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Eating habits and appetite control: a psychobiological perspective

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An individual's eating behaviour is shaped by factors ranging from economic conditions and cultural practices to biological influences. The physiological system controlling appetite appears to be adapted to solving the problem of an unevenness of food supply across time, and is fairly permissive in its response to undereating and overeating. Consequently, when food is abundant, the diet is energy dense and energy expenditure is low, there is a strong tendency to become obese (i.e. obesity is better viewed as due to a 'toxic' environment than to faulty physiological control of appetite). Under such conditions the most common method of avoiding obesity is through the cognitive control of eating. However, dietary restraint and dieting are demanding tasks, and are associated with psychological costs, including significant impairment of cognitive performance. Restraint is also prone to disinhibition, with the result that it can sometimes undermine eating control, even leading to the development of highly disordered eating patterns. In part, these difficulties are due to the self-perpetuating nature of dietary habits: for example, hunger tends to be diminished during strict unbroken dieting, but increased in individuals having a highly variable eating pattern (such as occurs when eating is frequently disinhibited). These features of appetite control provide both barriers and opportunities for changing behaviour. Accordingly, there is a need for future research to focus on the psycho-social factors and the dieting practices predicting successful eating and weight control, with the objective of identifying the actual cognitive and behavioural strategies used by the many dieters and restrained eaters who are able to achieve weight loss and maintain long-term weight stability.

Appetite: Satiety: Obesity: Dieting: Cognitive function

In theory and practice in the fields of nutrition, dietetics, medicine and psychology it is common to hear the argument that human eating behaviour is shaped by many factors, ranging from physiological to cognitive and socio-cultural influences. At the same time it has to be said that researchers, in particular, generally adopt a highly focused approach in the work that they actually carry out. The present paper attempts to present a broader perspective on eating habits and appetite control, in which cognitive and learned influences on behaviour are integrated with biological mechanisms contributing to energy balance. This is discussed primarily in relation to the control of energy intake and the problem of overweight and obesity, although the principles are also relevant to food choice. A more detailed account of the main arguments can be found in Mela & Rogers (1998).

Some biological background

Homeostasis and the control of appetite and body weight

An assumption of most biological perspectives on eating behaviour is that appetite is controlled by a homeostatic system that serves to maintain energy and/or nutrient balances. Such homeostatic models of motivation use ideas derived from engineering control theory describing regulatory systems capable of maintaining relatively stable states.

Abbreviation: DEBQ, Dutch eating behaviour questionnaire.

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Very often these models incorporate a set point and negative feedback (Toates, 1986), and a textbook analogy for such a system is the regulation of room temperature by a thermostatically-controlled heater (Carlson, 1994).

Set-point models can account for the maintenance of energy balance and the observation that in adulthood there is rather little long-term variation in body weight; but other evidence is clearly inconsistent with the existence, in a strict sense, of a set point for body weight or body fat. Crucially, certain simple dietary manipulations can markedly affect energy intake and body weight. Although these studies have been carried out on non-human animals, mainly rats, the results are clearly relevant to human eating behaviour and obesity. When switched from a standard laboratory diet to a high-fat diet or to a 'cafeteria diet' (consisting of a variety of foods such as bread, chocolate, cheese and breakfast cereals) rats overeat and can become markedly obese. The main features of these diets that promote the overeating and obesity appear to be their palatability, variety and macronutrient composition (for example, see Sclafani, 1980; Louis-Sylvestre *et al.* 1984; Rogers & Blundell, 1984).

