NEUROSCIENCE OF REWARD, MOTIVATION, AND DRIVE

Morten L. Kringelbach and Kent C. Berridge

ABSTRACT

Drive and motivation are central to affective neuroscience. Here, we describe the development of conceptualizations from early behaviorist theories to contemporary theories linking motivation closely to reward. Current experimental data suggest key roles of drive and motivation in the wanting, liking, and learning processes underlying the pleasure cycle supporting survival of individuals and species. In particular, the underlying functional neuroanatomy of drive and motivation is now becoming clearer in humans and other mammals, which provides hope for novel more effective interventions for the pervasive problems of drive and motivation in affective and addictive disorders.

Keywords: Motivation; pleasure; brain; addiction

INTRODUCTION

There is a long history in psychology and neuroscience of considering concepts of motivation and drive to understand our behavior; in particular to help better understand the variability of behavior as well as the short-term

Recent Developments in Neuroscience Research on Human Motivation
Advances in Motivation and Achievement, Volume 19, 23–35

ISSN: 0749-7423/doi:10.1108/S0749-742320160000019020

stability of goal-directed and threat-avoidant behaviors (Berridge, 2004). The main goals of this research have been trying to answer important questions of (1) why do individuals react to affectively important stimuli, (2) why do the individuals seek out specific things at particular times, and (3) why do the individuals choose to do different things at different times when faced with identical and constant stimuli?

Initially, progress was slow to adequately address these complex questions but over the last two decades affective neuroscience has made significant progress possible for understanding how motivation is driven by interactions over time between specific networks in the brain. In particular, it has become clear how motivation plays a significant role in the pleasure cycles that help allocate the necessary brain resources for behaviors promoting survival (Fig. 1) (Berridge & Kringelbach, 2008). In addition, we now have much better understanding of the functional neuroanatomy of underlying wanting, liking, and learning processes which have been linked

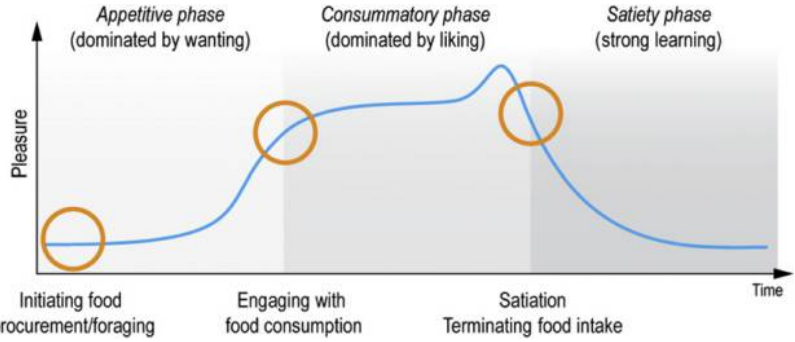

Fig. 1. The Pleasure Cycle. *Notes*: The cyclical processing of rewards has classically been proposed to be associated with appetitive, consummatory, and satiety phases (Craig, 1918; Sherrington, 1906). Research has demonstrated that this processing is supported by multiple brain networks and processes, which crucially involves *liking* (the core reactions to hedonic impact), *wanting* (motivational processing of incentive salience), and *learning* (typically Pavlovian or instrumental associations and cognitive representations) (Finlayson, King, & Blundell, 2007; Robinson & Berridge, 1993, 2003). These components wax and wane during the pleasure cycle and can co-occur at any time. Importantly, however, wanting processing tends to dominate the appetitive phase, while liking processing dominates the consummatory phase. In contrast, learning can happen throughout the cycle.

to dissociable networks of specific subcortical and cortical regions (Berridge & Kringelbach, 2015).

Here, we first briefly review a number of early behaviorist theories of drive and motivation and discuss their relative merits. We then turn our attention to contemporary theories linking motivation closely to reward, which have overturned earlier, erroneous theories proposing that drive reduction supposedly was the chief mechanism of reward. We show how the current experimental data suggest a key role of motivation in the wanting, liking, and learning processes underlying the pleasure cycle. In particular, we sketch the underlying brain circuitry supporting these processes over time.

