The aims of this paper are to describe the rules of the cortical processing of taste and smell, how the pleasantness (or affective or reward value) of the taste, smell and texture of food are represented in the brain, how cognitive factors and attention modulate these affective representations, and how these food reward representations play an important role in the control of appetite, food intake and obesity. To make the results relevant to understanding the control of human food intake, complementary evidence is provided by neurophysiological studies in non-human primates and by functional neuroimaging studies in human subjects. A broad perspective on brain processing involved in emotion and in hedonic aspects of the control of food intake is provided by Rolls in *Emotion and Decision-Making Explained*[^1][^2].

A reason why it is important to understand the brain systems for food reward is that the reward value of food (i.e. whether we will work for a food), measures our appetite for a food, and whether we will eat a food. Thus normally we want food (will work for it and will eat it) when we like it. ‘We want because we like’: the goal value, the food reward value, makes us want it. For example, neurons in the orbitofrontal cortex (OFC) and lateral hypothalamus described later respond to the reward value of a food when it is for example shown, and these neuronal responses predict whether that food will be eaten[^3][^4][^5][^6]. Similarly in a whole series of studies on sensory specific satiety in human subjects based on these discoveries, the reported pleasantness in human subjects of a food is closely correlated with whether it will then be eaten.

[^1]: ACC, anterior cingulate cortex; MSG, monosodium glutamate; OFC, orbitofrontal cortex.
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The Winter meeting of the Nutrition Society was held at the Royal College of Physicians, London on 6–7 December 2011

70th Anniversary Conference on ‘Body weight regulation – food, gut and brain signalling’

Plenary Lecture III

Taste, olfactory and food texture reward processing in the brain and the control of appetite

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Complementary neuronal recordings and functional neuroimaging in human subjects show that the primary taste cortex in the anterior insula provides separate and combined representations of the taste, temperature and texture (including fat texture) of food in the mouth independently of hunger and thus of reward value and pleasantness. One synapse on, in the orbitofrontal cortex (OFC), these sensory inputs are for some neurons combined by learning with olfactory and visual inputs, and these neurons encode food reward in that they only respond to food when hungry, and in that activations correlate with subjective pleasantness. Cognitive factors, including word-level descriptions, and attention modulate the representation of the reward value of food in the OFC and a region to which it projects, the anterior cingulate cortex. Further, there are individual differences in the representation of the reward value of food in the OFC. It is argued that over-eating and obesity are related in many cases to an increased reward value of the sensory inputs produced by foods, and their modulation by cognition and attention that over-ride existing satiety signals. It is proposed that control of all rather than one or several of these factors that influence food reward and eating may be important in the prevention and treatment of overeating and obesity.

Sensory-specific satiety: Fat: Food texture: Taste
eaten, and even with how much is eaten\(^6–8\). (The situation when it has been suggested that wanting is not a result of liking\(^9\) is when behaviour becomes a habit, that is, becomes a stimulus–response type of behaviour that is no longer under control of the goal, but of an overlearned conditioned stimulus\(^1,2\).) The concept here is that food reward normally drives appetite and eating, and it is therefore important to understand the brain mechanisms involved in food reward, the main subject of this paper, in order to understand the control of food intake. Moreover, individual differences in these reward systems may lead to differences in appetite, overeating and obesity; and environmental factors such as the palatability and variety of food available in our modern environment may tend to drive overeating, as described in the section ‘Implications for understanding, preventing and treating obesity’.

**Taste processing in the primate brain**

**Pathways**

A diagram of the taste and related olfactory, somatosensory, and visual pathways in primates is shown in Fig. 1. The multimodal convergence that enables single neurons to respond to different combinations of taste, olfactory, texture, temperature and visual inputs to represent different flavours produced often by new combinations of
sensory input is a theme of recent research that will be described.

The primary taste cortex

Rolls and co-workers have shown that the primary taste cortex in the primate anterior insula and adjoining frontal operculum contains not only taste neurons tuned to sweet, salt, bitter, sour and umami as exemplified by monosodium glutamate (MSG) but also other neurons that encode oral somatosensory stimuli including viscosity, fat texture, temperature and capsaicin. Some neurons in the primary taste cortex respond to particular combinations of taste and oral texture stimuli, but do not respond to olfactory stimuli or visual stimuli such as the sight of food. Neurons in the primary taste cortex do not represent the reward value of taste, that is, the appetite for a food, in that their firing is not decreased to zero by feeding the taste to satiety.

