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Effects of exercise and restrained eating behaviour on appetite control

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Obesity is a global epidemic; increased consumption of energy-dense food and reduced physical activity levels are likely to be the main drivers. Previous cross-sectional research has shown that sedentary males, unlike their active counterparts, are unable to compensate for previous energy intake (EI). Using a longitudinal design a 6-week exercise intervention was found to improve short-term appetite control, leading to a more 'sensitive' eating behaviour in response to previous EI, both acutely at a test meal and for the next 24 h. Although the mechanisms whereby acute and chronic exercise improves short-term appetite remain unknown, post-ingestive satiety peptides are likely to be involved. Acute exercise was found to increase postprandial levels of polypeptide YY, glucagon-like peptide-1 and pancreatic polypeptide but to have no impact on ghrelin, suggesting that exercise can trigger physiological changes in satiety hormone secretion that could help in appetite control and weight maintenance. In the context of an increased availability of highly-palatable food, dietary restraint may be increasingly important. Although restraint has been associated with abnormal eating behaviour, in the laboratory no counter-regulation was found in restrained eaters when presented with a buffet meal 60 min after a high-energy preload or when a pasta-meal was presented 3 h after preloading. Although restraint was not found to impact on polypeptide YY or TAG, lower postprandial glucose and insulin plasma levels were observed in restrained eaters, together with increased feelings of fullness. In conclusion, short-term appetite control seems to be favourably modified by exercise, while the impact of restraint on appetite seems to be more complex.

Exercise: Appetite: Restraint: Gut peptides

Obesity has become a global epidemic especially in (although not restricted to) developed countries, with >1·6 billion overweight adults and ≥400 million individuals who are clinically obese worldwide[1]. In England the prevalence of obesity increased almost threefold between 1980 and 2004, with present numbers indicating that >60% of the population are overweight and approximately 24% are obese[2]. If this rate of growth continues it is expected that by 2010 more than one in four adults in England will be obese, approaching the levels of obesity currently seen in the USA[3]. This steady increase in the prevalence of obesity has been accompanied, on one hand, by an increase in the consumption of energy-dense food and, on the other hand, by a reduction in physical activity (PA) levels[4,5].

It is a paradox that obesity is increasing while overall energy intake (EI) has been falling over the past 20 years in the UK[6]. It has been proposed that a reduction in PA levels together with an inability of individuals to down-regulate EI to a similar extent in order to match the decreased energy expenditure (EE) may be the dominant factors in promoting obesity[7]. It has been shown that the adoption of a sedentary lifestyle is not followed, at least in the short to medium term, by a compensatory decrease in EI, with consequent positive energy balance (EB)[8,9]. This outcome does not mean, however, that physical inactivity alone explains this epidemic, or that diet has no role in the aetiology of obesity[10–12]. However, it is undeniable that PA has decreased over the past few decades, driven by dramatic changes in lifestyle[6]. The current recommendations for adults, shared by the Department of Health in the UK and the American College of Sports and Medicine, are to accumulate at least 30 min of moderate PA on most, preferably all, days of the week[13–15].

Abbreviations: PA, physical activity; EI, energy intake; EE, energy expenditure; EB, energy balance; GLP-1, glucagon-like peptide-1; HEP, high-energy preload; LEP, low-energy preload; PYY, polypeptide YY; PP, pancreatic polypeptide; RRS, restrained revised scale; DEBQ, Dutch eating-behaviour questionnaire; TFEQ, Three-factor eating questionnaire.

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However, in England only 25% of women and 37% of men were achieving that target in 1998[15]. The situation in other European countries is no better[4].

Eating behaviour is a complex phenomenon encompassing the size and frequency of eating episodes and everyday food choices, which together determine total energy and macronutrient intake, and is the result of constant, physiological and environmental inputs[16,17]. The latter are especially important, and it is accepted that the physiological mechanisms that control food intake can be easily overridden by strong social and environmental factors[10]. Appetite is a subjective concept used to explain the control of food intake and can be defined as a range of variables associated with food consumption that predict normal eating behaviour[16,18].

At the physiological level appetite and food intake are under the control of both the brain and a plethora of hormones produced mainly by the gastrointestinal tract, but also by the pancreas, adrenal glands and adipose tissue[19]. These two types of regulation are known respectively as central and peripheral. It is well established that the hypothalamic region of the brain plays a key role in the central regulation of eating behaviour in human subjects and is constantly receiving and processing neural, metabolic and endocrine signals from the periphery, enabling it to adjust not only EI but also EE. The area within the hypothalamus most actively involved in the regulation of feeding is the arcuate nucleus, which expresses receptors for many of the hormones and peptides known to be involved in eating behaviour[20].

While fasting leads to the release of the orexigenic hormone ghrelin, feeding stimulates the coordinated production and release of several satiety hormones such as glucagon-like peptide-1 (GLP-1), peptide YY (PYY) and pancreatic polypeptide (PP). Some of these hormones are directly involved in gastric emptying, while others have longer-lasting postprandial effects that will affect not only satiation (or meal termination) but also satiety (inter-meal interval)[21,22].

A second category of peripheral signals that influences food intake includes hormones, the secretion of which is directly proportional to the amount of body fat: insulin released by the pancreas; leptin released mainly by the adipose tissue. In contrast to satiety signals these adiposity signals are tonically active, providing constant information to the hypothalamus about the state of energy stores. Leptin has been shown to reduce food intake and body weight and increase EE[21]. When fat stores are reduced leptin production is down regulated, leading to the stimulation of orexigenic neurons expressing neuropeptide Y and agouti-related peptide and the inhibition of anorectic neurons expressing propiomelanocortin[23,24]. Apart from acting directly as adiposity signals, both leptin and insulin can regulate body weight indirectly, by modulating the sensitivity of the brain to satiety signals and therefore determining the total amount of food eaten at an individual meal[25].

