Chapter 7
Towards a Neuroscience of Well-Being: Implications of Insights from Pleasure Research

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7.1 Introduction

The study of well-being or positive psychology is part of a long tradition reaching back to Aristotle, where well-being or happiness has been usefully proposed to consist of at least two ingredients: *hedonia* and *eudaimonia* (Aristotle 350 B. C. 2009; Seligman et al. 2005). Definitions by philosophers and psychologists have varied, but most generally agree that hedonia corresponds psychologically to pleasure. By comparison, eudaimonia has been less easy to define, but for most it corresponds to some aspect of a life well lived and not to any particular emotional state. In this review, we take eudaimonia to mean essentially a life experienced as valuably meaningful and as engaging.

Hedonic processing and eudaimonic meaningfulness may thus appear very different in terms of definition and conceptualization. At the same time, empirical findings have been found well-being to involve both together. Questionnaire scores for hedonia and eudaimonia typically converge in the same individuals (Diener et al. 2008; Kuppens et al. 2008). Thus, if a person self-reports to be hedonically happy, then that same person is also likely to report a high sense of positive meaningfulness in life.

The tendency for coherence between ratings of pleasure and meaningfulness opens a potential window of opportunity for the neuroscientific study of both

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aspects of well-being (Kringelbach and Berridge 2009; Urry et al. 2004). If both ingredients occur in the same people, then the neurobiological bases for both coexist in the same brains. If both cohere, then identifying neural markers of one may give a toehold into identifying the other. Still, most would probably agree that eudaimonic happiness poses harder challenges to psychology and neuroscience. It is difficult even to define life meaningfulness in a way as to avoid dispute, let alone to tie a happy sense of meaningfulness to any specific brain patterns of activation. The difficulties of approaching eudaimonic meaning are not insurmountable in principle, but for the foreseeable short term seem likely to remain obstacles to affective neuroscience.

We have therefore chosen to focus mostly upon the hedonia or pleasure aspect of well-being. The pleasure aspect is far more tractable, and can be inspected against a growing background of understanding of the neural foundations for specific pleasures. Supporting a hedonic approach to happiness, happy people typically take more pleasure from life. Indeed it has been suggested that the best and simplest measure of well-being may be to merely ask people how they hedonically feel right now—again and again—so as to track their hedonic accumulation across daily life (Kahneman 1999a). Such repeated self-reports of hedonic *states* could also be used to identify more stable neurobiological hedonic brain *traits* that dispose particular individuals toward happiness.

Conversely, most will agree that the capacity for pleasure is essential to normal well-being. The pathological loss of pleasure, anhedonia, which is found in many affective disorders is devastating and precludes well-being. Our aim in this review is to highlight findings from recent research on brain mechanisms of pleasure and to ask how to higher states of hedonia might be generated to produce well-being, and conversely what might go wrong in affective disorders (Berridge and Kringelbach 2008; Kringelbach and Berridge 2010b; Leknes and Tracey 2010; Smith et al. 2010).

In passing, we 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 below, which does not necessarily involve much actual pleasure. We also note that while our focus is mainly on mechanisms of stimulus-bound sensory pleasure, this reflects merely current experimental research, and the evidence appears to show that pleasure generators can be independent of sensory input as found, for example, in locked-in patients (Bruno et al. 2011). Further, 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 (Diener et al. 2008; Gilbert 2006; Kahneman 1999a; Seligman et al. 2005).

7.2 A Science of Pleasure

Pleasure has been proposed to be evolution's boldest trick allowing species and organisms to ensure survival and procreation in both individuals and species (Kringelbach 2009). Substantial mechanisms for pleasure would be selected for and conserved only if they ultimately served a central role in fulfilling Darwinian imperatives of gene proliferation via improved survival and procreation, suggesting the capacity for pleasure must have been fundamentally important in evolutionary fitness (Cabanac 2010; Darwin 1872; Nesse 2002; Panksepp 1998).

Pleasure is never merely a sensation, even for sensory pleasures (Frijda 2010; Kringelbach 2010; Kringelbach and Berridge 2010b; Ryle 1954). Instead pleasure always requires the recruitment of specialized brain systems to actively paint an additional "hedonic gloss" onto a sensation. Active recruitment of brain pleasure-generating systems is what makes a pleasant experience 'liked' (Fig. 7.1).

The capacity of certain stimuli, such as a sweet taste or a loved one, to reliably elicit pleasure—to nearly always be painted with a hedonic gloss—reflects the privileged ability of such stimuli to engage these hedonic brain systems responsible for manufacturing and applying the gloss. Hedonic brain systems are well-developed in the brain, spanning subcortical and cortical levels, and are quite similar across humans and other animals.

Some might be surprised by high similarity across species, or by substantial subcortical contributions, at least if one thinks of pleasure as uniquely human and as emerging only at the top of the brain. The neural similarity indicates an early phylogenetic appearance of neural circuits for pleasure and a conservation of those circuits, including deep brain circuits, in the elaboration of later species, including humans.