The secondary taste cortex

A secondary cortical taste area in primates was discovered by Rolls et al. in the OFC, extending several millimetres in front of the primary taste cortex. Neurons in this region respond not only to each of the four classical prototypical tastes sweet, salt, bitter and sour but also to umami tastants such as glutamate (which is present in many natural foods such as tomatoes, mushrooms and milk) and inosine monophosphate (which is present in meat and some fish such as tuna). This evidence, taken together with the identification of glutamate taste receptors, leads to the view that there are five prototypical types of taste information channels, with umami contributing, often in combination with corresponding olfactory inputs to the flavour of protein. In addition, other neurons respond to water and other to somatosensory stimuli including astringency as exemplified by tannic acid and capsaicin. Taste responses are found in a large mediolateral extent of the OFC.

The pleasantness of the taste of food, sensory-specific satiety, and the effects of variety on food intake

The modulation of the reward value of a sensory stimulus such as the taste of food by motivational state, for example hunger, is one important way in which motivational behaviour is controlled. The subjective correlate of this modulation is that food tastes pleasant when hungry, and tastes hedonically neutral when it has been eaten to satiety. Following Edmund Rolls’ discovery of sensory-specific satiety revealed by the selective reduction in the responses of lateral hypothalamic neurons to a food eaten to satiety, it has been shown that this is implemented in a region that projects to the hypothalamus, the orbitofrontal cortex, the taste, odour and sight of food.

This evidence shows that the reduced acceptance of food that occurs when food is eaten to satiety, the reduction in the pleasantness of its taste and flavour, and the effects of variety to increase food intake are produced in the OFC, but not at earlier stages of processing where the responses reflect factors such as the intensity of the taste, which is little affected by satiety. In addition to providing an implementation of sensory-specific satiety (probably by habituation of the synaptic afferents to orbitofrontal neurons with a time course of the order of the length of a course of a meal), it is likely that visceral and other satiety-related signals reach the OFC (as indicated in Fig. 1; from the nucleus of the solitary tract, via thalamic and possibly hypothalamic nuclei) and there modulate the representation of food, resulting in an output that reflects the reward (or appetitive) value of each food.

The representation of flavour: convergence of olfactory, taste and visual inputs in the orbitofrontal cortex

Taste and olfactory pathways are brought together in the OFC where flavour is formed by learned associations at the neuronal level between these inputs (see Fig. 1). Visual inputs also become associated by learning in the OFC with the taste of food to represent the sight of food and contribute to flavour. The visual and olfactory as well as the taste inputs represent the reward value of the food, as shown by sensory-specific satiety effects.

The texture of food, including fat texture

Some OFC neurons have oral texture-related responses that encode parametrically the viscosity of food in the mouth (shown using a methyl cellulose series in the range 1–10 000 centiPoise), others independently encode the particulate quality of food in the mouth, produced quantitatively for example by adding 20–100 μm microspheres to methyl cellulose, and others encode the oral temperature of fat as illustrated in Fig. 2. Somatosensory signals that transmit information about capsaicin and astringency are also reflected in neuronal activity in these cortical areas.

In addition, we have shown that some neurons in the OFC reflect the temperature of substances in the mouth, and that this temperature information is represented independently of other sensory inputs by some neurons, and in combination with taste or texture by other neurons.

Imaging studies in human subjects

Taste

In human subjects it has been shown in neuroimaging studies using functional MRI that taste activates a region of the anterior insula/frontal operculum which is probably the primary taste cortex, and part of the OFC, which is probably the secondary taste cortex. Within individual subjects separate areas of the OFC are activated by sweet (pleasant) and by salt (unpleasant) tastes. The primary taste cortex in the anterior insula of human subjects represents the identity and intensity of taste in that activations there correlate with the subjective intensity of the taste, and the orbitofrontal and anterior cingulate...
cortex (ACC) represents the reward value of taste, in that activations there correlate with the subjective pleasantness of taste\(^\text{(58,59)}\).

We also found activation of the human amygdala by the taste of glucose\(^\text{(55)}\). Extending this study, O’Doherty et al.\(^\text{(56)}\) showed that the human amygdala was as much activated by the affectively pleasant taste of glucose as by the affectively negative taste of NaCl, and thus provided evidence that the human amygdala is not especially involved in processing aversive as compared with reward-inducing stimuli. Zald et al.\(^\text{(60)}\) had shown earlier that the amygdala as well as the OFC respond to aversive (saline) taste stimuli.

