism to try to restore EB, TG has been shown to return to baseline levels with sustained weight loss maintenance, both in children[91] and adults[97], suggesting that TG may be involved in the regulation of acute, but not chronic, changes in EB. It remains to be seen if the same is true for AG, since changes in AG do not necessarily parallel changes in TG.

Evidence regarding the impact of chronic exercise on the release of anorexigenic gut peptides is relatively scarce. Long-term exercise has been shown not to change fasting CCK in active men [98], but induce a slight increase in fasting and postprandial PP, in previously sedentary NW men[99] and a significant increase in fasting PYY plasma levels in overweight adolescents.[94] Exercise-induced weight loss has no impact on fasting PYY or GLP-1 plasma levels, but leads to a tendency towards an increase in the delayed release of GLP-1 (90-180 min) after a meal, in overweight/obese adults.[96] Moreover, no change in fasting GLP-1, but a significant increase in GLP-1 release in the first 30 min post-prandially was reported in NW and overweight adolescents in response to five consecutive days of exercise.[100]

Similar to gastric emptying, habitual diet can influence appetite related peptides profiles. A high-fat/low-carbohydrate diet attenuates the secretion and action of gut peptides including CCK, PYY, GLP-1 and ghrelin.[101-104] The interaction of exercise with habitual diet is important to consider as these effects may undermine any potential beneficial effects of exercise on appetite control and therefore contribute to the variability in compensatory responses.

To the best of our knowledge no studies have systematically examined the complex interactions of changes in gastric emptying and gut peptides and alterations in subjective and objective measures of appetite in response to acute or chronic exercise. Therefore, this is an area which needs to be explored because variability in compensatory eating responses could be explained by gut-related activity.

### iii) Substrate oxidation

Consistent with a psychobiological approach to appetite control, in which physiological mediators act as drivers of behaviour [40], it is possible that exercise-induced changes in substrate metabolism play an important role in changes in appetite and food intake. Substrate metabolism has long been implicated in the energostatic control of EI, in which fatty acid oxidation (FAO) is thought to mediate EI via the maintenance of post-meal satiety.[105] While the mechanisms are poorly understood, changes in hepatic energy status (hepatocellular ATP/ADP ratio) resulting from altered FAO may influence EI via the stimulation of vagal afferent nerve activity.[106] Aerobic exercise is known to alter substrate utilization and availability during and following exercise. This may influence the regulation of EI, as it has been suggested that short-term feeding behaviour is designed to maintain the body's glycogen stores at a specific set point e.g. the Glycogenostatic theory.[107-108]. Due to its limited capacity for storage, challenges to glycogen availability (via diet or exercise) may act an internal biological cue that elicits feeding in order to restore glycogen levels.[107] However, evidence to support a direct link between substrate metabolism during exercise and compensatory eating is limited[109] and contradictory.[110] Recent evidence suggests that the maintenance of CHO balance plays a role in the short-term regulation of EI. Burton et al[111] reported that a positive CHO balance at the end of a six hour 'high energy turnover' condition (involving exercise and the immediate restoration of energy balance) was associated with lower *ad libitum* EI at a subsequent buffet meal than following a 'low energy turnover' (non-exercise) condition. Despite differing nutrient balances during the conditions, CHO balance following the buffet meal was identical, suggesting that feeding was driven by the need to restore CHO balance to a set level. This is

consistent with the finding that EI following a day of dietary CHO deprivation was designed to restore CHO balance rather than energy balance.[112] Where CHO balance has been measured over longer periods (1-3 days) following the manipulation of glycogen availability, a negative CHO balance (indicating greater CHO oxidation than intake) has been shown to be predictive of greater *ad libitum* EI over subsequent days.[111, 113-116] Furthermore, a negative carbohydrate balance has been shown to be predictive of weight gain over a four year follow-up period.[117] While inferences concerning glycogen availability based on short-term CHO balances should be made cautiously, the partitioning of dietary CHO for storage rather than oxidation appears to be associated with lower EI and more stable body weight.

