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From Dopamine to Desire: Distinguishing Between Wanting and Liking in Human Behavior

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Abstract. From fulfilling basic needs to driving addiction, rewards are central to human behavior. For a long time, it was assumed that "wanting" and "liking" were interchangeable; it seemed logical that we want what we enjoy. However, modern neuroscience challenges this idea, revealing a perspective that these experiences rely on separate brain circuits and can often pull in different directions. Drawing considerably from the incentive salience theory the paper explores why we need to differentiate between motivational drive and hedonic pleasure. This article reviews evidence that mesolimbic dopamine mechanisms relate to wanting, and also how liking is generated by certain hedonic hotspots in which μ-opioid/opioid or related circuitry are active within nucleus accumbens and ventral pallidum. Some crucial experimental results from human and animal models show how these processes can become disconnected, especially in addiction, when people continue to seek rewards even though they get little pleasure from them. The report also incorporates control-theoretic models that explain how dopaminergic signals could regulate behavioral policies in dynamic environments. By distinguishing 'wanting' from 'liking,' this paper clarifies why motivation fails and sets a new roadmap for psychological, neurological, and clinical research.

Keywords: Reward, Motivation, Dopamine, Wanting, Liking

1. Introduction

From basic drives, such as hunger and thirst, to complex behaviors like addiction and consumerism, there is no doubt that rewards play a crucial role in shaping human behavior. The pursuit of reward shapes our everyday actions. Traditionally, psychological and neuroscientific models have often treated "wanting" and "liking" as inseparable components of reward, assuming that what we desire is necessarily what we enjoy. However, recent decades of research have challenged this assumption, particularly by Kent Berridge and other colleagues [1,2]. These studies indicate that "wanting" (the motivational drive to obtain a reward) and "liking" (the pleasurable experience derived from its consumption) are mediated by distinct neural systems, and can become dissociated, especially under pathological conditions such as addiction. This report aims to explain the theoretical and empirical foundations that differentiate "wanting" from "liking", supported by key experimental findings in both animal and human models. By examining their neural substrates and behavioral consequences,

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we may gain insight into how these components interact, or sometimes conflict, to shape decision-making and behavior.

2. Theoretical distinction: wanting vs. liking

It was once believed that "wanting" and "liking" were the same, but Berridge & Robinson's experiment [1] showed fundamental differences between them. Their incentive salience theory suggests "wanting" corresponds to the motivational desire for reward, while "liking" relates to the hedonic pleasure achieved from consuming it. Both regions contribute to reward, but they are supported by distinct neural systems and can dissociate under specific requirements. From the neurological perspective, "wanting" is primarily mediated by dopaminergic circuits, particularly the mesolimbic dopamine pathway, which involves the ventral tegmental area (VTA) and the nucleus accumbens (NAc).

This circuit assigns incentive salience to reward-associated cues, thereby stimulating behavior and directing attention, even in the absence of conscious desire or updated cognitive expectations [1]. Unlike reward learning models, which link dopamine to associative prediction errors, the incentive salience hypothesis posits that dopamine reflects the motivational "wanting" properties of cues, separable from their hedonic or cognitive attributes. In contrast, "liking" involves hedonic hotspots within the nucleus accumbens shell and ventral pallidum in which opioid and endocannabinoid signaling (but not dopamine or glutamate modulation) modulate liking reactions to sweetness. Importantly, this effect is contingent on both the anatomical specificity of hotspots and also the precise neurochemical form of stimulation [2,3]. These sites elicit detectable signs of hedonic reactions in animals through the taste reactivity test.

This theoretical difference is not just semantic; it has profound implications for understanding addiction, eating disorders, and the divergence between behavioral motivation and actual enjoyment. Critically, these dissociations are rooted in distinct neural substrates: dopaminergic circuits for incentive motivation ("wanting") versus localized opioid/endocannabinoid systems for hedonic pleasure ("liking"). As later sections of this paper will demonstrate, these components can be experimentally separated, where wanting often outlasts or even opposes liking.

3. Neural basis of wanting

The neurological foundations of "wanting" are predominantly linked to the mesolimbic dopamine system, specifically in the ventral tegmental area (VTA) and the nucleus accumbens (NAc). A classic study by Olds and Milner [4] demonstrated that rats would compulsively self-stimulate by pressing a lever to receive electrical stimulation in specific brain regions, particularly the septal area. For example, the research result shows that some animals exceeded 7,500 presses in 12 hours. More importantly, researchers found that this pressing behavior persisted even when rats had free access to food and water, and no signs of pain or deprivation were observed. The acquisition trajectories observed following septal stimulation were not only similar to those produced by primary rewards, but were dissociated from energetic demands and hedonic consequences. These results support that dopamine-containing circuits (eventually identified as the mesolimbic pathway) can produce pure motivational drive ("wanting") in the absence of "liking"—a distinction later codified by Berridge and Robinson's incentive salience theory [1].

