CHAPTER 2

THE NEUROBIOLOGY OF PLEASURE AND HAPPINESS

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Introduction

Happiness is an elusive state, difficult to define, and therefore challenging to measure—partly due to its clearly subjective, and perhaps uniquely human, nature. But how can one get a scientific handle on such a slippery concept?

Since Aristotle, happiness has been thought of as consisting of at least two aspects: hedonia (pleasure) and eudaimonia (a life well-lived) (Waterman 1993). In contemporary psychology these aspects are usually referred to as pleasure and meaning, and scientists have recently proposed to add a third distinct component of engagement related to feelings of commitment and participation in life (Seligman et al. 2005).

Using these definitions scientists have made substantial progress in defining and measuring happiness in the form of self-reports of subjective well-being (Kahneman 1999; Ryan and Deci 2001; Diener et al. 2003; Seligman et al. 2005). This research shows that while there is clearly a sharp conceptual distinction between pleasure versus engagement-meaning components, hedonic and eudaimonic aspects empirically cohere together in happy people.

For example, in happiness surveys over 80% of people rate their overall eudaimonic life satisfaction as “pretty to very happy”, and comparably, 80% also rate their current hedonic mood as positive (e.g. positive 6–7 on a 10-point valence scale where 5 is hedonically neutral) (Kesebir and Diener 2008). A lucky few may even live consistently around a hedonic point of 8—although excessively higher hedonic scores may actually impede attainment of life success, as measured by riches, education, or political participation (Oishi et al. 2007).

While these surveys are interesting indications of mental well-being, they offer little evidence of the underlying neurobiology of happiness. In this review we will therefore focus on the substantial progress in understanding the psychology and neurobiology of sensory pleasure that has been made over the last decade (Berridge and Kringelbach 2008; Kringelbach and Berridge 2010).
These advances make the hedonic side of happiness most tractable to a scientific approach to the neural underpinnings of happiness. Supporting a hedonic approach, it has been suggested that the best measure of subjective well-being may be simply to ask people how they hedonically feel right now—again and again—so as to track their hedonic accumulation across daily life (Kahneman 2000; Diener et al. 2003; Gilbert and Wilson 2007). These repeated self-reports of hedonic states could also be used to identify more stable neurobiological hedonic brain traits that dispose particular individuals toward happiness. Further, a hedonic approach might even offer a toehold into identifying eudaimonic brain signatures of happiness, due to the empirical convergence between the two categories, even if pleasant mood is only half the happiness story (Kringelbach and Berridge 2009).

It is important to note that our focus on the hedonia component of happiness should not be confused with hedonism, which is the pursuit of pleasure for pleasure’s own sake, and more akin to the addiction features we describe later. Also, to focus on hedonics does not deny that some ascetics may have found bliss through painful self-sacrifice, but simply reflects that positive hedonic tone is indispensable to most people seeking happiness.

A SCIENCE OF PLEASURE

The link between pleasure and happiness has a long history in psychology. It was stressed in the early writings of Sigmund Freud (Freud and Riviere 1930), when he posited that people “strive after happiness; they want to become happy and to remain so. This endeavor has two sides, a positive and a negative aim. It aims, on the one hand, at an absence of pain and displeasure, and, on the other, at the experiencing of strong feelings of pleasure” (Freud and Riviere 1930, p. 76). Emphasizing a positive balance of affect to be happy implies that studies of hedonic brain circuits can advance the neuroscience of both pleasure and happiness. A related but slightly different view is that happiness depends most chiefly on eliminating negative “pain and displeasure” to free an individual to pursue engagement and meaning. Positive pleasure by this view is somewhat superfluous. This view may characterize the 20th century medical and clinical emphasis on alleviating negative psychopathology and strongly distressing emotions. It fits also with William James’s early quip that “Happiness, I have lately discovered, is no positive feeling, but a negative condition of freedom from a number of restrictive sensations of which our organism usually seems the seat. When they are wiped out, the clearness and cleanness of the contrast is happiness. This is why anaesthetics make us so happy. But don’t you take to drink on that account.” (James 1920, vol 2, p. 158).

