OXFORD

Cerebral Cortex, 2025, **35**, bhaf248
https://doi.org/10.1093/cercor/bhaf248
Advance access publication date 16 September 2025
Original Article

Reward-specific satiety and reward-specific motivation: neural bases and significance

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How choices are made between rewards is fundamental to understanding the behavior of humans and most other vertebrates. A key factor in the choices is reward-specific satiety, which is the sensory-specific decrease in the reward value of a particular reward when it is consumed to satiety. Another key factor is reward-specific motivation, the increase in the reward value of a reward when it is first provided. Here, we develop the theory based on experimental evidence in humans and other primates, that reward-specific satiety is implemented in orbitofrontal cortex reward value neurons by adaptation in the synapses from visual and taste cortical regions in which the neuronal firing is not influenced by reward-specific satiety. Correspondingly we develop the theory that reward-specific motivation (or incentive motivation) is implemented by shorter-term synaptic facilitation in the same synapses on to orbitofrontal cortex reward value neurons. We complement the theories with an integrate-and-fire neuronal network model of how these reward value computations are performed in the orbitofrontal cortex by synaptic adaptation and synaptic facilitation in the afferent connections to orbitofrontal cortex reward value neurons, to implement a profound influence on behavioral choice that has great adaptive value for humans and many other animals.

Keywords: food appetite control; incentive motivation; orbitofrontal cortex; reward value; sensory-specific satiety.

Introduction

Reward-specific satiety is the decrease in the reward value that occurs while a reward is being consumed until its value decreases in a way that is specific to the reward being consumed. An example of reward-specific satiety is eating one food, such as chicken, to satiety, which leaves the reward value and pleasantness of other foods (such as bananas) high, which can lead to overconsumption if a variety of rewards is offered (Rolls 2016a). Reward-specific motivation is the reward-specific increase in the reward value and pleasantness of a rewarding stimulus such as food soon after it is presented. An example of reward-specific motivation (or incentive motivation) is the smell of bread cooking in a bakery, which can increase the reward value of fresh bread, and encourage an individual to want to buy the fresh bread. These changes of reward value are not only fundamental to understanding appetite control, but also to most rewarded and emotional behavior, for reward-specific satiety and reward-specific motivation are properties of almost all of our reward systems, and none of our punishment systems (Rolls 2014, 2016b, 2023a, 2023b, 2025c, 2025b).

Here we present a theory based on the experimental evidence of how reward-specific satiety and reward-specific motivation are implemented in the orbitofrontal cortex of humans and other primates, add to this an integrate-and-fire computational model of the implementation by synaptic adaptation and facilitation, and consider some of the important implications for the choices that we make of these aspects of the design of reward systems in our brains. The processes described here are key to understanding many aspects of human decision-making between rewards.

In addition, there is an increase in reward value that occurs early on after a reward is offered that is called incentive motivation, or here, reward-specific motivation (Rolls 2014, 2023a) [and the salted nut phenomenon (Hebb 1949)]. Reward-specific motivation has biological adaptive value by locking an animal onto a reward for at least some time, which is a much more efficient foraging strategy than changing behavior to find a different reward whenever the reward value drops a little, as the two rewards might be far apart (Rolls 2014, 2023a). Reward-specific motivation, and reward-specific satiety, may it is proposed be a key part of the neural mechanism involved in "exploit vs explore" in foraging (Rolls 2023a), in which the frontal pole cortex is also implicated (Rolls et al. 2024a).

In the research described here, a theory is developed from the experimental evidence about how reward-specific satiety and reward-specific motivation are implemented in the brain, and then an integrate-and-fire neuronal network model of how reward-specific satiety and reward-specific motivation are computed in the orbitofrontal cortex is presented. This is the first theory and model we know of how these reward-specific modulations of reward value are implemented in our brains, and it is emphasized that these modulations are fundamental to understanding much behavior including emotion, motivation,

Received: May 5, 2025. **Revised:** July 31, 2025. **Accepted:** August 14, 2025

reward-related decision-making, and foraging, because they apply to most reward systems, and to no punishment systems (Rolls 2023a, 2023b).

The significance and importance of the research described here is that reward value is fundamental in understanding the choice behavior and reward-related decision-making of humans and other vertebrates. Understanding the temporal properties of our reward systems, and their biological adaptive value, is fundamental to understanding much human rewarded, motivated, and emotional behavior (Rolls 2014, 2023b, 2023a). Here a simple mechanism that can produce much of the richness of how the reward systems of humans and other primates operate in the short to medium time-scale is proposed, based on the experimental evidence.

A theory of the synaptic mechanism of the reward-specific decrease in reward value produced by satiety

Experimental evidence

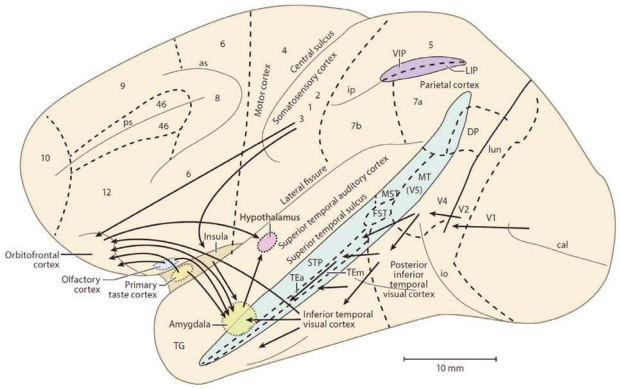
The experimental evidence on which the theory is based includes the following, some included in Fig. 1. Sensory-specific or reward-specific satiety was discovered during recordings from single neurons in the primate lateral hypothalamus (Rolls 1981; Rolls et al. 1986), and then in the orbitofrontal cortex (Fig. 1a), where neurons show reward-specific satiety-related decreases of their response to the taste of food (Rolls et al. 1989) (see example in Fig. 1b), to the sight and smell of food (Critchley and Rolls 1996), and to the oral texture of fat such as cream in the mouth (Rolls et al. 1999). Consistent findings for the primate orbitofrontal cortex have been reported in terms of subjective value signals during multicomponent economic choice (Pastor et al. 2021). These discoveries were followed up with human functional magnetic resonance imaging, in which we showed that the sensory-specific decrease in the subjective ratings of the pleasantness of food produced by eating a food to satiety were related to the sensory-specific decrease of the activations in the orbitofrontal cortex produced by feeding that food to satiety (Kringelbach et al. 2003). There is a comparable effect of a decrease in the neuronal responses and activations in the orbitofrontal cortex to water in the mouth and of the pleasantness ratings to water in the mouth when water is drunk to satiety (Rolls et al. 1989; de Araujo et al. 2003). The discovery of reward-specific satiety by neurophysiology was followed up by demonstrations of its specificity at the human subjective pleasantness and food intake levels in humans, and how as a result variety can increase food intake (Rolls et al. 1981a; Rolls et al. 1981b; Rolls et al. 1982a; Rolls et al. 1983; Hetherington 1996; Cunningham et al. 2023; Rolls 2025a, 2026). These studies show that reward-specific satiety is one of the most important factors that influence how much food is eaten in a meal, but even more importantly, is a key way in which humans and other animals are led to eat a variety of food, which has the evolutionary adaptive value of promoting eating of a range of different nutrients, but the risk of promoting overeating and obesity if a wide variety of food is available (Rolls 2016a).