Umami taste stimuli, of which an exemplar is MSG and which capture what is described as the taste of protein, activate the insular (primary), orbitofrontal (secondary) and anterior cingulate (tertiary\(^\text{(30)}\)) taste cortical areas\(^\text{(61)}\). When the nucleotide 0.005 M inosine 5’-monophosphate was added to MSG (0.05 M), the blood oxygenation-level dependent signal in an anterior part of the OFC showed supralinear additivity, and this may reflect the subjective enhancement of umami taste that has been described when inosine 5’-monophosphate is added to MSG\(^\text{(26)}\). (The supra-linear additivity refers to a greater activation to the combined stimulus MSG + inosine 5’-monophosphate than to the sum of the activations to MSG and inosine 5’-monophosphate presented separately. This evidence that the effect of the combination is greater than the sum of its parts indicates an interaction between the parts to form in this case an especially potent taste of umami, which is part of what can make a food taste delicious\(^\text{(26)}\).) Overall, these results illustrate that the responses of the brain can reflect inputs produced by particular combinations of sensory stimuli with supralinear activations, and that the combination of sensory stimuli may be especially represented in particular brain regions, and may help to make the food pleasant.

### Odour

In human subjects, in addition to activation of the pyriform (olfactory) cortex\(^\text{(62–64)}\), there is strong and consistent activation of the OFC by olfactory stimuli\(^\text{(55,65)}\), and this region appears to represent the pleasantness of odour, as shown by a sensory-specific satiety experiment with banana v. vanilla odour\(^\text{(66)}\). Further, pleasant odours tend to activate the medial, and unpleasant odours the more lateral, OFC\(^\text{(67)}\), adding to the evidence that it is a principle that there is a hedonic map in the OFC, and also in the ACC, which receives inputs from the OFC\(^\text{(32,68)}\). The primary olfactory (pyriform) cortex represents the identity and intensity of odour in that activations there correlate with the subjective intensity of the odour, and the orbitofrontal and ACC represents the reward value of odour, in that activations there correlate with the subjective pleasantness of odour\(^\text{(32,68,69)}\).

**Olfactory-taste convergence to represent flavour, and the influence of satiety**

Supradditive effects indicating convergence and interactions were found for taste (sucrose) and odour (strawberry) in the orbitofrontal and ACC, and activations in these regions were correlated with the pleasantness ratings given by the participants\(^\text{(70–72)}\). These results provide evidence on the neural substrate for the convergence of taste and olfactory stimuli to produce flavour in human subjects, and where the pleasantness of flavour is represented in the human brain.
are represented, is strongly activated by the texture of fat in the mouth and also by oral sucrose\textsuperscript{(75)}. We have recently shown that the pleasantness and reward value of fat texture is represented in the mid-orbitofrontal and ACC, where activations are correlated with the subjective pleasantness of oral fat texture\textsuperscript{(26,74,76)} (Fig. 3). This provides a foundation for studies of whether activations in the fat reward system are heightened in people who tend to become obese.

\textbf{The sight of food}

O’Doherty \textit{et al.}\textsuperscript{(77)} showed that visual stimuli associated with the taste of glucose activated the OFC and some connected areas, consistent with the primate neurophysiology. Simmons, Martin & Barsalou\textsuperscript{(78)} found that showing pictures of foods, compared with pictures of locations, can also activate the OFC. Similarly, the OFC and connected areas were also found to be activated after presentation of food stimuli to food-deprived subjects\textsuperscript{(79)}.

\textbf{Cognitive and selective attentional effects on representations of food}

To what extent does cognition influence the hedonics of food-related stimuli, and how far down into the sensory system does the cognitive influence reach? To address this, we performed a functional MRI investigation in which the delivery of a standard test odour (isovaleric acid combined with cheddar cheese odour, presented orthonasally using an olfactometer) was paired with a descriptor word on a screen, which on different trials was ‘Cheddar cheese’ or ‘Body odour’. Participants rated the affective value of the test odour as significantly more pleasant when labelled ‘Cheddar cheese’ than when labelled ‘Body odour’, and these effects reflected activations in the medial OFC/rostral ACC that had correlations with the pleasantness ratings\textsuperscript{(80)}. The implication is that cognitive factors can have profound effects on our responses to the hedonic and sensory properties of food, in that these effects are manifest quite far down into sensory processing, so that hedonic representations of odours are affected\textsuperscript{(80)}. Similar cognitive effects and mechanisms have now been found for the taste and flavour of food, where the cognitive word level descriptor was for example ‘rich delicious flavour’ and activations to flavour were increased in the OFC and regions to which it projects including the pregenual cingulate cortex and ventral striatum, but were not influenced in the insular primary taste cortex where activations reflected the intensity (concentration) of the stimuli\textsuperscript{(58)} (see Fig. 4).