Focusing on eliminating negative distress seems to leave positive pleasure outside the boundary of happiness, perhaps as an extra bonus or even an irrelevancy for ordinary pursuit. In practice, many mixtures of positive affect and negative affect may occur in individuals (Ryff et al. 2006) and cultures may vary in the importance of positive versus negative affect for happiness. For example, positive emotions are linked most strongly to ratings of life satisfaction overall in nations that stress self-expression, but alleviation of negative emotions may become relatively more important in nations that value individualism (Kuppens et al. 2008).

By either view, psychology seems to be moving away from the stoic notion that affect states such as pleasure are simply irrelevant to happiness. The growing evidence for the
importance of affect in psychology and neuroscience shows that a scientific account will have to involve hedonic pleasures and/or displeasures. To move towards a neuroscience of happiness, a neurobiological understanding is required of how positive and negative affect are balanced in the brain.

Given the potential contributions of hedonics to happiness, we now survey developments in understanding brain mechanisms of pleasure (Berridge and Kringelbach 2008; Leknes and Tracey 2008). The scientific study of pleasure and affect was foreshadowed by the pioneering ideas of Charles Darwin, who examined the evolution of emotions and affective expressions, and suggested that these are adaptive responses to environmental situations. In that vein, pleasure “liking” and displeasure reactions are prominent affective reactions in the behavior and brains of all mammals (Steiner et al. 2001), and likely had important evolutionary functions (Kringelbach 2009). Neural mechanisms for generating affective reactions are present and similar in most mammalian brains, and thus appear to have been selected for and conserved across species (Kringelbach 2010). Indeed, both positive affect and negative affect are recognized today as having adaptive functions (Nesse 2004), and positive affect in particular has consequences in daily life for planning and building cognitive and emotional resources (Fredrickson et al. 2008; Dickinson and Balleine 2010).

Such functional perspectives suggest that affective reactions may have objective features beyond subjective ones (Kringelbach 2004a). Progress in affective neuroscience has been made recently by identifying objective aspects of pleasure reactions and triangulating toward underlying brain substrates. This scientific strategy divides the concept of affect into two parts: the affective state, which has objective aspects in behavioral, physiological, and neural reactions; and conscious affective feelings seen as the subjective experience of emotion (Kringelbach 2004a). Note that such a definition allows conscious feelings to play a central role in hedonic experiences, but holds that the affective essence of a pleasure reaction is more than a conscious feeling.

Evidence so far available suggests that brain mechanisms involved in fundamental pleasures (food and sexual pleasures) overlap with those for higher-order pleasures (for example, monetary, artistic, musical, altruistic, and transcendent pleasures) (Small et al. 2001; Kahneman et al. 2004; Kringelbach 2005; Peciña et al. 2006; Gottfried 2010; Kringelbach 2010; Kringelbach et al. 2010; Veldhuizen et al. 2010).

From sensory pleasures and drugs of abuse (Robinson and Berridge 2003) to monetary, aesthetic, and musical delights, all pleasures seem to involve the same hedonic brain systems, even when linked to anticipation and memory (Skov 2010; Vuust and Kringelbach 2010). Pleasures important to happiness, such as socializing with friends (Kahneman 1999; Ryan and Deci 2001; Diener et al. 2003; Kahneman et al. 2004; Seligman et al. 2005), and related traits of positive hedonic mood are thus all likely to draw upon the same neurobiological roots that evolved for sensory pleasures. The neural overlap may offer a way to generalize from fundamental pleasures that are best understood and so infer larger hedonic brain principles likely to contribute to happiness.

We note the rewarding properties for all pleasures are likely to be generated by hedonic brain circuits that are distinct from the mediation of other features of the same events (e.g. sensory, cognitive) (Kringelbach 2005). Thus pleasure is never merely a sensation or a thought (Frijda 2010), but is instead an additional hedonic gloss generated by the brain via dedicated systems.
The neuroanatomy of pleasure

How does positive affect arise? Affective neuroscience research on sensory pleasure has revealed many networks of brain regions and neurotransmitters activated by pleasant events and states (Figures 2.1 and 2.2). Identification of hedonic substrates has been advanced by recognizing that pleasure or “liking” is but one component in the larger composite psychological process of reward, which also involves “wanting” and “learning” components (Smith et al. 2010). Each component also has conscious and non-conscious elements that can be studied in humans—and at least the latter can also be probed in other animals.