To examine where the first stage in neural processing is at which reward-specific satiety is represented, we recorded from the primate primary taste cortex which provides the taste inputs to the orbitofrontal cortex (Baylis et al. 1995), and found that neurons in the insular (Yaxley et al. 1988) and frontal opercular (Rolls et al. 1988) primary taste cortex are not influenced by feeding to satiety, and continue to respond to the taste after devaluation. Consistent with this, in humans the activations measured to taste with functional magnetic resonance imaging (fMRI) in the orbitofrontal cortex are related to the pleasantness of the taste, whereas activations in the insular taste cortex are related to the intensity of the taste and not its pleasantness (Grabenhorst and Rolls 2008). Thus the insular taste cortex is in tier 1 in Fig. 1c, where the identity of a sensory stimulus is represented independently of its reward or punishment value. Further, we showed that in the inferior temporal visual cortex, which provides visual inputs to orbitofrontal cortex neurons, sensory-specific satiety does not reduce the responses of neurons to the sight of food (Rolls et al. 1977). Further, inferior temporal cortex neurons do not represent the reward value of stimuli, in that their responses remain unaltered when a visual stimulus is reversed from being rewarding to punishing (Rolls et al. 1977; Aggelopoulos et al. 2005). Thus the inferior temporal visual cortex is in tier 1 in Fig. 1c, where the identity of a sensory stimulus is represented independently of its reward or punishment value. In contrast, orbitofrontal cortex neurons show effects of devaluation by feeding to satiety and sensory-specific satiety, and reverse the visual and olfactory stimuli to which they respond in as little as one trial when the reward value of the stimuli is reversed (Thorpe et al. 1979; Rolls et al. 1996; Rolls 2015). Moreover, damage to the human orbitofrontal cortex impairs reward value learning in reward reversal tasks, and impairs behaviors associated with reward such as emotion and motivation (Rolls et al. 1994; Hornak et al. 1996; Bechara et al. 2000; Fellows and Farah 2003; Hornak et al. 2003; Berlin et al. 2004; Berlin and Rolls 2004; Hornak et al. 2004; Berlin et al. 2005; Fellows and Farah 2005; Heberlein et al. 2008; Wheeler and Fellows 2008; Camille et al. 2011; Fellows 2011; Noonan et al. 2017; Rolls 2023a, 2023b). Thus the orbitofrontal cortex is in tier 2 in Fig. 1c, where the reward or punishment value of stimuli is represented (Rolls 2023a, 2023b). Importantly, in primates including humans, the orbitofrontal cortex represents reward value and not actions, but has outputs directed to different response and action systems in tier 3 (Fig. 1c), as described elsewhere (Rolls 2023a, 2023b, 2025b). In contrast, the rodent orbitofrontal cortex is very much less developed, may not be the first cortical region in which reward value is represented, and also may encode behavioral responses and actions, so rodents provide a poor model of the systems-level organization of reward systems in primates including humans (Rolls 2023a, 2023b).

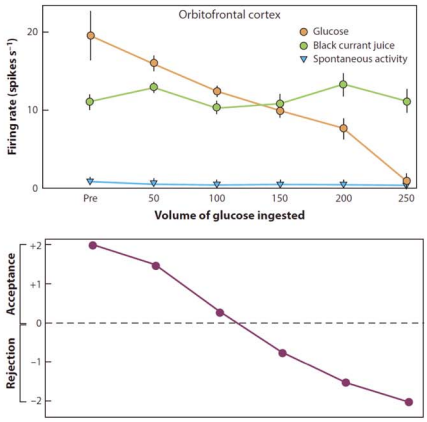
This evidence indicates that reward-specific satiety is represented in orbitofrontal cortex taste and visual reward value neurons which is where reward value is represented in primates including humans, but not in the previous stages of processing, the insular taste cortex and the inferior temporal visual cortex, where there is a perceptual representation in primates including humans that is not related to reward value. fMRI evidence in humans shows a similar situation for olfactory stimuli, with the pleasantness of olfactory stimuli represented in the orbitofrontal cortex, but not in the preceding pyriform olfactory cortex where intensity was represented (Rolls et al. 2008). Of course, reward value and reward-specific satiety may be represented in brain regions to which the orbitofrontal cortex projects, and indeed that is what we showed for lateral hypothalamic neurons that respond to the sight or taste of food (Rolls et al. 1986).

Further relevant evidence is that some sensory-specific reduction in the pleasantness of the smell of a food can be produced by simply smelling the food for about as long as the food would be eaten in a meal with no ingestion or tasting of the food, providing evidence that prolonged sensory stimulation by food can produce some decrease in its pleasantness (Rolls and Rolls 1997). In all these cases, it was the pleasantness and reward value that was decreased, and not the intensity of the sight, taste, or smell of

a. Sensory-specific satiety is found in the responses of orbitofrontal cortex neurons, but not in the taste and visual etc cortical regions that connect to the orbitofrontal cortex



b. Sensory-specific satiety related firing of an orbitofrontal cortex neuron when fed glucose to satiety. The neuron still responded to blackcurrant juice.



c. Sensory, reward, and output tiers of processing in primates including humans.