In addition, we have found that with taste, flavour and olfactory food-related stimuli, selective attention to pleasantness modulates representations in the OFC (see Fig. 5), whereas selective attention to intensity modulates activations in areas such as the primary taste cortex\textsuperscript{(79,81)}. Thus, depending on the context in which tastes and odours are presented and whether affect is relevant, the brain responds to a taste and odour differently. These findings show that when attention is paid to affective value, the brain systems engaged to represent the stimulus are different from...
those engaged when attention is directed to the physical properties of a stimulus such as its intensity. This differential biasing by prefrontal cortex attentional mechanisms\(^{82,83}\) of brain regions engaged in processing a sensory stimulus depending on whether the cognitive demand is for affect-related vs. more sensory-related processing may be an important aspect of cognition and attention which have implications for how strongly the reward system is driven by food, and thus for eating and the control of appetite\(^{59,68,81}\).

**Beyond reward value to decision-making**

Representations of the reward value of food and their subjective correlate the pleasantness of food are fundamental in determining appetite. But after the reward evaluation, a decision has to be made about whether to seek for and consume the reward. We are now starting to understand how the brain takes decisions as described in *The Noisy Brain*\(^{10}\), and this has implications for whether a reward of a particular value will be selected\(^{10,11,32,68,84,85}\). A tier of processing beyond the OFC, in medial prefrontal cortex area 10, becomes engaged when choices are made between odour stimuli based on their pleasantness\(^{86–89}\). The choices are made by a local attractor network in which the winning attractor represents the decision, with each possible attractor representing a different choice, and each attractor receiving inputs that reflect the evidence for that choice. (The attractor network is formed in a part of the cerebral cortex by strengthening of the recurrent collateral excitatory synapses between nearby pyramidal cells. One group of neurons with strengthened synapses between its...
members can form a stable attractor with high firing rates, which competes through inhibitory interneurons with other possible attractors formed by other groups of excitatory neurons\(^{(11,90)}\). The word attractor refers to the fact that inexact inputs are attracted to one of the states of high firing that are specified by the synaptic connections between the different groups of neurons. The result in this non-linear system is that one attractor wins, and this implements a mechanism for decision-making with one winner\(^{(10,11,85,91)}\). The decisions are probabilistic as they reflect the noise in the competitive non-linear decision-making process that is introduced by the random spiking times of neurons for a given mean rate that reflect a Poisson process\(^{(10,89)}\). The costs of each reward need to be subtracted from the value of each reward to produce a net reward value for each available reward before the decision is taken\(^{(11,32,68)}\). The reasoning or rational system with its long-term goals (introducing evidence such as ‘scientific studies have shown that fish oils rich in omega 3 may reduce the probability of Alzheimer’s disease’) then competes with the rewards such as the pleasant flavour of food (which are gene-specified\(^{(1,2)}\), though subject to conditioned effects\(^{(1,92)}\)) in a further decision process which may itself be subject to noise\(^{(1,10,11)}\). This can be described as a choice between the selfish phene (standing for phenotype) and the selfish gene\(^{(2,84,93)}\). In this context, the findings described in this paper that the cognitive system can have a top–down influence on the food reward system are important advances in our understanding of how these decisions are reached.

**Synthesis**

These investigations show that a principle of brain function is that representations of the reward/hedonic value and pleasantness of sensory including food-related stimuli are formed separately from representations of what the stimuli are. The pleasantness/reward value is represented in areas such as the OFC and pregenual cingulate cortex, and it is here that hunger/satiety signals modulate the representations of food to make them implement reward. The satiety signals that help in this modulation may reach the OFC from the hypothalamus, and in turn, the OFC projects to the hypothalamus where neurons are found that respond to the sight, smell and taste of food if hunger is present\(^{(32,33)}\). We have seen earlier some of the principles that help to make the food pleasant, including particular combinations of taste, olfactory, texture, visual and cognitive inputs.

