Cortical Processing of the Reward Value of Food

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The Pleasantness of the Taste of Food

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 behavior is controlled. The subjective correlate of this modulation is that food tastes pleasant when a person is hungry, and tastes hedonically neutral when a person has eaten to satiety. To make the results relevant to understanding these processes and to the control of food intake in humans, complementary evidence is provided by neurophysiological studies in nonhuman primates, and by functional neuroimaging studies in humans.

The macaque taste and olfactory system may be particularly relevant to understanding these processes in humans, for the primate taste system does not have a pontine taste area and direct subcortical connections, but instead relies on thalamic-to-cortical processing.

Rolls and colleagues have found that the modulation of taste-evoked signals by motivation is not a property found in early stages of the primate gustatory system. The responsiveness of taste neurons in the nucleus of the solitary tract and in the primary taste cortex (in the frontal operculum and anterior insula) is not attenuated by feeding to satiety. (Figure 1 shows a schematic diagram of a lateral view of some of the primate brain taste, olfactory, visual, and somatosensory pathways.) In contrast, in the secondary taste cortex, in the caudolateral part of the orbitofrontal cortex, it has been shown that the responses of the neurons to the taste of glucose decreased to zero while the monkey ate it to satiety, during the course of which the behavior turned from avid acceptance to active rejection. This modulation of responsiveness of the gustatory responses of the orbitofrontal cortex neurons by satiety could not have been due to peripheral adaptation in the gustatory system or to altered efficacy of gustatory stimulation after satiety was reached, because modulation of neuronal responsiveness by satiety was not seen at the earlier stages of the gustatory system, including the nucleus of the solitary tract, the frontal opercular taste cortex, and the insular taste cortex.

Sensory-Specific Satiety

In the secondary taste cortex, it was also found that the decreases in the responsiveness of the neurons were relatively specific to the food with which the monkey had been fed to satiety. For example, in seven experiments in which the monkey was fed glucose solution, neuronal responsiveness decreased to the taste of the glucose but not to the taste of blackcurrant juice (see example in Figure 2). Conversely, in two experiments in which the monkey was fed to satiety with fruit juice, the responses of the neurons decreased to fruit juice but not to glucose. An example of this sensory-specific decrease in the responsiveness of a neuron after feeding to satiety is shown in Figure 2. This neuron had taste, olfactory, and visual responses to food, and the satiety was produced by a food, blackcurrant juice, with of all these components.

This evidence shows that the reduced acceptance of food which occurs when food is eaten to satiety, and the reduction in the pleasantness of its taste, are not produced by a reduction in the responses of neurons in the nucleus of the solitary tract or frontal opercular or insular gustatory cortices to gustatory stimuli. Indeed, after feeding to satiety, humans reported that the taste of the food on which they had been satiated tasted almost as intense as when they were hungry, though much less pleasant. This comparison is consistent with the possibility that activity in the frontal opercular and insular taste cortices, as well as in the nucleus of the solitary tract, does not reflect the pleasantness of the taste of a food, but rather its sensory qualities, independently of motivational state. On the other hand, the responses of the neurons in the caudolateral orbitofrontal cortex taste area and in the lateral hypothalamus are modulated by satiety, and it is presumably in areas such as these that neuronal activity may be related to whether a food tastes pleasant, and to whether the food should be eaten. 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 orbitofrontal cortex (as indicated in Figure 1) (from the nucleus of the solitary tract, via thalamic areas), and there modulate the representation of food, resulting in an output that reflects the reward (or appetitive) value of each food.

It is an important principle that the identity of a taste, and its intensity, are represented separately from its pleasantness. Thus it is possible to represent what a taste is, and to learn about it, even when we are not hungry.
Representation of the Pleasantness of the Smell and Sight of Food in the Brain

It has also been possible to investigate whether the olfactory representation in the orbitofrontal cortex is affected by hunger, and thus whether the pleasantness of odor is represented in the orbitofrontal cortex. In satiety experiments, Critchley and Rolls showed that the responses of some olfactory neurons to a food odor are decreased during feeding to satiety with a food (e.g., fruit juice) containing that odor. In particular, seven of nine olfactory neurons that were responsive to the odors of foods, such as blackcurrant juice, were found to decrease their responses to the odor of the satiating food. The decrease was typically at least partly specific to the odor of the food that had been eaten to satiety, potentially providing part of the basis for sensory-specific satiety. It was also found for eight of nine orbitofrontal cortex neurons that had selective responses to the sight of food that they demonstrated a sensory-specific reduction in their visual responses to foods following satiation. These findings show that the olfactory and visual representations of food, as well as the taste representation of food, in the primate orbitofrontal cortex are modulated by hunger. Usually a component related to sensory-specific satiety can be demonstrated. Hunger does not influence the responses of neurons in the inferior temporal visual cortex to visual stimuli, so that the representation of the pleasantness or reward