EARLY THEORIES OF HOMEOSTATIC DRIVE

In the 1920s, drawing heavily on the earlier work by Claude Bernard and others physiologists, Walter Cannon developed the idea of *homeostasis* as the ability to maintain a stable internal state (Cannon, 1932). Subsequent research came to regard homeostasis as a specific type of regulatory system that uses a setpoint or built-in goal value to maintain a stable physiological state. Constant comparisons between the current setpoint and the desired physiological state drive a mismatch regulator triggering a homeostatic mechanism for the correction of the current state. The temperature thermostat found in many homes is often used as a simple analogy of this principle, yet presumably homeostasis in the human body must have many more error detectors. Thus homeostasis for, say, hunger, would entail error detectors embedded in neural, hormonal, and physiological systems with setpoints, for example, body weight, blood glucose, nutrient storage, neural metabolism, and other physiological variables relevant for hunger and satiety. Such ideas have dominated the behavioral neuroscience of hunger, thirst, salt appetite, and other ingestive behaviors. Following this view, a better understanding of motivation and problems with motivation is simply a question of a more detailed understanding of homeostatic-deficit triggers and their receptors. Much interesting research has arisen from this, yet it has also become clear that homeostatic drive cannot alone account for many known problems such as our current pandemic of obesity (Kringelbach, 2004).

Instead, it has been become clear that what may appear to be a homeostatic balancing of physiological systems can be achieved without homeostatic

setpoints mechanisms, for example, through the use of anticipatory mechanisms (Fitzsimons & Le Magnen, 1969; Schulkin, 2003) (i.e., drinking and eating in anticipation of hunger and thirst) or through "settling points" (i.e., a stable balance between opposing forces) (Wirtshafter & Davis, 1977). Take the example of obesity, which is difficult to explain as a purely homeostatic drive (Bolles, 1980; Kringelbach, 2004). The homeostatic theory would hold that the regulation of eating behavior requires a body weight setpoint from which hunger would be triggered. Yet, such a setpoint has not been found. Instead, it would appear that body weight simply settles around a moderately stable point settled by many factors, including internal appetite and satiety mechanisms as well as availability and palatability of foods. In other words, the pleasure of food can lead to overeating in the absence of a homeostatic deficit (Kringelbach, 2004).

Interestingly, Cannon never actually wrote about homeostasis in terms of setpoints, setpoint comparisons, or error detection. These concepts instead came from cybernetic theories in engineering and computer science (Wiener, 1948). Cannon thought about homeostasis exclusively in terms of opposing reflexes, for example, a high stimulus triggered an internal reflex which reduced the stimulus and restored homeostasis, that is, essentially as a settling point rather than a setpoint.

Another way to think about drive and motivation came with the rise of behaviorism, where behaviorists were obsessed with only describing behavior in terms of observable stimuli and responses. However, this quickly gives rise to ever more complex relationships when adding mappings between more stimuli and responses. One way to simplify these relationships is to introduce drive as an intervening variable, where, for example, by adding thirst as an intervening variable, this becomes a common route through which stimuli and responses can be mapped. This is meant to be a purely objective relationship, yet it is also clear from the data that thirst can have very different effects on responses.

FLEXIBLE GOALS AND AFFECTIVE DISPLAYS

Rather than continuing with the fairly sterile behaviorist concept of drive and motivation as intervening variables, researchers began to explore the idea of *internal* motivational processes. In the 1960s and early 1970s Philip Teitelbaum suggested one interesting way to think about real motivation as that which helps to motivate flexible instrumental behavior

(Teitelbaum, 1966, 1977). This built on the insights from the beginning of the 20th century, where ethologists and physiologists such as Craig, Sherrington, and others had proposed that all motivated behavior can be divided into two sequential phases: an appetitive phase and a consummatory phase (Craig, 1918; Sherrington, 1906). The appetitive phase of motivated behavior contains the flexible approach behavior that an individual emits before the motivational goal is found. Similarly, the consummatory phase follows consisting of stereotyped and species-specific patterns, whether for ingestive behaviors or sex and aggression. Teitelbaum drew attention to the appetitive phase as essential for motivation to drive the interaction with instrumental associative learning shaping new operant responses.