I develop a hypothesis later (section ‘Brain processing of the sensory properties and pleasantness of food’) that obesity is associated with overstimulation of these reward systems by very rewarding combinations of taste, odour, texture, visual and cognitive inputs.

**Implications for understanding, preventing and treating obesity**

Understanding the mechanisms that control appetite is becoming an increasingly important issue, given the increasing incidence of obesity (a threefold increase in the...
UK since 1980 to a figure of 20% defined by a BMI > 30) and the realisation that it is associated with major health risks (with 1000 deaths each week in the UK attributable to obesity). It is important to understand and thereby be able to minimise and treat obesity because many diseases are associated with a body weight that is much above normal. These diseases include hypertension, CVD, hypercholesterolaemia and gall bladder disease; and in addition obesity is associated with some deficits in reproductive function (e.g. ovulatory failure), and with an excess mortality from certain types of cancer (96, 97).

There are many factors that can cause or contribute to obesity in human subjects (96, 97) that are investigated with approaches within or related to neuroscience and psychology (1, 33, 98, 99). Rapid progress is being made in understanding many of these factors at present with the aim of leading to better ways to minimise and treat obesity. These factors include the following (1, 2, 100).

**Genetic factors**

These are of some importance, with some of the variance in weight and RMR in a population of human subjects attributable to inheritance (96, 97). However, the ‘obesity epidemic’ that has occurred since 1990 cannot be attributed to genetic changes, for which the time scale is far too short, but instead to factors such as the increased palatability, variety and availability of food which are some of the crucial drivers of food intake and the amount of food that is eaten in our changed modern environment (1, 33, 98, 99, 101) and that are described later.

**Endocrine factors and their interaction with brain systems**

A small proportion of cases of obesity can be related to gene-related dysfunctions of the peptide systems in the hypothalamus, with for example 4% of obese people having deficient melanocortin 4 receptors for melanocyt stimulating hormone (96, 97). Cases of obesity that can be related to changes in the leptin hormone satiety system are very rare (97). Further, obese people generally have high levels of leptin, so leptin production is not the problem, and instead leptin resistance (i.e. insensitivity) may be somewhat related to obesity, with the resistance perhaps related in part to smaller effects of leptin on arcuate nucleus NPY/AGRP (neuropeptide Y/Agouti-related peptide) neurons (102). However, although there are similarities in fatness within families, these are as strong between spouses as they are between parents and children, so that these similarities cannot be attributed to genetic influences, but presumably reflect the effect of family attitudes to food and weight.

**Brain processing of the sensory properties and palatabness of food**

The way in which the sensory factors produced by the taste, smell, texture and sight of food interact in the brain with satiety signals (such as gastric distension and satiety-related hormones) to determine the palatabness and palatability of food, and therefore whether and how much food will be eaten, is described earlier and shown in Figs. 1 and 6. The concept is that convergence of sensory inputs produced by the taste, smell, texture and sight of food occurs in the OFC to build a representation of food flavour. The OFC is where the pleasantness and palatability of food are represented, as shown by the discoveries that these representations of food are only activated if hunger is present, and correlate with the subjective pleasantness of the food flavour (1, 2, 32, 33, 68, 98, 99). The OFC representation of whether food is pleasant (given any satiety signals present) then drives brain areas such as the striatum and cingulate cortex that then lead to eating behaviour.

The fundamental concept this leads to about some of the major causes of obesity is that, over the last 30 years, sensory stimulation produced by the taste, smell, texture and appearance of food, as well as its availability, have increased dramatically, yet the satiety signals produced by stomach distension, satiety hormones, etc. (103) have remained essentially unchanged, so that the effect on the brain’s control system for appetite (shown in Figs. 1 and 5) is to lead to a net average increase in the reward value and palatability of food which over rides the satiety signals, and contributes to the tendency to be overstimulated by food and to overeat.

In this scenario, it is important to understand much better the rules used by the brain to produce the representation of the pleasantness of food and how the system is modulated by eating and satiety. This understanding, and how the sensory factors can be designed and controlled so as not to override satiety signals, are important research areas in the understanding, prevention and treatment of obesity. Advances in understanding the receptors that encode the taste and olfactory properties of food (22, 104), and the processing in the brain of these properties (1, 99, 105), are also important in providing the potential to produce highly palatable food that is at the same time nutritious and healthy.