Hedonic hotspots

Despite having an extensive distribution of reward-related circuitry, the brain appears rather frugal in “liking” mechanisms that cause pleasure reactions. As shown in later paragraphs, some hedonic mechanisms are found deep in the brain (nucleus accumbens, ventral pallidum, brainstem) and other candidates are in the cortex (orbitofrontal, cingulate, medial prefrontal, and insular cortices) (Berridge 1996; Cardinal et al. 2002; Kringelbach et al. 2003; Kringelbach and Rolls 2004; Everitt and Robbins 2005; Amodio and Frith 2006; Kringelbach 2010; Watson et al. 2010). Pleasure-activated brain networks are widespread, but compelling evidence for pleasure causation (detected as increases in “liking” reactions consequent to brain manipulation) has so far been found for only a few hedonic hotspots in the subcortical structures. Each hotspot is merely a cubic millimeter or so in volume in the rodent brain (and should be a cubic centimeter or so in humans, if proportional to whole brain volume). Hotspots are capable of generating enhancements of “liking” reactions to a sensory pleasure such as sweetness, when stimulated with opioid, endocannabinoid, or other neurochemical modulators (Smith et al. 2010).

Hotspots exist in nucleus accumbens shell and ventral pallidum, and possibly other forebrain and limbic cortical regions, and also in deep brainstem regions including the parabrachial nucleus in the pons (Figure 2.2d) (Peciña et al. 2006). The pleasure-generating capacity of these hotspots has been revealed in part by studies in which microinjections of drugs stimulated neurochemical receptors on neurons within a hotspot, and caused a doubling or tripling of the number of hedonic “liking” reactions normally elicited by a pleasant sucrose taste (Smith et al. 2010). Analogous to scattered islands that form a single archipelago, hedonic hotspots are anatomically distributed but interact to form a functional integrated circuit. The circuit obeys control rules that are largely hierarchical and organized into brain levels. Top levels function together as a cooperative heterarchy, so that, for example, multiple unanimous “votes” in favor from simultaneously-participating hotspots in the nucleus accumbens and ventral pallidum are required for opioid stimulation in either forebrain site to enhance “liking” above normal (Smith and Berridge 2007).

In addition, as mentioned earlier, pleasure is translated into motivational processes in part by activating a second component of reward termed “wanting” or incentive salience, which makes stimuli attractive when attributed to them by mesolimbic brain systems (Berridge and Robinson 2003). Incentive salience depends in particular on mesolimbic
FIG. 2.1 Measuring reward and hedonia. Reward and pleasure are multifaceted psychological concepts. Major processes within reward (first column) consist of motivation or wanting (white), learning (gray), and—most relevant to happiness—pleasure liking or affect (light gray). Each of these contains explicit (top three rows) and implicit (bottom three rows) psychological components (second column) that constantly interact and require careful scientific experimentation to tease apart. Explicit processes are consciously experienced (e.g. explicit pleasure and happiness, desire, or expectation), whereas implicit psychological processes are potentially unconscious in the sense that they can operate at a level not always directly accessible to conscious experience (implicit incentive salience, habits and “liking” reactions), and must be further translated by other mechanisms into subjective feelings. Measurements or behavioral procedures that are especially sensitive markers of the each of the processes are listed (third column). Examples of some of the brain regions and neurotransmitters are listed (fourth column), as well as specific examples of measurements (fifth column), such as an example of how highest subjective life satisfaction does not lead to the highest salaries (top) (Haisken-De New and Frick 2005). Another example shows the incentive sensitization model of addiction and how “wanting” to take drugs may grow over time independently of “liking” and “learning” drug pleasure as an individual becomes an addict (bottom) (Robinson and Berridge 1993). ACC, anterior cingulate cortex; Ach, acetylcholine; OFC, orbitofrontal cortex; mPFC, medial prefrontal cortex; NAc, nucleus accumbens; PAG, periaqueductal gray; VP, ventral pallidum; VTA, ventral tegmental area.
Fig. 2.2 (Also see Plate 2). Hedonic brain circuitry. The schematic figure shows the brain regions for causing and coding fundamental pleasure in rodents and humans. (a) Facial "liking" and "disliking" expressions elicited by sweet and bitter taste are similar in rodents and human infants.
(b, d) Pleasure causation has been identified in rodents as arising from interlinked subcortical hedonic hotspots, such as in nucleus accumbens and ventral pallidum. Similar pleasure coding and incentive salience networks have also been elicited in humans. (c) "Pleasure" electrodes in rodents and humans are unlikely to have elicited true pleasure but perhaps only incentive salience or "wanting." (d) The cortical localization of pleasure coding may reach an apex in various regions of the orbitofrontal cortex, where differentially subjective pleasantness from valence processing of aspects of the same stimulus, such as a pleasant food.

