Is susceptibility to weight gain characterized by homeostatic or hedonic risk factors for overconsumption?

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Abstract

In any particular group of people—living in the same culture—some gain weight whilst others do not. Overconsumption of food is one factor contributing to this susceptibility to weight gain. Because all individuals are exposed to a similar range of environmental appetite-stimulating factors, the variability in overconsumption must be due to variability in intrinsic psychobiological processes. Such variability is an inevitable feature of living organisms. This essay explores whether susceptibility to weight gain is caused by variation in homeostatic processes—such as weak satiety responses to fat, or by hedonic processes—such as hyperresponsivity to the sensory properties of food. The question also arises whether the homeostatic or hedonic processes function separately and independently, or whether they interact. The answer to these questions can throw light upon the organization of behaviours associated with weight control, and can help to develop strategies to prevent weight gain. The theme of this essay was inspired by Gerry Smith’s conceptual and experimental work on both homeostatic and hedonic mechanisms implicated in the control of food intake.

Keywords: Overconsumption; Weight gain; Hedonic response

1. Susceptibility to weight gain

Susceptibility to weight gain is associated with metabolic [1] and behavioural [2] risk factors. Considering metabolic factors, the tendency to gain weight is associated with a low basal metabolic rate, low energy cost of physical activity, a low capacity for fat oxidation, high insulin sensitivity, low sympathetic nervous system activity, and a low plasma leptin concentration. However, when a steady state of obesity occurs, many of these predictors of weight gain are reversed. Behavioural risk factors can exist for energy intake (EI) and energy expenditure (EE) and both can lead to positive energy balance. However, whereas for EE behaviour (physical activity) can account for between 40% and 60% of total EE (depending on the amount of volitional activity, exercise, etc.), behaviour accounts for 100% of EI. Because changes in EI can be of greater magnitude than in EE (on a daily basis), it can be argued that behavioural aspects of EI (i.e., appetite control) constitute risk factors of greater magnitude than those for EE.

The behavioural risk factors may be patterns of eating behaviour, hedonic events which guide and reinforce behaviour, the strength of hunger and satiety sensations, or preferences for and selection of particular types of foods. These behavioural risk factors can be regarded as biological dispositions which create a vulnerability for weight gain and which manifest themselves through behavioural acts, or through physiological processes which promote or permit changes in behaviour. It is an important scientific issue whether these behavioural risk factors operate independently of each other or interact.

2. Hedonic response: a risk factor for weight gain?

A reasonable proposal is that an exaggerated hedonic response to the sensory features of foods constitutes a behavioural risk factor that could promote excessive EI. Although sedentariness is widespread, there is evidence that weight-gaining individuals do indeed consume excessive amounts of food [3] and that weight gain is associated with specific food habits, including the consumption of fatty foods, eating outside the home, and the accessibility of fast/junk foods, each of which is likely to be related to...
enhanced palatability of foods. Physiological satiety signals can be overwhelmed by the potency of high energy-dense, highly palatable foods [4] and strong preferences for these foods are expressed as behavioural traits that constitute risk factors (e.g., Ref. [5]).

Interestingly, the hedonic response, noted here as a risk factor, can be viewed differently. As noted in the recent National Nutrition—Health Programme (2001–2005) in France, an individual’s food choice is a ‘free act’ and eating is recognised as a moment of pure pleasure. Is it therefore necessary to ‘sacrifice the gentle principle of pleasure to the all-powerful precautionary principle?’ Consequently, the hedonic response is both a fundamental aspect of culinary traditions and eating habits, but also a risk factor for overconsumption. Is it possible to satisfy the first whilst preventing the second?

3. Homeostasis and hedonics: separate identities?

In the homeostatic approach to energy balance, drives (such as hunger) that arise in part from biological needs are balanced by physiological satiety signalling systems. The substrate comprises a network of neuropeptides and biogenic aminergic neurotransmitters which links peripheral and central components. This system has been well characterised (e.g., Ref. [6]) and involves insulin, leptin, NPY, AgRP, MSH, CART, GLP-1, orexins, ghrelin, PYY, and other peptides along with serotonin pathways and other aminergic systems. A biological substrate mediating the hedonic processes surrounding consumption is also being characterised and ostensibly involves glutamate, opioids, benzodiazepines, endocannabinoids, and dopamine pathways (e.g., Refs. [7,8]). The circuits for energy homeostasis and hedonic mediation correspond closely to the hypothesised neural systems involved in food reward, reflecting the psychological or functional components of ‘wanting’ and ‘liking’ [9]. If homeostatic and hedonic processes are indeed associated with distinct neural substrates, the extent to which they operate independently can be investigated.