However, further studies have clarified that dopamine does not directly mediate pleasure ("liking"), but helps energize and direct behavior toward goals. This distinction is well illustrated in the clinical case reported by Moan and Heath [5]: While septal stimulation in their patient produced

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marked behavioral changes, including the initiation of novel heterosexual behaviors, the treatment primarily enhanced motivation and approach behaviors rather than directly inducing hedonic states. Crucially, the patient's new sexual behaviors emerged in the context of improved mood and reduced anxiety, these suggesting dopamine's role in facilitating behavioral engagement with previously avoided stimuli, independent of direct pleasure induction.

Recent work by Yin [6,7] captures "wanting" as part of a control-theoretic model where the role of behavior can be seen as reducing discrepancy between current state and internal goals achieved via hierarchical feedback systems. For instance, hunger produces a reference signal (e.g., ideal blood glucose level), and behaviors are adjusted in order to minimize the accompanying error signal [6].

This error-reduction process requires dynamic coordination across neural systems. Yin [7] extends this framework by specifying how dopaminergic circuits implement such control, not as reward signals but as adaptive policy updates, modulating transition variables (e.g., reward rate, action rate) to optimize goal pursuit in dynamic environments.

The basal ganglia support this hierarchical goal-control system through spiraling striato-nigro-striatal circuits, in which activity flows from limbic regions (e.g., ventral striatum and accumbens) toward associative and sensorimotor networks (e.g., DMS and DLS) [7]. This anatomical organization enables motivational signals to shape action selection and motor execution progressively. Rather than coding for reward per se, dopaminergic signals adjust policy parameters such as action rate or expected outcomes, helping the organism adaptively refine goal-directed behavior under changing conditions. This model explains the persistence of "wanting": When environmental outcomes mismatch internal expectations (e.g., failed reward prediction), sustained error signals continuously drive motivation until the discrepancy is resolved. This pathway illustrates how motivation influences motor execution through a series of layered feedback loops (Figure 1).

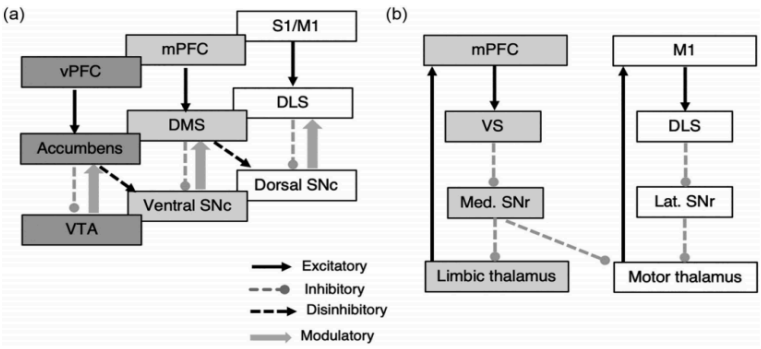


Figure 1. Cortico-basal ganglia circuits underlying motivational and motor control [7]

Together, these studies establish "wanting" as a hierarchically organized motivational process implemented through cortico-basal ganglia circuits [6,7]. The mesolimbic dopamine system serves as a computational engine that (a) translates physiological needs into reference signals, (b) detects mismatches between current and desired states, and (c) adjusts outputs to reduce error signals. This neural framework for motivated behavior sets the stage for understanding its distinct counterpart: the hedonic ("liking") system that evaluates outcomes.

4. Neural basis of liking

In contrast to the dynamic, goal-driven nature of "wanting," "liking" refers to the hedonic impact, the experience of pleasure that is elicited by rewards. Unlike the wide-reaching motivational

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circuitry involved in "wanting", "liking" appears to rely on a more localized and restricted set of neural mechanisms.

It has been demonstrated that liking is mediated by small brain structures known as "hedonic hotspots" localized in the nucleus accumbens (NAc) and the ventral pallidum (VP) [8]. For instance, when scientists use opioids to stimulate these areas, rats react more positively to sweets (such as by sticking out their tongues) but without increasing food-seeking behavior.

