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Combating excessive eating: a role for four evidence-based remedies

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Abstract
Objectives: Review the control of energy balance and outline some causes of and remedies for excessive energy intake.
Methods: Narrative review.
Results: There is negative feedback control of energy intake and body weight, but nonetheless energy intake is only loosely coupled with energy expenditure. Consequently, we are vulnerable to eating in excess of energy requirements. In this context, energy density, portion size and habitual meal patterns have strong influences on energy intake, and accordingly can be targeted to reduce energy intake. For example, energy density can be reduced without much affecting food reward (approximately the pleasure gained from eating), because their relationship is such that reward value is affected relatively little by increments in energy density above 1.5 kcal/g. This and other strategies that increase reward per calorie eaten may be superior to increasing the satiety effect of products because fullness is not inherently rewarding. Low-calorie sweeteners (LCS) provide a means to reduce energy density whilst largely preserving food or beverage reward value. Consistent with this, consumption of LCS compared with consumption of sugars has been found to reduce energy intake and body weight.
Conclusions: Understanding what causes excessive eating also provides insights into how to combat this problem.

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Introduction

The purpose of this article is to review the control of energy intake and body weight with the aim of explaining excessive eating and thereby provide evidence for ways to reduce it. Excessive eating, defined as energy intake in excess of that required to maintain a healthy body weight, is possible because there is no precise physiological balancing of energy intake against energy expenditure. In this context, energy density, portion size and habitual meal patterns have strong influences on energy intake. I also discuss the effects of using low-calorie sweeteners (LCS) to reduce the sugars content of foods and beverages. The example of LCS demonstrates potential unwanted effects, but ultimately it confirms that reduced dietary energy density benefits healthy weight management.

Appetite and energy balancing

Elsewhere we have described a simple model that outlines the main meal-to-meal and longer-term influences on eating (energy intake) behaviour (1,2). This model assumes that eating is, by default, rewarding, and more so when our upper gut is empty or fairly empty (3). For most of the time we are engaged in activities other than eating, but we are also ready to eat most of the time if the opportunity arises – for instance, if we are unexpectedly offered cake by a colleague during a break from work because it is their birthday. We eat the cake because it is delicious, and perhaps because it would be socially awkward to refuse it, and because we are not so full from our previous meal that it would be uncomfortable to eat again. In other words, our appetite is not coupled with current energy expenditure, rather it responds to opportunities to eat, which usually, but by no means always, arise at our planned meal times.

Appetite is stimulated by the anticipation and experience of food reward[1], but it is not uncontrolled, totally at the whim of excess food supply. First, during a meal food reward is reduced by fullness and by sensory-specific satiety (3,5), and although extreme fullness is usually avoided, ultimately this does limit the maximum amount that can be eaten in a single meal. This is a negative feedback system – the stimulating effect of food reward on food intake is counteracted by the filling (satiating) effect of food intake. A second, less apparent, negative feedback system adjusts food intake in relation to body fat stores (1). Evidence for this comes from the dynamics of changes in energy intake and body weight in rat dietary-induced obesity (6-8). When adult rats are switched from a conventional lab diet to a more palatable, energy-dense diet they increase their energy intake and gain weight. The weight gain comprises approximately 75% fat mass (FM) and 25% fat-free mass (FFM). As they fatten, however, their increased energy intake diminishes until a point at which their weight plateaus in parallel with, but above that, of control rats fed only the conventional lab diet throughout (6,8). Furthermore, when the obese rats are returned to

[1] By appetite I mean our desire to eat, and by food reward I mean 'the momentary value (utility) of food to the individual at the time of ingestion' (1,3). Food reward is experienced as pleasurable. However, there are effects on food reward separate from effects on its 'hedonic' component (e.g., effects on 'wanting' in Berridge's (4) model of food reward), therefore food reward can be only approximately equated to the pleasure gained from eating.

the conventional lab diet they eat substantially less than the (lean) control rats and they lose weight rapidly. But with weight loss their energy intake gradually recovers, eventually to control levels, and their weight stabilises again, now close to the weight of the control rats (7).