Apart from physiological processes, appetite is regulated by external stimuli arising from food and the surrounding environment. Environmental, psychological, social and cultural stimuli have been shown to exert powerful effects on food intake[26]. Moreover, food intake is also under hedonic control[27], activated by the availability of palatable food[28]. The increased availability of highly-palatable food in present-day society may lead to the chronic activation of the hedonic appetite system and explain why an increased number of individuals need to restrain their food intake in order to maintain or lose weight. Dietary restraint has, therefore, become an important behavioural concept.

In the face of the current obesity pandemic, the widespread levels of physical inactivity and the increased availability of highly-palatable energy-dense food, it is therefore important to understand how exercise (both in the short and long term) and dietary restraint impact on appetite and eating behaviour in order to achieve a better knowledge of the aetiology and/or potential treatments of obesity. The present review will be confined to these aspects.

Exercise and appetite

Effects of acute exercise on appetite, energy intake and energy balance

The majority of the studies have shown that acute exercise does not increase hunger or EI[29–34], even when vigorous exercise is taken[35], and so exercise is able to induce a short-term negative EB. In fact, vigorous exercise has been found to suppress hunger acutely, a phenomenon that has been described as 'exercise-induced anorexia'[31,36], although this phenomenon is short-lived and is unlikely to have any marked effect on EI[18,29,36,37]. However, some studies have shown an increase in appetite sensations[38], an increase in subsequent EI[39–41] or even a decrease in EI[32] in response to acute exercise. This lack of consistency is probably a result of differences in methodology, i.e. the intensity of exercise[29], nutritional state[42], gender[43], macronutrient composition of the test meal[44] and the time lag between exercise and eating[39].

King and colleagues have suggested that the inability of some studies to show a beneficial effect of acute exercise on EB derives from the fact that they do not account for the energy cost associated with exercise[35]. Even if acute exercise leads to a compensatory increase in absolute EI, a short-term negative EB can still be achieved if relative EI, i.e. EI after accounting for the EE induced by exercise, is reduced (when compared with a resting condition). A review paper by Blundell and colleagues has concluded that in the short (1–2 d) to medium term (7–16 d) exercise can produce a negative EB, with no substantial compensatory responses in EI being observed[45]. In the long term (>16 d) EI starts to increase, although the observed compensation is usually partial and incomplete, accounting for approximately 30% of the energy cost associated with exercise[45].

In fact, exercise has been shown to be more effective than dieting in inducing a negative EB in the short term. Hubert and colleagues have studied the effects of acute energy deficits created by dietary manipulation or exercise on appetite responses and subsequent EI in unrestrained women[46]. It was found that hunger ratings ($P<0{\cdot}005$),

ad libitum EI at a buffet lunch ($P<0·05$) and food cravings during the day ($P<0·05$) are significantly increased following a low-energy breakfast compared with a high-energy breakfast, whilst exercise fails to induce any significant effect on these variables, consequently resulting in a negative EB.

Effects of chronic exercise on energy intake, energy balance and body weight

Although in the short to medium term exercise can produce a negative EB, the general view in relation to the impact of long-term exercise on EB is that in the absence of energy restriction only very modest results are observed in terms of weight loss[47]. This finding does not mean, however, that exercise should not be present in all interventions intended to tackle the problem of obesity. In fact, although diet alone may seem at the first glance to be a more effective way of losing weight in the short term, its efficacy in the long term is questionable. In a recent comprehensive analysis of thirty-one long-term studies of the effect of energy restriction on weight loss it was found that despite an initial 5–10 % weight loss in the first 6 months of dieting one-third to two-thirds of the subjects regained more weight than they had originally lost within 4–5 years[48].

Many factors may help to explain the relative inefficacy of exercise as a weight-loss strategy. First, exercise leads to an improvement in body composition with the progressive substitution of fat by fat-free mass[49], which clearly underestimates its impact on weight loss. Second, when EB is disturbed by exercise, several physiological and behavioural compensatory mechanisms are activated in order to achieve a new steady-state[50]. Weight loss is associated with a compensatory reduction in total BMR, probably driven by the changes in body composition in favour of decreased fat-free mass[51]. Moreover, as maximal O_2 uptake increases with exercise training, a lower EE is achieved for the same volume of exercise. Finally, as body weight decreases the net exercise-induced EE is also reduced, especially for weight-bearing exercise[50]. However, it is unlikely that these physiological and metabolic mechanisms alone are responsible for the attainment of a new steady-state by reversing the uncoupling between EI and EE. Behavioural mechanisms are probably more important[34,52]. Two major behavioural mechanisms can reverse the energy imbalance created by exercise: a compensatory increase in EI and/or a reduction in EE, probably through down-regulation of spontaneous PA (non-exercise EE)[53,54]. The increase in EI may result from increased hunger or from a more relaxed dietary regimen, especially towards more-energy-dense foods, because of the widespread belief that the energy cost of exercise can offset dietary indulgences[46]. However, if EI does not increase, or the increase is insufficient, and EE is maintained constant a new steady-state may still be achieved (although it would take longer) through the changes in metabolism discussed earlier[54].

Despite the general view that exercise is a rather inefficient strategy to lose weight, a great variability is usually found among studies, with reports of weight loss, maintenance of weight or even a small weight gain after a PA intervention[55]. Although methodological differences among studies are likely to explain some of this variation, i.e. the macronutrient composition of the diet and the characteristics of the exercise intervention (type, duration, frequency and intensity)[55], it has been suggested that the large inter-individual variation in the response of body weight to exercise is a result of differences in the coupling between EI and EE[7]. Unfortunately, most of the studies investigating the impact of chronic exercise on EB are limited by the fact that neither EI nor EE were controlled or accurately measured. Free-living subjects tend to up regulate their EI in response to exercise, even if instructed not to, and often do not comply with the exercise prescription[56–58]. Moreover, an increase in total EE induced by exercise often rests on the assumption that normal activity throughout the rest of the day remains unchanged or increases[55], an area that remains controversial[59,60]. However, there is enough evidence to show that substantial weight loss can be achieved with exercise alone if EI is maintained constant and compliance with exercise is good[51,61].