Figure 1 Some of the pathways involved in sensory processing involved in the control of food intake and emotion are shown on this lateral view of the brain of the macaque monkey. Connections from the primary taste and olfactory cortices to the orbitofrontal cortex and amygdala are shown. Connections are also shown in the ‘ventral visual system’ from V1 to V2, V4, the inferior temporal visual cortex, etc., with some connections reaching the amygdala and orbitofrontal cortex. In addition, connections from the somatosensory cortical areas 1, 2, and 3 that reach the orbitofrontal cortex directly and via the insular cortex, and that reach the amygdala via the insular cortex, are shown. as, arcuate sulcus; cal, calcarine sulcus; cs, central sulcus; If, lateral (or Sylvian) fissure; lun, lunate sulcus; ps, principal sulcus; io, inferior occipital sulcus; ip, intraparietal sulcus (which has been opened to reveal some of the areas it contains); sts, superior temporal sulcus (which has been opened to reveal some of the areas it contains); AIT, anterior inferior temporal cortex; FST, visual motion processing area; LIP, lateral intraparietal area; MST, visual motion processing area; MT, visual motion processing area (also called V5); PIT, posterior inferior temporal cortex; STP, superior temporal plane; TA, architectonic area including auditory association cortex; TE, architectonic area including high-order visual association cortex, and some of its subareas TEa and TEM; TG, architectonic area in the temporal pole; V1–V4, visual areas V1–V4; VIP, ventral intraparietal area; TEO, architectonic area including posterior visual association cortex. The numerals refer to architectonic areas, and have the following approximate functional equivalence: 1, 2, 3, somatosensory cortex (posterior to the central sulcus); 4, motor cortex; 5, superior parietal lobule; 7a, inferior parietal lobule, visual part; 7b, inferior parietal lobule, somatosensory part; 6, lateral premotor cortex; 8, frontal eye field; 12, part of orbitofrontal cortex; 46, dorsolateral prefrontal cortex.
value of the sight of food is a special property of the orbitofrontal cortex, which receives visual inputs from the inferior temporal visual cortex.

These findings link at least part of the processing of olfactory and visual information in this brain region to the control of feeding-related behavior. This is further evidence that part of the olfactory representation in this region is related to the hedonic value of the olfactory stimulus, and in particular that at this level of the olfactory system in primates, the pleasure elicited by the food odor is at least part of what is represented.

As a result of the neurophysiological and behavioral observations showing the specificity of satiety in the monkey, experiments were performed to determine whether satiety was specific to foods eaten in humans. It was found that the pleasantness of the taste of food eaten to satiety decreased more than for foods that had not been eaten. One consequence of this is that if one food is eaten to satiety, appetite reduction for other foods is often incomplete, and this will lead to enhanced eating when a variety of foods is offered. Because sensory factors such as similarity of color, shape, flavor, and texture are usually more important than are metabolic equivalences in terms of protein, carbohydrate, and fat content, in influencing how foods interact in this type of satiety, this has been termed ‘sensory-specific satiety.’ It should be noted that this effect is distinct from alliesthesia, in that alliesthesia is a change in the pleasantness of sensory inputs produced by internal signals (such as glucose in the gut), whereas sensory-specific satiety is a change in the pleasantness of sensory inputs and is accounted for at least partly by the external sensory stimulation received (such as the taste of a particular food), in that it is at least partly specific to the external sensory stimulation received.

To investigate whether the sensory-specific reduction in the responsiveness of the orbitofrontal olfactory neurons might be related to a sensory-specific reduction in the pleasure produced by the odor of a food when it is eaten to satiety, Rolls and Rolls measured humans’ responses to the smell of a food which was eaten to satiety. It was found that the pleasantness of the odor of a food (but much less significantly its intensity) was decreased when persons ate the food to satiety. It was also found that the pleasantness of the smell of other foods (i.e., foods not eaten in the meal) showed much less decrease. This finding has clear implications for the control of food intake, for ways to keep foods presented in a meal appetitive, and for effects on odor pleasantness ratings that could occur following meals.