Recently, Polidori et al. (9) came to a similar conclusion about the negative feedback control of body weight from modelling the results of a trial of the sodium glucose transport 2 inhibitor canagliflozin given to people with type 2 diabetes. At the dose administered, canagliflozin results in an energy loss of 360 kcal/d due to increased urinary excretion of glucose. Compared with participants receiving placebo, canagliflozin-treated participants lost 2.6 kg in one year, with 67% of that weight loss occurring within the first 3 months, and plateauing over months 9 to 12. Based on this time course of weight loss and the daily energy loss due to canagliflozin treatment, the authors were able to quantify the feedback control of energy intake. Specifically, they calculated that this amounted to an increase in energy intake of 100 kcal/d per kg of weight lost. In other words, as weight loss ensued the negative feedback effect of body fat on appetite was reduced, causing a proportional increase in energy intake above the pre-intervention baseline. What is important about this study is that the energy deficit was imposed covertly, not through dietary restriction, so the increase in energy intake cannot have arisen through conscious compensation for eating less. However, it is not possible to rule out some conscious compensation in response to awareness of weight loss.

These observations on dietary-induced obesity in rats and the covert imposition of negative energy balance in humans point to a signal that reduces appetite proportional to body fat stores (1,9). A very strong candidate for this signal is leptin (10). Leptin is a cytokine produced mainly by adipose tissue and released into the blood stream in proportion to FM. It crosses the blood-brain barrier and the LepRb receptor, which mediates most of the physiological actions of leptin, is highly expressed in nuclei of the hypothalamus, itself known to control metabolism and appetite. Leptin therefore provides a link between fat stores, energy intake and metabolic regulation, and it helps resist the development of obesity. Possibly, decreased sensitivity to endogenous leptin ('leptin resistance') (10,11) could contribute to extreme obesity. That is, developing obesity, the diet consumed and/or other factors reduce sensitivity to leptin, thereby increasing the likelihood of further weight gain.

The concept of the feedback control of body weight is not new. It has its origins in Kennedy's lipostatic theory (12), which proposed that food intake was influenced by fat stores, and more completely in Wirtshafter and Davis (13) statement that 'an animal's feeding mechanism is activated by sensory stimuli arising from available food, which we will represent by the letter S, and inhibited by a feedback signal which is proportional to body weight which we will represent by the letter W' (p 76). While food intake affects body weight, body weight also affects food intake, so over time a balance occurs and weight remains stable, unless the 'available food' changes. Wirtshafter and Davis (13) coined the term 'settling point' to describe the value at which weight stabilises for a particular value of

S ('the sensory stimuli arising from the available food'). S is similar to palatability and part of what I call food reward but, in addition to food reward, it is clear that, for example, food accessibility (1,14) and food quantity (portion size – discussed below) also influence energy intake. Together, such features of the food supply might be called the 'food environment,' and thus according to the settling point model body weight will be a function of the food environment.

Relatedly, the term 'obesogenic environment' has been used widely to describe conditions that promote obesity. In the case of the dietary obese rats described above, the obesogenic environment comprised unfettered access to palatable, energy dense foods. For people, obesogenic environments also include the physical, economic, social-cultural and political conditions that influence their food intake, and their levels of physical activity (15). Some or all these conditions will change over shorter time scales (work versus non-work days, fasting versus feasting associated with religious festivals, etc.) and longer time scales (e.g., the family home versus college (16)).

