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Symposium on 'Nutrition: getting the balance right in 2010'

Session 1: Balancing intake and output: food *v.* exercise
The influence of physical activity on appetite control: an experimental system to understand the relationship between exercise-induced energy expenditure and energy intake

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Investigations of the impact of physical activity on appetite control have the potential to throw light on the understanding of energy balance and therefore, upon body weight regulation and the development of obesity. Given the complexity of the landscape influencing weight regulation, research strategies should reflect this complexity. We have developed a research approach based on the concept of the psychobiological system (multi-level measurement and analysis) and an experimental platform that respects the operations of an adaptive regulating biological system. It is important that both sides of the energy balance equation (activity and diet) receive similar detailed levels of analysis. The experimental platform uses realistic and fully supervised levels of physical activity, medium-term (not acute) interventions, measurement of body composition, energy metabolism (indirect calorimetry), satiety physiology (gut peptides), homeostatic and hedonic processes of appetite control, non-exercise activity, obese adult participants and both genders. This research approach has shown that the impact of physical activity on appetite control is characterised by large individual differences. Changes in body composition, waist circumference and health benefits are more meaningful than changes in weight. Further, we are realising that the acute effects do not predict what will happen in the longer term. The psychobiological systems approach offers a strategy for simultaneously investigating biological and behavioural processes relevant to understanding obese people and how obesity can be managed. This experimental platform provides opportunities for industry to examine the impact of foods under scientifically controlled conditions relevant to the real world.

Exercise: Physical activity: Appetite: Energy balance: Body weight

Background

For several years both research and theorising in the area of energy balance (EB) have been dominated by the issue of obesity. The so-called obesity epidemic has influenced funding for research as well as strategic health objectives set by the various governments. Underlying much thinking is the general view that obesity (weight gain) is a condition arising from a mismatch between energy expenditure (EE) and energy consumed. However, the neat simplicity of this statement takes on a different meaning when seen against the complexity of the Foresight Obesity Map. Despite the

Abbreviations: EB, energy balance; EE, energy expenditure; EI, energy intake; FM, fat mass; FFM, fat-free mass; NR, non-responders; PA, physical activity; RMR, resting metabolic rate.

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complicated nature of the interacting determinants that lead to weight gain, the prevailing concept of management is to exert control over dietary intake and physical activity (PA) (eat less, move more, for example). This is the case even though the consensus view seems to be that over consumption rather than under activity has been the major force leading to weight gain and the crisis in obesity[1–3]. It is important to keep in mind when considering this dual-approach that the PA contribution to total EE is approximately 20% to 30% behaviour (resting metabolic rate (RMR) and dietary induced thermogenesis, together make the largest contribution to total EE), but this varies somewhat according to the amount of PA (exercise plus non-exercise activity) achieved. On the other hand, dietary intake is 100% behaviour. This behaviour is called eating. Consequently, behaviour makes a huge contribution to the EB equation (EE *v.* energy intake (EI)). It is also worth noting that although PA and EI are two contributors to EB, they could also interact with each other to influence EB indirectly.

It therefore appears that exerting control over *behaviour* (dietary intake and PA) is the key to managing obesity. However, the behaviours in question are not isolated acts but are expressed as integrated sequences often maintained through a process of learning and referred to as habits. It is known that habitual forms of behaviour are extremely resistant to change[4]. The task of persuading people to change their rigid patterns of behaviour invokes the issues of compliance and ambivalence. Compliance is often far from perfect; people want to change their behaviour (diet or PA), they have an intention to change, but they fail to behave in accordance with their intentions. Therefore, from a public health perspective achieving compliance is the major issue.

