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A brief review of salient factors influencing adult eating behaviour

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Abstract

A better understanding of the factors that influence eating behaviour is of importance as our food choices are associated with the risk of developing chronic diseases such as obesity, CVD, type 2 diabetes or some forms of cancer. In addition, accumulating evidence suggests that the industrial food production system is a major contributor to greenhouse gas emission and may be unsustainable. Therefore, our food choices may also contribute to climate change. By identifying the factors that influence eating behaviour new interventions may be developed, at the individual or population level, to modify eating behaviour and contribute to society's health and environmental goals. Research indicates that eating behaviour is dictated by a complex interaction between physiology, environment, psychology, culture, socio-economics and genetics that is not fully understood. While a growing body of research has identified how several single factors influence eating behaviour, a better understanding of how these factors interact is required to facilitate the developing new models of eating behaviour. Due to the diversity of influences on eating behaviour this would probably necessitate a greater focus on multi-disciplinary research. In the present review, the influence of several salient physiological and environmental factors (largely related to food characteristics) on meal initiation, satiation (meal size) and satiety (inter-meal interval) are briefly discussed. Due to the large literature this review is not exhaustive but illustrates the complexity of eating behaviour. The present review will also highlight several limitations that apply to eating behaviour research.

Key words: **Appetite: Eating behaviour: Satiation: Satiety: Food intake**

Introduction

Eating behaviour is a broad term that encompasses several decisions regarding what to eat, when to eat and how much to eat. Understanding eating behaviour is important as our food choices have significant implications for the individual and society. For instance, overweight and obesity are leading public health problems throughout the world[1]. While the causes of the obesity epidemic are being debated and divergent views are held[2–5], these conditions are ultimately caused by a chronic positive energy balance[6]. This simple statement suggests that weight management is simply a matter of balancing energy in and energy out. From a thermodynamic perspective this is correct but this simple statement masks a complex and poorly understood relationship between energy in and out and energy balance. For instance, foods are not merely vehicles for energy. Foods differ in their macronutrient content, energy density or physical form. Each of these factors influences the rate of enzymic reactions or the postprandial metabolic and endocrine response and could alter the processes of satiation and satiety[7]. Moreover, factors such as eating rate, social facilitation or the environment in which a food is eaten also influence satiation or satiety. Consequently, while thermodynamically speaking, energy is energy, energy from different foods may have different effects on appetite, food intake and ultimately body weight. A consequence of this knowledge is that foods can be chosen or developed that reduce appetite and aid weight management. Moreover, food environments that augment food intake can be modified or avoided to reduce the risk of overeating. Differences in the effect on appetite raise the possibility that some diets will offer a metabolic advantage over what would be predicted by the energy content of a food[8]. However, the magnitude of a metabolic advantage in practice has yet to be fully established and may be modest[9,10].

In addition to obesity, our food decisions have the potential to influence the risk of developing other chronic diseases such as type 2 diabetes, CVD and some types of cancer[11,12]. Besides health concerns, climate change is seen as the biggest global threat of the 21st century[13] and its mitigation is a leading societal goal. Accumulating evidence indicates that our dietary choices are a significant contributor to climate change and that the modern food system is unsustainable[14,15]. Consequently, policies or dietary interventions that manipulate eating behaviour to achieve one societal goal should recognise that these may adversely influence other societal goals.

Considering that eating behaviour has the potential to exacerbate or mitigate several of the leading societal problems it is important that we understand the factors that influence eating behaviour and determine how it can be manipulated to suit societal goals. The aim of the present review is to provide a brief overview of several salient factors that influence several aspects of eating behaviour. In particular, this review focuses on factors

Abbreviations: CCK, cholecystokinin; CPR, cephalic phase response; GLP-1, glucagon-like-peptide-1; PYY, peptide YY; SSS, sensory-specifc satiety.

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that influence the amount eaten. Due to the large number of studies that have been conducted, this review is not exhaustive but seeks to illustrate that eating behaviour is determined by a number of interacting systems. These systems are dynamic and may respond to perturbations in energy balance by a robust physiological response to counteract these changes. Consequently, developing effective approaches to manipulate eating behaviour may not be a straightforward undertaking. This review will also include a brief discussion of several limitations of eating behaviour research. It is important to note these limitations when evaluating the literature relating to eating behaviour.

Eating behaviour

Eating behaviour is influenced by a cacophony of internal and external signals that influence our eating decisions[16,17]. While the primary goal of eating is to ingest sufficient nutrients to satisfy biological requirements, the types of food and the amount eaten to meet this goal are shaped by a multitude of factors including physiology, environmental[18], cultural[19], emotional[20], social[21], self-actualisation[22], economic and access pressures. Many of these will have a profound effect on eating behaviour. For instance, while physiological signals may signal the requirement to eat due to fluctuations in energy stores[23], the behavioural response will be strongly influenced by food accessibility or the types of available foods[24]. An example of this is provided by food-insecure individuals who may have limited food choices because of poor access to supermarkets or grocery stores[25,26] or food costs[27].