An important aspect of this hypothesis is that different human subjects may have reward systems that are especially strongly driven by the sensory and cognitive factors that make food highly palatable. In a test of this, we showed that activation to the sight and flavour of chocolate in the orbitofrontal and pregenual cingulate cortex were much higher in chocolate cravers than non-cravers (106). This concept that individual differences in responsiveness to food reward are reflected in brain activations in regions related to the control food intake (106, 107) may provide a way for understanding and helping to control food intake.

**Food palatability**

A factor in obesity (as described in section ‘Brain processing of the sensory properties and pleasantness of food’) is food palatability, which with modern methods of food production can now be greater than would have been the case during the evolution of our feeding control systems. These brain systems evolved so that internal signals from for example gastric distension and glucose utilisation could act to decrease the pleasantness of the sensory sensations produced by feeding sufficiently by the end of a meal to stop further eating (1, 99, 105). However, the
greater palatability of modern food may mean that this balance is altered, so that there is a tendency for the greater palatability of food to be insufficiently decreased by a standard amount of food eaten, so that extra food is eaten in a meal (see Fig. 6).

**Sensory-specific satiety and the effects of variety on food intake**

Sensory-specific satiety is the decrease in the appetite for a particular food as it is eaten in a meal, without a decrease in the appetite for different foods\(^{(1,99,105)}\), as shown earlier. It is an important factor influencing how much of each food is eaten in a meal, and its evolutionary significance may be to encourage eating of a range of different foods, and thus obtaining a range of nutrients. As a result of sensory-specific satiety, if a wide variety of foods is available, overeating in a meal can occur. Given that it is now possible to make available a very wide range of food flavours, textures and appearances, and that such foods are readily available, this variety effect may be a factor in promoting excess food intake.

**Fixed meal times and the availability of food**

Another factor that could contribute to obesity is fixed meal times, in that the normal control of food intake by alterations in inter-meal interval is not readily available in human subjects, and food may be eaten at a meal-time even if hunger is not present\(^{(114)}\). Even more than this, because of the high and easy availability of food (in the home and workplace) and stimulation by advertising, there is a tendency to start eating again when satiety signals after a previous meal have decreased only a little, and the consequence is that the system again becomes overloaded.

**Food saliency and portion size**

Making food salient, for example by placing it on display, may increase food selection particularly in the obese\(^{(108,109)}\), and portion size is a factor, with more being eaten if a large portion of food is presented\(^{(110)}\), though it is not yet clear whether this is a factor that can lead to obesity and not just alter meal size. The driving effects of visual and other stimuli, including the effects of advertising, on the brain systems that are activated by food reward may be different in different individuals, and may contribute to obesity.

**Energy density of food**

Although gastric emptying rate is slower for high-energy density foods, this does not fully compensate for the energy density of the food\(^{(111,112)}\). The implication is that eating energy dense foods (e.g. high-fat foods) may not allow gastric distension to contribute sufficiently to satiety. Because of this, the energy density of foods may be an important factor that influences how much energy is consumed in a meal\(^{(116,113)}\). Indeed, it is notable that obese people tend to eat foods with high-energy density, and to...
visits to restaurants with high-energy density (e.g. high-fat) foods. It is also a matter of clinical experience that gastric emptying is faster in obese than in thin individuals, so that gastric distension may play a less effective role in contributing to satiety in the obese. It is also important to remember that the flavour of a food can be conditioned to its energy density, leading over a few days to more eating of low than high-energy dense foods, in the phenomenon known as conditioned satiety(1,92).

Eating rate

A factor related to the effects described in section ‘Energy density of food’ is eating rate, which is typically fast in the obese, and may provide insufficient time for the full effect of satiety signals as food reaches the intestine to operate.

Stress

Another potential factor in obesity is stress, which can induce eating and could contribute to a tendency to obesity. (In a rat model of this, mild stress in the presence of food can lead to overeating and obesity. This overeating is reduced by anti-anxiety drugs.)