Smith & Berridge [8] claimed that this pleasure system is separate from the brain's motivation ("wanting") pathways, which means that both the NAc and VP hotspots must work together to produce pleasure; if blocking one cancels the other, it will be affected.

Moreover, these hedonic reactions persist even when dopaminergic signaling is severely impaired. Studies show that in animals with near-total dopamine depletion (to ~1% of normal levels), "liking" responses (e.g., tongue protrusions to sweetness) remain completely intact, while "wanting" behaviors (e.g., food-seeking) are abolished [2]. Similarly, Parkinson's patients with dopamine loss retain normal subjective pleasure ratings for sweet tastes [9]. These dissociations confirm dopamine's critical role in motivation rather than pleasure generation. Unlike the motivational drive of "wanting", "liking" is associated with pleasure systems involving opioids and GABAergic activity, rather than dopamine [2]. These responses tend to be immediate and reactive, triggered by the direct sensory qualities of rewarding stimuli.

5. Behavioral implications in real-life contexts

5.1. Drug addiction: excessive wanting without increased liking

One of the most obvious examples could show the distinction between "liking" and "wanting" in the real world is drug addiction. The incentive sensitization hypothesis explains that the drug's pleasurable effects either remain the same or decrease, but repeated drug exposure causes a pathological amplification of cue-triggered "wanting." This explanation is supported by evidence tested on humans: File et al. [10] found that the severity and frequency of use were linked to increased "wanting," but not to increased "liking." Similarly, Berridge and Robinson [11] argued that addiction is primarily driven by sensitized motivational systems, rather than increased pleasure. At the neural level, animal studies further support this dissociation. Calipari et al. [12] showed that consuming small amounts of cocaine could make dopamine more sensitive, but taking it in large quantities all at once makes it less sensitive. This suggests that motivation can increase independently of its pleasurable aspects. So, addiction reflects an excessive "wanting," not an increase in "liking."

5.2. Beyond addiction: wanting and liking in everyday pursuits

Addiction and extreme behavior are not the only situations when the difference between liking and wanting is apparent. People might find themselves craving for something in their daily lives, but they do not genuinely appreciate it. For example, a student may want academic recognition, such as good grades, but find little fulfillment when they get praise. Even if someone dislikes being online, they may still want attention on social media. These discrepancies indicate that cues, routines, or social conventions are possible to take control of desire, even if there is no actual pleasure. This is especially important in performance-driven societies, whenever internal emotional reward is separated from external reinforcement (such as grades, likes, and promotions). Berridge and Robinson [1] suggested that a cycle of pursuing without satisfaction can result from the brain's

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hypersensitive dopamine system, which can enhance desire but with no corresponding increase in liking. This occurrence extends the concept of wanting-liking into ordinary behavioral economics and motivation research.

5.3. Implications for human agency and self-understanding

A philosophical analysis of agency is also encouraged by the distinction between wanting and liking. Are we truly acting freely if we pursue things that we don't genuinely enjoy? From the traditional paradigm of rational decision-making, one chooses a course of action based on its alignment with one's values and goals. Wanting, however, has the potential to skew this process and influence behavior in ways that are inconsistent with wellbeing or conscious wishes. The consistency of "wanting something" as a motivation for action is called into question by this separation. Assisting people in recalibrating their objectives and identifying this misalignment through introspection, therapy, or neuroscience may help them regain agency. As a result, liking and desire have ontological and ethical meaning in addition to being physiological states.

6. Conclusion

This report shows that "wanting" and "liking" are two distinct brain systems that influence how we pursue rewards. "Wanting" is the drive that pushes us to act, powered by dopamine, while "liking" is the feeling of pleasure we get from the reward itself, which is controlled by other chemicals such as opioids. While dopaminergic circuits energize and direct behavior toward rewards, the experience of pleasure is mediated by localized hedonic hotspots involving opioids and endocannabinoids. Crucially, these two systems don't always work together; people can want something without actually enjoying it, as seen in cases of drug addiction or habitual consumer behavior. Integrating evidence from human and animal models, as well as control-theoretic frameworks, this dissociation challenges traditional models of rational agency and invites new approaches to understanding motivation. Recognizing the divergence between desire and enjoyment not only informs neuroscience and clinical interventions but also sheds light on everyday paradoxes of modern life, such as craving without satisfaction and consumption without pleasure. Distinguishing between wanting and liking provides a more precise roadmap for psychological research and a deeper understanding of human agency.

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