However, even if people comply fully with a requirement to increase PA, the theoretical reduction in body weight might not be achieved[5,6]. This is because the 'system' that embodies the processes of EB is an adaptive regulatory biological system. However, the EB system should not be conceived as a simple mechanical balance that automatically responds to additions or subtractions of energy. The system shows autoregulatory adjustments that occur whenever the dietary input or the level of PA is markedly shifted[7,8]. In the case of PA this raises the issue of *compensation*. There is an emerging interest in the various compensatory responses to PA which might partly explain the lower than expected weight loss[9,10]. There is strong evidence to suggest that food intake is more sensitive to manipulations in EI than EE[11,12]. One view of PA is that it is futile for weight loss since it automatically leads to an increase in hunger and food intake that compensates for the energy expended. For example, over 50 years ago it was asserted that ' ... the regulation of food intake functions with such flexibility that an increase in energy output due to exercise is automatically followed by an equivalent increase in caloric intake'[13]. This concept was given support by the findings on workers in the Bengal jute mills[14]. In addition, Edholm held a similar view, arguing that 'the mechanisms that relate intake to expenditure ... are what regulates appetite in fact'[15]. This issue of compensation formed the basis for negative articles in the media that appeared during 2009[16,17] that have been discussed elsewhere[18].

The attention given to compensation is probably appropriate since it can clearly be related to the operation of an actively regulating physiological system (as noted earlier) and is also prominent in the minds of people who undertake exercise for weight control (and possibly in the minds of those who do not). The idea that PA, EE and appetite control are intimately linked leads to the belief that exercise drives up food intake. It is this issue that has been at the focus of a research programme in Leeds for 18 years. The research concept is simple: apply and supervise PA, and measure EI. However, there are many experimental modalities for achieving this, and many forms that the research could undertake. The research has been designed with two considerations: first, to throw light on the processes believed to play a role in EB; and second, to achieve a level of validity that makes the outcome relevant to the management of appetite (and obesity) in the current (unhelpful) environment. Obtaining a realistic assessment of appetite is the key. Since there are many forms that the measurement of appetite can take, it is worth justifying the approach adopted here.

Note on appetite methodology

How appetite is measured under experimental circumstances depends upon the ideological and theoretical stance of the researcher, the resources available and the effort the researcher is prepared to invest to achieve validity. Of course not all research has the goal of contributing to understanding body weight or the control of obesity, but much research is justified by positioning appetite in relation to obesity, dieting and the obesogenic environment. Since a large component of appetite is a form of behaviour, the measurement of food intake suffers from most of the methodological contamination that has plagued measures of psychological and behavioural variables for well over 50 years. Unfortunately, current research is still subject to the limitations and shortcomings of early studies of behaviour and cognitions (thoughts in the head). Some of the weaknesses are as follows.

Use of undergraduate students (*often from psychology departments*)

Well over 50 years ago it was noted that 'The existing science of human behaviour is largely the science of the behaviour of sophomores'[19] with the implication that such participants severely restricted the usefulness of the data collected. Today between 10% and 20% of published studies on aspects of appetite are carried out on undergraduate students, many of whom are obliged to participate in experiments as a course requirement. This practice renders the research purely academic. As was noted 45 years ago 'The widespread practice of requiring students in various psychology courses to participate in a certain number of hours worth of experiments may in some cases permit the generalisation of research findings, at least to psychology students enrolled in certain courses. In many cases,

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however, even this generalisation may be unwarranted' (Rosenthal[20]). This view is reflected in the attitude of certain food companies which, when commissioning research in universities, stipulate that studies should not be carried out on students since the results are not relevant to the majority of consumers and purchasers of food products. The motivation for researchers to recruit undergraduate students seems to be the ease and convenience of getting data which overcomes the effort and trouble required to recruit more meaningful participants. This strategy clearly minimises the relevance of research for understanding social behaviour and motivation in the obesogenic environment[21]. It should be noted also that the European Food Safety Authority in assessing claims about foods, requires that studies have been conducted on the relevant population.