PAG, periaqueductal gray; VTA, ventral tegmental area.

"Liking" increases

"Disliking" decreases

"Liking" decrease

"Disliking" increase

"Wanting" hotspot

Eating
Importantly, incentive salience is not hedonic impact or pleasure “liking” (Berridge 2007). This is why an individual can “want” a reward without necessarily “liking” the same reward. Irrational “wanting” without liking can occur especially in addiction via incentive-sensitization of the mesolimbic dopamine system and connected structures (Robinson and Berridge 2003). At extreme, the addict may come to “want” what is neither “liked” nor expected to be liked, a dissociation possible because “wanting” mechanisms are largely subcortical and separable from cortically-mediated declarative expectation and conscious planning. This is a reason why addicts may compulsively “want” to take drugs even if, at a more cognitive and conscious level, they do not want to do so. That is surely a recipe for great unhappiness (Figure 2.2, bottom right).

Cortical pleasure

In cortex, hedonic evaluation of pleasure valence is anatomically distinguishable from precursor operations such as sensory computations, suggesting the existence of a hedonic cortex proper (Figure 2.2) (Kringelbach 2004b). Hedonic cortex involves regions such as the orbitofrontal (Kringelbach 2005), insula (Craig 2002), medial prefrontal (Amodio and Frith 2006), and cingulate cortices (Beckmann et al. 2009), which a wealth of human neuroimaging studies have shown to code for hedonic evaluations (including anticipation, appraisal, experience, and memory of pleasurable stimuli) and have close anatomical links to subcortical hedonic hotspots. It is important, however, to again make a distinction between brain activity coding and causing pleasure. Neural coding is inferred in practice by measuring brain activity correlated to a pleasant stimulus, using human neuroimaging techniques (Gottfried 2010), or electrophysiological or neurochemical activation measures in animals (Aldridge and Berridge 2010). Causation is generally inferred on the basis of a change in pleasure as a consequence of a brain manipulation such as a lesion or stimulation (Green et al. 2010; Smith et al. 2010). Coding and causation often go together for the same substrate, but they may diverge so that coding occurs alone.

Pleasure encoding may reach an apex of cortical localization in a mid-anterior subregion within the orbitofrontal cortex, where neuroimaging activity correlates strongly to subjective pleasantness ratings of food varieties (Kringelbach et al. 2003)—and to other pleasures such as sexual orgasms (Georgiadis et al. 2006), drugs (Völlm et al. 2004), chocolate (Small et al. 2001), and music (Blood and Zatorre 2001). Most importantly, mid-anterior orbitofrontal activity tracks changes in subjective pleasure, such as a decline in palatability when the reward value of one food was reduced by eating it to satiety (while remaining high to another food) (Kringelbach 2005) (Kringelbach et al. 2003). The mid-anterior subregion of orbitofrontal cortex is thus a prime candidate for the coding of subjective experience of pleasure (Kringelbach 2005).