Researchers have recently attempted to test the independence of the two systems by methodologically separating hedonic response from appetitive drive. For example, hedonic reactivity measured by subjective ratings and behavioural observation was dissociated from the incentive value of food by manipulating hunger state and measuring the reinforcing value of foods using a slot-machine-like progressive ratio computer task [10]. Moreover, a high-palatability food (chocolate) was reduced in rated pleasantness over 22 successive days of intake, despite the volitional quantity consumed remaining stable [11].

Pharmacological evidence also suggests that the processes operate independently. For example, in obese participants, administration of the serotonin drug D-fenfluramine [12] suppressed the sensation of hunger but had no effect on the appreciation of the pleasantness of food. Conversely, nalmefene, an opioid agonist, reduced the rated pleasantness of palatable foods but had no effect on hunger [13].

This double-dissociation concept indicates that appreciation of palatability is associated with a specific substrate that can be pharmacologically, and to an extent, methodologically dissected from the substrate-mediating hunger [14]. However, it is still possible for a functional interaction to occur when the manipulation is made through the natural commodity (selected food) rather than through a more selective artificial manipulation (such as a drug).

4. Hunger and pleasure—do they interact?

Considering that most studies have repeatedly shown that palatability has a positive effect on intake and meal size, this would suggest that the palatability of foods is involved in the process of satiation. However, it is less clear whether the palatability of food also has an influence on feelings of hunger and satiety [15]. The possibility of an interaction between palatability and hunger was specifically investigated almost 20 years ago. On separate occasions, participants consumed a fixed energy meal composed of either highly preferred foods or less preferred equivalent food items [16]. There was an enhancing effect of palatability (even the sight of a preferred food) on subsequent hunger which remained high during the meal. However, this fixed meal design did not allow the disclosure of any effect on EI. More recently, an overall increase in hunger following consumption of a palatable preload and an equivalent decrease in hunger following a bland preload with corresponding adjustments in ad libitum intake were revealed [17]. The authors reported this difference to occur irrespective of preload energy or macronutrient content.

Using repeated ratings of appetite within a meal whilst food intake is being constantly monitored, Martin Yeomans has produced a series of experiments that examined the interaction of hedonic response to food with hunger. Participants were presented with meals manipulated at three levels of palatability and showed differential effects of hedonic response on hunger ratings during ingestion. For the ‘strong’ flavoured test meal, rated hunger decreased monotonically with food consumed. For the ‘bland’ flavoured test meal, hunger was found to decrease at a slower rate compared to the strong meal, but increase with intake. In contrast to these conditions, the optimally flavoured ‘palatable’ test meal was associated with an initial short-lasting increase in hunger followed by the slowest decrease of all the conditions. Similar trends were observed by Yeomans et al. [18] using two levels of palatability.

Several researchers have examined the effect of food palatability on subsequent satiety. Foods eliciting an enhanced hedonic response appear to promote a more rapid recovery in hunger up to 3 h following consumption compared to a less palatable preload [19]. Moreover, there is evidence that some stimulation of intake occurs over the
24-h period following consumption of optimal preference sweetened yoghurt preloads [20,21]. In a more recent study, the effect of food palatability on the degree of energy compensation following preloads has been investigated [17]. The capacity of people to demonstrate compensation is usually regarded as a sign of the sensitivity of the system and indicates good appetite control. However, the presence of a more palatable food prevented compensation.

In one of the first free-living studies to examine palatability and intake responses, the extent to which hunger was an intermediary between palatability and meal size was assessed [22]. Comparing hunger with palatability in a two-factor multiple regression, the largest reduction in the palatability β coefficient occurred when it was paired with before meal hunger rating, suggesting that a portion of the effect of palatability on intake is mediated through hunger. Taken together these studies indicate that the response to the palatability of food does indeed exert an effect on EI, and this appears to be mediated via a modulation of hunger. This means that when the homeostatic and hedonic systems are stimulated simultaneously (by food), a synergy occurs that modulates the control of appetite. This process appears to constitute one way through which the hedonic potential of foods could lead to weight gain.