It would appear that physiology provides few absolute rules for eating and a vast array of eating choices are possible. For instance, there is no physiological reason why foods that are typically eaten at dinner cannot be eaten at breakfast. Moreover, there is no physiological reason why nutrition cannot be obtained from eating potential foods such as insects or worms. That these behaviours rarely occur is due to food culture or customs that strongly influence the types of foods that are eaten or the time of day that they are eaten[28,29]. While food culture may appear stable it can change markedly due to immigrants introducing new foods or methods of preparing foods, changes in lifestyles (for example, reduction in cooking skills, reduction in time to prepare foods at home), or changing ethical considerations (for example, desire to eat organic foods or vegetarian diets)[30–32]. Political factors can also change the food culture. For instance, it has also been proposed that Chinese food culture was influenced by the taste preferences of Mao Zedong for sweet potatoes[33].

At this time, significant progress has been made in understanding how physiology, culture, custom or socio-economic factors individually influence food decisions but how these factors interact to shape eating behaviour is less well understood. Due to the myriad of factors that influence eating each individual factor will probably only explain a small amount of the variation in eating behaviour. Consequently, it is possible that developing multidisciplinary approaches to understanding eating behaviour may yield new insights regarding individuals' food decisions and provide models that better explain decision making related to food.

Terminology

Blundell *et al.*[34] described a satiety cascade which integrated psychological and biological signals into a framework that integrates the processes of satiation and satiety. The satiety cascade model has been reviewed by other authors[35,36] and provides a useful model for understanding the various factors that influence eating behaviour and their temporal relationship.

It is important to note that the colloquial use of the terms hunger, satiation and satiety is frequently different from their scientific use. For the present review, the terminology proposed by Blundell *et al.*[37] will be used. Hunger is a 'construct or intervening variable that connotes the drive to eat. Not directly measurable but can be inferred from objective conditions'. Satiation is the 'process that leads to the termination of eating; therefore controls meal size', while satiety is the 'process that leads to inhibition of further eating, decline in hunger, increase in fullness after a meal has finished'. It should be noted that for logistical reasons most studies of satiety do not generally measure the length of the inter-meal interval but measure appetite sensations or biomarkers of appetite over a fixed period (typically 3–4 h) (for example, Zhu *et al.*[38,39] and Emilien *et al.*[40]). After this period, food intake at a test meal is measured and used as a marker of satiety[37].

Measurement of eating behaviour and food intake

When evaluating the literature, it is important to understand the various limitations of experimental approaches to investigating human eating behaviour. While a full discussion of experimental methodology is beyond the scope of this review, a recent paper provides an interesting discussion of the limitations of appetite research[41].

Humans can provide a subjective assessment of their motivation to eat which can be captured using questionnaires[37]. These appetite questionnaires predict meal initiation and food intake, and are sensitive to experimental manipulations[42]. However, it has been argued that the ability of questionnaires to predict meal size is modest, limiting their usefulness[43]. For instance, in a study of free-living individuals the correlation coefficient between subjective hunger and energy intake was 0·27[44]. Mattes[43] found that over a 7 d period the correlation coefficient between hunger and energy intake was 0·5. A major weakness of these studies is that food intake was self-reported rather than observed. Considering the well-documented problems with measuring food intake in free-living individuals[45], it is debatable that these studies provide an accurate assessment of the association between appetite ratings and meal size. Laboratory studies may provide a more accurate assessment of the association between appetite ratings and energy intake as food intake can be measured accurately. However, a meta-analysis of four short-term laboratory studies found that the correlation coefficient between hunger and energy intake was 0·16 and between fullness and energy intake was only –0·20[46].

From these studies it may be argued that the ability of questionnaires to predict meal size is modest and that directly measuring food intake is a more useful and relevant measure of appetite. This may be misguided as there are several reasons

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why food intake should not be viewed as an 'objective' or uncontaminated marker of appetite[47]. First, eating behaviour studies are generally conducted in highly contrived situations that do not reflect the environment in which study participants typically ingest food. Second, these studies frequently provide food portion servings in excess of what might usually be eaten which may augment consumption beyond what would usually be eaten[48]. Moreover, other factors may serve to promote overconsumption at a test meal including the availability of free food or so that a later meal does not need to be purchased. Third, while foods used in studies are generally palatable (the participants rating of the palatability of foods is typically measured during screening), there is a difference between 'liking' and 'wanting' and participants may not want to eat the test food at that particular occasion and reduce food intake[49]. Fourth, if participants are aware that they are being observed they may adjust their eating behaviour to meet social norms[50].