A recent review has demonstrated that exercise is crucial in preventing weight gain or regain in the long term[47], therefore reinforcing the importance of incorporating exercise in any plan designed to reduce or maintain body weight in the long term. It can be hypothesized that the beneficial effect of exercise on weight maintenance results from an improvement in appetite control. It may be that exercise leads to a better coupling between EI and EE in the long term that ensures that body weight is maintained[1] or to a more sensitive eating behaviour in response to previous EI[2].

Effects of exercise on appetite control

It has been shown that the coupling between EI and EE is dependent on habitual PA levels. As early as 1956 Mayer and collaborators examined the coupling between EI and EE in a male population of mill workers in West Bengal (India) by comparing EI in different groups according to the level of PA in their jobs[62]. A good correlation was found between EI and energy requirements, but the rise in EI with increasing levels of PA was only observed within a specific range of PA that the authors designated as 'normal activity range'. It was found that if PA is below that range, as happens in sedentary individuals, a decrease in EE is not followed by a proportional decrease in EI, but instead by an increase, therefore leading to a positive EB and weight gain. This finding provides an explanation for the increased body weight observed in the sedentary individuals compared with the more-active individuals[62]. It may be hypothesized, therefore, that the coupling between EI and EE, and in a broader context appetite control in general, is disrupted at low levels of PA. This outcome could help to explain why it is so hard to maintain body weight after a weight-loss intervention with a sedentary lifestyle, since the body is not able to down regulate EI to match the low levels of EE.

This and other cross-sectional studies showing a tight coupling between EI and EE at high levels of PA[18] suggest that exercise might sensitize the physiological

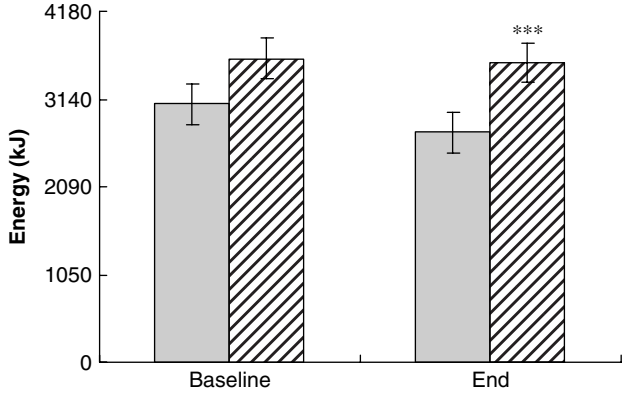

Fig. 1. The energy intake (kJ) at an *ad libitum* buffet lunch 1 h after a high-energy preload (HEP; □) and a low-energy preload (LEP; ///) was measured at baseline and at the end of a 6-week moderate-intensity exercise intervention in unrestrained normal-weight sedentary individuals (*n* 25; eleven men and fourteen women). Values are means with their standard errors represented by vertical bars. The effect of preload was significant (ANOVA; $P<0{\cdot}0001$), but the effect of exercise and the interactions were not significant. Mean value was significantly different from that after HEP: ***$P<0.0001$. (From Martins *et al.*[65].)

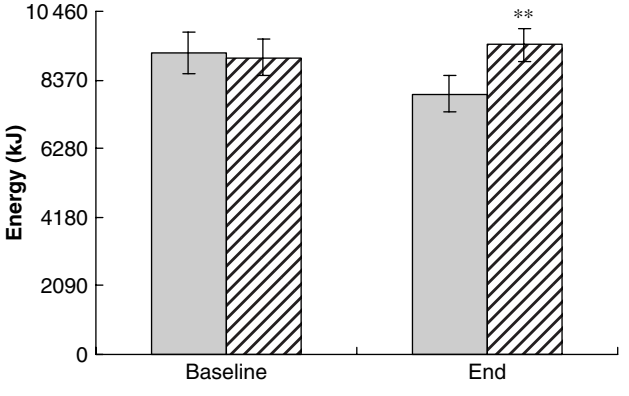

Fig. 2. The cumulative energy intake (kJ) over a 24 h period after a high-energy preload (HEP; □) and a low-energy preload (LEP; ///) was measured at baseline and at the end of a 6-week moderate-intensity exercise intervention in unrestrained normal-weight sedentary individuals (*n* 25; eleven men and fourteen women). Values are means with their standard errors represented by vertical bars. The exercise × preload interaction was significant (ANOVA; $P=0{\cdot}023$), but the main effects of exercise and preload and other interactions were not significant. Mean value was significantly different from that after HEP: **$P<0{\cdot}01$. (From Martins *et al.*[65].)

mechanisms involved in appetite control. Evidence for the role of exercise on short-term appetite control has been provided by King and colleagues, who have shown that active males are able to detect differences in the energy content of covertly-manipulated drinks after an exercise challenge and to adjust for the difference at a subsequent meal, achieving an almost perfect energy compensation[63].

Further support for this hypothesis has come from a study that has shown a better compensatory response to covert preload energy manipulation in active men compared with sedentary men[64]. Using a cross-over design healthy lean men were randomized to a low-energy (LEP) or high-energy (HEP) preload on two different occasions followed 60 min after the preload by an *ad libitum* buffet lunch. The buffet EI following the LEP and HEP was not significantly different in the sedentary group, denoting a deficient homeostatic feedback control of hunger and satiety that if maintained over time could lead to energy imbalances; in contrast, an almost perfect energy compensation (90 %) was observed in the exercise group, who down regulated the buffet EI after the HEP. However, a cross-sectional design does not prove causality and it is possible that the better energy compensation observed in the active group may be a result of other factors such as lifestyle or cognitive factors and be unrelated to their PA levels.