Experimental context

Most studies using students are also characterised by two other features: extremely short duration (often only a few hours) consistent with the needs of the students, and measurement inside specific types of laboratories. Such confines (spatial and temporal) place limitations on the value of the data collected. Often the measures may reflect thoughts (cognitions) or behaviour occurring over the course of a few hours (following the experimenter's instructions) and are contained by the stimulus-free environment. The spatial dimension is important since much of human eating is carried out in a turbulent and invasive obesogenic environment that contains a plethora of stimuli competing for control over the purchase, selection and, finally, consumption of foods. The short time periods covered by many experimental manipulations (usually <1 d and sometimes <1 h) allow only a brief extract of some parameter that is heavily dependent on the immediate contextual state. Given that participants (particularly psychology students) carry with them into the experimental situation their own thoughts, suppositions and beliefs about the meaning of the experiment (what has been called 'Demand Characteristics'[22]), the data collected are likely to be restricted in their meaning to the time and place where they were collected. The laboratory is effectively insulated (by design) from the real environment that influences eating and such data may have little to offer about the operations of thoughts and actions likely to occur in the 'real world'. Of course, laboratory studies are likely to be strong on internal validity (but very poor on external validity) and therefore, give information about specific determinants of the behaviour measured (but of course, only under artificial circumstances).

Experimental contrivance and deception

In order to investigate certain phenomena (often with participants and under circumstances similar to those described earlier) some researchers feel the need to employ deception to dislodge certain postulated motivational determinants of consumption. The deception often carried out in psychological studies (though discouraged by the Ethical Guidelines of the British Psychological Society) sometimes takes the form of deliberately misleading labels about the energy value of foods or beverages, or may employ devices such as the automatic refillable soup bowl. This contrivance or trickery may certainly fool participants into a form of counterintuitive behaviour (data will be collected) but may be the result of attempts to resolve ambiguous and incompatible demands. The interpretation of such outcomes is far from straightforward. While the outcome is usually concluded to reveal the operation of some powerful belief or thought process (cognition) on behaviour, it is only remotely connected to real-life eating under realistic natural circumstances. Also, as noted earlier by Rosenthal, the research findings may only be generalisable to other subjects drinking from refillable soup bowls, or eating soup with a deceptive label.

These notes on appetite methodology have been set down to illustrate the shortcomings of an approach to the measurement of behaviour that were apparent to methodologists over half a century ago and are still being perpetrated in many studies today. While the limitations are not confined to eating behaviour, the combination of extreme brevity, undergraduate 'volunteers', sanitised environments and weird equipment serves to isolate the outcomes from contact with happenings in the natural environment under which the behaviour is naturally expressed. Such studies therefore have very little to contribute to the understanding of obesity and weight regulation. Although they could of course serve as pilot studies and throw light on the mechanistic explanations under the specific limited circumstances of the experimental situation. It also follows that appetite studies alone, in the absence of any measure of energy expenditure (metabolic or behavioural), have limited capacity to make predictions about effects on body weight or obesity.

These methodological notes were also included in response to requests from nutritionists who are often baffled about the conceptual underpinnings of some appetite research and its contribution to understanding nutritional science and the natural eating patterns which most practicing nutritionists confront in their work. The relationship between nutrition and appetite is a sensitive and complicated area, but often made more difficult to understand by the strong rhetoric often attached to psychological studies on appetite. The view of the present authors is that, just as the European Food Safety Authority has stipulated that claims about foods should not go beyond the scientific evidence[23], so claims about appetite control should not go beyond the confines of the experimental circumstances.

Experimental platform to investigate physical activity and appetite control

The current methodological platform used in the University of Leeds has been developed from a series of studies started in 1992, and conducted in collaboration with James Stubbs and Stephen Whybrow of the Rowett Institute of Nutrition and Health at the University of Aberdeen. The approach is based on a number of conceptual principles and is designed to provide a comprehensive and flexible model for the study of energy expenditure and intake.

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Energy balance framework

Understanding the contribution of appetite to weight regulation and obesity can only be fully understood within the framework of EB. This is necessarily so because small changes in food intake occurring during a single meal or over a period of only a few hours may not exert any effect on daily EI and may therefore, not affect EB. Moreover, induced changes in EI via modulation of energy density, which do adjust EB [8], also induce adaptive compensatory adjustments to the regulatory system which oppose the direction of change. The net effect on EB, and therefore on body weight (or body composition) is not simply an effect of the imposed rise in EI but a consequence of EI and EE. This approach has also been promoted by other researchers[24–27]. Therefore, we do not claim that our approach is unique, but it is unusual for appetite researchers. It follows that the approach can only succeed if equal attention is given to the accuracy and precision of measurements of both EE and EI.