While dietary-induced obesity contradicts a body fat set-point model, it is not inconsistent with a negative feedback effect of body fat on appetite. Further results show that as body weight (fatness) increases in cafeteria-fed rats there is a decline in food intake, until a point is reached at which a new stable weight is maintained. Also, on return to the standard diet there is undereating and at least partial reversal of the weight gain (for example, see Rogers, 1985). These findings indicate a reduction in appetite with fattening which, physiologically, could be due to the increase in fat mobilization or a related signal that changes in proportion with the accumulation of body fat (Friedman, 1991). Accordingly, some authors have used the term 'settling point' to characterize the control of body weight (Wirtshafter & Davis, 1977; Pinel, 1993), the idea being that body fat appears to be regulated around a point at which the various external and internal factors that influence its level achieve equilibrium. Computer simulations with such models suggest that only a small background influence feeding back to suppress food intake is sufficient to provide a marked long-term stabilizing effect on weight (Booth & Mather, 1978).

It should also be noted, however, that even without negative feedback inhibition of appetite, changes in body weight (fat) tend to be self-limiting. This is because changes in body mass, including fat content, are accompanied by increases or decreases in energy expenditure due to changes in both resting metabolic weight and the energy cost of moving the body. Thus, a sustained increase in energy intake without any change in physical activity would lead to a self-limiting weight gain; the initial rate of weight gain progressively diminishing until a new asymptote is reached. As a contributor to body weight stability this 'passive feedback' influence is far from trivial (Van Itallie & Kissileff, 1990; Mela & Rogers, 1998).

A predisposition to become obese

Consideration of human dietary prehistory indicates that over the past tens of thousands of years our ancestors have subsisted on a wide variety of diets (Garn & Leonard, 1989). It is not possible, therefore, to be precise about how the nutritional composition of the 'natural' human diet (i.e. the diet to which modern human subjects are genetically adapted) compares with the contemporary Western diet. Nonetheless, it is fairly certain that we are descendants of omnivores, and that throughout much of our past cyclic food shortages and famine were commonplace (Brown & Konner, 1987; Garn & Leonard, 1989). In turn, the problem of obtaining enough to eat appears to have been significant in shaping human biology. For example, fossil and archaeological evidence together with anthropological studies point to the tendency of human subjects to gorge when the opportunity arose. This is characteristic of the hunter–gatherer lifestyle, where the subsistence component of the diet is of low to moderate energy density, supplemented relatively infrequently with highly-prized energy-dense foods (O'Dea, 1992). The capacity to store such excess intake efficiently into body fat which could then be mobilized during periods of food shortage would clearly provide a key survival advantage. Indeed, adipose tissue has an impressive ability to store large amounts of energy efficiently; it can expand to an enormous size, and compared with other body tissues it is highly energy dense. Subsequently, these biological and behavioural traits are likely to have been maintained or even selected for more strongly in the early agriculturists (Garn & Leonard, 1989).

The conclusion from these arguments is that human subjects (and other mammals) have evolved a predisposition to become obese. However, the behavioural, metabolic and anatomical traits which favour overeating and the storage of surplus energy in the form of body fat in times of plenty conflict with relatively very recent human socio-cultural and economic developments. In modern industrialized societies food availability rarely limits intake, and the prevalence of obesity is high and constitutes a major public health concern. Coupled with this are changes in the energy density and nutrient composition of the diet and in levels of energy expenditure, which further encourage the development of obesity. O'Dea (1992) has characterized the changes in food intake patterns from human prehistory to the present as a progression from feast and famine for the hunter–gatherer, to subsistence and famine for the early agriculturists, and finally to continuous feast for people from modern Westernized societies.

Because excess nutrient intakes are dealt with by metabolic transformation and storage (Frayn, 1996), the appropriate way to view the goal of nutritional behaviour is in relation to the anticipation of needs and the long-term sufficiency of nutrient supplies, rather than in terms of the maintenance of energy balance. Within this framework the next section discusses physiological and learned influences controlling eating from one meal to the next.

Physiological and learned influences on appetite

Why eating starts

As suggested previously, theories of appetite control have traditionally focused on the individual's internal state as the primary motivating factor for eating, a 'hunger drive'

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which increases in proportion to bodily need and is reduced by food intake. It is clear, however, that a simple depletion–repletion model of motivation cannot adequately account for eating behaviour. At the very least it is also necessary to consider the motivational effects of the presence of food, and the quality or palatability of that food, and the effort required to obtain it, which additionally influence the decision to eat and how much is eaten (for example, see Toates, 1986).