In an investigation of the mechanisms of this odor-specific, sensory-specific satiety, Rolls and Rolls...
allowed humans to chew a food without swallowing, for approximately as long as the food is normally in the mouth during eating. They demonstrated sensory-specific satiety with this procedure, showing that the sensory-specific satiety does not depend on food reaching the stomach. Thus at least part of the mechanism is likely to be produced by a change in processing in the olfactory pathways. It is not yet known which stage of olfactory processing is the earliest at which this modulation occurs. It is unlikely to be in the receptors, because the change in pleasantness found was much more significant than was the change in the intensity.

The increase of food intake that can occur when a variety of foods is available, as a result of the operation of sensory-specific satiety, may have been advantageous in evolution in ensuring that different foods with important different nutrients were consumed. However, today, humans have a wide variety of foods readily available, and this may be a factor that can lead to overeating and obesity. In a test of this in the rat, it has been found that variety itself can lead to obesity.

**Mouth Feel of Fat: Orbitofrontal Cortex, Primary Taste Cortex, and Amygdala**

Texture in the mouth is an important indicator of whether fat is present in a food, which is important not only as a high-value energy source, but also as a potential source of essential fatty acids. In the orbitofrontal cortex, Rolls, Critchley, and colleagues have found a population of neurons that responds when fat is in the mouth. An example of such a neuron is shown in Figure 3. The fat-related responses of these neurons are produced at least in part by the texture of the food rather than by chemical receptors sensitive to certain chemicals, in that such neurons typically respond not only to foods, such as cream and milk, containing fat, but also to paraffin oil (which is a pure hydrocarbon) and to silicone oil (Si(CH₃)₂O)n. Moreover, the texture channels through which these fat-sensitive neurons are activated are separate from viscosity-sensitive channels, in that the responses of these neurons cannot be predicted by the viscosity of the oral stimuli, as illustrated in Figure 3. Some of the fat-related neurons do, though, have convergent inputs from the chemical senses, in that in addition to taste inputs, some of these neurons respond to the odor associated with a fat, such as the odor of cream. Feeding to satiety with fat (e.g., cream) decreases the responses of these neurons to zero on the food eaten to satiety, but if the neuron receives a taste input from, for example, glucose taste, that is not decreased by feeding to satiety with cream. Thus there is a representation of the macronutrient fat in this brain area, and the activation produced by fat is reduced by eating fat to satiety.

For some neurons, a combination of taste, fat texture, oral viscosity, and temperature is represented in the macaque primary taste cortex in the rostral insula and adjoining frontal operculum. These oral sensory properties of food, and also the sight and smell of food, are also represented in the primate amygdala. Interestingly, the responses of these amygdala neurons do not correlate well with the preferences of the macaques for the oral stimuli, and feeding to

![Figure 3](image-url)
satiety does not produce the large reduction in the responses of primate amygdala neurons to food that is typical of orbitofrontal cortex neurons.

**Imaging Studies in Humans**

**Taste**

In humans it has been shown in neuroimaging studies using functional magnetic resonance imaging (fMRI) that taste activates an area of the anterior insula/frontal operculum, which is probably the primary taste cortex, and part of the orbitofrontal cortex, which is probably the secondary taste cortex. O’Doherty et al. 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 to rewarding stimuli.

Another study has shown that umami taste stimuli, of which an exemplar is monosodium glutamate (MSG) and which captures what is described as the taste of protein, activate cortical regions of the human taste system similar to those activated by a prototypical taste stimulus, glucose. A part of the rostral anterior cingulate cortex (ACC) was also activated. When the nucleotide 0.005 M inosine 5'-monophosphate (IMP) was added to MSG (0.05 M), the blood oxygenation-level-dependent (BOLD) signal in an anterior part of the orbitofrontal cortex showed supralinear additivity, and this may reflect the subjective enhancement of umami taste that has been described when IMP is added to MSG. 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.

**Odor**

In an investigation of where the pleasantness of olfactory stimuli might be represented in humans, O’Doherty et al. showed that the activation of an area of the orbitofrontal cortex to banana odor was decreased (relative to a control vanilla odor) after bananas were eaten to satiety. Thus activity in a part of the human orbitofrontal cortex olfactory area is related to sensory-specific satiety, and this is one brain region where the pleasantness of odor is represented.