Psychobiological system approach

For many years it has been argued that human appetite is the expression of a system[28,29]; an approach that was reflected in the construction of the Obesity Systems Map[30]. In part this represents what Richter termed the behavioural regulation of homeostasis[31]. Since eating clearly reflects demands for energy and nutrients to maintain the integrity and functioning of the bodily system, it is necessarily the case that a major drive for food arises from activity in tissue metabolism and energy utilisation. Such activity is likely to be functionally related to a signal arising from body composition and/or basal metabolism. Moreover, energy expended in PA (reflected in the PA Level as a multiple of RMR) will drive EI (with implications for the impact of volitional PA). It follows that an understanding of appetite can only take place against a background of measures of body composition (at least fat mass (FM) and fat-free mass (FFM)) and of RMR (from indirect calorimetry), since these elements set a minimal level for EI and therefore, exert control over appetite. Such control could be exerted via meal frequency or meal size, or via food selection (directional influence on taste and energy density of selected foods).

Another level of the psychobiological system is the pattern of episodes of eating and the control of this pattern by the Satiety Cascade[32]. Acting jointly, the processes of satiation and satiety are considered to control the pattern of eating. This level of the system requires that the act of eating is measured in a transparent and realistic manner, with the experimental methodology keeping close to forms of natural eating (subject to the need for scientific precision in measurement). The close relationship between the intensity and duration of satiety and the management of ingested food by the gastrointestinal tract requires the simultaneous measurement of key aspects of intestinal physiology. The prominent role of gastro-intestinal peptides in episodic food intake[33,34] means that measurement of specified key peptides (ghrelin, peptide YY, glucagon-like peptide-1 and cholecystokinin) form part of the systems approach together with tonic peptides leptin and adiponectin which are closely related to the amount of adipose tissue. A formula for this approach has been described elsewhere[35]. The Satiety Cascade also incorporates roles for subjective appetite sensations such as hunger and fullness; the four scales commonly used originate from work done three decades ago[36]. In recent years the measurement of these sensations has been subjected to technological improvement including hand-held computers[37] and Personal Digital Assistants[38–40]. A further level of analysis in a systems approach includes enduring dispositions or traits (measured by validated questionnaires) that have been shown to be related to obesity itself or to the tendency to gain weight. Key traits or factors arise from the Three Factor Eating Questionnaire and include restraint, disinhibition and hunger[41]. Disinhibition has been shown to be related to weight gain[42] and has been described as reflecting opportunistic eating[43], although disinhibition also interacts with restraint[44,45]. Other dispositions can be measured with the Binge Eating Scale[46]; the Power of Food Scale[47] and the Craving Inventory[48].

The psychobiological systems approach provides a comprehensive formula for investigating appetite control and EB. The joint assessment of body composition, metabolic factors, gastro-intestinal peptides, patterns of eating, appetite sensations and enduring traits allows the emergence of associations between different domains of knowledge which normally belong to separate disciplines. Within the context of an interaction between PA and appetite, this approach allows for the investigation of the impact of metabolism on behaviour and for the effect of behaviour on metabolism.

Homeostasis and Hedonics: combined influence on appetite

For over 50 years the major influence on the study of food intake arose from the tradition of homeostasis and the concepts of Claude Bernard[49]. This tradition was expressed through early theories of motivation[50]. More recently this ideology has been referred to by the term 'energy homeostasis'[51]. Although there always has been a movement postulating the influence of sensory factors on food intake (mainly food selection) it is within the last decade that it has been strongly recognised that overconsumption in the obesogenic environment is probably driven by hedonic aspects of food rather than by homeostatic principles of biology[52]. It has also focused attention on the hedonic aspects of eating and the influence of reward sensitivity[53], brain reward systems[54] and the functional properties of liking and wanting[55,56]. Given the importance attached to the role of food palatability in overconsumption and the power of hedonic processes, it is important to incorporate this aspect of eating into the systems approach. It then becomes possible to assess the impact of PA on both the homeostatic and hedonic aspects of appetite under identical conditions.