'Depletion' is, nevertheless, an important stimulus for meal initiation. This is demonstrated, for example, by observations on the relationships between meal size and inter-meal interval, which can be accounted for very well by models based on estimates of stomach capacity and emptying rates, or energy flows from the intestines (Booth, 1978; de Castro, 1981, 1988). Put another way, it can be said that the initiation of eating is increasingly likely to occur as the inhibitory effects of repletion dissipate, in which case hunger equates to the absence of satiety (see Stricker, 1984). While there is perhaps no real distinction here, this avoids the use of the term 'depletion of reserves', and emphasizes the fact that a dominant influence on satiety (absence of hunger) is the amount and composition of the food consumed in the previous meal. Note that the energy economy of the whole body probably has little impact in the short term, because for adult human subjects depletion from one meal to the next is typically very small compared with total bodily energy reserves (Mela & Rogers, 1998). The level of these reserves (body fat content) does exert an influence on appetite, but as discussed earlier (p. 60) the action of this relatively weak negative feedback effect will only be apparent over the longer term.

Experience strongly suggests that hunger can be aroused by external cues. For example, our appetite is stimulated by someone offering us an unexpected treat, or it can fade in the afternoon even though we were too busy to eat lunch. As well as the obvious effects of the sight and smell of food, eating can be motivated by learned contextual cues such as location and time of day. This has been confirmed experimentally in studies showing that arbitrary external stimuli previously associated with food consumption reliably stimulated eating in the absence of immediate deprivation or apparent nutritional 'need' (Weingarten, 1983; Birch *et al.* 1989).

A question arising from these observations concerns the specificity of the effects of external stimuli conditioned to eating. One possibility is that exposure to such stimuli triggers physiological responses in preparation for eating, including salivation, insulin release and gastric acid secretion (the so-called cephalic phase of digestion), the consequences of which feed back to the brain where they are interpreted as an internal signal for hunger. Against this, however, is the finding that blockade of cephalic-phase responses (with atropine) does not disrupt the initiation of eating in response to learned cues (Weingarten, 1984). Alternatively, rather than a general state of hunger, the presentation of stimuli which have become associated with consumption of a food may elicit a desire to eat that specific food (Weingarten, 1985). An analogy for this question is to consider visiting the cinema where in the past you have usually eaten popcorn. What is the effect of exposure to this eating-related setting? Does it trigger a general feeling of hunger, or a specific desire to eat popcorn? Recent results from studies on rats do indeed suggest that eating-related stimuli have specific effects on food choice (Day *et al.* 1998).

Another issue concerns the possibility that specific appetites might also become conditioned to salient internal stimuli, for example, accompanying particular emotional states. Actually, this would probably involve associations formed between eating and a configuration of both internal and external stimuli evoking the emotional response (Robbins & Fray, 1980; Wardle, 1990; Booth, 1994). Such a mechanism might underlie mood- and stress-induced eating, and in turn help explain effects of mood on eating at an individual level, since these relationships would be shaped according a person's own unique learning history. Furthermore, if specific appetites are based on learned associations, then presumably they can be unlearned (i.e. extinguished), for instance, through unreinforced exposure to the context in which the appetite or 'craving' is experienced. This technique, which is called cue exposure, has shown some success in the treatment of binge eating in bulimia nervosa (Jansen *et al.* 1992).

Why eating stops

During a meal, eating is controlled moment-to-moment predominantly by the oro-sensory and post-ingestive effects of the food consumed. These influences can be modelled as positive and negative feedback respectively; the overall contribution of the effects in the mouth usually being stimulatory, with the net effect of the entry of food into the stomach and intestines being inhibitory (Smith *et al.* 1990; Rogers, 1993).