Another coding site for positive hedonics in orbitofrontal cortex is along its medial edge that has activity related to the positive and negative valence of affective events (Kringelbach and Rolls 2004), contrasted to lateral portions that have been suggested to code unpleasant events (O’Doherty et al. 2001) (although lateral activity may reflect a signal to escape the situation, rather than displeasure per se (Iversen and Mishkin 1970; Kringelbach and
Rolls 2003; Hornak et al. 2004; Kringelbach and Rolls 2004)). This medial–lateral hedonic gradient interacts with an abstraction–concreteness gradient in the posterior–anterior dimension, so that more complex or abstract reinforcers (such as monetary gain and loss) (O’Doherty et al. 2001) are represented more anteriorly in the orbitofrontal cortex than less complex sensory rewards (such as taste) (Small et al. 2001). The medial region that codes pleasant sensations does not, however, appear to change its activity with reinforcer devaluation, and so may not reflect the full dynamics of pleasure.

Other cortical regions implicated in coding for pleasant stimuli include parts of the mid-insular (Craig 2009) and anterior cingulate cortices (Beckmann et al. 2009). As yet, however, it is not as clear as for the orbitofrontal cortex whether those regions specifically code pleasure or only emotion more generally (Feldman Barrett and Wager 2006). A related suggestion has emerged that the frontal left hemisphere plays a special lateralized role in positive affect more than the right hemisphere (Davidson and Irwin 1999), though how to reconcile left-positive findings with many other findings of bilateral activations of orbitofrontal and related cortical regions during hedonic processing remains an ongoing puzzle (Kringelbach 2005).

It remains still unknown, however, if mid-anterior orbitofrontal cortex or medial orbitofrontal cortex or any other cortical region actually causes a positive pleasure state. Clearly, damage to orbitofrontal cortex does impair pleasure-related decisions, including choices and context-related cognitions in humans, monkeys, and rats (Butter et al. 1963; Nauta 1971; Baylis and Gaffan 1991; Anderson et al. 1999; Baxter et al. 2000; Beer et al. 2003; Hornak et al. 2003; Pickens et al. 2003, 2005). But some caution regarding whether cortex generates positive affect states per se is indicated by the consideration that patients with lesions to the orbitofrontal cortex do still react normally to many pleasures, although sometimes showing inappropriate emotions (Damasio 1996; Anderson et al. 1999; Beer et al. 2003; Hornak et al. 2003). Hedonic capacity after prefrontal damage has not, however, yet been studied in carefully enough detail (e.g. using selective satiation paradigms (Kringelbach et al. 2003)), and it would be useful to have more information on the role of orbitofrontal cortex, insular cortex, and cingulate cortex in generating and modulating hedonic states.

Pleasure causation has been so far rather difficult to assess in humans given the limits of information from lesion studies, and the correlative nature of neuroimaging studies. A promising tool, however, is deep brain stimulation (DBS) which is a versatile and reversible technique that directly alters brain activity in a brain target and where the ensuing whole-brain activity can be measured with magnetoencephalography (MEG) (Kringelbach et al. 2007b). Pertinent to a view of happiness as freedom from distress, at least pain relief can be obtained from DBS of periaqueductal gray in the brainstem in humans (Gildenberg 2005), where specific neural signatures of pain have been found (Green et al. 2009), and where the pain relief is associated with activity in the mid-anterior orbitofrontal cortex, perhaps involving endogenous opioid release (Kringelbach et al. 2007a). Similarly, DBS may alleviate some unpleasant symptoms of depression, though without actually producing positive affect.

Famously, also, pleasure electrodes were reported to exist decades ago in animals and humans when implanted in subcortical structures including the nucleus accumbens, septum, and medial forebrain bundle (Olds and Milner 1954; Heath 1972) (Figure 2.2c). However, recently we and others have questioned whether most such electrodes truly caused pleasure, or instead, only a psychological process more akin to “wanting” without “liking”
In our view, it still remains unknown whether DBS causes true pleasure, or if so, where in the brain electrodes produce it.

**Loss of pleasure**

The lack of pleasure, anhedonia, is one of the most important symptoms of many mental illnesses including depression. It is difficult to conceive of anyone reporting happiness or well-being while so deprived of pleasure. Thus anhedonia is another potential avenue of evidence for the link between pleasure and happiness (Gorwood 2008).