The fundamental rewards afforded by biological evolution include food, sex and conspecifics. Food is one of the most universal routes to pleasure (Kringelbach 2004). Sex is another potent natural sensory pleasure which involves some of the same brain circuits (Georgiadis and Kringelbach 2012). Many other special classes of stimuli also appear tap into the same limbic circuits (Everitt et al. 2008; Kelley and Berridge 2002; Koob and Volkow 2010).

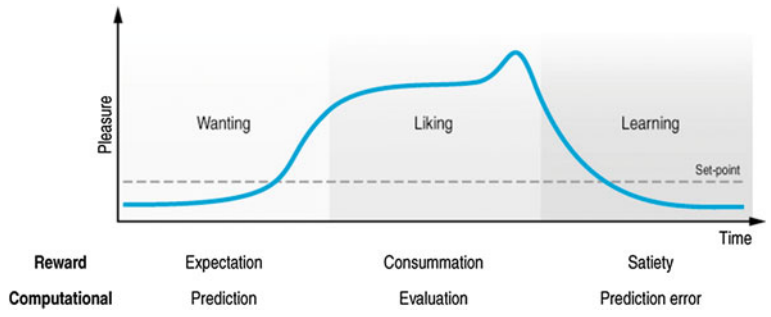

Fig. 7.1 Pleasure cycles

Also social interaction with conspecifics draws on overlapping neural systems (Frith and Frith 1999). In fact, it might well be even from an evolutionary perspective that in humans, at least, the social pleasures are often as pleasurable as the basic sensory pleasures.

Most uniquely, humans have many prominent higher order, abstract or cultural pleasures, including personal achievement as well as intellectual, artistic, musical, altruistic, and transcendent pleasures. While the neuroscience of higher pleasures is in relative infancy, even here there seems overlap in brain circuits with more basic hedonic pleasures (Frijda 2010; Harris et al. 2009; Leknes and Tracey 2010; Salimpoor et al. 2011; Skov 2010; Vuust and Kringelbach 2010). As such, brains may be viewed as having conserved and re-cycled some of the same neural mechanisms of hedonic generation for higher pleasures that originated early in evolution for simpler sensory pleasures.

7.3 The Neuroanatomy of Pleasure and Reward

Our subjective experience may suggest that a state of positive affect is a unitary process, but affective neuroscience analyses have indicated that even the simplest pleasant experience, such as a mere sensory reward, is actually a more complex set of cyclical processes containing several psychological components, each with distinguishable neurobiological mechanisms (Berridge et al. 2009; Kringelbach and Berridge 2009; Leknes and Tracey 2010). These include at least the three components of wanting, liking and learning. *Liking* is the actual pleasure component or hedonic impact of a reward, *wanting* is the motivation for reward and *learning* includes the associations, representations and predictions about future rewards based on past experiences (Fig. 7.2).

We distinguish between the conscious and non-conscious aspects of these subcomponents. Both exist in people (Winkielman et al. 2005), but the latter at least can also be studied in other animals in ways that help better reveal the underlying neural generating mechanisms. At the potentially non-conscious level, we use quotation marks to indicate that we are describing objective, behavioural or neural measures of these underlying brain processes. As such, 'liking' reactions result from activity in identifiable brain systems that paint hedonic value on a sensation such as sweetness. Similarly, 'wanting' includes incentive salience or motivational processes within reward that mirror hedonic 'liking' and make stimuli attractive when attributed to them by mesolimbic brain systems. Finally, 'learning' includes a wide range of processes linked to implicit knowledge as well as associative conditioning, such as basic Pavlovian and instrumental associations.

At the conscious level liking is the conscious experiences of pleasure, in the ordinary sense of the word, which may be elaborated out of core 'liking' reactions by cognitive brain mechanisms of awareness. Conscious wanting includes conscious desires for incentives or cognitive goals, while learning includes the updating of explicit and cognitive predictions (Friston and Kiebel 2009; Zhang et al. 2009).

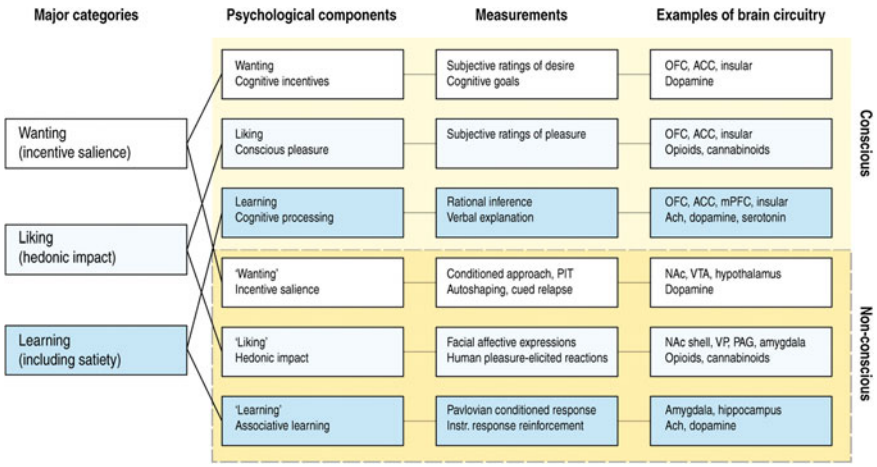

Fig. 7.2 Measuring reward and hedonia. Hedonic reward processes related to well-being are multifaceted psychological concepts that constantly interact and require careful scientific analysis to tease apart. Measurements or behavioral procedures that are especially sensitive markers of the each of the processes are listed (*third column*)