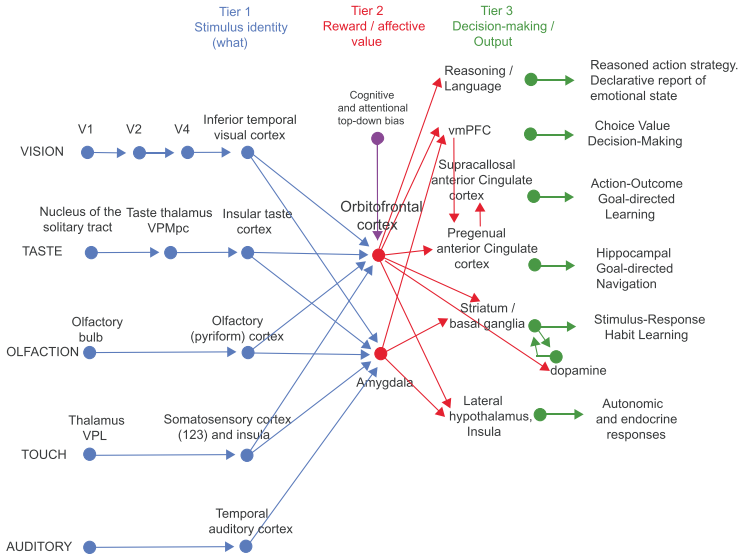


Fig. 1. a) Reward-specific satiety is found in neurons and activations in the primate including human orbitofrontal cortex, but not in the visual, taste, olfactory etc. cortical regions that provide inputs to the orbitofrontal cortex. Some of the neural pathways for food-related stimuli are shown on this lateral view of the macaque brain. Pathways to the orbitofrontal cortex and amygdala are shown from the primary taste and olfactory cortices. Connections are also shown in the "ventral visual system" from V1 to V2, V4, the inferior temporal visual cortex, which reach the orbitofrontal cortex and amygdala. Pathways from somatosensory regions 1, 2, and 3 reach the orbitofrontal cortex directly and via the insular cortex, and reach the amygdala via the insular cortex. Abbreviations follow from Rolls (2023b) *Brain Computations and Connectivity* (Open Access, Oxford University Press): "as, arcuate sulcus; cal, calcarine sulcus; cs, central sulcus; lf, 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,

the food, so that this is not sensory adaptation in the perceptual system where intensity is encoded, but instead a decrease in the reward value and pleasantness (Rolls 2023b).

A theory of reward-specific satiety

This experimental evidence, including what is summarized in Fig. 1, leads towards a theory of reward-specific satiety (sensory-specific satiety) for primates including humans that is presented here. Given that the orbitofrontal cortex reward value neurons remain responsive to another stimulus after feeding to satiety with one stimulus, the neural mechanism cannot be neuronal adaptation of the orbitofrontal cortex reward value neurons themselves. Given that the neurons in the taste and visual cortical regions that provide afferents to the orbitofrontal cortex do not decrease their responses after feeding to satiety (see above), the neural mechanism cannot be some change in the firing of neurons before the orbitofrontal cortex that provide the orbitofrontal cortex with taste, oral somatosensory, visual etc. inputs needed to compute taste, texture, visual etc. reward value.

Given these points of evidence, the neural mechanism that is now proposed for sensory-specific reductions in reward value is presynaptic depression (sometimes termed presynaptic adaptation, but not to be confused with sensory adaptation or habituation) that is produced by continuing activity of the presynaptic terminals (Mongillo et al. 2008). Presynaptic adaptation might relate for example to slow depletion of neurotransmitter in the presynaptic terminals. The computational architecture that is proposed here based on this evidence is illustrated in Fig. 2. Each sensory stimulus, for example a different type of food, activates a different subset of layer 1 neurons in sensory cortical regions such as the primary taste cortex or inferior temporal visual cortex, that synapse onto the same population of neurons in layer 2 in for example the orbitofrontal cortex. If one stimulus is repeated for about the length of time that a food is eaten in the meal, then gradually presynaptic adaptation sets in, and the result is that stimulus 1 gradually has less effect on the population of layer 2 neurons. But if another subset of neurons in layer 1 becomes active to represent a different type of food, then their synapses have not been active recently, and with no presynaptic adaptation initially, stimulus 2 when presented will produce an activation of the layer 2 reward neurons, providing a synaptic mechanism for reward-specific satiety. That neural mechanism is what accounts for the experimental evidence that neurons in the primate orbitofrontal cortex show sensory-specific satiety, and that taste and visual object neurons in the preceding cortical regions in tier 1 in Fig. 1c do not (Rolls 2023a, 2023b).

Given the experimental evidence just described, what is shown in Fig. 2 is the most parsimonious theory of how sensory-specific satiety is computed. Unless there is evidence against this theory, then this is the simple and adequate explanation for how this very important property of reward systems is computed. A very interesting point about the theory is that it proposes that the synaptic adaptation takes a long time to develop for these particular synapses in the orbitofrontal cortex. The modeling that is described later allows the parameters that influence the slow time-course of the synaptic adaptation to be estimated quantitatively. It is emphasized that this theory and model apply to primates including humans, and that the systems-level organization of reward value systems in the rodent brain is very different, with some effects of devaluation evident much earlier in sensory processing, as described in the discussion and elsewhere (Rolls 2015, 2023b). That makes the rodent a poor model of the operation of reward value systems in primates including humans, and indeed that is the case for the systems-level organization of many brain systems in rodents (Section 19.10 of Rolls (2023b)).

This theory accounts for the sensory-specific or reward-specific aspects of satiety, but for full satiety associated with feeding so that eating that food will stop, gut feedback is required, for example from gastric distension and from duodenal stimulation by food, as we showed (Gibbs et al. 1981; Rolls 2014, 2016a).

In order to demonstrate the synaptic mechanisms involved, and importantly to establish what parameters might be needed for the much longer time-course of synaptic adaptation needed here than considered in models of short-term memory (Mongillo et al. 2008; Deco et al. 2010), we next produced and analyzed an integrate-and-fire spiking model of the theory, with the model described in the Materials and Methods, and the results obtained with the model described later, in the results. This model is at a useful level, for it incorporates the stochastic dynamics that is typical of real neurons in the brain, and enables the computational implications of synaptic parameters such as conductance,

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." b) The neurophysiology of reward-specific satiety. Feeding to satiety with 20% glucose solution decreased the responses (firing rate ± sem) of a neuron in the secondary taste cortex in the orbitofrontal cortex to the taste of glucose (open circles) but not of blackcurrant juice (BJ). The spontaneous firing activity (SA) is shown. Below: The behavioral measure of the acceptance (+2) or rejection (−2) of the food is shown. Pre shows the firing rate before any glucose was fed. (After Rolls, E. T., Sienkiewicz, Z. J. And Yaxley, S. (1989) Hunger modulates the responses to gustatory stimuli of single neurons in the caudolateral orbitofrontal cortex of the macaque monkey. European journal of neuroscience 1: 53–60.) c) The systems level organization of the brain for reward value and emotion processing in primates including humans. In tier 1, representations are built of visual, taste, olfactory, and tactile stimuli that are independent of reward value and therefore of emotion. In tier 2, reward value and emotion are represented, in for example the orbitofrontal cortex. A pathway for top-down attentional and cognitive modulation of emotion is shown in purple. In tier 3 actions are learned in the supracallosal (or dorsal) anterior cingulate cortex to obtain the reward values signaled by the orbitofrontal cortex and amygdala that are relayed in part via the pregenual anterior cingulate cortex and vmPFC. Decisions between stimuli of different reward value can be taken in the ventromedial prefrontal cortex, vmPFC. In tier 3, orbitofrontal cortex inputs to the reasoning/language systems enable affective value to be incorporated and reported. In tier 3, stimulus–response habits can also be produced using reinforcement learning. In tier 3 autonomic responses can also be produced to emotion-provoking stimuli. Auditory inputs also reach the amygdala. V1—Primary visual (striate) cortex; V2 and V4—Further cortical visual areas. PFC—Prefrontal cortex. The medial PFC area 10 is part of the ventromedial prefrontal cortex (vmPFC). VPL—Ventro-postero-lateral nucleus of the thalamus, which conveys somatosensory information to the primary somatosensory cortex (areas 1, 2 and 3). VPMpc—Ventro-postero-medial nucleus pars parvocellularis of the thalamus, which conveys taste information to the primary taste cortex. (Modified from Rolls, E. T. (2023) Brain Computations and Connectivity. Oxford University Press: Oxford. Open access CC BY-NC-ND 4.0).