However, laboratory studies have several strengths and have undoubtedly made a substantial contribution to the understanding of human eating behaviour. Through the use of appropriate controls, a specific factor can be isolated so that its effect on appetite or food intake can be determined free from the influence of extraneous variables. In addition, the laboratory provides the opportunity to make precise and accurate measures of food intake or other appetite measures. It is also possible to collect biological samples or make physiological measurements that may provide mechanistic explanations for the observed results. Laboratory studies provide strong internal validity (for example, the ability to draw causal inferences). However, a key limitation of laboratory studies is that they do not reflect the environment in which human eating behaviour is expressed, which limits the generalisability of the data. Humans typically eat in environments that include features, noticed or unnoticed, that influence decision making (for example, food choices or energy intake) such as atmospherics, salience or social norms[51]. These features may potentiate or limit internal physiological appetite signals or weaken/strengthen self-control to influence eating behaviour. Moreover, evidence suggests that when participants are aware that their food intake is being monitored they consumer smaller meals[52]. Consequently, the results from laboratory studies may not reflect eating behaviour in typical eating situations.

An alternative approach to investigating eating behaviour is the field study. In this type of study, human subjects are observed in a typical eating environment. These studies have the advantage of having high ecological validity (for example, the results can be generalised to real-life settings) although participants in studies generally know that they are being observed which may influence their behaviour[53]. Moreover, field studies are frequently limited by poor control over experimental conditions. Perhaps the most significant drawback of field studies is the difficulty measuring food intake in free-living individuals[45].

Another key limitation of many studies of eating behaviour is that they are typically short term and are frequently less than 24 h in duration. They typically only observe behaviour at one or two meals. It should not be assumed that the results from short-term studies will persist over the medium to long term and lead to changes in body-weight change, as physiological changes will probably occur to oppose changes in body weight. A possible example of the body adapting to changes in food intake is provided by studies of energy-yielding beverages. Some short-term laboratory studies report that that there is no dietary compensation (i.e. reduction in the consumption of other energy sources to compensate for the energy provided by the test food) for energy consumed in liquid form[54]. Consequently, the increased consumption of energy-yielding beverages should lead to an amount of weight gain that would be predicted by the amount of energy provided by the beverage being regularly consumed. However, a longer-term study found that when energy-yielding beverages are added to the diet, weight gain is not as high as expected, suggesting that dietary compensation does occur[55]. Consequently, short-term studies may overestimate the influence of energy-yielding beverages on energy intake and exaggerate the potential impact on body weight. Still, short-term studies provide an opportunity to screen potential anti-obesity agents or strategies before conducting expensive and logistically challenging long-term studies. However, this may also mean that a potentially useful approach to reducing body weight may be abandoned prematurely if its effect on eating behaviour does not manifest itself in the short term. For instance, changes in the gut microbiota may influence the appetitive response due to a potential effect on satiety-related hormones[56,57]. Consequently, it may take several weeks for an intervention to alter the gut microbiota so that an effect on appetite can manifest itself.

The physiological regulation of eating behaviour

It has been proposed that multiple biological mechanisms act to regulate body fat[58]. This is known as the set-point theory[59]. In this model, perturbations in body fat are corrected for by changes in appetite that lead to a change in energy intake so that the perturbation is corrected[60]. For instance, leptin is secreted by the adipose tissue in direct proportion to adiposity[61]. Central administration of leptin has several metabolic and behavioural effects including increased energy expenditure, increased lipolysis and reduced food intake[62]. It is likely that these effects are mediated by hypothalamic neuropeptides including melonocortin-4 and neuropeptide Y[63]. Consequently, if an individual gains weight, circulating leptin levels would increase which would increase energy expenditure and reduce appetite until the perturbation in body weight is corrected. Conversely, if weight is lost, leptin levels are reduced leading to reduced energy expenditure and increased appetite until the perturbation in body weight is corrected. This set-point model accounts for observations that body weight remains remarkably constant over a long period of time[64] and for observations that periods of underfeeding are followed by periods of hyperphagia[65]. It has been argued that the discrepancy between energy intake and expenditure may be as little as 74 kJ/d and such precision points to a physiological regulatory system[60]. It should be noted that the set-point model is not universally accepted and others argue that body weight is not tightly regulated[60]. While it is acknowledged that body weight remains constant for extended periods and that

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physiological mechanisms contribute to this stability, there are non-physiological factors that also influence body weight[66–68]. That is, if environmental factors remain constant over a period of time this will result in a stable body weight.