More generally, Epstein proposed that three additional criteria are needed to distinguish motivated behavior: (1) flexible goal directedness, (2) goal expectation, and (3) affect (Epstein, 1982). The first criterion builds on Teitelbaum's operant learning idea by emphasizing the need to rule out simple forms of learning and simple drive activation of behavior. While instrumental learning is interesting, it is still much less complex than, for example, cognitive inferences. Similarly, various forms of expectation from classical conditioning of an anticipatory conditioned response to declarative, cognitive forms of goal expectation are central to motivation. Finally, Epstein proposed that real motivation is always accompanied by affective reactions to the goal itself. Motivation is always directed toward hedonically laden goals and the presence of hedonic reactions confirms that a given behavior is motivated.

Building on these insights, psychologist Richard Solomon proposed *opponent process theory* as a useful general concept for thinking about how many processes with different valences can interact (Solomon & Corbit, 1974). All hedonic stimuli elicit not only their own hedonic reactions but also an opponent process of opposite hedonic value, which helps maintain homeostasis. Yet, there are limits to the explanatory power of this theory in that the opposite processes do not occur for every affective event and are not always the chief motivational factor.

Another very popular concept in the history of motivational drives is the idea that *drive reduction* is the main mechanisms of reward (Hull, 1943; Mowrer, 1960; Spence, 1956). According to this concept – which in its day was seen so powerful as to be self-evident – food is a reward because it reduces hunger drive, while water is a reward because it reduces the thirst drive. Yet, the overwhelming evidence has clearly shown that this concept is false. Even for food and hunger, reducing physiological drive via intravenous feeding is not particularly effective

at stopping eating (Epstein & Teitelbaum, 1962). Similarly, evidence from brain stimulation reward showed that stimulation of brain sites associated with eating behavior would almost always coincide with sites where stimulation was rewarding (Valenstein, Cox, & Kakolewski, 1970), rather than with a reduction of reward as posited by the drive reduction theory.

INCENTIVE MOTIVATION CONCEPTS

These findings instead led to a revival of the old idea from ancient Greek philosophers such as Aristotle that hedonic reward or pleasure is at the heart what motivates us. Early pioneers such as Pfaffmann argued that neural encoding of rewards such as sweet taste and sex must be rewarding and motivating in and of itself, without the need for drive reduction (Pfaffmann, 1960; Pfaffmann, Norgren, & Grill, 1977). Similarly, later researchers such as Stellar asserted the need for behavioral neuroscience to study affective reactions (Stellar, 1982).

The failure of various theories of homeostatic drive, intervening variables and drive reduction to explain real motivation coupled with the realization of centrality of pleasure for understanding motivation, led to the development of incentive motivation concepts during the 1970s and 1980s. First, Bolles argued that individuals are motivated incentive expectancies rather than by drives or drive reduction (Bolles, 1972). Such incentive expectancies are learned expectations of hedonic reward. Bindra furthered this concept by rejecting expectation per se as the important factor for incentive motivation for rewards (Bindra, 1974, 1978). Instead, Bindra proposed that a conditioned stimulus for a reward evokes the same incentive motivational state caused by the reward, that is, it becomes a reward in and of itself. Toates then added to this concept by suggesting that physiological depletion states can enhance incentive motivation for appropriate incentives and cues (Toates, 1986). According to the theories of incentive motivation, individuals will select the available reward with the highest incentive value.

These concepts fit well with the alliesthesia concept coined by Michel Cabanac showing that physiological states can modulate the perceived hedonic value of rewards (Cabanac, 1971, 1979), for example, how a warm bath is delicious when we are cold but not on a very hot day.