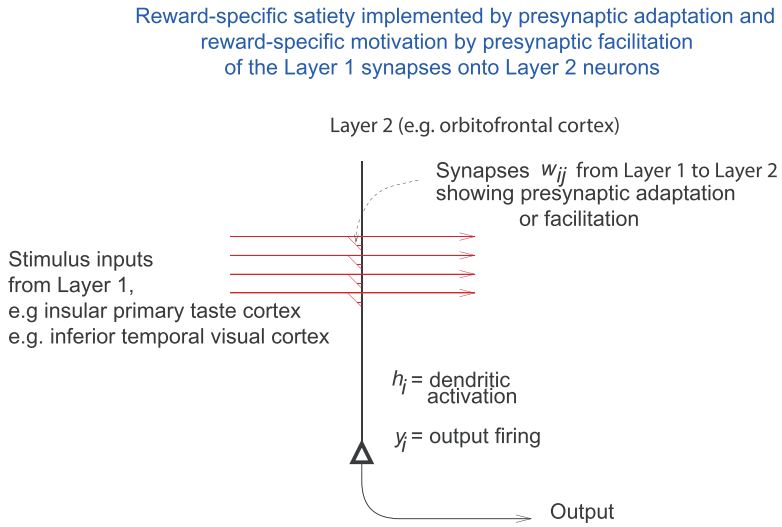


Fig. 2. Architecture for reward-specific satiety implemented by slow presynaptic adaptation in the synaptic terminals from layer 1 to layer 2. The same architecture implements reward-specific motivation by short-term presynaptic facilitation in the same synaptic terminals from layer 1 to layer 2. Layer 2 in primates including humans is the orbitofrontal cortex.

adaptation, and facilitation to be analyzed (Rolls and Deco 2010; Rolls 2023b).

A theory of the synaptic mechanism of reward-specific motivation, the increase in reward value that occurs soon after a new reward is made available

Reward-specific motivation [also termed incentive motivation (Rolls 2014, 2023a), and the "salted nut" phenomenon (Hebb 1949)], may it is proposed here be produced in the same neuronal architecture and neurons described here, and using analogous reasoning, but by shorter term presynaptic facilitation of the synapses of the layer 1 neurons onto the layer 2 neurons (Fig. 2). The presynaptic facilitation is implemented in the integrate-and-fire spiking model of the theory described here using the formalism described (Mongillo et al. 2008) and used previously (Rolls et al. 2013), and set out in the Materials and Methods.

Materials and Methods

A model of presynaptic adaptation/depression for use in integrate-and-fire simulations of reward-specific satiety

In the model of presynaptic adaptation or depression used to model reward-specific satiety, each action potential in a presynaptic terminal depletes the amount of transmitter, leaving less to be released by the next action potential (Mongillo et al. 2008). The amount of transmitter remaining in synaptic terminal j is x_j, starting with a value 1 and depleting as far as 0. x_j modulates the corresponding synaptic weight to model the decrease in the amount of transmitter released by each action potential. x_j recovers with a time constant τ_D (Mongillo et al. 2008). The parameter X defines the fraction of resources used by each action potential. When an action potential occurs, an amount Xx_j of the available resources is used to produce the postsynaptic current, thus reducing x_j. This process mimics the effects of the depletion of neurotransmitter. t_j^k is the time of the k'th presynaptic spike. The equation is as follows, which is different from Mongillo et al. (2008) in that there is no multiplication by u the parameter described below used for synaptic facilitation, and x in the implementation described here depends only on the previous history of x and the spikes being received:

$$\frac{dx_j(t)}{dt} = \frac{1 - x_j(t)}{\tau_D} - X\, x_j(t) \sum_k \delta\left(t - t_j^k\right) \quad (1)$$

In the research described here, we found that X had to be set to a low value of 0.0001 to produce a decay of x_j with a time-course of tens of seconds with realistic firing rates between 10 and 40 spikes/s. The recovery parameter time constant τ_D was also set to a high value (2000 s) to help produce a decay of x_j with a time-course of tens of seconds without fast recovery. The modulation by the presynaptic adaptation factor x_j is implemented by multiplying the synaptic weight by x_j to produce the adaptation part of the effective synaptic weight w_{eff}.

It is emphasized that these values of the parameters were those needed to produce the time-course of empirically measured sensory-specific satiety (Rolls et al. 1981a; Rolls et al. 1981b; Rolls et al. 1982a; Rolls et al. 1983; Rolls et al. 1986; Critchley and Rolls 1996; Hetherington 1996; Rolls and Rolls 1997; Cunningham et al. 2023), and as illustrated in Fig. 1a.

A model of presynaptic facilitation for use in integrate-and-fire simulations of reward-specific motivation

Presynaptic facilitation was used to help model reward-specific motivation. Synaptic facilitation is synapse-specific and provides for increasing efficacy of synaptic transmission especially early on in a neuronal response (Mongillo et al. 2008). Synaptic facilitation occurs commonly in higher cortical areas including the prefrontal cortex (Hempel et al. 2000; Zucker and Regehr 2002; Wang et al. 2006). Synaptic facilitation is caused by, for example, the increased accumulation of calcium at the presynaptic terminals, thereby increasing the probability of neurotransmitter release (Zucker and Regehr 2002). Short-term synaptic facilitation was implemented using a model of calcium-mediated transmission (Mongillo et al. 2008) used previously (Rolls et al. 2013). The synaptic efficacy of the synaptic connection of synapse j is modulated by the utilization variable u_j (the fraction of resources used), which reflects the calcium level. When an action potential reaches the presynaptic terminal, calcium influx in the presynaptic terminal causes an increase of u_j which increases the release probability of

transmitter and thereby the strength of that synapse. The time constant of the decay of the synaptic facilitation is controlled by a parameter τ_F which experimentally is around 1 to 2 s for some cortical regions (Wang et al. 2006; Mongillo et al. 2008). The value for τ_F (1.5 s) used here was similar to values reported experimentally and used elsewhere (Wang et al. 2006; Mongillo et al. 2008; Deco et al. 2010; Martinez-Garcia et al. 2011; Rolls et al. 2013). In detail, the strength of each excitatory synapse j is multiplied by the presynaptic utilization factor u_j (t), which is described by the following dynamics:

$$\frac{du_j(t)}{dt} = \frac{U - u_j(t)}{\tau_F} + U\left(1 - u_j(t)\right)\sum_k \delta\left(t - t_j^k\right), \quad (2)$$

where t_j^k is the time of the corresponding presynaptic spike k. The value of the baseline utilization factor U here, 0.01, was smaller than in those previous studies, to enable the presynaptic facilitation to build up over a longer timescale of several seconds to be in line with the time-course of reward-specific motivation. The first term shows how the synaptic utilization factor u_j decays to the baseline utilization factor $U = 0.01$ with time constant $\tau_F = 1.5$ s, and the second term shows how u_j is increased by each presynaptic action potential k to reach a maximum value of 1 when the neuron is firing fast. The modulation by the presynaptic utilization factor u_j is implemented by multiplying the synaptic weight by u_j to produce the facilitation part of the effective synaptic weight w_{eff}. This models the underlying synaptic processes.

The neuronal network integrate-and-fire architecture modeled

The architecture of the integrate-and-fire neuronal network modeled here is illustrated in Fig. 2. There were 100 excitatory neurons in layer 2. Neurons 51 to 60 were the neurons of interest in this model of the effects of reward-specific satiety and motivation on the effects of reward value, for these layer 2 neurons (corresponding for example to reward value neurons in the orbitofrontal cortex) each had 100 excitatory synapses from the 100 layer 1 excitatory neurons. Stimulus 1 was coded by high firing in neurons 1–10 of layer 1; stimulus 2 by high firing in neurons 11–20 of Layer 1, etc. It is emphasized that all the neurons 51 to 60 in layer 2 received from the same (100) layer 1 neurons.

The simulation protocol was to simulate 1 s of spontaneous activity, and to follow this by stimulus 1 applied until 400 s after which stimulus 1 was turned off; and to apply stimulus 2 from 400 to 800 s.

The integrate-and-fire neuronal network model

The integrate-and-fire neuronal network model was implemented using synaptic channels for AMPA, NMDA, and $GABA_A$ receptors in the same way as for attractor networks (Brunel and Wang 2001), and has been used and developed considerably (Wang 2002; Deco and Rolls 2006; Loh et al. 2007; Deco et al. 2009; Rolls and Deco 2010; Rolls et al. 2010b, 2010a; Rolls et al. 2012; Deco et al. 2013; Rolls and Deco 2015b, 2015a, 2016; Rolls 2023b). The details of the biophysical implementation are provided in the Supplementary Material, together with all the parameters that were used.

Results

An integrate-and-fire neuronal network model of reward-specific satiety

The network shown in Fig. 2 was modeled in an integrate-and-fire neuronal network simulation. The implementation of the synaptic and neuronal biophysics followed that described previously (Brunel and Wang 2001; Rolls and Deco 2010; Rolls 2023b) and in the Supplementary Material, but in the new neuronal network architecture illustrated in Fig. 2.

For reward-specific satiety, presynaptic adaptation, which reflects transmitter depletion in the presynaptic terminal, was modeled as described previously (Mongillo et al. 2008; Rolls et al. 2024b) and in the Materials and Methods, with the variable x representing the amount of depletion. For reward-specific motivation, presynaptic facilitation was modeled as described previously (Mongillo et al. 2008; Rolls et al. 2013) and in the Materials and Methods, with the variable u representing the amount of synaptic facilitation.

We found parameters for X in Equation (1) (the amount of synaptic adaptation produced by a single action potential) that produced a slow decay over several minutes of the firing rates of the neurons in layer 2, first when stimulus 1 was applied for 400 s, and then when stimulus 2 was applied for 400 s, as shown in Fig. 3. The key parameter X in Equation (1), the amount by which the synaptic transmission decreased for every action potential, that produced a time-course of several minutes, was $X = 0.0001$.

For the integrate-and-fire simulation, Fig. 3a shows the slow reductions of firing rates over many minutes while stimulus 1 was being applied for time = 1–400 s. These firing rates were from neurons 51 to 60 in the simulation that were activated by the inputs to the network, with the decrease in spiking shown for these 10 neurons in the red rastergrams. This mimics the approximate time-course of reward-specific satiety for eating a food to satiety. Importantly, when stimulus 2 was applied starting at time = 400 s, the neurons in the relevant population, neurons 51 to 60 in layer 2, had a large response to the new stimulus, stimulus 2, because the synapses for stimulus 2 had not been active recently. The response to stimulus 2 then gradually decayed as before for stimulus 1, and this emulates some of the key properties of the reward-specific reduction in reward value found in the orbitofrontal cortex and connected regions of the primate orbitofrontal cortex (Rolls et al. 1986; Critchley and Rolls 1996; Rolls 2014, 2016a, 2023a, 2023b).

Figure 3b shows the rastergrams that correspond to the data shown in Fig. 3a, to emphasize that these are spiking neurons with stochastic spike times.

Figure 3c shows the corresponding values of x in Equation (1), the presynaptic adaptation modulating variable, showing that as x decreases, so does the neuronal activity shown in Fig. 3a,b elicited by the inputs decrease.

The key values of the presynaptic adaptation parameters shown in Equation (1) were $X = 0.0001$ for Equation (1), and for the recovery parameter time constant $\tau_D = 2000$ s which helped to reveal the effects by slowing the recovery of x.

An integrate-and-fire neuronal network model of reward-specific motivation

Figure 4 illustrates that if shorter-lasting presynaptic facilitation is added to the integrate-and-fire simulation, an early increase in the firing rates (indicated by the red arrows) after a new stimulus was introduced was produced. This early period of extra-high firing occurred in the period 2–8 s in the simulation illustrated. The parameters were that the baseline utilization factor U in Equation (2) in the methods was 0.01, the time constant τ_F was 1.5 s, and the presynaptic facilitation was allowed to work in this way for the first 5 s of the simulation. Figure 4d shows the value of u the amount of synaptic facilitation from Equation (2) for the stimulus 1 inputs that accounted for the early increase in the firing rate of the neurons after a stimulus was applied, as