Other stabilising (and some destabilising) influences on body weight

In addition to the negative feedback effect of FM on appetite, there are other influences that assist in stabilising body weight. These include an increase in resting energy expenditure and physical activity energy expenditure with increased body weight. Energy expenditure also increases with increased food intake because of an increase in the energy cost of digestion, absorption and storage of dietary macronutrients (i.e., the 'thermic effect of food'). The increase in resting energy expenditure is the largest component of the total increase in energy expenditure associated with obesity (17). Nonetheless, as Polidori et al. (9) note, the changes in energy expenditure occurring with changes in weight are substantially smaller than weight-related changes in energy intake (i.e., for moderate weight loss approximately a 30 kcal/kg/day decrease in energy expenditure and a 100 kcal/day per kg increase in energy intake, respectively).

In humans, but not in rats, there is also the conscious inhibition of eating with the goal of avoiding weight gain or achieving weight loss (i.e., dietary restraint) (8,18). Relatedly, Booth (19) described an individual's preferred weight as their 'cognitive set point,' deviations from which are detected when they notice a change in their weight or the fit of their clothes. However, the extent to which dietary restraint is relevant or successful in obesogenic environments will vary with, for example, attitudes to overweight, knowledge of nutrition, and life stress and mood (1,20,21). Indeed, negative mood, as well as undermining restraint, may directly motivate food intake because it is relieved or 'soothed', at least temporarily, by eating (22).

In sum, a negative feedback effect of FM on appetite, together with changes in energy expenditure resulting from changes in food intake and body size, is sufficient to maintain constant body weight. For humans, dietary restraint, acting to resist the temptations of food reward, can be a further stabilising factor. Nonetheless, if there are enduring changes to components in this system, for example the amount of physical activity is reduced or dietary

restraint is increased, weight changes will also occur, but with weight eventually 'settling' at a new level. Crucially, though, if the perturbation is removed, say the increased dietary restraint is subsequently relaxed, weight will in due course revert to its previous level. It is thus inevitable that weight is regained after the cessation of weight loss interventions (23,24) – if the diet plan is no longer in place or adhered to, or the gastric band is removed, and no alternative intervention implemented, a brake is released and energy intake will increase over weeks or months until the previous equilibrium between the lure of food reward, the degree of body-fat-related inhibition of appetite and overall energy expenditure is re-established.

Effects of fat-free mass (FFM) and fat mass (FM)

It should be noted that the negative feedback effect of body fat stores on appetite has been questioned on the basis that across individuals energy intake is more strongly related to FFM than to FM (25-27). Based on the positive correlation between FFM and energy intake it is argued that there exists an unidentified signal related to FFM that drives appetite (27,28). This, however, is a positive feedback model, which predicts potential run-away increases or decreases in body weight. So, for example, a short-term environmentally-driven increase of energy intake in excess of energy expenditure would cause an increase in FFM (as well as an increase in FM) (28), which then in turn would drive a further increase in energy intake, and so on. Or consider the effect of an enforced period of bed rest with consequent loss of FFM (29). Energy intake would decrease with the loss of FFM, severely hampering the prospect of recovery. In other words, such a system would be unstable.

The reason that FFM is correlated with energy intake is because FFM is a major determinant of energy expenditure (9,30). Higher energy intake is required to maintain higher energy expenditure without loss of FFM. A human has more FFM than a rat or mouse, and requires greater energy intake to maintain that greater FFM. More subtly, this also holds true when comparing one human with another whilst adjusting amount of FFM for body length. A 'stockier' person requires greater energy intake to maintain their build. None of this demonstrates that FFM drives energy intake directly.