Adapting these insights, Berridge and colleagues realized in the 1990s that the incentive motivation value of reward can be further split into

incentive salience, "wanting" and the hedonic impact, "liking" with partly dissociable brain networks and neurotransmitters (Berridge, 1996; Berridge, Venier, & Robinson, 1989). Incentive salience makes a hedonic reward more attractive, attention grabbing, and ultimately available for Bindra-Toates goal-directed strategies. This is different from the hedonic impact, which triggers the brain reaction to pleasure. Normally, "liking" and "wanting" relate the same incentive value, but there are situations where they are different. It is possible, for example, to have "wanting" without "liking" as was the case for brain stimulation, where, for example, stimulation of the lateral hypothalamus in rats would trigger motivated eating behaviors without the associated hedonic face expressions (such as licking) associated with eating in rats (Berridge & Valenstein, 1991).

One important further insight from this body of research is the *incentive-sensitization theory of addiction* (Robinson & Berridge, 2003), which combines neural sensitization of dopamine-related brain systems with incentive salience to propose that sensitized "wanting" may explain compulsive long-lasting addiction and relapse. Many addictive drugs, such as cocaine, heroin, amphetamine, nicotine, and alcohol, cause neural sensitization in brain mesocorticolimbic systems which can trigger the brain systems into both sensitization (through increase in dopamine release) and tolerance (through decrease in dopamine receptors). The tolerance mechanisms usually recover within days whereas neural sensitization can last for years (Paulson, Camp, & Robinson, 1991). This may explain why some addicts may not like drugs but still want them, and why the relapse can occur in the context of drug-associated cues such as paraphernalia or places and social settings. We are thus beginning to understand why motivation and unbalancing of the associated brain networks can have devastating consequences but these insights also open up for the development of potential new treatments.

A growing body of evidence has started to map the underlying brain networks and transmitters involved in parsing reward in the pleasure cycle, linking "wanting," "liking," and "learning." This affective neuroscience endeavor has mapped "wanting" processes to changes in dopamine, which is not related to the "liking." On the other hand, "liking" processes have been linked strongly with opioids. In terms of brain regions, wanting for rewards is generated by a large and distributed brain system, while "liking" is served by a much smaller set of discrete hedonic regions with pleasure hotspot and coldspot regions in subcortical areas of the brain such as the nucleus accumbens and ventral pallidum (Peciña & Berridge, 2005; Smith & Berridge, 2007) (Fig. 2). Manipulations of these regions with opioids have been shown to causally change pleasure-elicited reactions (Berridge & Kringelbach, 2013).