An important issue is whether there are separate regions of the brain discriminable with fMRI that represent pleasant and unpleasant odors. To investigate this, we measured the brain activations produced by three pleasant and three unpleasant odors. The pleasant odors chosen were linalyl acetate (floral, sweet), geranyl acetate (floral), and Z-ionone (woody, slightly food related). The unpleasant odors chosen were hexanoic acid, octanol, and isovaleric acid. We found that these unpleasant odors activated disassociable parts of the human brain. Pleasant, but not unpleasant, odors were found to activate a medial region of the orbitofrontal cortex. Further, there was a correlation between the subjective pleasantness ratings of the six odors given during the investigation with activation of a medial region of the rostral orbitofrontal cortex. In contrast, a correlation between the subjective unpleasantness ratings of the six odors was found in regions of the left and more lateral orbitofrontal cortex. Activation was also found in the anterior cingulate cortex, with a middle part of the anterior cingulate activated by both pleasant and unpleasant odors, and a more anterior part of the anterior cingulate cortex showing a correlation with the subjective pleasantness ratings of the odors. These results provide evidence that there is a hedonic map of the sense of smell in brain regions such as the orbitofrontal cortex and cingulate cortex.

**Olfactory–Taste Convergence to Represent Flavor, and the Influence of Satiety**

To assess how satiety influences the brain activations to a whole food which produces taste, olfactory, and texture stimulation, we measured brain activation by whole foods before and after the food is eaten to satiety. The aim was to determine using a food that has olfactory, taste, and texture components the extent of the region that shows decreases when the food becomes less pleasant, in order to identify the different brain areas where the pleasantness of the odor, taste, and texture of food are represented. The foods eaten to satiety were either chocolate milk or tomato juice. A decrease in activation by the food eaten to satiety relative to the other food was found in the orbitofrontal cortex, but not in the primary taste cortex (see Figure 4). This study provided evidence that the pleasantness of the flavor of food, and sensory-specific satiety, are represented in the orbitofrontal cortex.

**Oral Viscosity and Fat Texture**

The viscosity of food in the mouth is represented in the human primary taste cortex (in the anterior insula), and also in a midinsular area that is not taste cortex, but which represents oral somatosensory stimuli. In these regions, the fMRI BOLD activations are proportional to the log of the viscosity of carboxymethyl cellulose in the mouth. Oral viscosity is also represented in the human orbitofrontal and perigenual cingulate cortices, and it is notable that the perigenual
cingulate cortex, an area in which many pleasant stimuli are represented, is strongly activated by the texture of fat in the mouth and also by oral sucrose.

The Sight of Food

Visual stimuli associated with the taste of glucose activate the orbitofrontal cortex and some connected areas, consistent with the primate neurophysiology. Consistent with these findings, Pelchat and colleagues found that after consuming a monotonous diet, study participants who were instructed to imagine foods that they craved showed more activation in some brain areas, including part of the insula, than did participants who had consumed a normal diet.

Satiety Signals

Satiety signals can be thought of as occurring in a sequence. In addition to sensory-specific satiety, which involves reduced activity in cortical areas that represent the pleasantness of food, further temporally overlapping signals include gastric distension, duodenal chemosensory signals, glucose utilization, and hormonal (including leptin) signals. Many of these signals are likely to have influences, in some cases via the lateral hypothalamus, on the orbitofrontal cortex, and by modulating sensory activity there, to set the current pleasantness and reward value of food. Investigation of hunger and satiety signals has been a major and difficult issue for many years, but whatever the details of these signals, they must influence processing in brain areas such as the orbitofrontal cortex. The outputs of the orbitofrontal cortex then reach brain regions such as the striatum, cingulate cortex, and dorsolateral prefrontal cortex, where behavioral responses to food may be elicited, because these structures produce behavior which makes the orbitofrontal cortex reward neurons fire, as they represent a goal for behavior. At the same time, outputs from the orbitofrontal cortex and amygdala, in part via the hypothalamus, may provide for appropriate autonomic and endocrine responses to food to be produced, including the release of hormones such as insulin.

See also: Central Gustatory System and Ingestive Behavior; Conditioned Taste Aversion; Flavor Physiology; Food and Water Intake: Regulation; Gastrointestinal Signals: Stimulation; Gastrointestinal Signals: Satiety;
Neuropeptides: Food Intake; Olfactory Cortex Physiology; Retronasal Olfaction; Reward Neurophysiology and Orbitofrontal Cortex; Sensory Aging: Chemical Senses; Taste: Vertebrate Taste Bud Physiology; Taste: Vertebrate Central Pathways.

Further Reading