The effect of long-term exercise on the sensitivity of compensation in response to previous EI has recently been assessed using a more robust longitudinal design[65]. Short-term appetite control, using the preload–test meal paradigm, was measured before and after a 6-week moderate-intensity exercise programme in unrestrained normal-weight sedentary individuals (eleven men and fourteen women). Energy compensation was measured using a protocol similar to that described by Long and colleagues[64], with the participants being given a HEP or LEP on two separate occasions and EI measured 1 h later at an *ad libitum* buffet meal. An improvement in appetite control was observed with the exercise intervention, with a more-sensitive eating behaviour in response to previous EI, both acutely at lunch time and for the next 24 h. While at baseline participants were unable to adjust their subsequent EI in response to preload-energy manipulation, after the exercise intervention their EI after the HEP was found to be significantly lower than that after the LEP (Figs 1 and 2)[65]. These findings suggest that the role of exercise on EB extends beyond its ability merely to increase EE, and includes an indirect effect that modulates food intake towards a more 'sensitive' eating behaviour in response to previous EI.

The mechanisms targeted by exercise that may help to explain this improvement in short-term appetite control are likely to fall into three categories: long term, including leptin and insulin; intermediate, including post-absorptive signals associated with macronutrient oxidation such as glucose and NEFA levels; short-term satiety signals arising from the gastrointestinal tract in response to food intake[16], such as cholecystokinin, GLP-1 and PYY[21]. However, no significant changes in fasting insulin, glucose, TAG or NEFA levels or insulin sensitivity were found with the exercise intervention, and leptin levels are unlikely to have changed[65] as no significant changes in body weight or composition were observed with the 6-week exercise intervention[66]. Changes in the release of satiety hormones by the gastrointestinal tract are, therefore, likely to be involved in the improvement in short-term appetite control observed in this study.

Effects of exercise on appetite-related hormones

Although the effects of exercise on leptin and ghrelin have been extensively studied and the conclusion drawn that

Table 1. Studies looking at the effect of acute and chronic exercise on gut peptides involved in appetite control

Reference	Subjects	Intervention	Outcome
Hilsted *et al.*[72]	Healthy men	3 h exercise (cycle ergometer) at 40% V_{O2max} *v.* resting	Significant increase in fasting PP ($P<0·05$)
Sullivan *et al.*[71]	Male athletes	Marathon running	Significant increase in fasting PP ($P<0·01$)
Greenberg *et al.*[73]	Non-obese healthy men and women	45 min exercise (cycle ergometer) at 50% V_{O2max}, 30 min after a 1780 kJ breakfast *v.* resting	Significant increase in postprandial levels of PP ($P<0·01$)
Hurley *et al.*[75]	Normal-weight sedentary males	10-week exercise programme (20 min jogging at 70% V_{O2max}, three times per week) Blood samples were taken before and after the intervention in fasting and after a 1340 kJ breakfast	Slight increase in PP fasting levels and postprandial peak
O'Connor *et al.*[69]	Athletes (men and women)	Marathon running	Significant increases in fasting GLP-1 ($P<0·0001$)and PP ($P<0·01$)
Bailey *et al.*[68]	Physically-active normal-weight men	Cycling test to exhaustion 4-week cycling exercise programme (three times per week with incremental duration and intensity)	Significant increase in fasting CCK levels ($P<0·05$) in relation to resting No effect on fasting CCK levels
O'Connor *et al.*[70]	Male athletes	2 h treadmill run at 60% Vo_2 max *v.* resting	Significant increase in fasting GLP-1 during exercise, but also during resting ($P<0·001$)

CCK, cholecystokinin; PP, pancreatic polypeptide; GLP-1, glucose like-peptide-1.

exercise, in the absence of weight loss, does not induce any significant increase in the plasma levels of these two hormones[66,67], few studies have looked at the impact of both acute and chronic exercise on circulating levels of satiety gut peptides known to be involved in appetite control (Table 1).

The little evidence currently available suggests that acute exercise increases fasting plasma levels of cholecystokinin[68] and GLP-1[69,70], although these latter two studies were performed in athletes and in the study of O'Connor and colleagues an identical increase in GLP-1 plasma levels was also observed during resting[70]. Acute exercise has also been shown to increase plasma PP levels, not only whilst fasting[69,71,72] but also postprandially[73], although the increase is dependent on the intensity of exercise[74]. Chronic exercise, on the other hand, has been shown not to increase fasting cholecystokinin levels in active men[68] but to induce a slight increase in fasting PP plasma levels as well as in its postprandial peak[75] in previously-sedentary males. Interestingly, the magnitude of the increase observed in plasma PP levels with acute exercise seems to decrease with training[76]. However, most of these studies have looked only at the impact of exercise on the fasting levels of these gut peptides. Cholecystokinin, GLP-1 and PP are satiety hormones released in the postprandial state, and therefore changes in fasting levels provide very little information. Moreover, appetite measurement was not the primary outcome in any of these studies. To the authors' knowledge, there are no published studies that relate changes in the plasma levels of these appetite-related hormones in response to both acute and chronic exercise to alterations in subjective and objective measures of appetite and net EB.

To try to address some of these questions, a study has been designed to investigate the effects of an acute bout of moderate-intensity exercise when performed in the fed state on the plasma levels of appetite-related hormones and metabolites, and to correlate potential alterations with changes in subjective feelings of hunger and fullness and prospective food intake at a subsequent meal. Ghrelin, PYY, GLP-1 and PP were measured both in the fasted state and postprandially in healthy unrestrained normal-weight volunteers using a randomized cross-over design. At 1 h after a standardized breakfast subjects either cycled for 60 min at 65% of their maximal heart rate or rested. Subjective appetite was assessed throughout the study period using visual analogue scales, and subsequent EI at a buffet meal was measured at the end of the meal (3 h post-breakfast, 1 h post-exercise)[77].