A resolution to this debate is required because the approach to reducing the number of overweight or obese individuals would differ depending on the correct model. The set-point theory essentially denies a role for environment or social factors in determining body weight and subsumes everything to physiology. Consequently, efforts to alter the food environment would have little effect on body weight. However, if non-physiological models are correct, changes to the environment may be a useful strategy for reducing obesity. As neither model fully explains human eating behaviour and body-weight changes, the development of new models is required. The development of new models may be facilitated by an increased focus on multi-disciplinary research that integrates the most salient impacts on eating behaviour (both physiological and environmental).

Hunger and meal initiation

In the 1950s, Mayer[69] proposed that hunger sensations were due to a decrease in glucose utilisation which was detected by glucose-sensitive sites in the brain. The increase in hunger sensations would lead to meal initiation. This became known as the glucostatic theory. Later studies found that the administration of exogenous insulin or pharmacological agents that prevent the cellular oxidation of glucose (2-deoxy-D-glucose) causes animal to eat[70,71]. However, these studies reduce blood glucose to levels that are not normally encountered and their relevance to meal initiation in normal situations is limited. In studies where blood glucose levels have been continuously monitored it has been demonstrated that meal initiation is preceded by a fall in blood glucose concentration shortly before eating begins[72,73]. In humans, it has also been found that meal requests correlate with a transient drop in blood glucose[74]. However, when individuals are in negative energy balance there was no correlation between meal initiation and a drop in blood glucose[75]. Furthermore, in a study that used a euglycaemic clamp to keep blood glucose levels steady it was found that human subjects still spontaneously requested food, indicating that a decline in glucose utilisation rates was not a necessary precondition for meal initiation[76]. Moreover, the current data supporting a link between blood glucose and meal initiation are based on correlation and do not prove a causal link.

It has been hypothesised that ghrelin, a hormone secreted by the stomach, is a factor in meal initiation[23]. To date, ghrelin is the only peripheral orexigenic hormone that has been identified. Studies have shown that ghrelin rises before a meal is initiated[23,77] and decreases after feeding[78]. Moreover, the intravenous administration of ghrelin to rodents results in the stimulation of food intake[79]. Plasma ghrelin concentration has also been found to correlate with hunger ratings[77]. However, mice lacking ghrelin receptors do not exhibit altered meal patterns, suggesting that that ghrelin does not have an essential role in meal initiation[80]. Other research has raised the possibility that ghrelin rises in anticipation of a meal rather than as a stimulus for a meal[81].

Indeed, while an argument can be made that each of these metabolic factors are causally related to meal initiation it may also be argued that these are anticipatory reflexes initiated because of the expectation of food intake. It has been proposed that while under certain circumstances physiological signals can initiate a meal, these are rarely encountered during normal life and under most situations it is environmental factors (for example, access to food, habitual meal times based on work schedule) that cause an eating episode to be initiated[82].

Satiation

Before food is ingested a collection of responses, known as the cephalic phase response (CPR), prepares the body for the ingestion of food[83]. While the CPR is small and transient it may have implications for satiation[84] and it has been proposed that a stronger CPR will lead to reduced meal size[85]. While it has been demonstrated that bypassing the CPR by introducing food directly into the stomach results in larger meal sizes[86], further research is required to understand the role of the CPR in satiation. Still, the importance of taste in the correct metabolic response to nutrient intake has also been demonstrated by Spetter *et al.*[87] and this may lead to changes in satiation. Another study has found modest effects of the CPR on hormones related to appetite[88]. Little is currently known about how the CPR influences satiation and a better understanding of what factors influence the CPR and how this makes an impact on meal size is required.

Another key contributor to meal termination is sensory-specifc satiety (SSS). SSS describes the reduction in the pleasantness of a food due to its continued consumption while the pleasantness of uneaten foods remains unchanged[89]. One study found that SSS is a key influence in meal termination[90] and that providing a variety of foods in a meal can delay satiation and increase meal size[91,92]. It is not clear how palatability influences SSS although consuming foods with stronger flavours does not influence the process of SSS[93]. At this time, the physiological basis for SSS is poorly understood although changes in neuron firing rates in areas of the brain associated with the hedonic evaluation of foods may be involved[94]. It is interesting to note that older adults do not develop SSS[95], possibly due to the effect of sensory losses[96]. The effect of this on food intake in older adults has not been fully established. While it may be predicted that the reduction in SSS would reduce the stimulatory effect of food variety on food intake in older adults, this does not appear to be the case[91].

Gastric distention has been found to contribute to meal termination. In a study by Geliebter[97], participants were asked to swallow a balloon that was then filled with varying amounts of water[97]. As the balloon volume increased, causing gastric distention, meal size was reduced, suggesting that gastric distention is involved in satiation. Further studies have shown that causing gastric distention is related to sensations of hunger and fullness and the response may be mediated by cholecystokinin (CCK)-8[98].