This universal experience of pleasure as a consciously felt feeling is perhaps the reason why pleasure has seemed purely subjective to many thinkers. But related to the notion that pleasure naturally evolved, we suggest that pleasure also has objective aspects that can be detected in brain and mind. Note again, however, the underlying similarities of brain mechanisms for generating sensory pleasures in the brains of most mammals, both humans and nonhumans alike (Fig. 7.3). It seems unlikely so much neural machinery would have been selected and conserved across species if it had no function. Basic pleasure reactions have always had objective consequences, and brain mechanisms for hedonic reactions have long been functionally useful—even before any additional mechanisms appeared that characterize any human-unique aspects of subjective feelings of pleasure. In a sense, we suggest hedonic reactions have been too important to survival for pleasure to be exclusively subjective.

Pleasure Generators: Hedonic Hotspots in the Brain

How does pleasure actually arise in a brain? The brain appears frugal in mechanisms that are causally sufficient to generate 'liking' or magnify pleasure to high levels. These few mechanisms are candidate brain wellsprings for hedonic happiness.

Compelling evidence for pleasure causation as increases in 'liking' reactions has so far been found for only a few brain substrates, or hedonic hotspots. Those hedonic hotspots mostly reside—surprisingly, if one thought pleasure to be

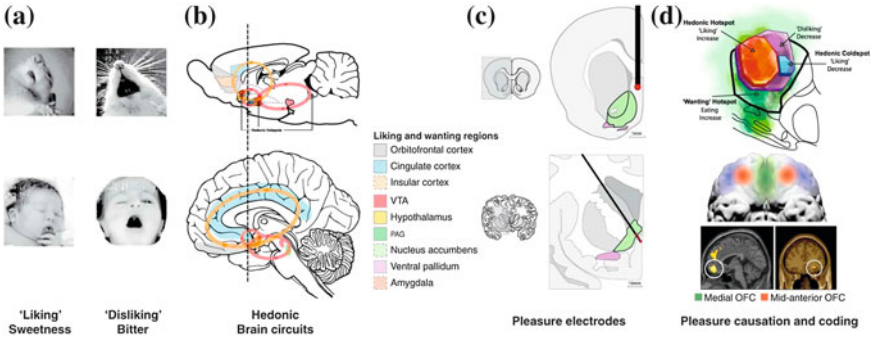

Fig. 7.3 Hedonic brain circuitry. The schematic figure shows the brain regions for causing and coding fundamental pleasures in rodents and humans. **a** Facial 'liking' and 'disliking' expressions elicited by sweet and bitter taste are similar in rodents and human infants. **b**, **d** Pleasure causation has been identified in rodents as arising from interlinked subcortical hedonic hotspots, such as in nucleus accumbens and ventral pallidum, where neural activation may increase 'liking' expressions to sweetness. Similar pleasure coding and incentive salience networks have also been identified in humans. **c** We believe the so-called 'pleasure' electrodes in rodents and humans were unlikely to have elicited much 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, which differentiate subjective pleasantness from valence processing of aspects the same stimulus, such as a pleasant food

cortical—deep below the neocortex in subcortical structures. Our strategy to find such neural generators of pleasure gloss has relied on activating neural mechanisms underlying natural 'liking' reactions to intensely pleasant sensations. An example of 'liking' is the positive affective facial expression elicited by the hedonic impact of sweet tastes in newborn human infants (Fig. 7.3a), such as tongue protrusions that can lick the lips. By contrast, nasty bitter tastes instead elicit facial 'disliking' expressions of disgust such as gapes, nose and brow wrinkling, and shaking of the head. Many of these affective expressions are similar and homologous (sharing features such as identical allometric timing laws) in humans, orangutans, chimpanzees, monkeys, and even rats and mice (Steiner et al. 2001). Homology in origin of 'liking' reactions implies that the underlying hedonic brain mechanisms are similar in humans and other animals, opening the way for an affective neuroscience of pleasure generators that bridges both.