By contrast, as well as stabilising FM, the negative feedback settling-point model is compatible with fuelling (and stabilising) FFM. This is because energy expenditure driven by FFM in excess or in deficit of energy intake will affect body fat stores accordingly to cause a counteracting change in energy intake. For example, if energy expenditure increases above energy intake through increased FFM resulting from physical training, then FM will be depleted somewhat, causing a weakening of the negative feedback signal on appetite and a consequent compensatory increase in energy intake. In this example of an individual or group of individuals over time, there is a correlation between an increase in FFM and an increase in energy intake, but no appreciable change in FM. And like the description of the dynamics of energy intake and weight gain in dietary-induced obesity described above, this change over time is critical to determining the nature of the control mechanisms at play. Cross-sectional measurements (i.e., single point measurements of energy intake, FFM and FM in different individuals) reveal not much more than a positive relationship between

energy intake and body size. In fact, what is somewhat surprising is that in these studies FM is not more clearly positively correlated with energy intake (26,27), as FM contributes to energy expenditure, albeit less kg-for-kg than does FFM (9). The probable explanation is that people with a relatively high FM are prone to undereat in laboratory tests and undereat and/or underreport when recording their free-living food intake (31).

In other words, FM can be viewed as a large energy reservoir depleted by energy expenditure and repleted by energy intake operating via a negative feedback loop. Negative feedback is a powerful principle. Indeed, it is an indispensable stabilizing influence in biological and physical systems (32). More specifically, the existence of weight-related negative feedback control of appetite is supported by convergent evidence from physiological and behavioural studies in humans and non-human animals. The various influences on energy intake and energy expenditure discussed above are listed in Figure 1. The dashed line indicates that these two aspects of energy balance are only 'loosely coupled' in the short to medium term. Energy intake affects energy expenditure, but energy intake must consistently exceed or fall short of energy expenditure for days or weeks for body mass to change appreciably. Conversely, energy expenditure in excess of energy intake will have little or no effect on energy intake in the short term (33) as, even for a lean person, the energy content of FM is many times greater than daily energy expenditure (34).

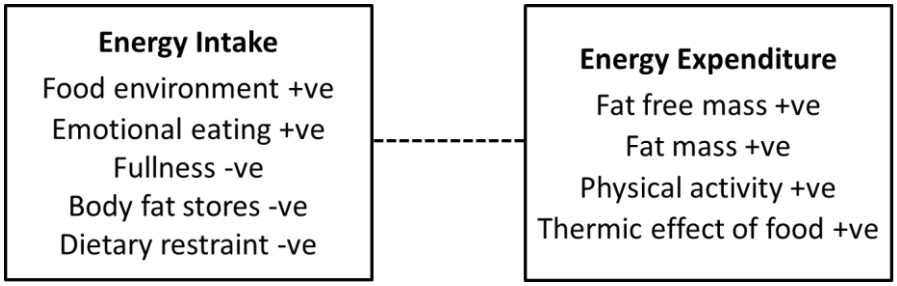

Figure 1 Influences on energy intake and energy expenditure discussed in the preceding text. +ve means that the influence increases energy intake or energy expenditure. -ve means that the influence decreases energy intake.

More on meal-to-meal determinants of energy intake

As discussed above, food intake and body fatness each have a negative-feedback effect on appetite. They differ, however, in that the effect of food intake is strong but acute and the effect of body fatness is weak but chronic. This is because the gut has a relatively limited capacity to accommodate and process food compared with the large capacity of the body to store energy, as glycogen and fat (1). The negative-feedback effect of food intake is, of course, experienced as fullness. However, fullness wanes fairly rapidly during digestion, and typically we are ready to eat again after only a few hours at most. A consequence of this is that food intake in one meal has rather little effect on food intake in the next meal. For example, Levitsky (35) found that energy intake increased at lunch when participants missed breakfast compared with when they ate breakfast, but the increase (135 kcal) compensated

for only 22% of the energy consumed at breakfast (625 kcal). Furthermore, there was no additional compensation in energy intake during the rest of the day. Similarly, we found little compensation (12%) after 2 hours for energy consumed in a food or in beverages versus consumption of water at breakfast (36). By contrast, there was substantial compensation (82%) if participants were permitted to eat again almost immediately (36) (see also (37)). In other words, the legacy of a meal, both in respect of physiological satiety signals and memory for recent eating (38,39), appears to be short-lived and insufficient to come close to balancing energy intake from one meal to the next.