The brain regions necessary for pleasure—but disrupted in anhedonia—are not yet clear. Core “liking” reactions to sensory pleasures appear relatively difficult to abolish absolutely in animals by a single brain lesion or drug, which may be very good in evolutionary terms. Only the ventral pallidum has emerged among brain hedonic hotspots as a site where damage fully abolishes the capacity for positive hedonic reaction in rodent studies, replacing even “liking” for sweetness with “disliking” gapes normally reserved for bitter or similarly noxious tastes (Cromwell and Berridge 1993; Aldridge and Berridge 2010). Interestingly, there are extensive connections from the ventral pallidum to the medial orbitofrontal cortex (Öngür and Price 2000).

On the basis of this evidence, the ventral pallidum might also be linked to human anhedonia. This brain region has not yet been directly surgically targeted by clinicians but there is anecdotal evidence that some patients with pallidotomies (of nearby globus pallidus, just above and behind the ventral pallidum) for Parkinson’s patients show flattened affect (Parkin et al. 2002) (T. Z. Aziz, personal communication), and stimulation of globus pallidus internus may help with depression (Kosel et al. 2007). A case study has also reported anhedonia following bilateral lesion to the ventral pallidum (Miller et al. 2006).

Alternatively, core “liking” for fundamental pleasures might persist intact but unacknowledged in anhedonia, while instead only more cognitive construals, including retrospective or anticipatory savoring, becomes impaired. That is, fundamental pleasure may not be abolished in depression after all. Instead, what is called anhedonia might be secondary to motivational deficits and cognitive misappraisals of rewards, or to an overlay of negative affective states. This may still disrupt life enjoyment, and perhaps render higher pleasures impossible.

Other potential regions targeted by DBS to help with depression and anhedonia include the nucleus accumbens (Schlaepfer et al. 2008) and the subgenual cingulate cortex (Mayberg et al. 2005). In addition, lesions of the posterior part of the anterior cingulate cortex have been used for the treatment of depression with some success (Steele et al. 2008).

**Bridging pleasure to meaning**

It is potentially interesting to note that all these structures either have close links with frontal cortical structures in the hedonic network (e.g. nucleus accumbens and ventral pallidum) or belong to what has been termed the brain’s default network which changes over early development (Fransson et al. 2007; Fair et al. 2008) (Figure 2.3).
FIG. 2.3 (Also see Plate 3). The brain's default network and eudaimonic–hedonic interaction. (a–c) The brain's default network (Gusnard and Raichle 2001; Addis et al. 2007) has been linked to self-awareness, remembering the past and prospecting the future (Addis et al. 2007). Some components overlap with pleasure networks, including midline structures such as the orbitofrontal, medial prefrontal, and cingulate cortices.
Figure 2.3 (continued) We wonder whether happiness might include a role for the default network, or for related neural circuits that contribute to computing relations between self and others, in evaluating eudaimonic meaning and interacting with hedonic circuits of positive affect. Examples show (d) key regions of the default network such as the anterior cingulate and orbitofrontal cortices that have a high density of opiate receptors (Willoch et al. 2004), (e) have been linked to depression (Drevets et al. 1997), and (f) its surgical treatment (Steele et al. 2008). (g) Subregional localization of function may be indicated by connectivity analyses of cingulate cortex (Beckmann et al. 2009) and related structures, (h) important in pleasure-related monitoring, learning, and memory (Kringelbach and Rolls 2004), (i) as well as self-knowledge, person perception, and other cognitive functions (Amodio and Frith 2006). (j) The default network may change over early life in children and pre-term babies (Fransson et al. 2007; Fair et al. 2008), (k) in pathological states including depression and vegetative states (Laureys et al. 2004), (l) and after lesions to its medial orbitofrontal and subgenual cingulate cortices that disrupt reality monitoring and create spontaneous confabulations (Schnider 2003).

Mention of the default network brings us back to the topic of eudaimonic happiness, and to potential interactions of hedonic brain circuits with circuits that assess meaningful relationships of self to social others. The default network is a steady-state circuit of the brain which becomes perturbed during cognitive tasks (Gusnard and Raichle 2001). Most pertinent here is an emerging literature that has proposed the default network to carry representations of self (Lou et al. 1999), internal modes of cognition (Buckner et al. 2008), and perhaps even states of consciousness (Laureys et al. 2004). Such functions might well be important to higher pleasures as well as meaningful aspects of happiness.