Subcortical Hedonic Hotspots in Nucleus Accumbens, Ventral Pallidum and Brainstem

Some insight into pleasure-causing circuitry of human brains has been gained by affective neuroscience studies in rodents in which the hedonic hotspots are activated to magnify a sensory pleasure, and so reveal the location and neurotransmitter identity of the generating mechanism for intense 'liking'. A hedonic hotspot

is capable of generating enhancements of 'liking' reactions to a sensory pleasure such as sweetness, when opioid, endocannabinoid or other hedonic neurochemical circuits within the hotspot are stimulated (Mahler et al. 2007; Peciña and Berridge 2005; Peciña et al. 2006; Smith and Berridge 2005). In rodent studies, the hotspots can be activated by painless microinjections of drug droplets that stimulate neurotransmitter receptors on neurons nearby. Within the hotspot, drug microinjections magnify the hedonic impact of a sweet pleasure, whereas outside the border of the hotspot the same microinjections fail to elevate 'liking'.

The results of such studies reveal a network of brain hedonic hotspots, distributed as a chain of 'liking'-enhancing islands of brain tissue across several deep structures of the brain. The network of hedonic hotspots acts together as a coordinated whole to amplify core pleasure reactions. Each brain hotspot may be merely a cubic-millimeter or so in volume in the rodent brain (and would be expected to be a cubic-centimeter or so in you, if proportional to the larger human volume of whole brain). The small size of each anatomical hotspot indicates a surprisingly localized concentration of sufficient-cause mechanisms for generating an intense pleasure in the brain. The network properties reveal a fragile substrate for pleasure enhancement that requires unanimity across the several parts in order to elevate hedonic 'liking' (Peciña 2008; Peciña and Smith 2010; Smith et al. 2010).

A major hotspot has been found in the nucleus accumbens, a brain structure at the bottom font of the brain, specifically in its medial shell region near the center of the structure. Other hotspots have been found further back in the brain. For example, a very important hedonic hotspot lies in the ventral pallidum, which is near the hypothalamus near the very bottom center of the forebrain and receives most outputs from the nucleus accumbens. Still other hotspots may be found in more distant parts of the rodent brain, possibly as far front in limbic regions of prefrontal cortex, and almost certainly as far back as deep brainstem regions including the parabrachial nucleus in the top of the pons.

Analogous to scattered islands that form a single archipelago, the network of distributed hedonic hotspots forms a functional integrated circuit, which obeys control rules that are largely hierarchical and organized into brain levels (Aldridge et al. 1993; Berridge and Fentress 1986; Grill and Norgren 1978; Peciña et al. 2006). At the highest levels, the hotspot network may function as a more democratic heterarchy, in which unanimity of positive votes across hotspots is required in order to generate a greater pleasure. For example, any successful enhancement that starts in one hotspot involves recruiting neuronal activation across other hotspots simultaneously, to create a network of several that all vote 'yes' together for more pleasure. Conversely, a pleasure enhancement initiated by opioid activation of one hotspot can be vetoed by an opposite vote of 'no' from another hotspot where opioid signals are suppressed. Such findings reveal the need for unanimity across hotspots in order for a greater pleasure to be produced, and the potential fragility of hedonic enhancement if any hotspot defects (Smith and Berridge 2007; Smith et al. 2010).

But all of these findings on brain pleasure generators are focused on making pleasures *nicer than usual*. Neurochemical activation of hedonic hotspots creates a brain wellspring for intense pleasure when candidate sensations are encountered, generating high hedonic peaks of sensory pleasure.

Yet well-being is a more continuous and quotidian state of *hedonic normalcy* in a slightly positive balance. What in the brain is required for creating the daily continual baseline level of a normal pleasure gloss? It turns out that only some of the hotspots that amplify pleasure are necessary for normal hedonic levels of 'liking' to pleasant sensations, and particularly the one in ventral pallidum.

In both the clinical literature and in our experiments, normal core 'liking' reactions to pleasure are relatively difficult to abolish absolutely by any single event, condition, brain lesion or drug (Bruno et al. 2011; Peciña 2008; Peciña and Smith 2010; Smith et al. 2010). Resilience of brain circuits for normal baseline pleasures may be very good in evolutionary terms.

Hedonic resilience may also be related to why many people can eventually regain a reasonably happy state even after catastrophic events (Diener et al. 2006; Gilbert 2006; Kahneman 1999b). Strikingly, hedonic balance may be retained even in the most extreme situations. One of the most extreme situations must surely be locked-in syndrome, a brain condition that leaves the person fully aware and cognitively intact but completely paralyzed to the extent of being able only to make slight movements of an eye or eyelid. Yet in the face of even this devastating degree of paralysis, locked-in patients may often still be happy. A recent study found that 72 % of locked-in respondents did report themselves to be moderately happy. The average response of this happy yet massively incapacitated group was +3 out of a hedonic scale from −5 to +5, where +3 corresponded to 'very well' (between +2 = 'well', and +4 = "almost as well at the best period in my life prior to having locked-in syndrome"). The remaining 28 % of locked-in respondents, who were much more likely to also be experiencing pain, reported themselves to be unhappy at −4, but even this corresponded only to "almost as bad as the worst period in my life before locked-in syndrome" (and not quite as bad as −5 = "as bad as the worst period in my life before"); only 7 % wished for euthanasia (Bruno et al. 2011). Hedonic resilience can apparently often persist with seemingly little to go on, still generated by hedonic circuits within the person.