The positive feedback is the stimulation of eating by eating, and its strength is influenced by food palatability (for example, see Yeomans, 1996), which is defined as the individual's hedonic or affective response to the taste, flavour, texture, etc. of a food or drink (Rogers, 1990). Palatability, in turn, is determined both by innate responses (e.g. there is an inborn liking for sweet tastes and dislike of bitter tastes) and learning. The latter occurs through association of the oro-sensory and post-ingestive effects of eating and drinking. For example, animals rapidly learn to avoid food when consumption of that food is paired with nausea and gastrointestinal discomfort (Garcia *et al.* 1974), and the same basic Pavlovian conditioning processes appear to underlie food aversions acquired by human subjects (Bernstein, 1994). As a result, strong aversions are sometimes formed despite the person's awareness that the food did not cause the illness. Similarly, the positive after-effects of food ingestion can increase liking for a food. This has been demonstrated convincingly by Sclafani and his colleagues (see Sclafani, 1995) who reported that rats acquired strong preferences for flavours paired with intragastric starch infusions. Other results suggested that these changes in preference were indeed due to increased palatability of the starch-paired flavour. Studies on human volunteers have confirmed significant reinforcing effects of carbohydrate, fat and protein (for example, see Kern *et al.*

1993), as well as increased liking for flavours paired with the consumption of caffeine and alcohol (for example, see Rogers *et al.* 1995).

Note that palatability is not the same as food preference. Consider a person choosing between margarine and butter; she may eat more margarine because of price or perceived health benefits, but she may like the 'taste' of butter more than that of margarine. As measured by the amount consumed she would show a preference for margarine, although on a hedonic measure butter would score higher. Thus, although preference will be affected by palatability, it can also be strongly influenced by cognitive factors. Palatability, nonetheless, plays a central role in motivating eating behaviour. It guides the choice of foods in relation to their biological utility, and acts to stimulate and maintain consumption during a meal through a positive feedback effect on eating.

The operation of negative feedback during normal eating is demonstrated, for example, by the results of studies on sham feeding. Rats fitted with a chronic gastric fistula eat (drink) vastly increased amounts when the liquid food is allowed to drain out of the open fistula, compared with when the fistula is closed (Smith *et al.* 1974). In other words, satiety does not occur if ingested food fails to distend the stomach or enter the small intestine, thus excluding taste, other oral stimuli, pharyngeal and oesophageal movements, and the contact of food with the gastric mucosa as potent stimuli for satiation. Although the sham-fed rat does eventually stop eating, this is likely to be due to fatigue or the effects of the digestion and absorption of a portion of the ingested nutrients (Sclafani & Nissenbaum, 1985). Other evidence shows that the post-ingestive and post-absorptive effects of food ingestion which influence meal size and the maintenance of satiety in the post-meal interval include filling of the gastrointestinal tract, release of regulatory hormones such as cholecystokinin, insulin and glucagon, and the detection of nutrients absorbed into the systemic circulation (for example, see Forbes, 1988; Mela & Rogers, 1998).

Results from sham-feeding experiments also show that learning contributes to the control of meal size. A large increase in meal size is not seen on the first occasion that rats are sham fed. Instead there is a suppression of eating during initial sham feeds due to learned satiety (Weingarten & Kulikovsky, 1989). During normal feeding the visual and oro-sensory properties of the food become associated with the post-ingestive effects experienced, and this provides anticipatory control of meal size. On the first sham feed, meal size is modulated according to the 'expected' post-ingestive effects, but with continued sham feeding this learning extinguishes and meal size increases. The learned control of meal size by flavour cues has also been demonstrated in studies on human adults and children (Booth *et al.* 1982; Birch & Deysher, 1985). Potentially, the learned anticipatory control of meal size is a refinement which could help overcome the problem posed by the delay between the moment of eating and the major post-ingestive and post-absorptive effects of food (Booth, 1977), although this mechanism might be undermined when meals are composed of many tastes and flavours. There is additionally the problem that there is not always a consistent relationship between the oro-sensory characteristics and the energy content of foods or drinks. For instance, a food sweetened with an intense sweetener can have a much lower energy content than the similar tasting food sweetened with sugar. Furthermore, because an empty plate is a strong cue for satiety, it is clear that decisions about meal size are often made in advance of eating.