### **III) Does exercise alter food and taste preferences?**

A contributor to the compensatory response to exercise could be alterations in food and taste preference.[118] In a recent review, Elder & Roberts[119] identified 12 studies investigating the acute effect of exercise on food palatability and taste perception.[9, 120-130] We have since identified a further 6 studies that contribute to this literature.[131-136] In terms of taste perception and preference, the outcomes are rather variable and show increases, no change and decreases in acuity of taste perception and rated preference for tastes after exercise. This between-study variation may be explained by differences in the exercise protocols adopted. Those studies employing longer and higher intensity exercise sessions (120-180 minutes) tended to find effects of exercise[124, 130], while shorter, lower intensity exercise (10-30 minutes) studies did not report significant changes.[123, 125] Effects were more likely to be reported for perception and preference of salt than sweet or bitter taste. This suggests any effect of exercise on taste perception and preference is

likely to be small or subject to a threshold of physical exertion or energy expenditure. Currently there are no studies that have independently examined the roles of exertion and expenditure on taste perception and taste preference after exercise. Other studies have tested the effect of exercise on the palatability of whole meals or food items more generally.[122, 126] King et al[122] found increased palatability ratings after high fat and low fat test meals, while Lluch et al[126] found increased ratings for low fat foods only. Finlayson et al[137] measured hedonic response to images of food immediately before and after acute exercise at baseline and following 12 weeks of daily moderate intensity exercise. The authors reported an overall decrease in palatability ratings over the 12 weeks, however acute increases in food preference after the exercise session was associated with poorer weight loss response to the intervention. Finlayson et al[132] measured reaction times – an implicit measure of wanting for food - in response to foods varying in fat content and sweet taste after 50 minutes moderate intensity cycling (compared to no exercise) followed by a test meal. The authors observed increased reaction times (increased food wanting) after exercise among those participants who increased their EI compared to those whose EI did not change. The findings that individual differences in the hedonic evaluation of food after exercise are linked with food intake and poor weight loss outcome suggest that altered food preferences are a stable characteristic that may partially explain compensatory responses to exercise.

Exercise-induced changes in food preference imply a change in the hedonic or motivational response to food. These processes can be understood as separable psychological components of liking and wanting that can be dissociated in the brain[138] and behaviourally in response to acute exercise.[132] It has been hypothesised that exercise may act as a buffer for reward driven eating.[139] In rats, chronic exercise has been shown to decrease the reinforcing value of self-administered cocaine.[140] However it is also possible that exercise at the acute level has an enhancing effect on reward (similar to a low dose of an addictive drug) via a sensitizing action.[141] The deliberate choice of foods with high hedonic value (e.g. fatty or sweet tasting 'treats') to reward virtuous behavior or to regulate changes in psychological states could be associated with exercise-induced changes in mood (e.g.[135]) or stress.[136] The effects of exercise on food preference can be linked with equal importance to the metabolic and cognitive consequences of engaging in physical activity. For example, the effect of exercise on food preference can depend

on eating behaviour traits of the population studied such as Restraint and Disinhibition.[122, 126, 142-143]

Exercise-induced changes in food reward could be an important consideration in the efficacy of exercise as a means to reduce overweight. In particular, an enhanced motivational drive or wanting for food after exercise may help to explain why some people overcompensate during acute eating episodes.[132] Some individuals could be resistant to the beneficial effects of exercise due to a predisposition to compensate for exercise-induced energy expenditure as a result of physiologically or psychologically modulated changes in food preference.

#### **IV) Psychological processes and behavioural traits**

There is evidence to suggest that eating behaviour traits measured using the Three Factor Eating Questionnaire[144] exert an influence on food intake and that they also play a role in weight loss interventions.[143] The factors of Disinhibition and Restraint in particular, have emerged as important eating behaviour traits which influence weight gain, weight loss and weight maintenance, thus can be deemed as psychological markers of appetite regulation. On the one hand, there are data to suggest that individuals with a high level of Disinhibition, are more susceptible to overcompensate for the energy expended during exercise[145], whereas exercise can also exert a positive influence over appetite control in individuals who show susceptibility towards opportunistic eating. For example, in lean women, an acute bout of exercise has been found to reduce motivation to eat and increase preference for low fat foods.[146] Conversely, in lean women with a high level of Restraint, an acute bout of exercise increased the perceived pleasantness of low-fat food and reduce the motivation to eat.[126] In concordance with this, lean and overweight males with high Restraint, did not show a counter-regulatory eating response (an overeating response initiated by the breakdown of cognitive Restraint) following a bout of moderate intensity exercise.[147] Therefore the influence of an acute bout of exercise appears to be beneficial, at least in the short-term, for men and women who exhibit a high Restraint score.