Perhaps not surprisingly then, only one brain lesion has been found in our lab studies to destroy a normal sensory pleasure, and convert sweetness into a nasty experience: the ventral pallidum hotspot. This site is still preserved in locked-in patients, perhaps contributing to their remaining well-being. Damage to this unique brain site abolishes hedonic 'liking' reactions to sweetness and replaces instead with disgust or 'disliking' reactions (e.g., gapes) as though the sweet taste had turned bitter (Berridge et al. 2010; Cromwell and Berridge 1993; Smith et al. 2010). The ventral pallidum is the chief recipient of output from the nucleus accumbens and part of a corticolimbic circuit that extends from prefrontal cortex to nucleus accumbens to ventral pallidum, before looping up via thalamus to begin the circuit all over again in prefrontal cortex (Smith et al. 2010).

An important question is how similar or different the ventral pallidum role in pleasure might be in humans compared to in rodents. Currently we do not have much available data on the hedonic consequences of human hotspot loss, because a human stroke or tumor lesion rarely damages the ventral pallidum on both sides of the brain without also damaging hypothalamus and related structures in between, producing severe incapacitation that precludes much psychological assessment. Yet, in a rare human case report of a brain lesion that did rather selectively damage the ventral pallidal region on both sides without much else, positive affect and craving for rewards was reported to be much reduced. The patient's brain had incurred damage to ventral pallidum (and nearby medial globus pallidus) due to oxygen starvation when the patient stopped breathing during an enormous drug (Miller et al. 2006). Afterwards the pallidal-lesion patient reported that his feelings became dominated by depression, hopelessness, guilt, and anhedonia. Even formerly craved and hedonic sensations like drinking alcohol lost their feelings of pleasure for him, and he no longer craved the many drugs of abuse that he had previously avidly consumed. Even this lesion probably did not fully destroy his ventral pallidum, and perhaps this is why he was not as strongly seized by disgust as a rat would be if it had complete lesions of the ventral pallidum hotspot. Instead, the patient still continued to eat and drink normally after his lesion, and even gained weight. But his apparent dramatic decline in hedonic well-being suggests impairment in normal pleasure, and helps confirm a continuity between the ventral pallidum hotspot and human hedonia. We have also encountered anecdotal evidence that in some patients with pallidotomies (of nearby globus pallidus, just above and behind the human ventral pallidum) for Parkinson's patients, this led to severely flattened affect or anhedonia (Aziz, personal communication).

The striking restriction of brain substrates where damage converts 'liking' to 'disliking' seems a testimonial to the robustness of the brain's capacity for a basic pleasure reaction, and also perhaps an insight into what pathological mechanisms result in true anhedonia.

Additional Pleasure Codes in the Brain

The occurrence of pleasure is coded by neural activity in many additional forebrain sites beyond the hotspots mentioned above, including in amygdala and in the cortex: especially prefrontal cortical regions such as orbitofrontal cortex, anterior cingulate cortex, and insular cortex, (Grabenhorst and Rolls 2011; Kringelbach 2010; Salimpoor et al. 2011) (Fig. 7.4).

But not all brain structures that *code* for pleasure actually help to *cause* it. *Coding* of pleasure in the brain can reflect not only pleasure causation but also the neural consequences of pleasure: brain activity that results from pleasure enhancement but causes another function, such as cognition or learning. This implies that some brain activity may both cause and code pleasure reactions, whereas others do not cause pleasure but may code it while causing other