It should be recognized that the various effects of eating described previously have different but overlapping time-courses. Food intake can be seen, therefore, as triggering a cascade of events, some of which will exert inhibitory influences primarily during eating, thereby bringing the meal to an end, while others will be more important in maintaining satiety in the post-meal interval (for example, see Stricker & Verbalis, 1987; Blundell & Rogers, 1991). For example, at the time lunch is started most of the food eaten at breakfast will have emptied from the stomach. Satiety during this inter-meal interval is maintained by the effects of food in the intestines, and then increasingly by the continued action of absorbed nutrients. Finally, perhaps when the flow of nutrients from the intestines reaches a certain minimum, events such as a transient decline in blood glucose level may signal the end of satiety (Campfield *et al.* 1996). Usually, however, the timing of the next meal will not be determined simply by internal signals, because the opportunity to eat is often constrained or stimulated by external factors (see p. 61). Under these circumstances the amount eaten in the previous meal will exert its influence mainly on satiation in the current meal.

In relation to dietary influences on eating and obesity, a critical question is what characteristics of food influence satiation and satiety, or determine satiating efficiency (i.e. the degree of inhibition of eating per kJ consumed). These characteristics include macronutrient composition, energy density, physical properties such as osmolarity, viscosity and particle size, and palatability (for example, see Kissileff & Van Itallie, 1982; Holt *et al.* 1995; Prentice, 1995). Current evidence points to energy density, normally determined primarily by the fat content of the diet, as the dietary factor having the greatest effect on long-term food intake and energy balance (Mela & Rogers, 1998).

The cognitive control of eating

Externality and dietary restraint

Against the background of imprecise physiological control of energy intake in relation to expenditure, there has been a recognition of the critical role played by dieting and dietary restraint in influencing human eating behaviour. Weight loss or the avoidance of weight gain can be achieved through the deliberate control of food intake. Indeed, an individual's preferred weight, shape, waist size etc. (derived from cultural norms) can be viewed as a 'cognitive set point' (Booth, 1978). Deviations from this set point are detected when the individual notices, for example, a change in fit of their clothes or an increase in measured weight, causing her or him to try to eat less in an attempt to eventually restore weight to the desired level. However, as discussed later (pp. 63–64), dieting can sometimes undermine successful eating control, and it is also a significant psychological stressor.

Research on dietary restraint originally developed as a result of studies designed to test the so-called externality theory of obesity (Schachter, 1971). This theory proposed that obese people are more reactive to external food-related cues, and less sensitive to internal hunger and satiation cues than lean individuals. It was further argued that externality is a predisposing factor in the aetiology of obesity; high external responsiveness predisposes individuals living in environments where food is abundant and highly palatable to overeat and, therefore, to gain excessive weight.

Subsequently, however, it was suggested that the poor control of eating displayed by obese subjects in laboratory tests, such as overeating palatable foods and failing to adjust their intake in compensation for food 'preloads', is linked to dieting rather than externality (Nisbett, 1972; Herman & Mack, 1975; Herman, 1978). The preload was a fixed amount of food that subjects were required to eat as part of their participation in the experiment. Classifying individuals according to their degree of dietary restraint, measured by the revised restraint scale, was found to predict a very striking phenomenon, i.e. highly restrained subjects actually ate more instead of less following a food preload. This 'counter-regulatory' behaviour was interpreted in terms of a process of disinhibition. The preload, by forcing the perceived intake of energy above a critical threshold or 'diet boundary', causes normally restrained eaters to suspend their self-imposed restraint, thereby releasing their underlying desire to eat. Other disinhibitors of eating in restrained eaters, including emotional events, the consumption of alcohol, the behaviour of others and even anticipated future overeating, have also been identified (Ruderman, 1986). Partly on the basis of these results it was argued that restrained eating is a direct precursor of binge eating and bulimia nervosa (Polivy & Herman, 1985).