However, the system does appear to operate asymmetrically; although enhanced hedonic response to foods can augment hunger (and therefore, food intake), the presence of strong satiety (developing over the course of a meal) does not down-regulate palatability (e.g., Ref. [23]). Indeed, it has been documented that participants in a state of satiety can be induced to prolong their eating by offering highly palatable foods [24]. It is also noticeable that the reasons people give for stopping eating emphasise fullness and loss of hunger rather than a decline in the pleasantness of the food [25,26]. In other words, participants do not generally perceive pleasantness to be the most important factor in terminating meals. However, this may only hold for foods in which the hedonic value is above some minimum threshold [27].

5. Food choice, hedonic response, and obesity

In addition to an interaction between palatability and hunger, the perceived pleasantness or hedonic response to foods could also modulate appetite control indirectly by influencing the choice of foods. There is considerable evidence that this is the case. In an experiment in which participants sampled a range of foods containing varying amounts of fat and rated their sensory preferences, there was a positive relationship between the rated pleasantness of the fat content of the foods and measures of the adiposity of the participants [28]. The fatter the participant, the greater was his/her rated pleasantness of the fatty foods. More recently, the food choices of monozygotic twins discordant for body weight have been assessed [29]. The twins with the highest degree of fatness displayed a significantly higher preference for fatty foods. If it is assumed that the expression of food preferences is influenced at least in part by the pleasure yielded by the foods, then these studies have demonstrated that levels of body fat are associated with greater rating of pleasantness of fat-containing foods.

It should be noted that food palatability can also affect the reward value of food during ingestion. The phenomenon called ‘sensory-specific satiety’ describes the habituation of specific sensory neurons in response to the prolonged application of the same, but not a novel stimulus [30]. Sensory-specific satiety is shown by a reduction in hedonics of a food being consumed, with little change in hedonics of foods not consumed [31]. From this, it has been argued that being given the opportunity to consume novel foods in a meal can result in a greater intake than if participants are offered more of the same food they have recently consumed [32]. Alternatively, it has been proposed [14,33] that the initial exposure to sensory characteristics of a new food should be viewed as a method to stimulate eating (e.g., sensitization) rather than being an important factor of satiation. Consequently, exposure to new foods can augment food consumption during a meal rather than the pleasantness of foods decreasing due to prior consumption [14].

However, the contradiction of stimulating and inhibiting effects of exposure to the sensory properties of food on eating can be integrated in that the sensory properties of food may provide positive and negative feedback for eating through orosensory stimulation of the preabsorptive receptors along the gastrointestinal tract that are sensitive to the chemical, colligative, or mechanical stimuli of food [34]. Therefore, as eating ensues, positive feedback dominates the system, causing consumption to increase. As more stimulation occurs, negative feedback increases in strength, until the potencies of the positive and negative feedbacks are equal, causing consumption to decrease. This supports the idea that sensory (and hedonic) properties of food are important for the promotion and inhibition of eating. It could be that the sheer variety of highly palatable foods available to an individual during any one meal may increase the amount of time required to balance the positive and negative feedbacks, causing a longer, and larger, eating bout [34].

Recent research has explored the link between vulnerability for addiction and its relation to brain dopamine activity and potential excessive weight gain. Sensitivity to reward (STR), a psychobiological trait reportedly linked to the neurobiology of the mesolimbic dopamine pathway, was used to examine how (an)hedonia, the capacity to experience pleasure, might relate to overconsumption and subsequent weight gain [35]. Contrary to predictions of a linear relationship between BMI and STR, an inverted U relationship was revealed with overweight participants (BMI > 25 < 30) scoring higher than obese and normal-weight participants. Is it possible that low anhedonia (high STR) promotes a life style of hedonically driven overconsumption, which, over time, down-regulates mesolimbic dopamine availability to compensate for its overstimulation? In
other words, high hedonic responsiveness would be a risk factor for weight gain (the achievement of obesity) but would not necessarily characterise an obese person.