A significant increase in buffet EI was observed with exercise. However, once the energy expended during exercise had been accounted for, a reduction in relative EI was observed with exercise, with the attainment of a short-term negative EB. This outcome was associated with the absence of any significant difference in hunger sensations or in circulating appetite-related hormones between the exercised leg and the control leg immediately before the buffet meal[77]. Moreover, a transitory increase in the plasma levels of satiety hormones was observed during exercise in the absence of an increase in the orexigenic peptide ghrelin, suggesting that acute exercise does not trigger any compensatory responses at the level of gastrointestinal hormones involved in appetite regulation that would lead to an increase in hunger and/or EI in the short term[77]. On the contrary, the transitory increase in circulating satiety hormones that occurred during exercise could be responsible for the well-documented phenomenon of

exercise-induced anorexia, which has been described previously[31,36]. It is possible that the increase in absolute EI observed in response to acute exercise is the result of cognitive factors, including attitudes and beliefs associated with exercise such as 'food rewards for exercising' and the belief that 'exercise increases hunger'[50].

Dietary restraint

Dietary restraint refers to the extent to which individuals are concerned with their body weight and attempt to control it by dieting[78]. It is characterized by a self-imposed resistance to the internal and external cues that regulate eating behaviour, motivated by the desire to maintain or suppress body weight[78]. Dietary restraint has been associated with an abnormal eating behaviour in response to preloading. While unrestrained individuals eat less after a preload, restrained individuals tend to eat more; two opposite behaviours that have become known as 'regulatory' and 'counter-regulatory eating' respectively[78–83].

It has become a common practice to exclude restrained eaters from appetite studies[31–33,84,85], based on the assumption that they exhibit an atypical eating behaviour in response to preloading. Moreover, it has also been suggested that the level of restraint can modulate the effects of exercise on EB[53]. Acute exercise has previously been shown to be more effective in creating a negative EB in restrained eaters compared with unrestrained eaters; while unrestrained eaters increase their EI after exercise, restrained eaters tend to decrease their EI[86]. However, the interaction between restraint and exercise in determining EI in the post-exercise period remains controversial[30,33] and is probably dependent on disinhibition (loss of restraint) levels.

The exclusion of restrained eaters from appetite studies, however, has been carried out somewhat arbitrarily and no consensus seems to exist either on the questionnaire or on the cut-off points used for exclusion. Rather surprisingly, closer analysis of the literature reveals that only a few studies have been able to show a true 'counter-regulatory eating behaviour' (with a significant preload × restraint interaction) in response to preloading in restrained eaters[78,80–83], with such behaviour not being observed in the majority of studies[87–93] (Table 2). Interestingly, in all these studies a single highly-palatable and 'diet-breaking' food (ice cream) was used as the test meal and restraint was always measured by the restrained revised scale (RRS)[94]. It seems, therefore, that the response to preloading, in terms of eating behaviour, is dependent on both the questionnaire used to measure restraint and the type of test meal presented.

The dependence on the use of the RRS to demonstrate counter-regulatory eating behaviour is not unexpected, since the RRS tends to identify individuals susceptible to disinhibition, and therefore 'unsuccessful dieters', while the restraint subscales of the three-factor eating questionnaire (TFEQ)[95] and the Dutch eating-behaviour questionnaire (DEBQ)[96] tend to identify individuals less susceptible to disinhibition, and therefore 'successful dieters'[97]. It has been reported previously that only those subjects who simultaneously present with high levels of both restraint and disinhibition show a counter-regulatory eating behaviour in response to preloading[98]. Dietary restraint has been shown not to be a homogeneous construct and two dimensions have been identified: 'rigid control of eating' and 'flexible control of eating'[99]. Those exhibiting a 'pure' rigid control are more likely to show counter-regulatory eating behaviour, since they tend to set their cognitive diet boundary as a rigid point, which if passed means that further attempts to diet are hopeless, with consequent disinhibition and overeating.

Several studies have tried to identify the best questionnaire to predict disinhibition in the laboratory[100–104], but the results have been inconclusive. However, they have all used the taste-test paradigm that involves tasting and rating a single highly-palatable food (usually seen as 'diet breaking'), e.g. ice-cream, cookies or crackers, none of which illustrates a 'typical' meal. The question remains, therefore, as to whether restrained eaters need to be excluded from appetite-related studies and, if so, which questionnaire and cut-off point should be used.

The predictive validity of three scales that assess dietary restraint (the RRS, a shortened version of the TFEQ (eighteen-item revised[105] and the DEBQ) have been investigated for their ability to predict disinhibited eating behaviour in normal-weight women in the laboratory (C Martins, E Tolhurst and LM Morgan, unpublished results). Using a cross-over design fifteen participants were randomly assigned to an HEP and an LEP, each clearly labelled with the exact energy content, and EI was measured 1 h later at an *ad libitum* buffet lunch comprising a variety of foods that included healthy (sandwiches, salad, yoghurt, fruit) and unhealthy options (cakes, biscuits, crisp, sauces). It was found that restrained women fail to show a counter-regulatory eating behaviour, independently of the questionnaire used to measure restraint (classification based on a median split). Indeed, both restrained and unrestrained individuals were found to show a regulatory eating behaviour, eating more after the LEP compared with after the HEP ($P<0{\cdot}01$), independently of the questionnaire used to measure restraint. Moreover, no significant differences in energy compensation (calculated as the difference in EI at the buffet lunches between the two study days (LEP–HEP) divided by the difference in preload energy (1506 kJ) and expressed as a percentage) were found between restrained and unrestrained women. Interestingly, a significant negative correlation ($r-0{\cdot}653$, n 15, $P<0{\cdot}01$) was observed between energy compensation and restraint when using the RRS, but not the other scales (C Martins, E Tolhurst and LM Morgan, unpublished results). It seems, therefore, that despite the inability of the RRS to predict disinhibition in this study, the loss of compensation observed with increasing levels of restraint was best forecasted by this scale.