Food craving

Binge eating has some parallels to addiction. In one rodent model of binge eating, access to sucrose for several hours daily can lead to binge-like consumption of the sucrose over a period of days114. The binge-eating is associated with the release of dopamine. This model brings binge eating close to an addictive process, at least in this model, in that after the binge-eating has become a habit, sucrose withdrawal decreases dopamine release in the ventral striatum (a part of the brain involved in addiction to drugs such as amphetamine), altered binding of dopamine to its receptors in the ventral striatum is produced, and signs of withdrawal from an addiction occur including teeth chattering. In withdrawal, the animals are also hypersensitive to the effects of amphetamine. Another rat model is being used to investigate the binge eating of fat, and whether the reinforcing cues associated with this can be reduced by the gamma-aminobutyric acid B receptor agonist baclofen114.

Energy output

If energy intake is greater than energy output, body weight increases. Energy output is thus an important factor in the equation. However, studies in human subjects show that although exercise has health benefits, it does not have very significant effects on body weight gain and adiposity in the obese or those who become obese115,116. These findings help to emphasise the importance of understanding the factors that lead to overeating, including factors such as increased responsiveness of the reward system for food in some individuals, and the effects described here that contribute to reward signals produced in modern society being greater than the satiety signals, which have not changed from those in our evolutionary history(101).

Cognitive factors and attention

As described earlier, cognitive factors, such as preconceptions about the nature of a particular food or odour, can reach down into the olfactory and taste system in the OFC which controls the palatability of food to influence how pleasant an olfactory, taste, or flavour stimulus is58,80. This has implications for further ways in which food intake can be controlled by cognitive factors, and this needs further investigation. For example, the cognitive factors that have been investigated in these studies are descriptors of the reward value of the food, such as ‘rich and delicious’. But it could be that cognitive descriptions of the consequences of eating a particular food, such as ‘this food tends to increase body weight’, ‘this food tends to alter your body shape towards fatness’, ‘this food tends to make you less attractive’, ‘this food will reduce the risk of a particular disease’, etc., could also modulate the reward value of the food as it is represented in the OFC. If so, these further types of cognitive modulation could be emphasised in the prevention and treatment of obesity.

Further, attention to the affective properties of food modulates processing of the reward value of food in the OFC59,81, and this again suggests that how attention is directed may be important in the extent to which food over-stimulates food intake. Not drawing attention to the reward properties of food, or drawing attention to other properties such as its nutritional value and energy content, could reduce the activation of the brain’s reward system by the food, and could be another useful way to help prevent and treat obesity.

The neuroscience and psychology of compliance with information about risk factors for obesity

It is important to develop better ways to provide information that will be effective in the long term in decreasing food intake while maintaining a healthy diet, and in promoting an increase in energy expenditure by for example encouraging exercise. In this respect, the individual differences in the brain’s response to the reward value of a food, found for example in our study with chocolate cravers and non-cravers106, is one type of factor that may influence whether an individual can comply. But there are individual differences in other factors that may influence compliance, such as impulsiveness, and the OFC is implicated in this117–119. It is important to better understand possible individual differences in the ability for an individual to stop, and be influenced by the reasoning system with its long-term interests in comparison with the immediate rewards specified by genes11,12,118. It could also be that substances such as alcohol shift this balance, making an individual temporarily or possibly in the long term more impulsive and less under control of the reasoning executive system120, and therefore more likely to eat, and to eat unhealthily. These effects of alcohol on impulsiveness may be complemented by hormonal processes121. Understanding these processes, and enabling individuals to benefit from this understanding, may also be useful in the prevention and treatment of obesity.
Overall, I suggest that understanding of all the afore-mentioned processes, and their use in combination rather than purely individually, may provide new avenues to the control of overeating and body weight. I have outlined in this paper a number of factors that may tend to promote overeating and obesity in our modern society, for example by increasing the impact of reward signals on the brain’s appetitive control system, or by making it difficult for individuals to resist the increased hedonic value of food. It is possible that any one of these, or a few in combination, could produce overeating and obesity. In these circumstances, to prevent and treat obesity it is unlikely to be sufficient to reduce and focus on or test just one or a few of these factors. As there are many factors, there may always be others that apply and that tend to promote overeating and obesity. The conclusion I therefore reach is that to prevent and treat obesity, it may be important to address all of the afore-mentioned factors together, given that any one, or a few, could tend to lead to overeating and obesity. The science I have described suggests that taking this overall approach, minimising the impact of all these factors, could be an important aim for future research and strategy.

Acknowledgements

This research was supported by the Medical Research Council. The participation of many colleagues in the studies cited is sincerely acknowledged. The author declares no conflict of interest.

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