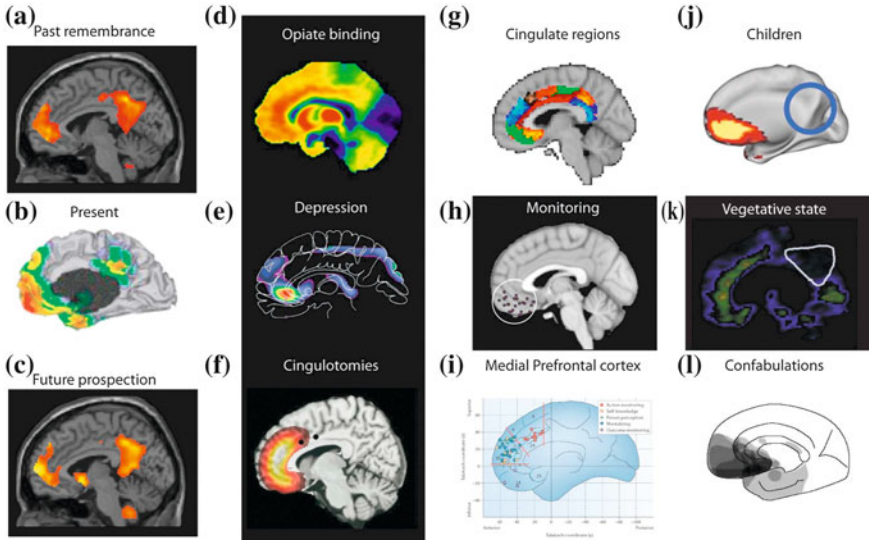

Fig. 7.4 The brain's default network and eudaimonic—hedonic interaction. **a–c** The brain's default network has been linked to self awareness, remembering the past and prospecting the future. Some components overlap with pleasure networks. We wonder whether happiness might include a role for the default 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 key regions of the default network such as **d** the anterior cingulate and orbitofrontal cortices that have a high density of opiate receptors, **e** have been linked to depression, and **f** its surgical treatment **g** have been implicated by connectivity analyses, **h** are implicated in pleasure-related cognitive functions such as monitoring, learning and memory, **i** or in self-knowledge, person perception and other cognitive functions. **j** The default network may change over early life in infants and children, **k** in pathological states including depression and vegetative states, **l** and after cortical lesions that disrupt reality monitoring and create spontaneous confabulations

psychological or behavioral processes. Neural *coding* is inferred in practice by measuring brain *activity correlated to a pleasure*, using techniques such as PET, fMRI and MEG neuroimaging in humans, or electrophysiological or neurochemical activation measures in animals presented with a rewarding stimulus. Causation is generally inferred on the basis of a *change* in pleasure as a *consequence of a brain manipulation* such as lesion or stimulation.

As a general rule, we suggest that brains operate by the principle of 'many more codes than causes' for pleasure. In part, the greater number of hedonic coding sites results from the tendency of signals to spread beyond their source, as well as from the massive need for brain systems to translate pleasure signals into many other psychological functions, such as learning and memory, cognitive representations, decisions, action, and consciousness.

Code-but-not-cause systems might nonetheless be reliable indicators that a pleasant event is occurring, because they must take pleasure signals as inputs to achieve other component processes in reward and related. We distinguish here

between the cognitive representations and memories of reward (reward learning) and the motivational value appraisals or decisions (reward wanting). For example, parts of the prefrontal cortex regions sensitively code reward and hedonic impact, as described below. Yet damage to ventromedial region of prefrontal cortex may impair the cognitive use of emotional reactions without necessarily impairing the capacity to experience the hedonic impact of those emotional reactions (Bechara et al. 1997; Damasio 2004; Kringelbach 2005). The difference between coding and causing poses challenges to human affective neuroscience studies, where lesions, stimulations or other causal tools are rarely available.

7.4 Cortical Cognition and Pleasure

In humans, evidence suggests that pleasure encoding may reach an apex of cortical localization in a subregion of orbitofrontal cortex: this hedonic-coding site is placed in the mid-anterior and roughly mid-lateral zone of the orbitofrontal region. Here neuroimaging activity in people particularly correlates strongly to subjective pleasantness ratings of food varieties—and to other pleasures such as sexual orgasms, drugs, chocolate, and music (Geogiadis and Kortekaas 2010; Kringelbach and Berridge 2010a; Leknes and Tracey 2010; Veldhuizen et al. 2010; Vuust and Kringelbach 2010). Most importantly, activity in this special mid-anterior zone of orbitofrontal cortex 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). The mid-anterior subregion of orbitofrontal cortex is thus a prime candidate for the coding of subjective experience of pleasure (Kringelbach 2005).

Another potential 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, contrasted to lateral portions that have been suggested to code unpleasant events (although lateral activity may reflect a signal to escape the situation, rather than displeasure per se) (Kringelbach 2010; Kringelbach and Rolls 2004). This medial–lateral hedonic gradient in orbitofrontal cortex interacts with an abstraction-concreteness gradient in the posterior-anterior dimension, so that more complex or abstract reinforcers (such as monetary gain and loss) are represented more anteriorly in the orbitofrontal cortex than less complex sensory rewards (such as taste). The medial region that codes pleasant sensations does not, however, appear to change its activity with reinforcer devaluation as effectively as the mid-anterior subregion that best codes hedonics, and so the medial region may not reflect the full dynamics of pleasure.

A malfunction of these hedonic mechanisms in the orbitofrontal cortex could explain the profound changes in eating habits (escalating desire for sweet food coupled with reduced satiety) that are often followed by enormous weight gain in patients with frontotemporal dementia. This progressive neurodegenerative disorder is associated with major and pervasive behavioural changes in personality

and social conduct resembling those produced by orbitofrontal lesions (although it should be noted that more focal lesions to the orbitofrontal cortex have not to date been associated with obesity) (Rahman et al. 1999). It has become clear recently that the orbitofrontal cortex also has an important role in emotional disorders such as depression and addiction (Kringelbach 2005).

Beyond orbitofrontal cortex, other cortical regions implicated in coding for pleasant stimuli include parts of the mid-insular (Craig 2009) and anterior cingulate cortices. 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 et al. 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 2004). Most specifically related to well-being, resting EEG activity in left prefrontal cortex has been reported to be higher in individuals with greater eudaimonic and hedonic well-being (Urry et al. 2004). How to reconcile left-positive findings with many other findings of bilateral activity in orbitofrontal and related cortical regions during hedonic processing remains an ongoing puzzle.