Although highly speculative, we wonder whether the default network might deserve further consideration for a role in connecting eudaimonic and hedonic happiness. At least, key regions of the frontal default network overlap with the hedonic network discussed earlier, such as the anterior cingulate and orbitofrontal cortices (Kringelbach and Rolls 2004; Amodio and Frith 2006; Steele et al. 2008; Beckmann et al. 2009), and have a relatively high density of opiate receptors (Willoch et al. 2004). And activity changes in the frontal default network, such as in the subgenual cingulate and orbitofrontal cortices, correlate to pathological changes in subjective hedonic experience, such as in depressed patients (Drevets et al. 1997).

Pathological self-representations by the frontal default network could also provide a potential link between hedonic distortions of happiness that are accompanied by eudaimonic dissatisfaction, such as in cognitive rumination of depression (Williams et al. 1996; Schnider 2003; Addis et al. 2007). Conversely, mindfulness-based cognitive therapy for depression, which aims to disengage from dysphoria-activated depressogenic thinking, might conceivably recruit default network circuitry to help mediate improvement in happiness via a linkage to hedonic circuitry (Teasdale et al. 2000).

Concluding remarks

The most difficult questions facing pleasure and happiness research remain the nature of its subjective experience and the relation of hedonic components (pleasure or positive affect)
to eudaimonic components (cognitive appraisals of meaning and life satisfaction). While some progress has been made in understanding brain hedonics, it is important not to over-interpret. In particular we have still not made substantial progress towards understanding the functional neuroanatomy of happiness.

In this review we have, however, identified a number of brain regions that are important in the brain’s hedonic networks, and speculated on potential interaction with eudaimonic networks. While it remains unclear how pleasure and happiness are exactly linked, it may be safe to say at least that the pathological lack of pleasure, in anhedonia or dysphoria, amounts to a formidable obstacle to happiness.

In social animals like humans, social interactions with conspecifics are fundamental and central to enhancing the other pleasures. Humans are intensely social, and data indicate that one of the most important factors for happiness is social relationships with other people. Social pleasures may still include vital sensory features such as visual faces, touch features of grooming and caress, as well as in humans more abstract and cognitive features of social reward and relationship evaluation (Adolphs 2003).

In particular, adult pair bonds and attachment bonds between parents and infants are likely to be extremely important for the survival of the species (Kringelbach et al. 2008). The breakdown of these bonds is all too common and can lead to great unhappiness. And even bond formation can potentially disrupt happiness, such as in transient parental depression after birth of an infant (in over 10% of mothers and approximately 3% of fathers (Cooper and Murray 1998)). Progress in understanding the hedonics of social bonds could be useful in understanding happiness.

Social neuroscience is beginning to unravel some of the complex dynamics of human social interactions. One of its major challenges is to map the developmental changes in reward processing over a lifespan. Another challenge is to understand how brain networks underlying fundamental pleasure relate to higher pleasures such as music, dance, play and flow, and to happiness.

Further, so far as positive affect contributes to happiness, then considerable progress has been made in understanding the neurobiology of pleasure in ways that might be relevant. For example, we can imagine several possibilities to relate happiness to particular hedonic psychological processes discussed previously. Thus, one way to conceive of hedonic happiness is as “liking” without “wanting.” That is, a state of pleasure without disruptive desires, a state of contentment (Kringelbach 2009). Another possibility is that moderate “wanting” matched to positive “liking” facilitates engagement with the world. A little incentive salience may add zest to the perception of life and perhaps even promote the construction of meaning, just as in some patients DBS may help lift the veil of depression by making life events more appealing. However, too much “wanting” can readily spiral into maladaptive patterns such as addiction, and is a direct route to great unhappiness. Finally, happiness might spring from higher pleasures, positive appraisals of life meaning, and social connectedness, all combined and merged by interaction between the brain’s default networks and pleasure networks. Achieving the right hedonic balance in such ways may be crucial to keep one not just ticking over but perhaps even happy.

Future scientific advances may provide a better sorting of psychological features of happiness and its underlying brain networks. If so, it remains a distinct possibility that more among us may be one day shifted into a better situation to enjoy daily events, to find life meaningful and worth living—and perhaps even to achieve a degree of bliss.
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REFERENCES