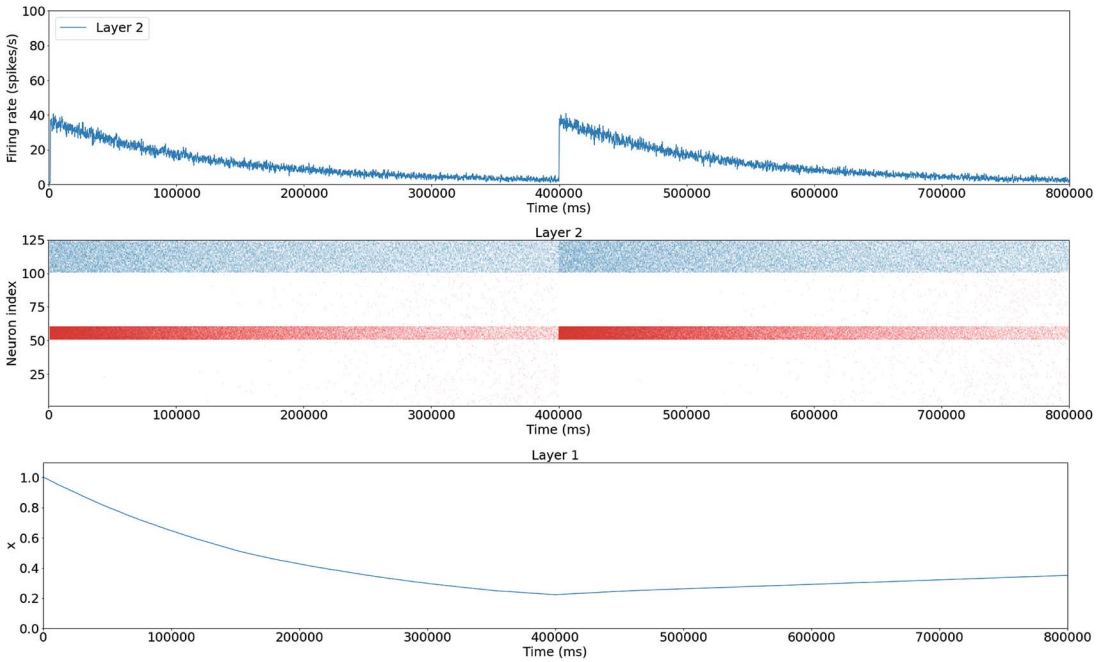


Fig. 3. The simulation of reward-specific satiety in an integrate-and-fire neuronal network. The firing rates for layer 2 reflect the reward value of the input stimuli. a) the firing rate of the layer 2 neurons with stimulus 1 applied as input from 0 to 400 s, and with stimulus 2 applied from 400 to 800 s. The firing rate of the neurons to stimulus 1 gradually decreased over 400 s due to presynaptic adaptation (depression). When stimulus 2 was applied at 400 s, the neurons showed a large response to stimulus 2 initially, as there was no presynaptic adaptation of the synaptic terminals that provided stimulus 2 as input, as they had not been active recently. In the period 400–800 s, the response to stimulus 2 also gradually decreased due to presynaptic adaptation of those synaptic terminals. b) Rastergram for the 100 excitatory neurons and 25 inhibitory neurons in layer 2. The stimulus inputs 1 and 2 were applied to neurons 51 to 60 in layer 2. c) the time-course of the variable x which models the presynaptic adaptation, shown for one neuron in layer 1 that responds to stimulus 1. A value of 1 indicates no adaptation, and of 0 complete adaptation and no transmitter release at the synapses.

indicated by red arrows, that models reward-specific motivation (also known as incentive motivation). No change of the u value for these stimulus 1 synapses occurs of course when stimulus 2 is applied, and activates the same output neurons, but through different synapses.

What is shown in Fig. 4 apart from that is reward-specific satiety which occurs much more slowly and was implemented by presynaptic adaptation as in Fig. 3. Overall the results are very similar to what is shown in Fig. 3, except that reward-specific motivation-related effects implemented by presynaptic facilitation are evident in the first 2–8 s of the start of each stimulus where indicated by the red arrows.

Discussion

The theory and model of the reward-specific decrease of reward value as reward-specific satiety develops that is based in presynaptic adaptation/depression is able to account for some of the behavioral and subjective properties of reward-specific satiety. The model shows a gradual decrease in the responsiveness of the reward value neurons over many minutes, and also shows that the reward neurons remain sensitive to a different reward (Fig. 3). This is achieved with an architecture of the type shown in Fig. 2, in which the neurons in layer 1 are not affected in their firing rates by reward-specific satiety, but the neurons in layer 2 decrease their firing rates only to stimuli that are applied for several minutes, because of the presynaptic adaptation in the layer 1 synaptic terminals onto layer 2 neurons. Layer 1 in the model shown in Fig. 2 might be the primary taste cortex in the anterior insula, or the inferior temporal visual cortex, or the pyriform olfactory cortex, as set out in the Introduction. Layer 2 might be the orbitofrontal cortex. As set out in the introduction, the properties of the neuronal activity in these cortical regions measured in macaques at the neuronal level and with fMRI in humans matches what the theory and model describe, and indeed the theory and model were developed explicitly in the light of those neurophysiological discoveries (Rolls 2016a, 2023a, 2023b). As far as we know, this is the first neural theory and model of the change of reward value with reward-specific satiety, which is one of the major factors influencing behavioral choice and reward-related decision-making (Rolls 2023a, 2023b).

Because the decrease of reward value with reward-specific satiety is a relatively slow process that takes place typically over minutes, the underlying presynaptic adaptation time-course must be relatively slow, and it was found that a value of $X = 0.0001$ in Equation (1) worked well. The implication of the proposed mechanism is that there is a specialized neural system for inputs to the reward neurons in the primate orbitofrontal cortex with an especially slow time-course for their presynaptic adaptation/depression, and that is exactly what we propose has been developed in evolution in the orbitofrontal cortex of primates including humans (Rolls 2023b). The parameter τ_D can be altered as necessary to fit the slow reward-specific recovery of reward value after reward-specific satiety has been reached.

The theory and model of the reward-specific increase of reward value when a reward is initially made available, reward-specific motivation or incentive motivation, is that presynaptic facilitation of the inputs to reward value neurons in for example the orbitofrontal cortex is able to account for the important facilitating effect on behavioral choice and the pleasantness of a reward stimulus when it is initially delivered. Here a critical parameter is U in Equation (2) in the methods.