In longer term interventions there is a consistent and robust finding that successful weight loss is associated with a decrease in Disinhibition and Hunger, and an increase in Restraint (e.g. ([148-152])). Independent of the type of energy balance perturbation, individuals who are successful in losing weight, respond by increasing their control over eating (i.e., restraint) and reducing their opportunistic eating behaviour. More specifically, Butryn et al[153] found that individuals who showed a larger reduction in their level of Internal Disinhibition (e.g. eating in response to negative affect), experienced the greatest weight loss.[153] Two studies have examined the influence of exercise over a longer term on psychological aspects of appetite regulation. Keim et al[154] found that following a four-month exercise intervention (aerobic exercise or resistance training 5d/week), reduced-obese women could be separated into over- and under-compensators. Those who overate were characterised by a high Disinhibition and Hunger scores on the TFEQ, whereas the under-eaters showed a decrease in Disinhibition and Hunger scores and an increase in Restraint score during the intervention. In line with this, a recent 3 month exercise intervention, designed to expend 500kcal/day, with no dietary intervention[22, 24], demonstrated that overweight and obese individuals with a higher baseline Disinhibition experienced a greater weight loss. In addition, those individuals who experienced a decrease in their level of Disinhibition and an increase in Restraint had a higher weight loss.[155] Figure 4 shows data originating from King et al[24] demonstrating how changes in TFEQ eating behaviour traits are affected differentially – depending on the actual weight loss experienced. That is, those who lost less weight than expected (non-responders) experienced lower decreases in Disinhibition and increases in Restraint than those who lost equal to, or more than the expected weight (responders). Of course, it is not clear whether it is exercise *per se*, or weight loss which is driving these changes – but it is clear that exercise can be used to modify eating behaviour traits which are associated with susceptibility to weight gain. By identifying and characterising psychological markers of eating behaviour the efficacy of exercise on weight loss could be improved.

Figure 4 about here

## **Summary**

This review has identified the current state of play regarding the interaction between exercise and appetite – with a focus on individual variability and compensatory responses. It is clear that although the phenomenon of exercise-induced inter-individual variability in responses was highlighted over 20 years ago - it hasn't been exploited - and the characterisation of this variability can be used to identify resistance to exercise-induced weight loss and for more efficient weight management strategies. Resistance to exercise-induced weight loss is partially explained by orexigenic responses in eating behaviour, which are mediated by physiological and psychological processes. A better understanding of how these underlying processes collectively contribute to a lower than expected weight loss, will determine why for some, exercise is not the most effective weight loss method.

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### ***Information Box***

#### **What is already known on this topic?**

Although there are several reviews of the effects of exercise on appetite, there is no information on the proposed mechanisms on the relationship. Therefore, there is information on the summary of how exercise affects appetite, but nothing on the various behavioural, physiological and psychological processes involved.

#### **What this study adds:**

This review adds value by providing a collective discussion of how the behavioural, physiological and psychological processes might influence compensatory responses to exercise, and partially explain the individual variability.

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### **Table and Figure Legends**

Figure 1. Individual variability in changes in body weight in overweight and obese men and women (n=58) after 12 weeks of supervised exercise. Each histogram represents an individual's change in weight. There was no difference between males (■) and females (□). Data from King *et al*, 2009a.

Figure 2. Daily energy and nutrient intakes, and orocecal transit times in 20 physically active males. Data shows a negative correlation between mouth to large intestine (orocecal) transit time and daily energy intake. Data from Harris *et al*, (1991).

Figure 3. Total ghrelin (A) and acylated ghrelin (B) plasma levels (pg/mL) during the post-breakfast postprandial period, before (◆) and after (□) a 12-week exercise intervention in overweight/obese individuals (n=15). Despite no change in TG, there was an exercise-induced significant increases in fasting (p=0.01) and postprandial suppression (p=0.009) of AG plasma levels. Values represent means ±SEM. Adapted from Martins *et al*, 2010.

Figure 4. Mean (±SEM) changes in Three Factor Eating Questionnaire (TFEQ) scores of Restraint, Disinhibition and Hunger after 12 weeks of supervised exercise. Data is displayed for responders and non-responders who lost more than, and less than the expected weight respectively.

Table 1. Changes in body mass, fat mass, waist circumference, blood pressure and  $VO_{2max}$  in responders and non-responders after 12 weeks of supervised exercise. Adapted from King *et al*, 2009b.