Psychometric analysis of the questions making up the revised restraint scale showed that these measure principally 'concern with dieting' and 'weight fluctuation' (Wardle, 1986), and in later research two other questionnaires, the three-factor eating questionnaire (Stunkard & Messick, 1985) and the Dutch eating behaviour questionnaire (DEBQ; van Strein *et al.* 1986), were developed with items which relate more directly to restrained eating and the conscious restriction of food intake. Crucially, individuals scoring high on the three-factor eating questionnaire and DEBQ were found to be relatively less susceptible to disinhibited eating, leading to the suggestion that the revised restraint scale tends to identify unsuccessful dieters, whereas the three-factor eating questionnaire and DEBQ restraint scales largely identify successful dieters (Heatherton *et al.* 1988; Lowe, 1993; Mela & Rogers, 1998). An explanation for this predictability of successful *v.* unsuccessful dietary control is that these outcomes are due to certain self-perpetuating patterns of eating behaviour.

This is illustrated by a study that investigated food-induced salivation as a function of two extreme styles of 'dietary restraint', i.e. strict and unrelenting dieting exemplified by a group of restricting anorexic patients, and variable dieting exemplified by a group of bulimic patients (LeGoff *et al.* 1988). These participants were asked to identify various odours with their eyes closed. Compared with age-matched control subjects, anticipatory salivation to food odours, but not to non-food odours, was reduced among the anorexics and exaggerated among the bulimics. Additionally, the anorexics reported lower levels of hunger. Two further results were also very revealing. First, when the anorexic and bulimic patients' food intake patterns were to a large extent normalized after 60 d of intensive in-patient treatment, the differences in salivation and hunger responses disappeared or were markedly reduced. Second, an analysis which pooled data from all the anorexic, bulimic and control subjects showed a strong positive correlation between energy variability (i.e. 'variability of the energy content of meals') and food-induced salivation.

LeGoff *et al.* (1988) conclude that these different appetite responses are a direct consequence of the different eating patterns adopted by restricting anorexic and bulimic patients. The explanation for the reduced anticipatory salivation and hunger associated with the unrelenting anorexic style of dietary restriction is that these conditioned responses have been extinguished, because typically little or nothing is consumed on occasions when food-related stimuli are present (Herman *et al.* 1981; LeGoff *et al.* 1988). This implies that, despite their undernourished weight, the presence of food and food-related stimuli will have a relatively weak stimulatory effect on appetite for such individuals. Paradoxically, therefore, they may experience reduced rather than enhanced appetite as dieting progresses, which will then contribute to further restriction of eating. This conclusion is consistent with the view that external stimuli conditioned to eating play a major role in the control of appetite (see p. 61), and also with the results of studies on the effects of weight-reducing diets showing that, although hunger and food-induced salivation are increased following short-term food deprivation, after longer-term food restriction these responses are more likely to be diminished (for review, see Mela & Rogers, 1998). In contrast, appetite may be enhanced in bulimic individuals, particularly in situations where overeating has occurred in the past, again with the result that the behaviour tends to become self-perpetuating (for example, see Jansen *et al.* 1992).

Although anorexia and bulimia are characterized by pathological patterns of eating behaviour, in less-extreme forms such patterns are probably typical of the behaviour of restrained eaters and dieters. Furthermore, these different eating styles would appear to be selected predominately by respectively the DEBQ and three-factor eating questionnaire restraint scales *v.* the revised restraint scale, and an important factor underlying less successful restraint is high eating variability. The prediction is that individuals with highly variable eating patterns will encounter cues associated with eating significantly more frequently than individuals with relatively invariant eating habits. As consequence, they will experience greater levels of hunger and will need to exert greater effort to restrain their eating.