Contextual aspects: participants, time period, nutrients

Given the criticism of appetite methodology in an earlier section, it is important that an experimental platform for

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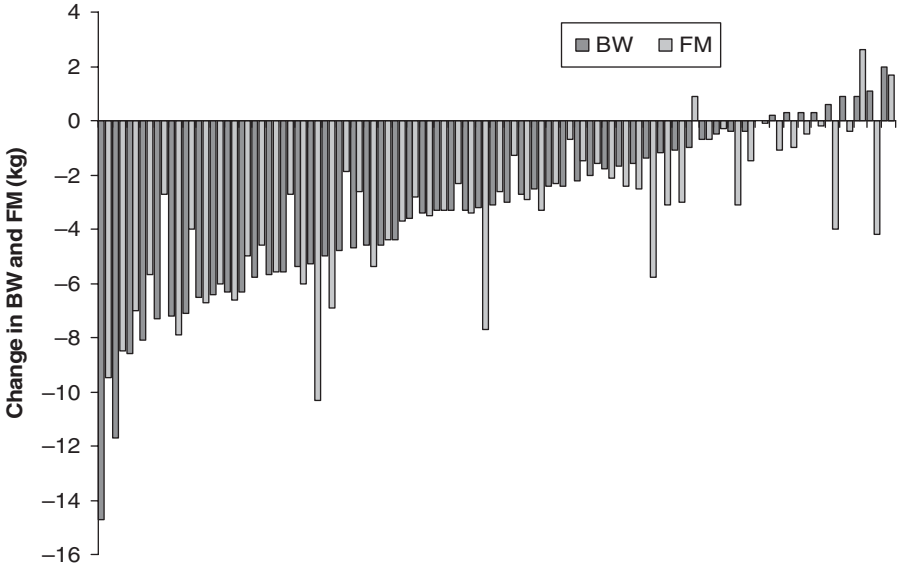

Fig. 1. Individual changes in body weight (BW) and fat mass (FM) following 12 weeks of supervised aerobic exercise in a cohort of fifty-eight overweight and obese individuals (data from King *et al.*[6]).

investigations related to obesity incorporates plausible contextual features. Therefore, an important aspect of the platform is the use of overweight and obese adults drawn from the community. This gives the results greater relevance and applicability (i.e. external validity) to those individuals impacted by the obesogenic environment.

The time period for study should also be realistic in relation to the operations of adaptive metabolism and physiology. Experiments that last only a few hours can only assess transient changes which may have no long-term consequences. We have therefore, designed the platform around a 12-week intervention in which participants engage several days each week with research staff. Over this period, all variables forming part of the psychobiological systems approach are measured periodically. The longitudinal approach allows the emergence of adaptive changes that can be observed in various multi-dimensional layers of the system. An extended time period means that acute effects can be compared with effects emerging over the longer term. This longitudinal approach also allows the investigation of mediators and modulators on the outcome variables; that is, those factors that predict changes and those which are associated with the process of change[57–59].

It is also important that research designed to have a high degree of relevance should reflect certain features of the obesogenic environment, and especially the high-risk factors. This requirement creates a dilemma since comprehensive measurement of the obesogenic landscape is not compatible with accurate scientific measurement. The issue has been partially resolved by recognising energy density as a major (nutritional) aspect of the obesogenic environment and a key risk factor for overconsumption[60,61]. The platform therefore incorporates exposure to high-fat and low-fat probe days to assess the phenomenon of passive overconsumption[62,63] and to assess the impact of this on EB under the influence of PA.

The nature of the experimental platform has been described at some length since it is proposed that this framework provides a plausible model for appetite control (within the context of a psychobiological system and with respect to EB). The platform also respects the complexity of the system (biological, psychological and environmental) that influences weight regulation and the obesogenic nature of the system in which obese people (and the non-obese) are obliged to manage their lives.

Procedural aspects of the platform

Several cohorts of participants have now completed investigations using the platform. The major focus of research has been to evaluate the impact of PA on appetite control, and the consequences of this for body weight. The core procedure involves mandatory supervised sessions of PA of a sufficient frequency (five per week), volume (60 min at 70% VO_{2max}) and period (12 weeks). The platform is therefore not used as a measure of compliance, but is used to assess the effect of PA that has definitely been performed. The details of the procedure have been described in several publications[6,64,65] and will not be described here. However, it is useful to provide a synopsis of certain conclusions that can be drawn from using the extensive experimental platform described earlier.

