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Current perspectives on incentive salience and applications to clinical disorders

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Affective neuroscience research has revealed that reward contains separable components of 'liking', 'wanting', and learning. Here we focus on current 'liking' and 'wanting' findings and applications to clinical disorders. 'Liking' is the hedonic impact derived from a pleasant experience, and is amplified by opioid and related signals in discrete sites located in limbic-related brain areas. 'Wanting' refers to incentive salience, a motivation process for reward, and is mediated by larger systems involving mesocorticolimbic dopamine. Deficits in incentive salience may contribute to avolitional features of depression and related disorders, whereas deficits in hedonic impact may produce true anhedonia. Excesses in incentive salience, on the other hand, can lead to addiction, especially when narrowly focused on a particular target. Finally, a fearful form of motivational salience may even contribute to some paranoia symptoms of schizophrenia and related disorders.

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Introduction

A fundamental question in affective neuroscience is how reward is generated in the brain. Answers may provide valuable insight not only into normal reward experiences, but also into how dysfunction in reward mechanisms contributes to neuropsychological disorders such as drug and behavioral addictions, major depressive disorder (MDD), Parkinson's disease (PD), and schizophrenia. Reward contains major components of 'liking' (hedonic pleasure), 'wanting' (incentive salience or motivation), and learning, and we will focus here especially on relations between 'liking' and 'wanting'. Research has indicated these two components are dissociable, and mediated by separable neural substrates. Here, we consider these components, and their application to reward dysfunctions.

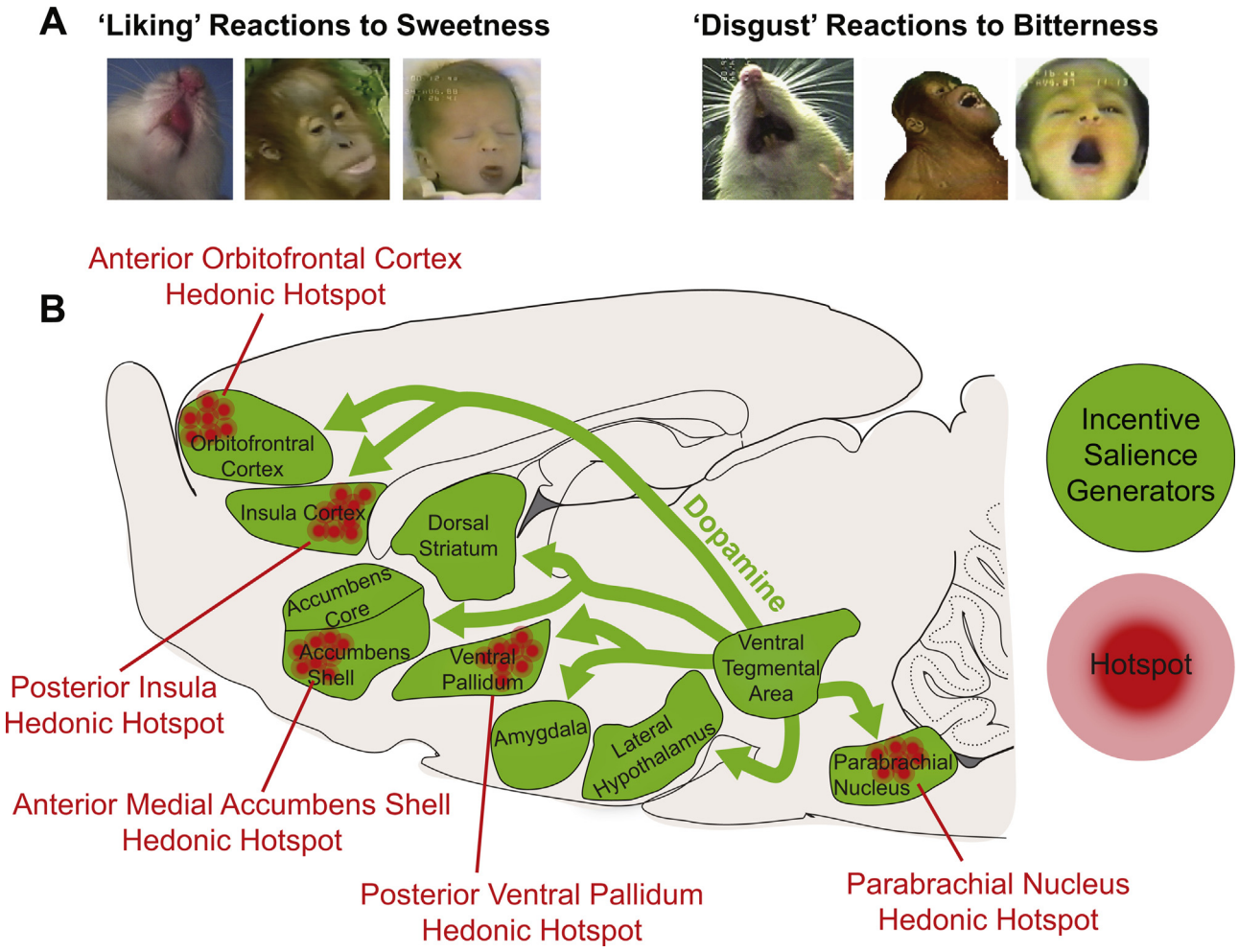
'Liking' and 'wanting' as separate aspects of reward