Dieting is cognitively effortful

Recent research has shown that, in addition to its impact on the (dys)control of eating patterns and food intake, there are broader effects arising from self-imposed dietary restraint. For instance, dieting is associated with depression, and

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some evidence implicates altered brain serotoninergic function during dieting as a possible mechanism for this effect (Goodwin *et al.* 1990; Cowen *et al.* 1992). Current dieters have also been found to display relatively impaired cognitive performance (Green *et al.* 1994; Green & Rogers, 1995). These impairments included slowed reaction time, poorer immediate memory and poorer vigilance performance. On a cognitively undemanding tapping task, however, dieters tended to perform somewhat better than non-dieters, suggesting that the differences were not due simply to lowered motivation in the dieters. Furthermore, because the same individuals showed impaired performance when they were dieting compared with when they were not dieting, it appears that this is related to dieting *per se* rather than to pre-existing differences between dieters and non-dieters.

A further result from these experiments (Green *et al.* 1994; Green & Rogers, 1995) was that individuals reporting high dietary restraint (DEBQ) but who were not currently dieting to lose weight performed at a level intermediate between dieters and individuals with low restraint scores. This would be consistent with an effect of energy restriction as the primary cause of the poorer performance (Laessle *et al.* 1990); however, other evidence suggests that this is not the case. For example, short-term food deprivation (missing up to three consecutive meals) was found not to have significant effects on cognitive efficiency (Green *et al.* 1995) and, crucially, current dieters performed poorly even in the absence of weight loss. A more likely explanation is that the impaired performance of dieters is related to greater 'distractibility' due primarily to their increased preoccupation with thoughts about food and weight. Support for this possibility comes from several rather separate lines of investigation (for example, see Green & Rogers, 1993; Herman *et al.* 1978), and is consistent with a theory originally proposed as an account of aspects of drug use and drug craving (Tiffany, 1990, 1995).

Tiffany (1990, 1995) proposes that drug use is largely controlled by automatic processes and involves no significant urges or cravings, except when drug use is prevented or resisted. Drug use, and in the present case eating, is viewed as the performance of a series of cognitive and motor tasks which with repeated practice become increasingly effortless, efficient and stimulus bound (i.e. triggered by external cues), and which are carried out with little or no awareness. It is generally acknowledged that this development of automatized skills regulates most 'routine' activities, including complex skills such as driving a car. Further characteristics of automatized skills are that their performance is cognitively undemanding, they are capable of being initiated and completed without intention, and they are difficult to impede. In relation to drug use, this theory perhaps applies best to the regular cigarette smoker, for whom strong urges or cravings do not usually precede lighting and smoking a cigarette. Equally, on most occasions, eating is not accompanied by cravings or intense hunger or desire to eat, and it would appear to involve minimal non-automatic cognitive processing, so that even the choice of foods and initiation of eating requires little cognitive effort. Crucially, however, for the individual who is dieting to lose weight, or who is aware of having to restrain their eating in order to avoid gaining weight (or the smoker who is attempting to 'give up the habit'), attempting to resist eating (or smoking) activates non-automatic processing and accompanying behavioural and emotional responses, including reports of cravings and urges to eat. In other words, Tiffany (1990, 1995) views the processes controlling cravings and urges as largely separate from the proximate cause of drug use. This is similar to the proposal that chocolate is the most frequently craved food because it is the food that people most often try to resist eating (Rogers, 1994). A specific appetite for chocolate may be triggered by external cues or emotional states previously associated with eating chocolate (see p. 61), but ambivalence towards chocolate ('nice but naughty') leads to attempts to restrict intake, only to cause the desire for chocolate to become much more prominent and intense.