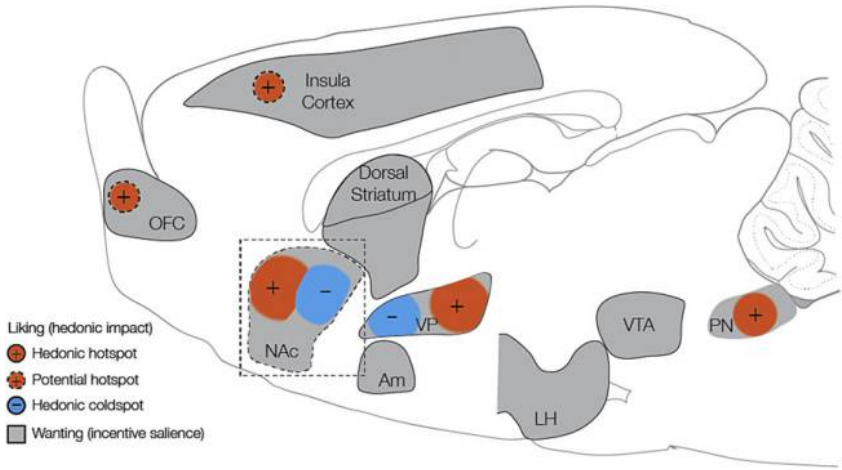

Fig. 2. Liking and Wanting Circuits in the Rat Brain. *Notes:* The figure shows sagittal view of wanting (incentive salience, in gray) and liking (hedonic impact) regions in the rat brain. The large spread wanting regions include dorsal striatum, lateral hypothalamus (LH), ventral tegmental area (VTA), parabrachial nucleus (PN), amygdala (Am), nucleus accumbens (NAc), ventral pallidum (VP), insula cortex, and orbitofrontal cortex (OFC). Within these regions there are hedonic hotspots in much smaller regions of NAc, VP, and PN, as well as, putatively, insula cortex and OFC. Hotspots (+) depict sites where opioid stimulation enhances "liking" reactions elicited by sucrose taste (with a stippled line indicating putative hotspots). Coldspots (−) show sites where the same opioid stimulation oppositely suppresses "liking" reactions to sucrose.

Other regions involved in wanting and liking have been found using human neuroimaging in the orbitofrontal, cingulate, medial prefrontal, and insular cortices (Amodio & Frith, 2006; Berridge, 1996; Cardinal, Parkinson, Hall, & Everitt, 2002; Everitt & Robbins, 2005; Kringelbach, 2010; Kringelbach, O'Doherty, Rolls, & Andrews, 2003; Kringelbach & Rolls, 2004; Watson, Shepherd, & Platt, 2010). These interacting networks do not act in splendid isolation but are embedded within much larger brain networks (Fig. 3). We are beginning to understand the metastable nature, topological and functional features of these networks using advances in network science and graph theory together with advanced whole-brain computational models (Cabral, Kringelbach, & Deco, 2014; Deco & Kringelbach, 2014; Kringelbach, McIntosh, Ritter, Jirsa, & Deco, 2015).