In ordinary experience, 'liking' and 'wanting' seem conjoined. For example, we eat cake because we enjoy it, and often eat more than intended if the cake tastes good. However, brain processes underlying liking and wanting can sometimes cause these components to diverge in certain circumstances.

'Liking' (in quote marks) refers to the hedonic impact derived from a stimulus (which can be conscious or unconscious) [1,2]. Consciously, it corresponds to subjectively experienced pleasure commonly denoted by liking (without quote marks). An objective form of 'liking' reaction has been measured in affective neuroscience studies of rodents using the taste reactivity (TR) test based on hedonic facial expressions to taste, first developed in human infants by Steiner [3] and later adapted for rats by Grill and Norgren [4]. The taste-elicited orofacial reactions are homologous across many species, including rodents, apes, and humans [3,5]. For example, sweetness causes positive, appetitive orofacial reactions (e.g. tongue protrusions), which represent a 'liked' tastant. Alternatively, bitterness elicits aversive, negative orofacial reactions (e.g. gapes) and reflects 'disgust' (Figure 1a). Importantly, 'liking' can be distinguished from the sensory properties of a stimulus, such as sweetness. For example, when a sweet food that was once 'liked' is now disliked after being paired with visceral illness — a phenomenon known as conditioned taste aversion (CTA) [6–9]. Similarly, physiological states may shift hedonic tone, called alliesthesia [10], in which food may become more 'liked' when one is hungry [10] or even tasty chocolate can become less 'liked' when satiated [11].

'Wanting', or incentive salience, refers to attention-grabbing and motivational features of rewards and their learned cues [12]. Reward cues have the ability to often trigger bursts of reward-seeking motivation [13,14••], and the cue itself becomes attractive as a 'motivational magnet'. Cue 'wanting' can be experimentally tested using the Pavlovian autoshaping or sign-tracking test [15,16], or in conditioned reinforcement tests where

Figure 1



Reward systems in the brain. **(a)** Examples of positive hedonic orofacial reactions ('liking') in response to the taste of sweet sucrose (left). Negative affective reactions ('disgust') in response to the taste of bitter quinine solution (right). These affective orofacial reactions to tastes are conserved across species in rodents, non-human primates, and humans infants. **(b)** Sagittal view of a rat brain depicting brain reward systems. Discrete sites, known as hedonic hotspots (clusters of red circles), can enhance 'liking' through the actions of opioids, endocannabinoids and orexins, but not dopamine. 'Wanting' is derived from dopamine signaling (green arrows), originating from the ventral tegmental area, acting in brain areas that generate incentive salience (purple structures). Hyperactivity in this 'wanting' circuit underlies many conditions characterized by excessive motivation, such as addiction, whereas hypoactivity (depicted by the smaller, darker arrows) may produce avolition seen in depression. Finally, aversive dysfunction of this 'wanting' circuit, particularly signaling onto the nucleus accumbens shell, may promote fearful salience to otherwise neutral stimuli — a trait commonly observed in patients with paranoid psychosis.

instrumentally working can earn cue presentations [17], and cue-triggered spurts of reward seeking are often experimentally tested using the Pavlovian Instrumental Transfer (PIT) test [13,18].

Separate neural substrates mediate 'liking' and 'wanting'

Liking in the brain

Research in our laboratory has identified a network of discrete sites, called 'hedonic hotspots', within limbic-related brain structures in which pleasure amplification mechanisms are localized (Figure 1b) [5]. For example, one hedonic hotspot can be found in the anterior–dorsal portion of the medial shell of the nucleus accumbens (NAc). Within hedonic hotspots, microinjection of opioid-stimulating drugs or a few other agents can enhance positive 'liking' reactions to sucrose taste [19–23]. Other subcortical hedonic hotspots have been found in the posterior portion of the ventral pallidum (VP) [24,25], and near the brainstem parabrachial nucleus of the pons (PBN) [26]. Recent findings have also identified two cortical hotspots: one in the anterior orbitofrontal cortex (OFC) and another in posterior insula [27••], consistent with human neuroimaging findings that implicate these cortical regions in pleasure and/or disgust [11,28,29].