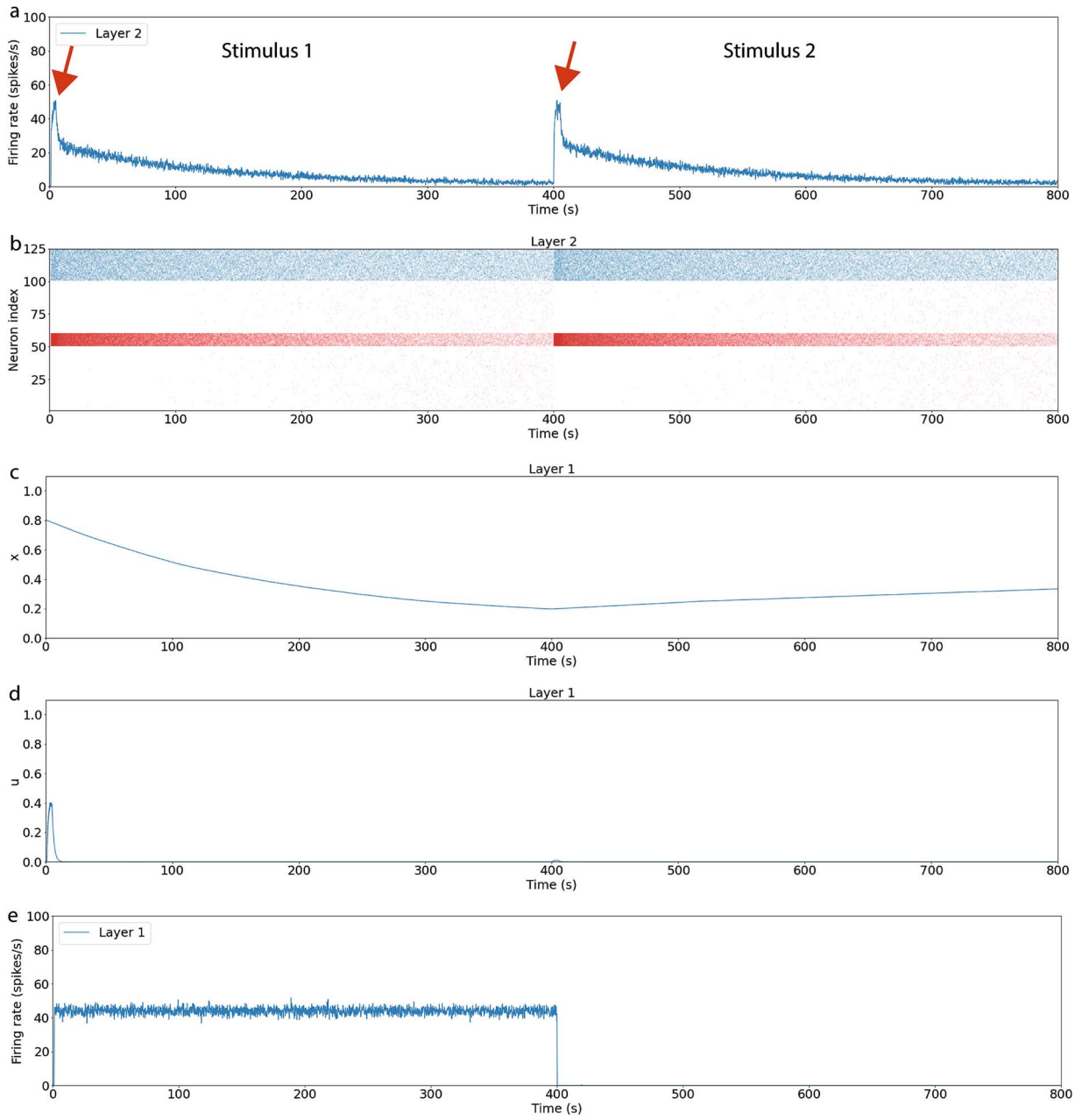


Fig. 4. The simulation of reward-specific motivation as well as reward-specific satiety. The firing rates for layer 2 reflect the reward value of the input stimuli. a) The firing rate of the layer 2 neurons with stimulus 1 applied as input from 1 to 400 s, and with stimulus 2 applied from 400 to 800 s. The firing rate of the neurons to stimulus 1 first shows an increase for approximately 6 s (indicated by a red arrow) due to presynaptic facilitation to model reward-specific motivation (incentive motivation), followed by a gradual decrease over 400 s due to presynaptic adaptation (depression) to model the slower reward-specific satiety. When stimulus 2 was applied at 400 s, the neurons showed a large response to stimulus 2 initially for about 6 s, partly because of presynaptic facilitation modeling reward-specific motivation (red arrow); and partly because the different synapses activated by stimulus 2 had not been recently active and were not in a state of presynaptic adaptation. In the period 400–800 s, the response to stimulus 2 then gradually decreased due to presynaptic adaptation of those synaptic terminals modeling reward-specific satiety. b) Rastergram for the 100 excitatory neurons and 25 inhibitory neurons in layer 2. The stimulus inputs 1 and 2 were applied to neurons 51 to 60 in layer 2. c) The time-course of the variable x which models the presynaptic adaptation. A value of 0.8 indicates no adaptation, and of 0 complete adaptation and no transmitter release at the synapses. e) The time-course of the variable u which models the presynaptic facilitation used for the stimulus 1 synapses from layer 1 to layer 2 to model reward-specific motivation. The synapses are those activated by stimulus 1 and not by stimulus 2, so there was no change of u when stimulus 2 was started at 400 ms. e) For completeness, the firing of the layer 1 neurons active for stimulus 1 is shown.

Something else is evident in the neurophysiology that may be of interest to model in future is that after reward-specific satiety has been reached for one stimulus, if a different stimulus eg another food, is delivered, then there may be some recovery of the sensory-specific satiety for the first stimulus, but that recovery is quickly reset to zero reward value with a small further amount of stimulus 1 (Rolls 1981; Rolls et al. 1986; Rolls et al. 1989; Critchley and Rolls 1996; Rolls et al. 1999). These details may imply some interactions between different stimuli, with a second, different, reward stimulus producing a minor return of pleasantness to the first stimulus.

The whole neural architecture of reward systems appears to be very different in rodents, with some effects of reward and satiety found even early on in sensory systems, with a much

less clear separation of sensory/perceptual representations from the representation of the reward value of stimuli (Rolls 2015, 2023b). But in rodents, even if reward-specific satiety is computed earlier on in the neural pathways than in primates including humans, nevertheless it is proposed that the same presynaptic adaptation/depression mechanism as described here is used.

As shown in Fig. 1c, the amygdala is a brain region in primates that has appropriate connectivity to reflect reward value and sensory-specific satiety. The macaque amygdala has neurons that can be activated by taste, oral somatosensory stimuli including viscosity, fat texture, grittiness, and temperature (Kadohisa et al. 2005a; Kadohisa et al. 2005b; Rolls et al. 2018), and visual stimuli (Sanghera et al. 1979; Leonard et al. 1985), and both rewarding and aversive tastes activate the human amygdala (O'Doherty et al. 2001). We found that although some amygdala visual neurons responded to the sight of food, their food selectivity was not as complete as orbitofrontal cortex neurons, and they did not reverse their reward-related responses well in a visual discrimination reversal task (Sanghera et al. 1979), which has been confirmed (Saez et al. 2017). Some orbitofrontal cortex neurons respond to any visual stimulus associated with a food reward, and other neurons are object-selective in that they respond to only some visual stimuli when they are associated with reward ("conditional reward neurons") (Thorpe et al. 1979; Deco and Rolls 2005). What may be similar neurons have been described in the macaque amygdala ("any object being currently viewed" vs object-selective reward neurons (Grabenhorst et al. 2023; Grabenhorst and Baez–Mendoza 2025). But less is known about sensory-specific satiety for primate amygdala neurons, and this is a topic that it would be useful to investigate.