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Recent studies have raised the possibility that RMR and fat-free mass are key drivers of energy intake[99]. Caudwell *et al.*[41] found that meal size was correlated with fat-free mass but not fat mass. An implication of this finding that these data are not consistent with adipocentric models of appetite control was noted by the authors. A subsequent study reported a correlation between RMR and energy intake[41]. The authors propose that RMR could be a useful marker for energy intake and possibly represents a physiological marker for hunger. However, further research is required to demonstrate a causal relationship between fat-free mass, RMR and meal size.

Other studies have found a correlation between body temperature[100] or metabolic rate[101] and meal size. It has also been reported that lower ambient temperatures are associated with increased meal size[102]. Further research is required to clarify the role of these and other potential influences on food intake that have gained little attention[103].

Satiety

Many physiological factors contribute to satiety. A growing body of research has identified several hormones that are secreted by the gastrointestinal tract and influence eating behaviour. This raises the possibility that pharmacological agents or dietary supplements could be developed that increase the secretion of the hormones to increase satiety. Multiple gut hormones have been linked to the expression of satiety[104] and a full discussion of these is beyond the scope of the present review. Only three hormones, CCK, glucagon-like-peptide-1 (GLP-1) and peptide YY $(PYY)_{3–36}$, will be discussed due to their widespread measurement in appetite studies.

CCK is a hormone secreted by I-cells located predominantly in the proximal duodenum[105]. CCK has an effect on meal size or early-stage satiety although its role in eating behaviour may be dispensable[106]. Studies have generally shown that infusing CCK into dogs, rodents or human subjects reduces food intake or suppresses appetite in a dose-dependent manner[107,108]. Moreover, when a CCK-1 antagonist is administered before a meal is eaten this leads to increased meal sizes[109,110]. In human subjects, studies have been conducted where CCK has been infused and the effect on food intake or appetite measured. These studies generally support a role for CCK on meal size. Schick *et al.*[107] found that infusing CCK reduced food intake but only at supra-physiological levels. These results were mirrored by a study that infused CCK at physiological levels and had no statistically significant effect on food intake or appetite sensations[111]. Different results were obtained by Ballinger *et al.*[112] who found that a physiological dose of CCK reduced food intake by approximately 1350 kJ. Similar findings to Ballinger *et al.*[112] were reported by Lieverse *et al.*[113], Gurtzwiller[114,115] and Brennan *et al.*[116]. Fatty acids and protein appear to be potent stimulators of CCK secretion while carbohydrate has a minor effect[117]. While there is evidence that CCK is causally involved in satiation it is not clear that its administration or increasing its plasma concentration will lead to a change in body weight. Rodent studies report that while the repeated administration of CCK reduced meal size, the rodents ate more frequently, meaning there was little effect on overall food intake[118].

GLP-1 is a hormone secreted by cells in the ileum. After secretion, the active form $(GLP\text{-}1_{7–36})$ is rapidly converted to the inactive form $(GLP\text{-}1_{9–36})$ by dipeptidyl peptidase-4. GLP-1 is thought to have a role in the 'ileal break' mechanism which slows the entry of nutrients into the large intestine to facilitate absorption in the small intestine[119]. It has been proposed that GLP-1 may influence satiety by slowing gastric emptying and prolonging gastric distention[120]. GLP-1 may also directly interact with the brain and GLP-1 receptors have been found in the hypothalamus[121]. There is a correlation between post-prandial GLP-1 concentration and activation of areas in the hypothalamus associated with satiety[122]. While this evidence suggests a role for GLP-1 in satiety this has still to be confirmed. Several studies have infused GLP-1 and examined the effect on food intake in appetite but have provided mixed results. While some studies report that infusing GLP-1 reduces food intake and/or appetite[114,123–125], other studies have found no effect on the same outcome measures[116,120,126,127]. It has also been argued that the studies demonstrating an effect of GLP-1 on food intake or appetite used supra-physiological doses of GLP-1 and its relevance to satiety under normal conditions remains unclear[128].

$PYY_{3–36}$ is released primarily in the distal gastrointestinal tract and acts as a agonist on the Y2 receptor in the hypothalamus[129,130]. A potential effect of $PYY_{3–36}$ on food intake was first reported in 2002 by Batterham *et al.*[129] who found that food intake was reduced by 33 % in the 24 h after $PYY_{3–36}$ was infused for 2 h. While subsequent studies report that infusing $PYY_{3–36}$ reduces food intake or increases satiety this only occurs at higher doses or when infused with GLP-1[127,131–134]. Some authors have suggested that the effect of $PYY_{3–36}$ on food intake may be due to feelings of nausea rather than an effect on satiety[131,134]. In a review, it was concluded that there was no overlap between the circulating concentration of $PYY_{3–36}$ following a meal and following infusion of $PYY_{3–36}$[128]. This raises questions about the role of $PYY_{3–36}$ in the normal satiety process. Further research is required to fully characterise the role of $PYY_{3–36}$ in the satiety process.