Outcomes

Individual variability

Figure 1 shows the spectrum of individual changes in body weight and FM that occurs during 12 weeks of supervised exercise. This degree of variability has been observed in three previous cohorts using overweight and obese adult participants[6,64,66]. It is clear that the imposed PA exerted has markedly differing effects in different participants.

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Table 1. Anthropometric variables (mean values and standard deviations) in those participants who gained weight, measured at four time periods across the 12-week exercise intervention (data from King *et al.*[6])

	Body mass (kg)	SD	% body fat	SD	Body fat (kg)	SD	Fat-free mass (kg)	SD	Waist circumference (cm)	SD
Week 0	91·4	9·6	32·3	7·5	29·2	5·7	62·5	12·1	101·3	8·0
Week 4	91·6	10·1	33·0	7·4	29·9	5·7	62·0	12·1	99·7	8·7
Week 8	91·9	9·8	32·8	7·8	30·4	6·0	62·2	11·9	98·6	8·4
Week 12	92·4	10·1	31·7	8·1	28·8	6·3	63·6	12·8	98·8	9·0

Table 2. Anthropometric measures (mean values and standard deviations) at weeks 0, 4, 8 and 12 for a complete cohort including those participants who lost, maintained or gained weight (*n* 58) (data from King *et al.*[6])

	Body mass (kg)	SD	% body fat	SD	Body fat (kg)	SD	Fat-free mass (kg)	SD	Waist circumference (cm)	SD
Week 0	91·1	12·2	34·9	7·8	32·3	9·6	59·2	9·7	101·5	12·4
Week 4	90·0	12·3	33·9	8·3	31·0	9·8	59·3	10·0	99·8	12·2
Week 8	88·8	12·5	33·7	8·4	30·4	9·8	58·8	10·1	98·2	11·7
Week 12	87·9	12·4	32·5	8·3	29·1	9·6	59·2	10·0	96·5	11·8

Indeed this heterogeneity is one of the most striking outcomes and demonstrates underlying differences in the way that the biological system responds to the physiological challenge imposed by the exercise sessions. This degree of variability has been observed 20 years ago in response to both an energy surfeit (imposed by overfeeding)[67] and an energy deficit (imposed by exercise) in groups of twins[68]; however, the phenomenon of individual variability has not been exploited.

Since then it has been emphasised that individual variability in response to nutrition[69] and to exercise[70] is a major outcome variable of experimental interventions. Clearly it can be concluded that when individuals take up PA (of this type, intensity and duration) many different outcomes are possible.

Responders and non-responders

The spectrum of body weight and body composition changes clearly indicate the extremes of the response to an extended PA intervention. Because the cohorts always display such heterogeneity, we have developed an analysis based on the amount of body composition change relative to the amount of exercise-induced EE which in turn allows us to identify responders (R) and non-responders (NR). In essence, R experience changes in FM equal to or more than the expected amount, whereas the NR lose less FM than is expected. The NR show a much greater degree of food intake compensation and increase their daily EI by approximately 1 MJ[6]. This means that at the end of the 12-week programme the difference in the daily EI between the NR and R is approximately 1 MJ/d. This difference in EI is sufficient to account for the changes in body composition observed between the R and NR. These distinct categories of response to exercise suggest the existence of specific phenotypes which are susceptible or resistant to weight loss.

Does exercise induce compensation?

The observation (Fig. 1) that over 50% of participants failed to lose the amount of weight that should have ensued from the amount of energy expended over 12 weeks, indicates that some form of compensatory process is operating[64]. Such compensation, to offset the anticipated weight loss, could arise due to increased hunger and food intake, decreased PA (non-exercise activity) in the non-exercising parts of the day, or metabolic adjustment through RMR. All of these were measured objectively throughout the study. Neither non-exercise activity nor RMR significantly changed, whereas there was an overall significant increase in measured hunger (fasting level and area under the curve) and in daily EI[71]. This indicates that, in general, when people undertake this level of PA, the system does induce a compensatory response; this is primarily via food intake and it is very variable; some people compensate strongly whereas others do not. However, there may also be some diminution in the level of non-exercise activity[72].