Similar results were observed in another study with a mixed sample of normal-weight volunteers (twenty-one women and twelve men). Dietary restraint was measured by the DEBQ and TFEQ (eighteen-item revised version) and for women restrained eaters were defined as scoring ≥ 18 on the restraint subscale of the TFEQ (eighteen-item revised version) and/or $\geq 3{\cdot}7$ on the restraint subscale of

Table 2. Studies assessing the effects of restraint on food intake after preloading

Reference	Tool	Cut point for restraint	Design	Preload	Test meal	Results
Herman & Mack[78]	RRS	Median split (8·5)	Parallel study No preload One milkshake Two milkshakes	Milkshake (213 g)	Taste test Ice cream	Significant preload × restraint interaction ($P<0·005$) Restrained counter-regulated Unrestrained compensated
Polivy[87]	RRS	Median split (11·5)	Parallel study ↑ Energy told ↑ energy ↑ Energy told ↓ energy ↓ Energy told ↓ energy ↓ Energy told ↑ energy	High- and low-energy chocolate pudding (227 g)	Taste test Sandwiches	Tendency to a significant preload × restraint interaction ($P = 0·07$) Restrained eaters counter-regulated, but only when perceiving the preload as ↑ energy Unrestrained eaters regulated only when perceiving the preload as ↑ energy
Hibscher & Herman[83]	RRS	Median split (14)	Parallel study No preload Preload	Milkshake (425 g)	Taste test Ice cream	Significant preload × restraint interaction ($P<0·05$) Restrained eaters counter-regulated Unrestrained eaters compensated
Spencer & Fremouw[82]	RRS	Median split (16)	Parallel study Told ↑ energy Told ↓ energy	Milkshake (445 g; 2092 kJ)	Taste test Ice cream	Significant perceived preload × restraint interaction ($P<0·05$) Restrained eaters counter-regulated Unrestrained eaters compensated
Woody *et al.*[80]	RRS	Median split (16·5)	Parallel study No preload Good or bad tasting preload (presented as ↑ energy (1673 kJ) or ↓ energy (280 kJ)) followed by ice cream (good- or bad-tasting)	Good- or bad-tasting milkshake (284 g)	Taste test Good- or bad-tasting ice cream	Counter-regulation only found when preload was perceived as ↑ energy and test meal was good tasting ($P<0·05$)
Ruderman & Christensen[88]	RRS	Median split (13)	Parallel study No preload Preload	Milkshake (amount not stated)	Taste test Ice cream	No significant preload × restraint interaction Restrained eaters ate more after preloading compared with no preload, but not significant
Wardle & Beales[89]	DEBQ	Median split (?)	Parallel study No preload Preload	Milkshake (250 ml, 1840 kJ)	Taste test Ice cream	No significant preload × restraint interaction
Jansen *et al.*[100]	RRS DEBQ	Median split (12) Median split (2·9)	Parallel study No preload Preload	Milkshake (1255 kJ; amount not stated)	Taste test Ice cream	No significant preload × restraint interaction

Polivy *et al.*[81]	RRS	Median split (15)	Parallel study No preload Preload	Milkshake (425 g)	Taste test Ice cream	Significant preload × restraint interaction ($P<0·01$) Restrained eaters counter-regulated Unrestrained eaters compensated but also preload × restraint × self-esteem ($P = 0·057$) (only restrained eaters with ↓ self-esteem counter-regulated)
Lowe & Kleifield[90]	TFEQ	Median split (10)	Parallel study No preload Preload	Milkshake (425 g)	Taste test Ice cream	No significant preload × restraint interaction
Ogden & Wardle[137]	DEBQ	Median split (?)	Cross-over HEP told ↑ or ↓ energy LEP told ↑ or ↓ energy	High- (1255 kJ) and low-(209 kJ) energy drinks (about 150 ml)	Sandwiches and cookies	No significant preload × restraint interaction
Ogden & Wardle[91]	DEBQ	Median split (?)	Parallel study	Mars bar *v.* plain cracker	Taste test Biscuits	No significant preload × restraint interaction
McCann *et al.*[92]	RRS	Median split (24) (obese participants)	No preload One milkshake Two milkshakes	Milkshake (213 g)	Taste test Ice cream	No significant preload × restraint interaction Significant effect of preload ($P<0·01$); participants ate more after the no preload conditions compared with the preload conditions Both restrained and unrestrained eaters counter-regulated (not surprisingly since even unrestrained eaters had relatively-high restraint scores)
Dritschel *et al.*[104]	RRS TFEQ DEBQ	Median split (?) Median split (?) Unrest $<2·75$ Rest $>$mean + 1 SD	Parallel study Water Preload	Milkshake (340 g)	Taste test Biscuits	No significant preload × restraint interaction (regardless of the questionnaire used to measure restraint)
Van Strien *et al.*[103]	RRS TFEQ DEBQ	Restraint was treated as a continuous variable	Parallel study Water Preload	Milkshake (200 ml)	Taste test Ice cream	Ice cream consumption was positively correlated with the scores from the RRS and restraint subscale of the DEBQ
Rotenberg & Flood[79]	RRS	Restraint was treated as a continuous variable	Parallel study Water Preload	Chocolate milk shake (142 g)	Taste test Cookies	The amount of cookies eaten significantly increased as a function of restraint in the preload condition ($P<0·05$)
Ouwens *et al.*[102]	RRS TFEQ DEBQ	Restraint was treated as a continuous variable	Parallel study No preload Preload	Milkshake (400 ml, 1108 kJ)	Taste test Cookies	Cookies consumption was not associated with restraint (regardless of the questionnaire)

DEBQ, Dutch eating-behaviour questionnaire; RRS, restrained revised scale; TFEQ, three-factor eating questionnaire; ?, the exact median split was not stated; ↑, high; ↓, low; HEP, LEP, high- and low-energy preload respectively.

the DEBQ (for men ≥16 and/or ≥3·1 respectively) and unrestrained eaters were defined as scoring ≤12 on the restraint subscale of the TFEQ (eighteen-item revised version) and ≤2·3 on the restraint subscale of the DEBQ (for men ≤11 and ≤1·8 respectively). Participants were randomly assigned to one of two preloads with a similar energy density but double the volume (250 ml and 2092 kJ or 500 ml and 4184 kJ hot chocolate drink) and EI was measured 3 h later at a pasta meal (tomato-based pasta meal with cheese), simulating a second meal effect (C Martins, MD Robertson and LM Morgan, unpublished results). Restrained eaters were not found to counter-regulate in response to preloading; in fact, both groups were observed to compensate.