Wherever sensory-specific satiety is found, it is likely to be generated by the synaptic adaptation mechanism described here. Moreover, whenever a primate including human is fed to satiety, it is likely that mechanisms of the type described apply, and it is proposed that the same reward-specific satiety applies to most natural rewards (Rolls 2014, 2023a, 2023b, 2025b). In addition, incentive motivation and sensory-specific satiety mechanisms of the type described here are likely to be involved when different food rewards are offered (Tremblay and Schultz 1999), and when adaptation occurs to the statistical distribution of available rewards (Padoa-Schioppa 2009; Kobayashi et al. 2010).

Neurons in the primate insular taste cortex that bring taste inputs to the orbitofrontal cortex (Baylis et al. 1995) can respond to different sets of taste and oral somatosensory stimuli including viscosity, fat texture, grittiness and temperature, allowing discrimination between oral sensory inputs, and most of these neurons can respond to the components to which they are responsive when the components are presented separately (Verhagen et al. 2004; Kadohisa et al. 2005b; Rolls 2016c). The same is found in the orbitofrontal cortex (Rolls and Baylis 1994; Rolls et al. 1999; Rolls et al. 2003; Verhagen et al. 2003; Kadohisa et al. 2004; Kadohisa et al. 2005b). The implication of this for sensory-specific satiety is that after sensory-specific satiety with one complex food with taste, oral texture, olfactory and visual components, the synaptic inputs to the orbitofrontal cortex will adapt for those components, and so there will be some generalization of sensory-specific satiety to other foods with similar components. That matches what is found experimentally (Rolls et al. 1981a; Rolls et al. 1981b; Rolls et al. 1982b; Rolls et al. 1982c; Rolls et al. 1983; Rolls et al. 1986).

When decisions are made between competing rewards, there is evidence that attractor networks in the more anterior parts of the orbitofrontal cortex and ventromedial prefrontal cortex are involved (Rolls and Grabenhorst 2008; Rolls et al. 2010b, 2010a; Grabenhorst and Rolls 2011; Rolls 2023b). In these attractor decision-making circuits, two or more inputs to the attractor network compete to push the attractor into the basin that represents the decision for one of the inputs (Wang 2002; Rolls and Deco 2010; Rolls 2023b). The synaptic mechanisms described here will influence such decision-making, for the inputs to the decision-making attractor network will reflect the recent history of the presentation of each of the reward inputs by reward-specific incentive motivation and reward-specific satiety. Similarly, the same synaptic mechanisms described here are likely to be involved whenever the recent history of receiving different rewards is involved in reward value guided choice (Huang et al. 2021; Huang and Grabenhorst 2023; Cui et al. 2025).

As described in the introduction, reward-specific motivation has biological adaptive value by locking the individual onto a reward for at least some time, which is a much more efficient foraging strategy than changing behavior to find a different reward when the reward value drops a little, as the two rewards might be far apart (Rolls 2014, 2023a). This is the first theory and model we know of how reward-specific motivation is implemented in the brain. Reward-specific motivation, and reward-specific satiety, may it is proposed be a key part of the neural mechanism involved in "exploit vs explore", that is, stay with the current reward or try a new one to see whether it is more rewarding, in foraging (Rolls 2023a), in which the frontal pole is also implicated (Rolls et al. 2024a).

Reward-specific motivation, and reward-specific satiety, are key components of almost all reward systems of humans and most other vertebrates, and are key to understanding much of human reward-related, motivation-related, and emotional behavior, including reward-related decision-making and foraging (Rolls 2014, 2023a). This is the first neural theory and model that we know for either process, and emphasizes how the recent history of the rewards received influences future choice of rewards and behavior. It is also proposed that each type of reward (e.g. food reward, water reward, social reward, the rewards associated with reproductive behavior, etc.) each have their own time-course, tuned using parameters of the type described here to produce an appropriate time-course to maximize reproductive success (Rolls 2014, 2023a). For example, feeding should not occupy too much of our time, so that time is left for other rewarded behaviors all of which should occur with a frequency that is adaptive in terms of reproductive success (Rolls 2014, 2023a). Moreover, part of the basis of personality is that in the course of evolution by natural selection there is a search for the parameters that control rewarded behavior to be optimized, leading to variation in the reward systems of different individuals. These variations are important in understanding personality (Rolls 2014, 2023a). Moreover, part of what is built in our brains during evolution is that no punishment system should have incentive motivation or sensory-specific satiety built into it (Rolls 2014, 2023a).

In summary, reward value is fundamental in understanding the choice behavior and reward-related decision-making of humans and other vertebrates. Here a synaptic theory and model are presented for how reward-specific motivation is computed by synaptic facilitation, and reward-specific satiety is computed by synaptic depression, in reward value systems such as the orbitofrontal cortex of humans and other primates. Understanding these phenomena of our reward systems, and their biological adaptive value, is fundamental to understanding much human rewarded, motivated, and emotional behavior (Rolls 2014, 2023b, 2023a, 2025c, 2025b).

Author contributions

Edmund T. Rolls (Conceptualization, Data curation, Formal Analysis, Investigation, Methodology, Project administration, Resources, Software, Supervision, Validation, Visualization, Writing—original draft, Writing—review & editing), Chenfei Zhang (Methodology, Software, Visualization, Writing—review & editing), and Jianfeng Feng (Funding acquisition, Writing—review & editing).

Supplementary material

Supplementary material is available at *Cerebral Cortex* online. The Supplementary Material shows how the synaptic dynamics for AMPA, NMDA, and GABA receptors and the neuronal dynamics were implemented in the integrate-and-fire simulation.

Funding

The work was supported by the following grants. Professor J.F.: 111 Project (No. B18015); ZJLab, and Shanghai Center for Brain Science and Brain-Inspired Technology. The funding sources had no role in the study design; in the collection, analysis and interpretation of data; in the writing of the report; and in the decision to submit the article for publication.

Conflict of interest statement: None declared.

Code availability

The implementation of the pattern association, attractor, and competitive networks was as described by Rolls (2016d, 2021, 2023b), and Matlab code for each of these classes of network as described there is made available open access at https://www.oxcns.org/NeuronalNetworkSimulationSoftware.html. Additional code used in the simulations is available from the corresponding author.

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Cerebral Cortex, 2025, **35**, bhaf248
https://doi.org/10.1093/cercor/bhaf248
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