Surprisingly, about 15% of participants gained weight over the 12-week intervention, despite the fact that they had actually performed the exercise prescribed. The characteristics of the weight gainers are shown in Table 1. Noticeable is the fact that most of these participants still lost body fat and the weight gained comprised FFM (probably lean tissue). This effect, which has been demonstrated in every cohort that has been studied using this experimental platform, indicates that for some people exercise is an anabolic experience which drives the build up of tissue. The weight gainers also show large increases in food intake in response to exercise.

Body composition and body weight

The effect of exercise on the body composition of the weight gainers represents the strongest effect of exercise on lean tissue. However, considering the entire cohort, it is clear that the overall mean reduction in body weight (approximately 3·3 kg) is accounted for entirely by a reduction in FM while lean mass remains unchanged (Table 2). Consequently, in contrast to weight lost by dietary restriction (which normally reduces both FM and FFM in a proportion of 70:30) exercise preserves lean tissue. This is an important consideration particularly when

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overall body weight change may appear trivial. Recent evidence to support this comes from a study which manipulated PA by imposing inactivity in the form of being confined to a bed for 60 d[73]. The negligible effect 60 d of imposed inactivity had on body weight (0·4 kg) was due to maintenance of FM but attrition of FFM. In PA interventions however, the reverse usually occurs; that is maintenance of FFM and reduction in FM. The loss of FM and the preservation of FFM, strongly implies that body composition is a far more useful measure of the efficacy of PA interventions than gross change in body weight. This implication is enhanced when it is recognised that exercise also leads to a significant reduction in waist circumference (see Table 2). A reduction in waist circumference is an indicator of a lowering of the risk for heart disease[74–76] and is a more relevant marker of health than body weight[71].

Dual-process of appetite control

One major objective for the development of this experimental platform is to allow investigation of mechanisms of appetite control. The physiological impetus created by a significant level of EE can be used to drive changes in appetite processes occurring over the 12-week period. The methodology for the intense probe days comprised a combination of self-selected, self-determined meals together with meals of fixed size accompanied by ratings of hunger and other sensations throughout the whole day. With this arrangement it is possible to measure the strength of an orexigenic drive (fasting hunger together with overall volume (area under the curve) of hunger), the effect on satiation (meal size) and the effect on satiety (post-prandial action measured by the satiety quotient[77–79]. Together these measures indicate a dissociation of processes underlying the expression of appetite. Exercise induces an increase in fasting hunger, but this is significantly greater (and enduring) in NR than R. Hunger is not driven up equally in all participants. Exercise also improves post-prandial satiety reflected by changes in the satiety quotient, and this finding is consistent with previous studies[80,81]. This effect seems to reflect an up-regulation in the sensitivity of the satiety signalling mechanisms induced by the physiological stimulus of exercise; it occurs in R and NR. The experimental platform has therefore revealed two processes underlying appetite control; the overall orexigenic drive and post-prandial satiety[6]. Taken together these effects determine the degree to which an individual will increase food intake in response to exercise which, in turn, contributes to the degree of compensation displayed.

Acute v. *long-term outcomes*

The effects derived from the experimental platform described here (12 week formula within an EB framework) can be compared with a long series of experiments of various durations ranging from 1 d to 19 d which we have conducted over the last 15 years. These studies have shown that a single acute exercise session has little effect on subsequent food intake[11,82–84] and no consequences are observed even in response to a significant amount of high-intensity exercise[85]. These outcomes are consistent with conclusions drawn by Edholm[15] in his classic EB studies, and suggested a loose coupling between EE and EI[86]. However, when our exercise intervention was extended to 7 d in participants observed in a residential unit, a degree of food intake compensation began to occur, but more strongly in women[87] than in men[72]. Subsequent studies under carefully controlled conditions in a human calorimeter demonstrated that for an additional 2 MJ of exercise per day, an average compensatory response of about 0·3 MJ EI was observed after the end of 1 week[7,8]. A subsequent study in which total EE was measured by doubly labelled water over a period of 14 d, EI began to track EE in a dose-dependent manner and reached approximately 30 % of the induced additional EE after 2 weeks[88] but it was very variable. Taken together this series of studies indicate that the outcome of a single acute intervention is not a good predictor of what happens when exercise persists for days or weeks. This is a good example of single acute experiments (of brief duration and carried out within a single day) having little relevance for what happens in the long term.