Cortical Causation of Human Pleasure?

Despite the evidence above for hedonic coding, however, it still remains unknown if even the mid-anterior pleasure-coding site of orbitofrontal cortex actually *causes* a positive pleasure state. It would be of considerable interest to investigate whether any of these sub-regions of the orbitofrontal cortex are necessary or sufficient causes of pleasure, or alternatively whether their role is restricted to cognitive elaboration of value, and translation of hedonic affect into goal-directed plans.

The proposed link to subjective hedonic processing might make the orbitofrontal cortex an important gateway for neuroscientific analyses of human subjective conscious experience. Some have even suggested that the orbitofrontal and anterior cingulate cortices could be viewed as part of a global workspace for access to consciousness with the specific role of evaluating the affective valence of stimuli (Dehaene et al. 1998; Kringelbach and Berridge 2010a). In this context, it is interesting that the medial parts of the orbitofrontal are part of a proposed network for the baseline activity of the human brain at rest (Gusnard et al. 2001), as this would place the orbitofrontal cortex as a key node in the network subserving consciousness. This could potentially explain why all our subjective experiences have an emotional tone and perhaps even why we have conscious pleasure.

One way of investigating this causation question would be to ask whether the orbitofrontal cortex is actually required for normal pleasure reactions or conscious feelings. Only scattered data are available, primarily from historical and case study sources. Prefrontal lobotomies were performed on thousands of human patients in the 1950s, and may provide some insights. If orbitofrontal or other prefrontal areas are necessary for basic 'liking' reactions, these lobotomy patients should no longer

have been able to feel pleasure. Yet perhaps surprisingly from this perspective, prefrontal lobotomy may not produce a catastrophic loss of pleasure feelings as far as one can tell from the available literature. Although many subtle emotional deficits occur in how patients describe or act upon their emotions after damage to prefrontal cortex the capacity for basic 'liking' reactions appeared to remain intact. Lobotomy patients were by no means oblivious to the pleasures of food, sex or other rewards.

Modern analyses of more focal prefrontal lesions report deficits in cognitive-emotional processing of decisions of human patients, similarly do not indicate a total loss of the capacity for pleasures (Bechara et al. 2000; Damasio 1999; Damasio 2004; Hornak et al. 2003). Decisions are often profoundly imbalanced in such patients but positive hedonia does not seem abolished by medial prefrontal or orbitofrontal cortex lesions.

Such considerations suggest that orbitofrontal cortex might be more important to translating hedonic information into cognitive representations and decisions than to generating a core 'liking' reaction to pleasant events (Burke et al. 2010; Dickinson and Balleine 2010).

Such evidence leads us to suggest that that the human prefrontal cortex might not actually be needed to cause pleasure, or at least not all pleasures. It is possible that the main role of the prefrontal cortex in pleasure is to act as the interface of higher order processing such as consciousness and attention to the non-conscious pleasure generators in primarily sub-cortical regions.

At its extreme, this position might view hedonic reactions as arising from subcortical structures even when the subcortical brain is on its own and unable to interact with neocortex. Some further evidence from humans, as well as much from animals, supports a subcortical emphasis for pleasure generation. For example, Shewmon et al. described several hydranencephalic cases, including a 6-year old boy with congenital "absence of cerebral tissue rostral to the thalamus, except for small mesial temporal-lobe remnants" (Shewmon et al. 1999, p. 364) and a tissue-lined cyst, who nevertheless "smiled when spoken to and giggled when played with. These human interactions were much more intense than, and qualitatively different from, his positive reactions to favorite toys and music" (Shewmon et al. 1999, p. 366). Similarly, Merker suggested that hydranencephalic children "express pleasure by smiling and laughter, and aversion by "fussing," arching of the back and crying (in many gradations), their faces being animated by these emotional states. A familiar adult can employ this responsiveness to build up play sequences predictably progressing from smiling, through giggling, to laughter and great excitement on the part of the child." (Merker 2007, p. 79).

Such cases of emotional reaction without (much) cortex raise fascinating questions for future consideration about the relative roles of cortical regions versus subcortical structures in human pleasures. However, no matter what conclusion is reached regarding pleasure generation, there seems general consensus that neocortex is crucial to link affect with complex cognition and introspection about hedonic states.

Controversial Subcortical Pleasure Generators

Several other particular limbic substrates, even subcortical ones, which were once thought to cause pleasure have now turned out not to do so after all. These include the mesolimbic dopamine system and many so-called pleasure electrodes in related brain substrates.

Mesolimbic dopamine was long regarded as a pleasure neurotransmitter, but now is increasingly thought by many neuroscientists to fail to live up to its pleasure label. Instead, dopamine is currently thought by many to facilitate some psychological valuation process besides either learning or pleasure 'liking'. Suggestions have included motivational incentive salience, arousal, motivation, and memory consolidation. We think it safe to sum up by saying that the consensus among affective neuroscientists today is that brain mesolimbic dopamine is not, after all, primarily a pleasure neurotransmitter.