The capacity to be ready to eat again soon after even a fairly large meal is adaptive if energy requirements are high; however, if they are not, this makes us vulnerable to excessive eating. Moreover, consumption of energy dense foods adds to the risk of overeating. This is primarily because the ratio of satiety to energy content ('Satiety Index') decreases as energy density increases (40). As we have noted previously (1), this also explains why energy dense foods are particularly palatable – from a biological perspective the major goal of eating is energy intake, not satiety (i.e., fuel not fullness). Indeed, in the short term, fullness prevents further energy intake. Accordingly, energy dense foods have greater biological utility and hence greater reward value. In other words, energy dense foods encourage excessive eating because they are, relatedly, highly palatable (rewarding) and low in satiety kcal for kcal. Thus, key features of the obesogenic environment are the almost effortless access to energy dense foods, together with low levels of physical activity (14,41).

Below I discuss several dietary strategies for combating excessive eating in the context of the omnipresence of energy dense food. First, though, it is worth remembering that weight loss is resisted. The model of weight control described in the previous section predicts that appetite will increase as energy stores decrease, and there is also the reduction in energy expenditure resulting from reduced food intake and reduced body weight (1,8,9,17,18). Therefore, the same effort at reducing energy intake will be rewarded by a slowing of weight loss over time, with weight eventually reaching a new, but lower plateau. Similarly, weight gain is resisted, as there is no in-built asymmetry in these controllers of body weight (negative feedback effect of FM on appetite and changes in energy expenditure associated changes in energy intake and body weight). Nonetheless, severe restriction of food intake, and especially severe restriction of carbohydrate intake, will risk hypoglycaemia, which will in turn cause hunger, and seemingly specifically hunger for carbohydrate-rich foods (42). This internally-driven ('true') hunger can be contrasted with the absence of fullness and related increase in anticipated food reward that we commonly call hunger, but which arguably is no more than a sign that we are ready to (over)eat again (1).

A role for reducing energy density

Given the role of energy density in promoting food intake described above, it follows that providing relatively energy dilute foods will reduce overall energy intake, and indeed this has been demonstrated very convincingly (e.g., (43)). The problem with this strategy, of course, is that energy dilute foods are less attractive than energy dense foods. Crucially, however, a recent study shows that the relationship between food energy density and

reward value (indexed by choice) is non-linear, with the relationship plateauing at energy densities above 1.5 kcal/g (44). So, for example, carrot (0.24 kcal/g) was chosen more often than celery (0.1 kcal/g), and banana (0.89 kcal/g) more often than pear (0.47 kcal/g), but among high-energy-dense foods, including salted peanuts (6.2 kcal/g) chocolate confectionery products (5.05 and 4.85 kcal/g) and apple pie (3.65 kcal/g), there was not consistent relationship between choice and energy density. This suggests that there is considerable scope for manufacturers to reduce the energy content of energy-dense products, and for consumers to enjoy those products. In view of the expectation that 'diet' or reduced energy foods will not taste good (e.g., (45)), and consistent with their still intrinsically high reward value, such products might be more successful if still categorised as 'luxury' or 'indulgent'. Of course, consumer acceptance would also be higher if it were possible to reduce energy density with minimal impact on the oro-sensory characteristics of the product. For example, creaminess and viscosity can be manipulated to a significant extent independently of energy density (46,47). Furthermore, rather than disrupt appetite control (48), increasing creaminess together with viscosity has been reported to enhance the satiating effect of the product's energy content (46,47). Even small decreases in the energy density of manufactured food and beverage products would be a step in right direction.