Environmental factors that influence eating behaviour

Hunger and meal initiation

It is likely that under normal circumstances, where food is reliably available, meal initiation is largely influenced by our schedules or the opportunistic access to foods[82,135]. In a study that asked obese participants why they initiated a meal the most common response (32·7 % of respondents) was that it was a meal time[136] and only 20 % of meals were initiated in response to hunger. It appears that hunger is not a necessary stimulus for meal initiation, with a study finding that the exposure to a palatable food was sufficient to cause meal initiation even when the individual was sated[137]. Moreover, the sight and proximity of food have also been found to stimulate food intake independent of hunger[24].

Another potentially key contributor to meal initiation is food cravings. Food cravings are experienced by 21–97 % of the population and are defined as an intense desire to consume a

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specific food independent of hunger[138]. Food cravings typically involve the desire to eat energy-dense, high-fat foods[139]. Data indicate that obese people experience more frequent food cravings than their lean counterparts[140,141]. A study that used diet records has reported an association between food cravings and energy intake[142] while a laboratory study found that specific food cravings were associated with intake of a corresponding food[143]. It has been suggested that cravings and other forms of food cue reactivity should lead to increased food intake and consequently weight gain. However, studies provide inconsistent results and further study is required. For instance, while several studies have found that food cue exposure can increase eating in adults[144–146] and is associated with weight gain[147,148], other studies have failed to show any associations[149–151]. A recent meta-analysis combined the results from forty-five studies and found a statistically significant but moderate effect of food cue reactivity and craving on eating (r 0·33)[152].

Satiation

In general, it seems that in the short term meal size is determined by environmental factors, although physiology clearly places limits on the amount that is eaten in a meal. However, it must be remembered that long-term body-weight regulatory systems may exert an increasing influence on meal size if body weight is being gained or lost. Therefore, caution should be used when interpreting the results gained from the short-term studies that are discussed in this section.

It has been suggested that the role of physiology in determining meal size has been overstated and that the largest influence on meal size is the pre-ingestive decisions regarding meal size[153]. Observational studies suggest that humans plan the amount of food that they are going to eat in advance[154,155]. Other studies have shown that memory of a recent meal can reduce food intake at the subsequent meal[156,157]. Disrupting memory through the use of distractors increases food intake at that meal but also increases food intake at subsequent meals[158]. This suggests that the memory of how much was eaten at a meal has consequences for food intake over the short term. In a series of studies by Brunstrom *et al.*[159,160] it was reported that individuals gauge the expected satiation of a meal and the amount of energy served correlates with this expectation. It is potentially interesting to note that expected satiation of a product remains stable over time and repeated exposure to lower-energy-density alternatives has no effect on expected satiety[161]. This information may have implications for the creation of new dietary products to aid weight management.

Evidence suggests that the portion size of foods has increased over the past three decades[162–164]. These observations are of interest, as serving a larger portion size increases food intake[48,165,166]. A noteworthy observation by Rolls *et al.*[48] was that when a large portion of food was served participants consumed 30 % more energy compared with the small portion. However, appetite ratings were similar following both meals and only 45 % of the participants noticed that the portion sizes differed. These observations have public health implications as people frequently eat away from home and are not able to control the portion size served to them.

The palatability of the meal can also increase meal size. A cross-sectional study found that meals in the highest palatability rating were 40 % higher than at the lowest rating[167]. Intervention studies have generally supported this observation, showing that palatable versions of a food are eaten in higher amounts than bland or unpalatable versions[168,169]. The relevance of this information to normal eating behaviour may be questioned, as few people choose to eat unpalatable foods. However, it has been reported that food deprivation increases the rated palatability of foods, which results in larger meal sizes[169]. It has been proposed that we live in an obesogenic environment that provides access to cheap, energy-dense and palatable foods[170]. Therefore, the frequent exposure to palatable foods may provide a strong incentive to consume food, leading to the overconsumption of foods that potentially have low satiating potential[171].

Accumulating evidence suggests that eating rate may influence satiation. In a meta-analysis it was found that slowing eating rate by any method reduced meal size[172]. This included studies that slowed eating rate by eating slowly[173], increasing the number of masticatory cycles before swallowing[174,175], or manipulating food form[176]. A precise mechanism has not yet been identified but could involve increased sensory exposure[177] or a longer meal time which allows nutrients to enter the gastrointestinal tract and stimulate cells that secrete CCK or other satiety-related hormones[178,179]. It is also possible that mastication has an independent effect on satiation. Studies using rodent models suggest that mastication activates areas of the brain associated with satiation through an increase in histamine production[180]. This is initiated by stimulation of the periodontal ligaments[181]. At this time, it is not clear that this pathway has an important role in human eating behaviour. An answer to this question would be useful as it may provide an explanation for observations that tooth loss (which would result in lower stimulation of periodontal ligaments even with dentures) is associated with higher body weight[182].