Similarly, 'pleasure electrodes' in the brain for 50 years were supposed to tap directly into brain pleasure circuits (Olds 1956). However, we believe that many so-called 'pleasure electrodes' may actually have failed to truly cause significant pleasure at all (Kringelbach and Berridge 2012). Instead we suggest most electrodes more exclusively activated only the 'wanting' component of reward (similar to mesolimbic dopamine stimulation; which indeed is typically activated by such electrodes). Such electrode activations may be sought out, or may stimulate seeking of external rewards (food, sex, gambling, shopping, etc.), yet need not be pleasant themselves.

7.5 Towards a Balanced Brain

It is interesting to note that all brain structures discussed above or being targeted for brain-based treatments of pathological mood disorders today either have close links with the hedonic network we have considered (e.g., orbitofrontal cortex, nucleus accumbens and ventral pallidum, etc.) or belong to what has been termed the brain's default network which changes over early development (e.g., additional regions of prefrontal cortex, or of cingulate cortex, temporal cortex, and parietal cortex) (Fair et al. 2008; Fransson et al. 2007) (Fig. 7.4).

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 et al. 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 above, such as the anterior cingulate and orbitofrontal cortices, and have a relatively high density of opiate receptors. Eudaimonic well being may be correlated with activity in the anterior cingulate and in left prefrontal cortex, perhaps though the ability to suppress negative emotions (Urry et al. 2004; Urry et al. 2006; van Reekum et al. 2007). 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 (Davidson et al. 2002).

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. 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.

Beyond the default network are other cortical networks in which activations may correspond with evaluations of self, others, and meaningful themes related to life satisfaction (Heller et al. 2009; Schacter et al. 2007). These include dorsolateral prefrontal, and other parietal and temporal areas of cortex and related networks. In short, the default network and networks whose activation encodes evaluations of self and life meaning stand among the brain candidates for a substrate that might mediate eudaimonia appraisals. How these networks might embody eudaimonia components, and link evaluations of life meaningfulness and satisfaction with pleasurable states of hedonia, remains a major challenge to psychological neuroscience for the future.

7.6 Conclusions

As shown in this review, the main role of pleasure is to help initiate, sustain or terminate the phases involved in pleasure cycles of reward. Pleasure can thus be said to play a crucial role in guiding the survival-related decision-making involved in optimizing resource allocation of brain resources. From this perspective *optimization* rather than *maximization* of pleasure processing is the most sensible strategy since this leads to the most optimal brain resource allocation.

It is not straightforward, however, to bring this balancing view of hedonia a step further to understand the relation of sensory pleasure to the more enduring hedonia of well-being, the interaction between hedonia (pleasure or positive affect) and eudaimonia (cognitive appraisals of meaning and life satisfaction), within underlying brain systems, and the nature of their subjective experience.

While some progress has been made in understanding brain hedonics, it is important not to over-interpret the findings. In particular we have still not made substantial progress towards understanding the functional neuroscience specifically of well-being or happiness. We have merely aimed to sketch out the beginnings of a hedonic approach.

Further, when all is done, one may still question our entire effort, based as it is largely on evidence from sensory pleasures. Some will demur that pleasure, our chief focus here, is irrelevant after all to true happiness. For many, this view might be well expressed by the words of John Stuart Mill, "It is better to be a human being dissatisfied than a pig satisfied; better to be Socrates dissatisfied than a fool satisfied" (Mill et al. 1998, p. 57). By the view expressed in this quotation, a life filled with the most intense pleasures of pigs or fools would never be enough for happiness. That is because true happiness hinges on a superior kind of psychological or eudaimonic richness that is unique to the enlightened, though hedonically dissatisfied, Socrates. But note that Mill, however, seemed to say elsewhere that hedonic pleasure was important to happiness too.

At the opposite extreme, Sigmund Freud seemed to take a purely hedonic view of happiness, more likely to favor our endeavor. Freud wrote, in response to his own question about what people demand of life and wish to achieve in it, the reply "The answer to this can hardly be in doubt. They 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 1930, p. 76). Freud's answer equates hedonic pleasure with happiness. According to this view, the more pleasure you have (while avoiding displeasure), the happier you are. Modern psychologists tend to fall in between these poles. Yet relatively few today would deny that hedonic pleasure is at least relevant to a final state of well-being.

We do not pretend to see deeper into the nature of happiness than such major figures of earlier times, but simply point again to the empirical convergence of hedonic and eudaimonic features together in most people who are actually happy. And we note in conclusion, that so far as positive affect contributes to happiness, then at least some progress has been made in understanding the neurobiology of pleasure in ways that might be relevant.

In finishing, we can imagine several possibilities to relate happiness to particular hedonic psychological processes discussed above. 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 and Berridge 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 therapeutic deep brain stimulation 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, all might agree that happiness springs

not from any single component but from the interplay of 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 moving forward through life—but even to achieve high state of well-being.

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