A role for reducing portion size

It is clear that food portion size has a strong effect on overall energy intake (43,49). This is assisted by our tendency to eat all of the served portion (50). The difficulty of reducing food portion size, though, lies in loss of reward value and fullness of smaller meals and snacks. Our current approach to this is to focus on reward value, specifically on how to offset the decrease in reward value of smaller servings through increased variety and intensity of the tastes and flavours within the meal (51). In other words, prioritising quality over quantity (52). Preliminary results show that this does indeed compensate, in terms of meal enjoyment, for reduced portion sizes, especially when supported by appropriate 'hedonistic' labelling (51). Provided that this largely prevents consumption of additional food items within the meal, a reduction in overall energy intake should follow due to the weak meal-to-meal influences on intake described above. Furthermore, consumer acceptance of smaller food portions can be expected to increase over the longer term as repeated exposure causes them to be perceived as 'normal' sized (53).

Another way to conceptualise the prioritisation of oro-sensory reward to compensate for reduced portion size is that it maximises 'reward per calorie'. We have described this metric elsewhere (51) as a personal assessment of the reward (or approximately, pleasure) per calorie one estimates one would gain from consuming an individual meal, food or beverage. Its purpose is to guide choices between similarly preferred products that maximise reward per calorie and thereby potentially reduce energy intake whilst at least maintaining, or even increasing, eating pleasure. Cornil and Chandon (52) also discuss the merits of 'pleasure as a substitute for size.' They found that asking participants to imagine the taste, smell and texture of a liked food caused them to subsequently choose smaller portions of another liked food. Moreover, the participants also anticipated enjoying the chosen food more and

were willing to pay more for it. The authors' explanation as to why smaller portions suffice when attention is drawn to the oro-sensory aspects of eating is that it causes a food to be evaluated on the basis of the pleasure of eating, which peaks during the early part of the meal. This is supported by the further observation that the average portion chosen as just right for sensory pleasure was smaller than the average portion chosen as just right for fullness (52). Furthermore, it is consistent the well-established phenomenon of sensory-specific satiety (5) and our proposal of an acute negative-feedback effect of fullness on food reward described above. In relation to the latter, it is also worth noting that the discomfort of over-fullness may cause very large portions to become significantly devalued (54). So, in sum, it seems that from several perspectives it is not the case that 'bigger is better'.

A role for missing meals

The maximum reduction in portion size is, of course, to eat nothing – to miss a meal or a snack. As described above, this results in a reduction in energy intake over the whole day (e.g., (35)). Moreover, missing a meal does not adversely affect cognitive performance (1). If anything, performance may be superior compared with the decrements in performance that occur acutely after food consumption (1). A potential advantage of missing a meal is that no eating is taking place, so there is no contact with food and consequently no direct reminder or experience of the pleasure of eating. If this is done repeatedly, for example not eating breakfast or not eating a mid-morning snack, it will become habitual, just as eating an energy dense snack with coffee becomes habitual. While cutting out an unwanted habit initially requires cognitive effort, the largely subconscious nature of habits means that, once it is established, the new behaviour comes to be largely self-sustaining (18,55). Missing a meal, and even extending this to regular short-term fasting (56), therefore marries two principles, namely (1) an empty stomach does not compromise energy supply to brain (or muscle), and (2) it may be easier, in terms of cognitive restraint, to eat nothing than to eat only a small amount.

More generally, this highlights the potential benefit of adopting a relatively invariant pattern of eating (18). Whilst, as discussed above, the negative feedback control of appetite and effects of body weight on energy expenditure act to resist weight loss, repetition of a behaviour works to increase the future likelihood of that behaviour, whether that is eating less at lunch by omitting the potato chips, substituting fresh fruit for an energy dense dessert, or no longer pairing eating with drinking coffee during work breaks.

A role for low-calorie sweeteners

In relation to the preceding arguments, it would seem that LCS exemplify a food ingredient with significant potential to reduce energy intake and body weight. Most obviously, by replacing or partly replacing sugars in beverages and foods, LCS reduce energy density, and they do this whilst largely preserving the products' reward value. The latter follows from the innately rewarding nature of sweetness (2,57). As a low-calorie or calorie-free reward, LCS products might be used to replace some non-sweet, energy-containing products in the diet, or even used as a substitute for missed meal. While the effects of these latter, perhaps

contentious, uses of LCS are largely unexplored, there is substantial evidence concerning the effects of LCS compared with sugars.