The form in which a food is consumed has also been found to influence meal size. In particular, foods that can be ingested rapidly (such as liquids or low-fibre foods) are associated with increased energy intake[176]. This observation is important, as the modern food supply is characterised by an ample supply of highly processed, low-fibre foods. Other aspects of food rheology such as viscosity have also been found to influence satiation[183,184]. One potential explanation for these results is that foods that require greater eating effort and are eaten more slowly increase oral exposure time which augments satiation[185].

Studies have shown that humans eat a constant weight of food[186–189]. Consequently, altering the energy density of the meal (kJ/g) would enable people to eat the same weight of food but consume less energy. Several studies have altered the energy density of the diet by increasing the water content of the food[190], increasing the air content of food[191] or increasing the fibre content of food[186]. These effects of reducing the energy density of food on energy intake persists over a 48 h period[186] and may lead to weight loss over the long term[192]. Another potential method to reduce the energy density of the diet is to drink water with a meal; however, there is little supporting evidence to suggest this influences food intake[190].

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Satiety

A number of studies have investigated the influence of various interventions on the processes of satiety. A large number of studies have investigated the role of nutrients on satiety. It is generally thought that there is a macronutrient hierarchy, with protein being more satiating than carbohydrate which is more satiating than fat[193]. However, a recent systematic review and meta-analysis found that when high-protein meals were compared with low-protein meals only 35 % reported a reduction in hunger while 55 % showed an increase in postprandial fullness[194]. Only 18 % of studies found that a high-protein test food reduced food intake at the next meal compared with a low-protein meal. The inconsistent results may be due to differences in the protein quality used in the test meals or the form of the test meal. It has also been proposed that to obtain an effect on satiety a protein threshold must be crossed. This has been estimated to be approximately 25–30 g[195] although insufficient data exist to specify a minimum amount of protein that may have an effect on appetite.

Much recent effort has focused on the effect of energy-yielding beverages on satiety. Several studies have found that beverages are less satiating than their solid equivalents[54,196–199], although conflicting data exist[200]. It has been hypothesised that it is the liquid medium that is responsible for the poor satiating effect and the macronutrient content of the beverage is not relevant[201]. Supporting this hypothesis is a well-controlled study where it was found that solid versions of high-carbohydrate, -fat and -protein foods was more satiating than the liquid equivalents[202]. However, other studies have demonstrated that beverages containing fibre produce satiety[203]. Moreover, studies have shown that beverages that are consumed as part of weight-loss programmes induce satiation, which may suggest that expectation has a role[204]. Supporting a role of expectation is a study that led participants to believe that a beverage or solid food would change form in the stomach to become a solid or liquid; perceptions of satiety and gastric emptying rate were altered[205]. Moreover, soup provides energy in a liquid form but studies have shown it to cause a robust satiety response[206,207]. It has also been found that a liquid soup may be more satiating than a soup containing solid pieces of food[39]. This may be due to an increased satiety hormone response due to the greater availability of nutrients.

Food form has also been shown to influence satiety. Some studies have found that increasing the viscosity of a semi-liquid food increases postprandial satiety[208,209]. This may be due in part to a slower gastric emptying rate[208,209] although gastric dilution may rapidly reduce the viscosity of a meal leading to minimal effects on gastric emptying rate[210]. Changing the form of a solid food using a food processer to form a purée has been shown to increase satiety compared with eating a solid equivalent[39] or induce reduced satiety compared with eating a solid equivalent[211]. The breaking of the plant cell walls may increase the availability of nutrients from the food[212] and increase altering the postprandial endocrine response in a manner that increases satiety[179]. However, processing foods also increased the postprandial insulin response[39]. In light of the widespread consumption of processed food, further research is warranted to fully understand the impact of processing on appetite and markers of chronic disease.

Emerging evidence suggests that in addition to an effect on satiation, eating rate or mastication may also influence satiety. However, data are inconsistent. A meta-analysis relating to eating rate did not find evidence that slowing eating rate has a robust effect on satiety[172]. Another meta-analysis found an association between eating fast and obesity[213]. However, three studies that have investigated the effect of mastication on satiety found that increased chewing activity increased postprandial satiety[178,179,214]. It is possible that increasing masticatory effort means that the swallowed bolus contains smaller particles, increasing the surface area available for digestive enzymes to act on. As many satiety-related hormones are secreted in response to nutrients in the small intestine, these changed digestion kinetics may be sufficient to cause an altered endocrine profile that is associated with increased satiety.