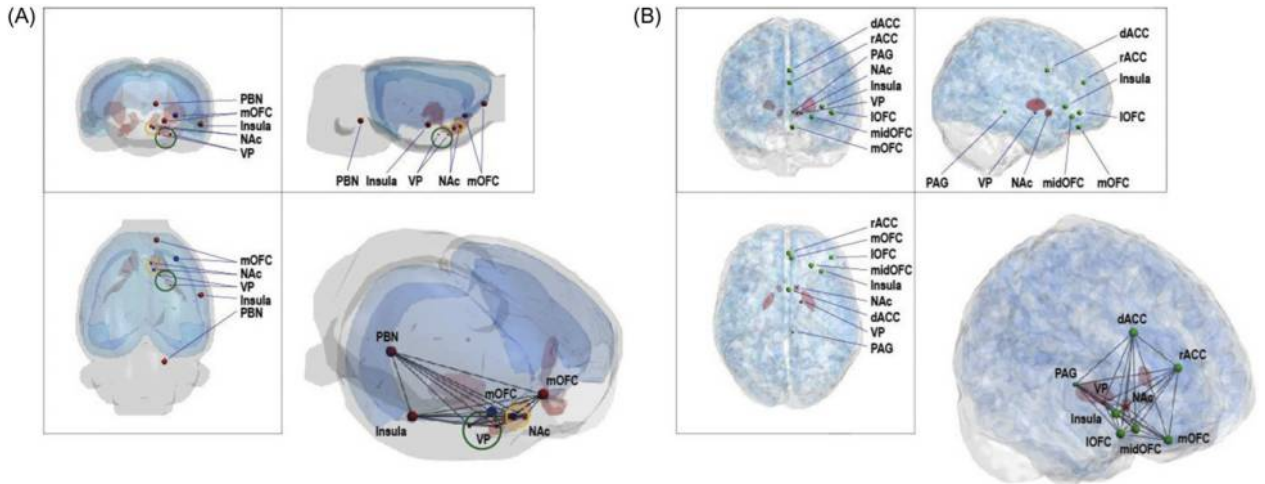

Fig. 3. Three-Dimensional Comparison of Hedonic Sites in Rat Brain (Left) and Human Brain (Right). *Notes:* (A) Rat brain shows hedonic hotspots (+) and coldspots (−) in coronal, sagittal, horizontal planes, and in 3D fronto-lateral perspective view (clockwise from top left). (B) Human brain shows extrapolation of rat causal hotspots to analogous human sites in NAc and VP (+), and shows fMRI coding sites for positive affective reactions in green (from text). Human views are also in coronal, sagittal, horizontal, and 3D perspective (clockwise from top left of B). The tentative functional networks between the different hotspots and coldspots have been added to give an impression of the topology of a pleasure network. The functional connection lines are not meant to imply direct anatomical projections between two connected structures, but rather a functional network in mediating hedonic "liking" reactions and subjective pleasure ratings. Abbreviations: VP, ventral pallidum; NAc, nucleus accumbens; PBN, parabrachial nucleus; mOFC, medial orbitofrontal cortex; lOFC, lateral orbitofrontal cortex; midOFC, mid-anterior orbitofrontal cortex; dACC, dorsal anterior cingulate cortex; rACC, rostral anterior cingulate cortex; PAG, periaqueductal gray.

CONCLUSION

Over the last century much progress has been made in understanding motivation in the human brain (Kringelbach & Berridge, 2010). Fascinating new insights have tied motivation closely the pleasure system of the brain and it has become clear that "wanting," "liking," and learning processes are carefully choreographed in the healthy human brain. Through this careful, growing body of brain research, we now have a much clearer idea about why individuals react to affectively important stimuli, as well why they seek out specific things at particular times and why they choose to do different things at different times when faced with identical and constant stimuli. We are beginning to understand how unbalancing of these processes can lead to the diverse symptoms of anhedonia found in affective disorders (Rømer Thomsen, Whybrow, & Kringelbach, 2015). Ultimately, carefully characterizing the interactions between brain networks involved in motivation during a healthy pleasure cycle may come to offer new, early biomarkers for affective disorders and perhaps novel, more effective interventions in the years to come.

ACKNOWLEDGMENT

Research in our laboratories has been supported by grants from the TrygFonden Charitable Foundation and European Research Council (ERC) Consolidator Grant to M.L.K. (CAREGIVING n. 615539) and from the NIH to K.C.B. (MH63644 and DA015188). This chapter is an abridged version of another book chapter (Kringelbach & Berridge, 2016).

REFERENCES

Amodio, D. M., & Frith, C. D. (2006). Meeting of minds: The medial frontal cortex and social cognition. *Nature Review Neuroscience*, *7*(4), 268–277.

Berridge, K. C. (1996). Food reward: Brain substrates of wanting and liking. *Neuroscience & Biobehavioral Review*, *20*(1), 1–25.

Berridge, K. C. (2004). Motivation concepts in behavioral neuroscience. *Physiology & Behaviour*, *81*(2), 179–209. doi:10.1016/j.physbeh.2004.02.004

Berridge, K. C., & Kringelbach, M. L. (2008). Affective neuroscience of pleasure: Reward in humans and animals. *Psychopharmacology*, *199*, 457–480.

Berridge, K. C., & Kringelbach, M. L. (2013). Neuroscience of affect: Brain mechanisms of pleasure and displeasure. *Current Opinion in Neurobiology*, *23*(3), 294–303. doi:10.1016/j.conb.2013.01.017

Berridge, K. C., & Kringelbach, M. L. (2015). Pleasure systems in the brain. *Neuron*, *86*, 646–664.

Berridge, K. C., & Valenstein, E. S. (1991). What psychological process mediates feeding evoked by electrical stimulation of the lateral hypothalamus? *Behavioural Neuroscience*, *105*(1), 3–14.

Berridge, K. C., Venier, I. L., & Robinson, T. E. (1989). Taste reactivity analysis of 6-hydroxydopamine-induced aphagia: Implications for arousal and anhedonia hypotheses of dopamine function. *Behavioural Neuroscience*, *103*(1), 36–45.

Bindra, D. (1974). A motivational view of learning, performance, and behavior. *Psychological Review*, *81*, 199–213.