As predicted by the lack of short-term balancing of energy intake against energy expenditure, recent meta-analyses of acute and longer-term randomised controlled trials in human participants demonstrate that consumption of LCS compared with consumption of sugars reduces energy intake and body weight (58,59)[2].

The acute studies compared the effects of consumption of LCS- and sugar-sweetened 'preloads' (mostly beverages) on energy intake in a subsequent ad libitum test meal. We found that overall in these studies (n = 62) test-meal energy intake compensated for 50% of the difference in energy content of the LCS- versus sugar-sweetened preloads (59). If anything, this is likely to be an overestimate of energy intake compensation (i.e., an underestimate of the energy deficit due to LCS consumption) that occurs in everyday life, as a majority (64%) of studies served the test meal between 20 and 60 minutes after the preload, which coincides with high sensitivity to the energy content of a preload (37,62). Whilst these studies confirm that the 'missing calories' in a LCS-sweetened food or beverage are not fully compensated for by an increase in subsequent energy intake, they also show that sugars (versus a LCS-sweetened sensorily-matched control) suppress appetite. That is, the compensation observed was significantly less than 100%, but it was also significantly greater than 0% (59). Furthermore, analysis of the data presented in Table S7 of the supplementary materials to our review (59) shows almost the same degree of compensation (means ± SDs) for sugars versus LCS in beverages (52 ± 52%) as in foods, such as yogurts, jello and puddings (50 ± 70%). This outcome is fully consistent with results of studies that have directly compared compensation for sugars in beverages with compensation for sugars in semi-solid and/or solid foods (63-65). So, although liquids, with the exception of soup (66), might be perceived as less filling than solids (36), it appears that the effects on short-term energy intake of sugars within beverages versus within foods do not differ.

Our meta-analysis of effects of LCS versus sugars on body weight included nine studies, comprising 1332 adults and children (59). The duration of the interventions and any follow-up varied from 4 weeks to 40 months. The effect sizes of LCS versus sugars were −1·41 (95% CI −2·62, −0·20) kg for adults and −1·02 (95% CI −1·52, −0·52) kg for children. Outcomes were similar for studies in which the test products, mostly beverages, were added to the diet and those in which LCS partially replaced sugars in the participants' diet.

Despite this substantial body of evidence from acute and sustained intervention studies, some of which date back to the 1980s, the role of LCS in weight management has been

[2] More recently, Azad et al. (60) concluded that 'Evidence from RCTs does not clearly support the intended benefits of nonnutritive sweeteners for weight management' (p E929). However, their analyses excluded all but one of nine relevant comparisons of LCS versus sugar (59), including the largest trial to date (61). Instead they included three comparisons of LCS versus water and two comparisons of LCS in capsules versus placebo capsules. Neither of these latter types of trial are relevant to determining the effects of LCS compared with the effects of sugar in the diet.

questioned on various grounds. One prominent claim is that by 'decoupling' sweetness from food and beverage energy content LCS undermine the learned control of energy intake (i.e., LCS cause sweetness to become an unreliable predictor of energy content), and thereby lead to increased risk of overeating and obesity. Widely reported results from research exposing rats intermittently to either additional food sweetened with glucose or additional food sweetened with a LCS have been used to support this claim (67). However, the logic of the decoupling argument can be challenged on the grounds that even when LCS, and for that matter all 'processed,' products are disregarded, sweetness does not reliably predict the energy content of different foods and beverages in the diet (2). There is also a question as to whether humans, or rats, rely much on simple taste-nutrient relationships to control energy intake. More likely, signals triggered by nutrients detected in the gut and post-absorptively dominate in influencing satiety (1). Furthermore, notwithstanding these difficulties, recent research has failed to replicate the effects of intermittent exposure to LCS versus glucose, finding instead that rats fed glucose-supplemented food gain the most body fat (2,68).