There is evidence to support this proposition. For example, PET scans of normal-weight healthy participants eating a favourite meal revealed an increase in dopamine release that correlated with the degree of experienced pleasure. Another study demonstrated that the availability of the dopamine D2 receptor was decreased in obese participants in proportion to their BMI [36]. These studies map onto the idea that there is an optimal level or inverted U relationship between hedonic predisposition and dopamine activation [37]. Participants administered the dopamine agonist methylphenidate found it either pleasant or unpleasant depending on their DRD2 receptor levels. Those participants reporting pleasant effects had significantly lower levels of the dopamine receptor than those who found it unpleasant [37]. It can therefore be construed that potent stimuli (including palatable foods) elicit a positive hedonic response through the stimulation of dopamine activity in people of an anhedonic predisposition. However, there is no evidence to date that tests the notion that particularly hedonic individuals respond aversively to highly palatable foods. It has been suggested that dopamine activity relating to excessive food consumption might only involve activation of brain reward circuitry within normal limits, with other psychological factors exerting stronger effects [38].

Recent investigations of behavioural phenotypes characterised by habitual food choices suggests that the hedonic response to palatable food can influence appetite control via effects on both food choice and EI. Groups of overweight and lean young males matched for age and the habitual high consumption of fat (high-fat phenotypes) were compared [39]. Although both groups were eating a diet known to favour a positive energy balance, the overweight phenotypes consumed greater amounts of the high-fat foods in a test meal and reported greater feelings of pleasantness, satisfaction, and tastiness for the foods consumed. One interpretation of these data is that, for at least this group of overweight people (susceptible to weight gain), they habitually self-select (fatty/palatable) foods with a high probability of generating a positive energy balance (on the basis of their energy density), consume these foods in greater amounts, and derive greater pleasure from this eating. This outcome also demonstrates that certain high-fat phenotypes—susceptible to weight gain—have a disposition to perceive foods as being more pleasant than their lean counterparts (who consume the same habitual high-fat diet). Given this capacity to obtain a high level of pleasure from foods (and eating), it is not surprising that obese people show a tendency to self-select high-palatability foods. It is yet to be determined whether this indicates a supersensitivity in components of the neural circuitry forming the substrate for hedonic properties of foods or whether it reflects a more global sensitivity in the ability to experience pleasure from natural reinforcers in the environment.

6. Pharmaceutical strategies

Although low levels of physical activity contribute to excess weight gain, there is no doubt that many weight-gaining and obese individuals display behavioural risk factors expressed as patterns of eating, food selection, weak satiety, and a recurring drive to eat that favour the attainment of a positive energy balance [2]. To these risk factors, enhanced hedonic capacity should be added. Currently pharmaceutical strategies for the treatment of obesity are concerned with the reduction of energy assimilated—by improving satiety (sibutramine) or by reducing fat intake and fat digestion (Orlistat). Given the evidence cited above, some consideration should be given to the concept of controlling the hedonic response to foods, particularly in the light of the increasing palatability of foods entering the marketplace. The strategy would nullify one significant risk factor that predisposes people to weight gain via a number of means. The identification of a neural substrate that mediates aspects of the hedonic response or reward value of foods, identifies pharmacological targets. Of particular interest are the endocannabinoids which are known to be involved in food consumption and especially the intake of highly palatable foods [40]. It can be noted that the action of endocannabinoids may be via incentive motivational properties of food stimuli as well as via effects on reward processes [41]. These mechanisms have implications for the way in which endocannabinoid activity could modify the pattern of eating and profile of food choices in humans. Interestingly, CB1 receptor antagonists, such as SR141716A (Rimonabant), may be effective in helping people to diminish consumption of high-risk foods and adapt to more appropriate food choices and resist cravings. This type of approach would add a useful dimension to pharmaceutical treatments and may be especially helpful for a subset of weight-gaining and obese people who overconsume because of a potent hedonic response to food. It should also be noted in passing that the endocannabinoids may also be involved in the homeostatic control of appetite [42]. Because of the distribution of CB1 receptors in the brain and periphery, an ‘endocannabinoid bridge’ could function as a link between homeostatic and hedonic processes controlling patterns of eating. We conclude that overconsumption and weight gain can be brought about by either homeostatic or hedonic risk factors, and that such risk factors can be colocalized in certain susceptible individuals, and subserved by overlapping neural circuits.

7. Postscript

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for the precision and clarity of conceptual issues and the insistence on rigour and accuracy in experimentation. Because science advances through the transmission of good ideas and strong methodology, it is intended that the inspiration of Gerry Smith will be inherited by GF. At another level, JB’s daughter, Joanna (3 years old at the time), still has fond memories of being taken by Gerry on a reverential visit to Sherrington’s laboratory in the Physiology Department of the University of Liverpool (1986). A fascination for science can be transmitted in diverse ways.

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