One of the predictions of Tiffany's (1990, 1995) theory and its extension to eating is that restraint will significantly disrupt cognitive functioning, because thoughts about, for instance, food, eating and weight will interfere with concurrent tasks also requiring non-automatic processing. This would provide an explanation for the impairment of cognitive performance during dieting, if it is assumed that dieters show a sustained increase in preoccupation with these issues; however, it can be expected that there will be an even greater impact when the dieter or restrained eater is attempting to cope with eating-related situations. This was observed in a recent study (Green *et al.* 1999) in which female participants were tested on a simple reaction time task, once while they imagined their favourite food and once while they imagined their favourite holiday. Current dieters and highly-restrained non-dieters (restraint measured by the DEBQ) displayed very substantially slower reaction times than low-to-medium-restrained non-dieters in the former situation, but not in the latter situation. There were no differences in self-reported vividness with which participants from the different groups imagined the food and holiday scenarios, indicating that performance was disrupted due to the intrusion of thoughts about restraint rather than to more elaborate visualization of the food by the highly-restrained eaters and dieters. These thoughts might have included concerns about, for example, the energy content of the food, how the food might threaten the diet, and what coping strategies could be used to avoid eating the food. Taken together, these various results strongly suggest that dieting and dietary restraint are cognitively effortful, and that consequently they can have a significant detrimental effect on psychological well-being.

Integration of influences on eating behaviour

The present review has focused on some of the basic psychobiological processes controlling appetite. In turn, these processes operate within social, cultural and economic contexts to shape the individual's experiences with food through constraints on food selection and eating behaviour. Thus, cultural forces are the major determinants of cuisine and food attitudes (Rozin, 1996). Additionally, there are many other psychological and environmental factors affecting eating which were not discussed previously. There are, for example, significant weekly and seasonal variations in food intake (de Castro, 1996). There is also strong

social facilitation of eating. Recent studies have found that individual intake is much greater in meals eaten with company than in meals eaten alone, and furthermore that intake increases as the number of other people increases (Redd & de Castro, 1992; Clendenen *et al.* 1994).

How then amidst the complexity of these external influences is eating regulated to achieve longer-term energy balance and stability of body weight, and what does this reveal about the causes of obesity? Physiological stabilizing factors include an inhibitory effect of body fat and recent energy intake on appetite, and a 'passive feedback' effect due to the greater energy cost associated with increased body mass. There are also limitations placed by the physical capacity of the digestive system to accommodate and process food. Nevertheless, it is clear that the physiological system does not exert precise control over energy intake in relation to expenditure. Indeed, the ability in times of plenty to 'overeat' and store the excess energy as body fat is an adaptive trait, but one which predisposes the susceptible individual to harm in environments where energy-dense food is always in surplus. Against this background, conscious dietary restraint and micro-environmental influences (i.e. short-term influences acting on the individual) have a major impact on eating, so that food intake may vary very substantially across the day, from day to day, and even in the longer term. Crucially, however, when viewed over sufficiently long periods of time their net effect will be constant (de Castro, 1996). This is because exposure to these external influences is regulated by the individual's lifestyle. It follows, therefore, that significant changes in adult body weight will tend to be associated with changes in lifestyle, such as occur when leaving home for college, after marriage or retirement, or when changing jobs (see Rodin & Slochower, 1976). Eating and body fatness can also be altered intentionally, through methods ranging from surgery (for example, gastric stapling), jaw wiring and the use of appetite suppressant drugs, to the most common method of self-imposed dietary restraint.

Attempts to increase dietary restraint and to significantly change eating habits frequently fail to achieve their goals. In part, this may be due to individuals having inadequate nutritional knowledge, misperceptions of their own diet, or exaggerated beliefs about the extent of dietary change already undertaken (Lloyd *et al.* 1993; Cox *et al.* 1998). However, dietary habits tend to be self-perpetuating, and making dietary changes or imposing dietary restraint are difficult tasks. This is illustrated by the counter-regulatory behaviour of restrained eaters; but as well as demonstrating reasons for the breakdown of eating restraint, there is a need to focus on the psycho-social factors and features of dieting which are associated with successful eating and weight control. Rather than view dieting only negatively, it should be possible, for instance, to identify the actual cognitive and behavioural strategies used by the many dieters and restrained eaters who are able to achieve weight loss and subsequently maintain long-term weight stability (see Blair *et al.* 1989; Brug *et al.* 1997). Unfortunately, such an approach is generally lacking in current research on human eating behaviour.

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