Exercise and dietary fat intake

The obesogenic environment provides a continuous stimulation for the appetite system and is widely agreed to provoke inappropriate eating and overconsumption. Some researchers argue that the food component of the obesogenic environment is responsible for the obesity epidemic[89]. The food environment is clearly complex and a number of routes to obesity can be envisaged. It would therefore be impossible to incorporate the full panoply of the food environment in any methodological approach; however, the experimental platform allows for systematic nutrition exposures at different times during the 12-week exercise period. Moreover, it has been argued that much overconsumption is not deliberate or intentional, but passive[63]. That is, the consumption occurs inexorably due to the high energy density of the diet. This type of diet (typified by high-fat foods) leads to the rapid intake of large amounts of food energy before satiety signals can operate to prevent the prolongation of eating. This leads to a phenomenon known as the 'fat paradox'[63]. Because of the potency of passive overconsumption, we have currently investigated the effect on self-determined consumption of a full day's exposure to a particular high- or low-fat diet. Interestingly, in obese participants, persistent exercise reduced the intake of high-fat (but not low-fat) foods and therefore, curtailed passive overconsumption[90]. This outcome demonstrates the sensitivity of the experimental platform to detect the effects of dietary manipulations, and indicates a further beneficial effect of exercise, namely, a tendency to reduce the self-selection of meals composed of fatty foods.

Exercise and food reward

Eating is a source of pleasure for most people, and reward plays an important role in the initiation, maintenance and cessation of eating. Food reward is perceived as an acute, conscious, sensation of pleasure or desire to eat but also operates at a level that can influence motivation to choose

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and consume food, without being readily perceived. These distinct components of reward are referred to as 'liking' (explicit evaluation of pleasure) and 'wanting' (implicit motivational forces) and are measured in our laboratory using a psychological tool[56]. By incorporating a measure of liking and wanting into the experimental platform it is possible to examine their role as underlying mechanisms involved in compensatory eating in the acute setting[91] and in resistance to weight loss in the longer term. We recently examined the effect of an acute bout of exercise on liking and wanting for foods in overweight and obese participants, before and following the 12-week exercise schedule[92]. We found that those who experienced an immediate post-exercise increase in liking for food and more specifically an increased wanting for high-fat sweet foods also demonstrated the least fat loss after 12 weeks. This acute response was not attenuated by regular exercise (or change in body composition) suggesting it is a strong habitual trait. The implication is that the effect of acute exercise on food reward is more implicit when it comes to high-risk foods and that vulnerable people are largely unaware of the influences determining the degree of compensatory eating. Furthermore, the results demonstrate that the tool can be used to identify hedonically susceptible individuals which in turn may help to optimise strategies for weight control with dietary modification or pharmacotherapy.

Overview

The psychobiological systems approach has demonstrated a potential to describe relationships between domains of biology and behaviour not normally measured simultaneously; it has already disclosed novel processes that influence the behavioural output of the human system under the challenge of persistent supervised exercise. The data presented earlier have provided a sample of the published outcomes that have been generated by the platform. Other features (not described here) include the influence of enduring traits such as disinhibition and binge eating on exercise-induced weight loss[93,94] and the determining effects of substrate oxidation on fat loss[95]. The experimental platform is versatile and can be adapted to evaluate the influence of other interventions superimposed upon the basic formulation. Of particular importance for research and development in the food industry is the potential to assess additional dietary manipulations (e.g. high *v*. low protein exposures) and to evaluate the response to general types of foods (or individual food products) inserted into the nutritional environment. This feature of the experimental platform can therefore provide a useful resource for food companies to monitor the effect of novel foods on human appetite and EB under realistic conditions that are relevant for overweight and obese people struggling to manage their body weight within an unhelpful obesogenic environment.

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