Discussion

The present review has focused on several salient physiological and environmental factors that have been found to influence eating behaviour. This list is not exhaustive but illustrates that there are several potential points of intervention, either at the individual or population level, to modify eating behaviour and contribute to societal public health or environmental goals. Further research is required to understand how multiple factors interact to determine eating behaviour. This will involve a greater focus on multidisciplinary research. However, there are considerable barriers to this approach that have been previously discussed[215]. An interesting review that discusses the multidisciplinary aspects of developing consumer products that augment satiety has been published[36]. It is likely that new approaches to training scientists will be required to overcome these barriers.

Several physiological mechanisms have been identified that influence eating behaviour and body weight. While physiological explanations are insufficient on their own, a key insight from this research is that body-weight regulation is strongly asymmetric in that it strongly resists weight loss but only weakly protects against overeating or weight gain. This would provide an explanation for why weight gain is relatively easy whereas attempts to lose weight frequently end in failure. Another key insight is that there are multiple levels of redundancy in the physiological system. Whole parts of the system can be made inoperative without significant effect, as other systems appear to take over, causing changes in behaviour to counteract the loss (for example, increases in feeding frequency). This suggests that pharmaceutical or functional food approaches that aim to aid weight loss by targeting a single mechanism (for example, augmenting CCK secretion to increase satiety) may have limited success. Further research is required to fully understand the physiological basis of eating behaviour and whether greater success in manipulating it may be achieved by targeting multiple systems.

As the physiological systems that influence eating behaviour do not appear to strongly oppose overeating, it could be argued that environmental changes that promote food intake are the

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key driver of overeating and weight gain. This means that there should be a strong focus on gaining a better understanding of how environmental factors interact to influence eating behaviour. A better understanding of how environmental factors interact to influence eating behaviour may require new approaches to research. For instance, a large percentage of laboratory research seeks to isolate one factor so that its effect on eating behaviour can be unambiguously determined. This approach has several strengths and has strong internal validity, but as people do not generally eat in such environments this type of research lacks external validity. Individuals eat in environments where multiple factors combine to influence eating behaviour. An individual's eating decisions may also be constrained by cultural, socio-economic or accessibility issues. It is not clear how all these factors interact. They may cancel each other out, have an additive effect to promote food intake or satiety, or there may be a synergistic effect which potentiates the influence of individual factors. Consequently, it is not possible to predict how many of the observed environmental influences on eating behaviour will operate in commonly encountered eating environments. It is possible that new technologies, such as virtual reality, could be employed to combine the advantages of laboratory studies and field studies[216] to better understand eating behaviour in realistic environments. Research is required to develop and test the validity of new approaches to eating behaviour research.

If environmental factors are a key influence on eating behaviour, another challenge will be keeping pace with societal and technological advances. We are in a period where technological and societal changes are occurring at an unprecedented pace. The effect of these changes on eating behaviour is poorly understood. For instance, societal changes such as increased urbanisation will influence the environment in which most people make eating decisions (for example, greater access to convenience stores, restaurants or supermarkets). Changes in social inequality also have the potential to influence eating decisions[217]. How socio-economic inequality interacts with the food environment to influence eating behaviour requires further study. Technological changes may also have a profound effect on our eating behaviour. Artificial intelligence approaches have been developed to create recipes (for example, IBM and Chef Watson). This technology could be potentially developed to aid healthy eating decisions, especially as advances in personalised nutrition are made. The degree to which our eating decisions could be made by artificial intelligence, its acceptability to the consumer and the effect on health is worthy of research. As it stands, it would appear that some people are willing to outsource some of their food decisions by using meal services that ship 'healthy' meals to their home. Moreover, as more people buy their foods online, research is required to determine how this approach influences food decisions and whether this provides new avenues to promote healthier eating choices. Of course, health advantages provided by technological advances have the potential to further entrench health inequalities unless there is widespread access to the relevant technology.

The wider effects of changes in eating behaviour should be considered when policy or dietary advice is being formulated. For instance, short-term appetite studies suggest that protein is the most satiating macronutrient. Dietary advice to increase protein consumption to aid weight management would therefore seem prudent as obesity is a leading public health problem. However, studies suggest that diets high in animal protein contribute to climate change and are unsustainable[218,219]. While more research is required to understand this complex problem, consideration to other societal problems should be made.

Eating behaviour is a complex yet fascinating area of study where much remains to be discovered. Considering that eating behaviour contributes to several of society's pressing problems, more focus should be placed on understanding eating behaviour and how it may be manipulated so that societal goals can be achieved.

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