It can be hypothesized, therefore, that in the absence of 'diet-breaking' food counter-regulation is not expected to occur, independently of the questionnaire used to measure restraint. The results of these two studies suggest a minor role for restraint in predicting disinhibition when a more 'natural' setting is created. The question as to whether to include or exclude restrained eaters from appetite studies, based on their atypical eating behaviour, deserves further investigation and none of the studies reported here can be used to address this issue. Although the present findings may support the inclusion of restrained eaters in appetite studies when 'diet-breaking' foods are not involved, as no counter-regulatory behaviour has been found in these circumstances, restrained eaters may compensate differently, even if they do not counter-regulate. Even though no significant differences were observed between restrained and unrestrained eaters in short-term energy compensation in the studies previously described (unrestrained eaters were found to compensate slightly better than unrestrained eaters in the first study, but the opposite was found in the second study) none of these studies was designed to look at such differences. More research is therefore needed in this area, and also on the impact of restraint on EI in response to exercise, for any firm conclusions to be reached.

The studies described here and other investigations[106,107] suggest that restraint is a spectrum and not a dichotomic variable; restrained eaters do not necessarily show a diametrically-opposite behaviour to unrestrained, but they may show a slightly or significantly different behaviour depending on the characteristics of the sample (levels of restraint and disinhibition) and the study design (type of preload and test meal and questionnaire used to measure restraint). Unrestrained eaters may be seen as 'less-restrained eaters' who present a higher threshold for counter-regulation compared with their restrained counterparts[106,108]. Moreover, counter-regulatory eating behaviour in response to preloading has been shown to be equally as dependent on disinhibition as on restraint levels[98], suggesting that only those scoring high on both restraint and disinhibition should be excluded from appetite studies.

Dietary restraint and abnormal physiology: is there a link?

As previously discussed, restrained individuals may under certain circumstances present an atypical eating behaviour in response to preloading in the laboratory environment. Moreover, in a free-living environment restrained eaters have been shown to consume less energy[109] (although that may be the result of underreporting[110]), take fewer meals and exhibit a higher preference for low-energy and healthy food[109], including fruits and vegetables[111], compared with unrestrained eaters. Interestingly, a link has been suggested between restrained eating behaviour and altered physiological pathways; restrained eating has been shown to impact on metabolic[83,109] as well as endocrine[112–114] functions.

A lower total EE has been described in restrained eaters[115], but it does not seem to be driven by a lower BMR[116]. Dietary restraint seems also to play a role in the magnitude of cephalic-phase reflexes, with restrained women showing larger cephalic-phase insulin[117] and salivary[118] responses compared with unrestrained women. Another study, however, has shown no significant differences in insulin, glucagon or PP cephalic-phase responses to the sham-feeding of a high-fat or low-fat cake between restrained and unrestrained individuals[119].

Increased levels of fasting TAG[109] and NEFA[83] and lower fasting insulin[112,113] and leptin levels, even after controlling for fat mass[120–122], have also been reported in restrained eaters. Moreover, higher levels of cortisol, indicative of increased psychological stress, probably in association with 'eating behaviour', has also been found in some[123,124] but not all studies[111]. The metabolic response to a meal seems also to be affected by the level of restraint. Restrained women have been shown to have a reduced diet-induced thermogenesis[125] and an increased carbohydrate oxidation after a mixed meal[112], as well as a reduced secretion of noradrenaline following a test meal[113]. It has been proposed that this change in fuel oxidation may be related to an increased insulin sensitivity in restrained individuals[112]. Another study, however, has found that high disinhibition rather than high restraint is associated with a lower thermic effect of food[116]. Finally, restraint also seems to impact on the postprandial release of gastrointestinal hormones involved in the control of appetite and food intake, with a blunted cholecystokinin release in response to a meal containing 40 % total energy from fat, reported in restrained eaters[114].

These altered patterns of reduced leptin levels, reduced total EE, reduced ability to oxidize fat and reduced levels and/or a blunted release of satiety hormones may put restrained individuals at an increased risk for weight gain and could explain why they need to cognitively restrict their food intake in order to maintain their body weight. However, it is also possible that this metabolic pattern is a consequence of the acute energy restriction characteristic of restrained eaters[126]. More research is needed in this area to establish more clearly whether the metabolic and endocrine abnormalities reported by some studies in restrained eaters are a cause or a consequence of their atypical eating behaviour.

Although restrained eating has been shown to be strongly associated with measures of adiposity in normal-weight subjects but not obese subjects[111,127,128], the effect of dietary restraint on body weight in the long-term remains controversial. Some studies show a positive

association between dietary restraint at baseline and weight gain 1 year later in women but not in men[129], while other studies show that restraint does not promote weight gain over a 2-year period[127]. The complexity of the association between restraint and adiposity may be explained, at least in part, by the fact that restraint is not a homogenous construct, as previously discussed. It has been shown that while the rigid construct of restraint is positively associated with BMI, the flexible construct of restraint is negatively associated with BMI[128]. Interestingly, higher values for adiposity at baseline have been shown to predict a bigger increase in cognitive restraint 2 years later, suggesting that restraint may be an adaptive mechanism to try to limit weight gain[127].