A second prominent claim is that exposure to sweetness encourages a 'sweet tooth' and therefore increased intake of sweet, energy-containing foods and beverages (e.g., (69)). This predicts that consumption of LCS beverages will increase energy intake and body weight compared with consuming water. Studies show that this does not occur (59,70). The latter study found no increase in sweet food intake with exposure to LCS beverages versus water for 5 weeks. If anything, there was a decrease in sugars intake with LCS consumption. This is consistent with findings from a 6-month intervention study in which consumption of LCS beverages versus water led to a reduction in energy intake from desserts (and from fruits and vegetables) (71). It is also noteworthy that in another study participants who consumed a low-sugars diet for 3 months showed an increase in perceived sweet-taste intensity (at low concentrations of sucrose) but no change in preference for sweetness in test products (72). The latter two studies are included in a recent systematic review of studies investigating the effects of dietary exposure to sweetness on the subsequent generalized acceptance, preference or choice of sweet foods and beverages (73). The authors conclude that overall the evidence from population cohort studies is 'equivocal' and that controlled studies indicate that higher sweet taste exposure tends to lead to reduced preferences for sweetness in the short term, with limited effects in the longer term.

A third claim is that there may be 'conscious' overcompensation for consumption of LCS (74), because at least sometimes consumers use inclusion of low-energy products in their diet as a licence to consume more of those products, or more of other foods and beverages (e.g., using LCS instead of sugars to sweeten my coffee, allows me to have coffee and a cookie). Relatively few studies on LCS have investigated this, but those that have, for example acute compensation studies comparing the effect of informing versus not informing participants of the sweetener and/or energy content of the preload, do not demonstrate conscious overcompensation (2). Furthermore, results of longer-term studies comparing effects of LCS and sugars on body weight were similar for studies in which participants were blinded versus not blinded to the intervention (59).

In sum, there is good evidence that LCS versus sugars consumption reduces energy intake and body weight, and that this occurs because the dilution of food and beverage energy content (energy density) achieved with LCS is not fully compensated for within the meal or at subsequent meals. As discussed, it is conceivable that LCS also have counterproductive effects. However, given the results of the intervention studies comparing LCS and sugars it appears that the sum of any such effects is relatively minor. Furthermore, it may be that the effect of exposure to sweetness is to satisfy, rather than to increase, desire for sweetness, and perhaps appetite more generally, so providing another effect of LCS consumption that reduces energy intake. There is some, though far from definitive, support for this from studies of the effects of repeated exposure to sweetness (73) and from studies comparing the effects of consuming LCS versus water (2). Nevertheless, it is important to recognise that impact of LCS on body weight will be limited by the amount of sugars they can replace in the diet, and by the counteracting effects of increased appetite and decreased energy expenditure that occur with weight loss. An illustration of the latter is that a 178 cm tall, 40-year-old man weighing 80 kg who maintains a moderate level of physical activity would need to reduce his energy intake by 310 kcal/d to achieve a weight loss of 5 kg in 6 months (75). To maintain his lower weight (75 kg) thereafter he would need to consume 150 kcal/d less than when he weighed 80 kg (75).

Conclusions

Humans have the capacity to eat substantially in excess of energy requirements, and especially so if there is an oversupply of energy dense food and physical activity levels are low. Understanding what causes excessive eating also provides insight into how to combat this problem. Remedies discussed here involve reducing energy density, reducing portion size and missing meals, which all work because instances of reduced energy intake are not fully compensated for at the next or subsequent eating occasions. Importantly, eating less energy need not be less rewarding if more attention is given to maximising taste and flavour quality. This is illustrated by the use of LCS, which provide the reward of sweetness without calories.

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