Bindra, D. (1978). How adaptive behavior is produced: A perceptual-motivational alternative to response-reinforcement. *Behavioral and Brain Sciences*, *1*, 41–91.

Bolles, R. (1972). Reinforcement, expectancy, and learning. *Psychological Review*, *79*, 394–409.

Bolles, R. W. (1980). Some functionalistic thoughts about regulation. In T. Toates & T. Halliday (Eds.), *Analysis of motivational processes* (pp. 63–75). New York, NY: Academic Press.

Cabanac, M. (1971). Physiological role of pleasure. *Science*, *173*(2), 1103–1107.

Cabanac, M. (1979). Sensory pleasure. *Quarterly Review of Biology*, *54*, 1–29.

Cabral, J., Kringelbach, M. L., & Deco, G. (2014). Exploring the network dynamics underlying brain activity during rest. *Progress in Neurobiology*, *114*, 102–131. doi:10.1016/j.pneurobio.2013.12.005

Cannon, W. (1932). *The wisdom of the body*. New York, NY: W.W. Norton and Company.

Cardinal, R. N., Parkinson, J. A., Hall, J., & Everitt, B. J. (2002). Emotion and motivation: The role of the amygdala, ventral striatum, and prefrontal cortex. *Neuroscience & Biobehavioural Reviews*, *26*(3), 321–352.

Craig, W. (1918). Appetites and aversions as constituents of instincts. *Biological Bulletin of Woods Hole*, *34*, 91–107.

Deco, G., & Kringelbach, M. L. (2014). Great expectations: Using whole-brain computational connectomics for understanding neuropsychiatric disorders. *Neuron*, *84*, 892–905.

Epstein, A. (1982). The physiology of thirst. In D. Pfaff (Ed.), *Physiological mechanisms of motivation* (pp. 25–55). New York, NY: Springer-Verlag.

Epstein, A., & Teitelbaum, P. (1962). Regulation of food Intake in absence of taste, smell, and other oropharyngeal sensations. *Journal of Comparative and Physiological Psychology Medicine*, *55*, 753–759.

Everitt, B. J., & Robbins, T. W. (2005). Neural systems of reinforcement for drug addiction: From actions to habits to compulsion. *Nature Neuroscience*, *8*(11), 1481–1489.

Finlayson, G., King, N., & Blundell, J. E. (2007). Liking vs. wanting food: Importance for human appetite control and weight regulation. *Neuroscience & Biobehavioural Reviews*, *31*(7), 987–1002.

Fitzsimons, T. J., & Le Magnen, J. (1969). Eating as a regulatory control of drinking in the rat. *Journal of Comparative and Physiological Psychology*, *67*(3), 273–283.

Hull, C. (1943). *Principles of behavior, an introduction to behavior theory*. New York, NY: D. Appleton-Century.

Kringelbach, M. L. (2004). Food for thought: Hedonic experience beyond homeostasis in the human brain. *Neuroscience*, *126*, 807–819.

Kringelbach, M. L. (2010). The hedonic brain: A functional neuroanatomy of human pleasure. In M. L. Kringelbach & K. C. Berridge (Eds.), *Pleasures of the brain* (pp. 202–221). Oxford: Oxford University Press.

Kringelbach, M. L., & Berridge, K. C. (2010). *Pleasures of the brain*. New York, NY: Oxford University Press.

Kringelbach, M. L., & Berridge, K. C. (2016). Drive and motivation in the brain. In H. Miller (Ed.), *Sage encyclopedia of theory in psychology* (pp. 240–244). New York, NY: Sage.

Kringelbach, M. L., McIntosh, A. R., Ritter, P., Jirsa, V. K., & Deco, G. (2015). The rediscovery of slowness: Exploring the timing of cognition. *Trends in Cognitive Science*, *19*(10), 616–628.

Kringelbach, M. L., O'Doherty, J., Rolls, E. T., & Andrews, C. (2003). Activation of the human orbitofrontal cortex to a liquid food stimulus is correlated with its subjective pleasantness. *Cerebral Cortex*, *13*(10), 1064–1071.