Similar findings to those described by Burton-Freeman[114] have been observed in relation to PYY, with moderately-restrained individuals presenting lower fasting levels of PYY, at the limit of significance ($P = 0{\cdot}05$), compared with unrestrained eaters (using the middle point of the DEBQ (2·5) as the cut-off point) and a trend towards lower levels of PYY in the postprandial state ($P = 0{\cdot}07$; C Martins, LM Morgan and MD Robertson, unpublished results). However, this study was not designed to look at the effect of restraint on PYY plasma levels[77], and highly-restrained subjects (>3·5 in the restraint subscale of the DEBQ) were excluded from the study, resulting in a very narrow range of restraint. PYY plasma levels have been shown to be sensitive to acute and chronic food restriction[130], to be modulated by gender (with higher levels in females)[131] and also may possibly be modulated by body weight[131] (with lower fasting and postprandial levels in obese subjects[132,133]). These confounding factors are, however, unlikely to explain the differences in PYY between restrained and unrestrained eaters observed in the authors' study, as males:females was similar for both groups and no significant differences were found between groups in body weight, energy or macronutrient intake in the 24 h preceding the study.

One of the limitations of most of the previously described studies that have shown an association between restrained eating behaviour and altered physiology is that they use a median split of the scale as the cut-off point to classify restraint. If restrained eating behaviour is a continuous rather a dichotomic variable[106,107], as previously discussed, this type of approach results in the arbitrary inclusion on either the restrained or unrestrained category of those scoring in the middle of the scale. A better approach would be to exclude those in the middle of the scale and select only those scoring very low or very high in restraint.

In order to overcome these limitations a study has been conducted to investigate the effects of dietary restraint on fasting and postprandial levels of appetite-related metabolites and hormones, subjective feelings of hunger and fullness and EI at a test-meal later in the day, in order to try to establish associations between potential alterations in the secretion of appetite-related hormones and metabolites and changes in subjective and objective measures of appetite in restrained eaters (C Martins, MD Robertson and LM Morgan, unpublished results). Normal-weight men and women were recruited and accepted for the study if they scored either high or low in restraint (cut-off points described earlier). Using a randomized cross-over design participants (twelve men and twenty-one women) were assigned to one of two breakfasts (preloads; a hot chocolate drink) with a similar energy density but double the volume (250 ml and 2092 kJ or 500 ml and 4184 kJ). Plasma levels of glucose, TAG, insulin and PYY and subjective hunger and fullness were measured for a period of 3 h. Participants were then presented with a pasta-based meal and EI was measured. No significant effect of restraint on the postprandial plasma levels of PYY or TAG was found, but glucose and insulin were found to be significantly lower ($P = 0{\cdot}045$ and $P = 0{\cdot}015$ respectively) in restrained eaters compared with unrestrained eaters, independently of the preload. Despite these findings, restrained eaters reported significantly higher fullness ratings ($P = 0{\cdot}033$) throughout the study period compared with unrestrained eaters regardless of the preload. However, this increased fullness in restrained eaters was not found to be reflected in the EI at the pasta meal, which was similar in both groups. Restrained eaters also presented with reduced fasting insulin levels and increased fasting insulin sensitivity (using the HOMA model), independently of the condition ($P<0{\cdot}05$ for all), despite there being no significant differences in body weight or composition between the two groups (C Martins, MD Robertson, LM Morgan, unpublished results).

Lower fasting insulin levels have previously been reported in restrained women[112,113], as well as lower insulin resistance (based on the HOMA model)[112]. However, body weight was found to be a confounder in the latter study[112], which showed increased fasting insulin sensitivity in restrained eaters. The findings of the present authors are in line with and reinforce those of the earlier studies[112,113]. The reason for the lower glucose and insulin plasma levels in the postprandial state observed in restrained eaters in the present study is not known. However, noradrenaline may be involved, as it plays a key role in energy metabolism, i.e. by increasing glycolysis[134] with consequent release of glucose into the bloodstream. Plasma noradrenaline levels have been reported to be lower in restrained normal-weight women after a meal[113], therefore offering a plausible explanation for the lower postprandial glucose plasma levels observed in restrained eaters in the present study.

It was originally proposed that restrained eaters suffer from a weak sensitivity to physiological cues that regulate food intake and an overreliance on cognitive cues[135], experiencing hunger only when very deprived and reaching satiety much later than unrestrained eaters[136]. This model was, however, soon challenged[137], when it was shown that restrained eaters are not less sensitive to internal cues, but do have an increased sensitivity to external cues. The increased fullness in restrained eaters compared with unrestrained eaters after the same meal, previously described in the authors' study, suggests an increased sensitivity to both internal and external cues that regulate food intake in restrained eaters; the two preloads carried both a different cognitive message (one or two mugs of hot chocolate) and generated a different physiological response (2092 and 4184 kJ).

Conclusions

Exercise has been shown to have beneficial effects on short-term appetite control by enabling a more 'sensitive' eating behaviour in response to previous EI. Moreover, it does not appear to prompt any acute physiological adaptations that would lead to an increase in hunger and/or EI in response to increased EE. The beneficial impact of exercise on the EB equation is therefore twofold, not only increasing EE but also modulating EI. These findings provide the foundation for future work in this area and have important implications in terms of the steadily-increasing prevalence of obesity in the UK and the current failure to meet the Department of Health recommendations for PA[15]. Insufficient studies have been performed in this area to draw any firm conclusions about the optimal level of exercise needed to improve appetite control. Moderate PA for 30 min on most, preferably all, days of the week proposed by both the American College of Sports Medicine[13, 14] and the UK Department of Health[15] has been shown to be effective[65] and is therefore at present the best recommendation for the general public in terms of good health. Further studies are needed to elucidate the mechanisms whereby exercise improves short-term appetite control.

Restrained eating behaviour does not seem to impact on PYY plasma levels, but is likely to be involved in glucose metabolism. Despite being associated with increased fullness in the postprandial state, restraint seems to exert a minor role in predicting disinhibition in the laboratory, at least when a less 'artificial' setting is created. More research is needed to clarify the role of restraint on glucose homeostasis and the relevance of the metabolic and endocrine abnormalities reported in restrained individuals to the aetiology of the current obesity pandemic.

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