Kringelbach, M. L., & Rolls, E. T. (2004). The functional neuroanatomy of the human orbitofrontal cortex: Evidence from neuroimaging and neuropsychology. *Progress in Neurobiology*, *72*(5), 341–372.

Mowrer, O. (1960). *Learning theory and behavior*. New York, NY: Wiley.

Paulson, P. E., Camp, D. M., & Robinson, T. E. (1991). Time course of transient behavioral depression and persistent behavioral sensitization in relation to regional brain monoamine concentrations during amphetamine withdrawal in rats. *Psychopharmacology (Berl)*, *103*(4), 480–492.

Peciña, S., & Berridge, K. C. (2005). Hedonic hot spot in nucleus accumbens shell: Where do mu-opioids cause increased hedonic impact of sweetness? *The Journal of Neuroscience*, *25*(50), 11777–11786.

Pfaffmann, C. (1960). The pleasures of sensation. *Psychological Review*, *67*, 253–268.

Pfaffmann, C., Norgren, R., & Grill, H. J. (1977). Sensory affect and motivation. *Annals of the New York Academy of Sciences*, *290*, 18–34.

Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: An incentive-sensitization theory of addiction. *Brain Research Reviews*, *18*(3), 247–291.

Robinson, T. E., & Berridge, K. C. (2003). Addiction. *Annual Review of Psychology*, *54*, 25–53.

Rømer Thomsen, K., Whybrow, P. C., & Kringelbach, M. L. (2015). Reconceptualising anhedonia: Novel perspectives on balancing the pleasure networks in the human brain. *Frontiers in Behavioural Neuroscience*, *9*, 49.

Schulkin, J. (2003). *Rethinking homeostasis: Allostatic regulation in physiology and pathophysiology*. Cambridge, MA: MIT Press.

Sherrington, C. S. (1906). *The integrative action of the nervous system*. New York, NY: C. Scribner's Sons.

Smith, K. S., & Berridge, K. C. (2007). Opioid limbic circuit for reward: Interaction between hedonic hotspots of nucleus accumbens and ventral pallidum. *The Journal of Neuroscience*, *27*(7), 1594–1605.

Solomon, R. L., & Corbit, J. D. (1974). An opponent-process theory of motivation: I. Temporal Dynamics of affect. *Psychological Review*, *81*(2), 119–145.

Spence, K. (1956). *Behavior theory and conditioning*. New Haven, CT: Yale University Press.

Stellar, E. (1982). Brain mechanisms in hedonic processes. In D. Pfaff (Ed.), *The physiological mechanisms of motivation* (pp. 377–408). New York, NY: Springer-Verlag.

Teitelbaum, P. (1966). The use of operant methods in the assessment and control of motivational states. In W. Honig (Ed.), *Operant behavior: Areas of research and application* (pp. 565–608). New York, NY: Appleton-Century-Crofts.

Teitelbaum, P. (1977). Levels of integration of the operant. In W. Honig & J. Staddon (Eds.), *Handbook of operant behavior* (pp. 7–27). Englewood Cliffs, NJ: Prentice-Hall.

Toates, F. (1986). *Motivational systems*. Cambridge: Cambridge University Press.

Valenstein, E. S., Cox, V. C., & Kakolewski, J. W. (1970). Reexamination of the role of the hypothalamus in motivation. *Psychological Review*, *77*(1), 16–31.

Watson, K. K., Shepherd, S. V., & Platt, M. L. (2010). Neuroethology of pleasure. In M. L. Kringelbach & K. C. Berridge (Eds.), *Pleasures of the brain* (pp. 85–95). New York, NY: Oxford University Press.

Wiener, N. (1948). *Cybernetics; or, control and communication in the animal and the machine*. Cambridge, MA: Technology Press.

Wirtshafter, D., & Davis, J. D. (1977). Set points, settling points, and the control of body weight. *Physiology & Behavior*, *19*(1), 75–78.