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Making the most of ourselves in the 21st century**

**State-of-Science Review: SR-E2
Neuroscience of Human Reward**

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Summary

Reward is an essential feature of most human behaviour: the need to obtain reward and avoid punishment is crucial to our motivation. This review explores this growth area of research. It begins with laboratory-based experiments to analyse reward-driven behaviour, using techniques such as gambling tasks. It goes on to summarise the impairments to motivation brought about by brain damage or pathology. Current work on functional neuroimaging is described. This technique enables us to identify the brain regions that control reward behaviour such as eating and drinking. These studies also provide insights into the underlying neural activities driving responses to financial reward or loss. There is discussion of social factors such as cooperation and altruism in shaping human reward mechanisms. Finally, we look ahead to future directions for research, including the use of genotyping, psychoactive drugs and the novel field of neuromarketing.

1. Introduction

Reward is crucial to most human behaviour. We do not act aimlessly for no reason; rather, we do things that will achieve outcomes that we need or want, or to avoid outcomes that would be harmful or unpleasant. The vast majority of our behaviour is motivated by either obtaining rewards or avoiding punishments. The study of human motivation has been a huge growth area in the last five to 10 years, in part due to the advent of brain imaging techniques that allow us to access motivational function in a way that was not previously possible. This has allowed us to develop brain-based models of reward processing in humans.

2. Important definitions

The study of motivation is associated with very precise definitions and concepts based on an extensive animal literature. Motivated behaviour is any behaviour or action performed to obtain reward or avoid punishment. A *reward* is defined as anything an animal will work for, while a *punishment* is anything an animal will work to avoid or escape. The process whereby behaviour is modified by rewards and punishments is termed *reinforcement*, and a *reinforcer* is any stimulus that elicits behavioural modification.

A reinforcer can be primary (or unconditioned) or secondary (or conditioned). A primary reinforcer elicits motivated behaviour without any learning, while a secondary reinforcer only elicits the response after learning has occurred. *Conditioning* is the process by which an originally neutral stimulus becomes associated with a primary reinforcer, while *instrumental learning* is a process whereby animals learn to perform an action to obtain a reward or avoid a punishment.

These basic principles govern human reward behaviour; we learn which cues signal positive and negative outcomes and we learn how our actions and behaviours can increase the probability of positive outcomes.

3. Measuring motivation

For various reasons, the huge literature on reinforcement processing in animals has not been paralleled in human studies. However, several approaches have been taken to studying reward using laboratory tasks. Over the last 10 to 15 years, gambling tasks have emerged as the most widely used means of exploring human reward-driven behaviour in the laboratory. For example, the Iowa Gambling Task (IGT) developed by Damasio, Bechara and colleagues (Bechara et al., 1994; 1999) assesses how rewards and punishments influence decision-making. Subjects are shown four decks of cards, two high-risk and two low-risk. The

high-risk decks provide immediate, large rewards but even larger penalties. The low-risk decks offer smaller immediate rewards, but the penalties are also smaller. Over a series of trials, subjects will gain most money by choosing cards from the low-risk rather than the high-risk decks. Normal subjects start the task by sampling from all decks. They then develop a short-lived preference for high-risk decks but start to prefer the safer, low-risk decks when intermittent punishments are experienced.

Another gambling task developed by Rogers and colleagues (Rogers et al., 1999) presents subjects with a number of coloured boxes, some red and some blue. Subjects are asked to predict in which colour box a token will be randomly hidden. They win or lose points depending on whether their guess is correct. An interesting extra feature of this task is that subjects are also asked to bet on how certain they are of the outcome. They can choose how many of their current points total they wish to bet. This task therefore provides several indices of reward-based behaviour. Normal subjects place lower bets when the numbers of red and blue boxes are approximately equal, and higher bets when the boxes are predominantly of one colour.

4. Abnormalities in reward-based behaviour

We know from animal studies that there are distinct circuits within the brain for reward-related behaviour. Critical regions of this circuitry include midbrain, ventral and dorsal striatum, the amygdala and regions of the prefrontal cortex. The function of this circuitry is modulated by the neurotransmitter dopamine.

Before the development of imaging techniques to study normal brain function, the neuroscience of human reward was largely based on observations and performance of patients with circumscribed brain damage, or patients whose core pathology influences relevant brain regions.

4.1. Ventral prefrontal lesions

Patients with damage to ventral and medial parts of the prefrontal cortex show impairments in motivated behaviour. The classic case is that of Phineas Gage (Harlow, 1848) who experienced a severe, penetrating injury to the ventromedial frontal cortex. In spite of relatively preserved cognitive function, Gage's social function was profoundly affected and he was no longer motivated by social norms, suggesting disrupted reward function. Similar deficits have been observed in more recent cases of ventral frontal damage (Damasio, 1994; Stuss et al., 1986).

Patients with ventral frontal damage have been tested empirically using the IGT. Bechara et al. (1994) demonstrated that patients continued to opt for high-risk decks even though they understood and could explain the contingencies. The researchers interpreted this failure to use knowledge of contingencies to guide behaviour as suggesting that patients are insensitive to future consequences of their actions. Gambling impairments in ventral frontal patients have also been reported by Rogers et al. (1999). Subjects were less accurate in their judgements of reward probabilities. They were also more willing to place risky bets on uncertain outcomes.

4.2. Amygdala lesions

In animals, the effects of amygdala lesions have been extensively described, but in humans focal damage to the amygdala is extremely rare. Surgical removal of all or part of the amygdala is sometimes used as a last resort treatment in intractable epilepsy. There is also historical evidence based on the use of amygdectomy to treat extreme aggression (Heimbürger et al., 1966). Studies of these patients provide

some evidence that amygdala lesions have temporary effects on primary motivated behaviours, such as eating and sexual behaviour.

More systematic investigation with laboratory tasks shows that patients with bilateral amygdala lesions are impaired on the IGT (Bechara et al., 1999). Subtle differences in the nature of their performance suggest that these patients are under-responsive to the outcomes of choices (rewards or punishments). Using a different paradigm based on conditioned learning, Johnsruide et al. (2000) showed that patients with amygdala damage do not exhibit normal conditioned learning. This paradigm paired certain visual stimuli with food reward in the context of a complex task. Normal subjects develop a subsequent preference for the rewarded stimuli; patients with amygdala damage do not.

4.3. Parkinson's disease

Fibiger (1984) and Cummings (1993) argue that the high incidence of anhedonia (lack of pleasure) and depression in patients with Parkinson's disease may be due to dysfunction in dopaminergic reward systems. Deficits in a rewarded learning task have been reported in patients with moderate to severe Parkinson's disease (Swainson et al., 2000). Further, Charbonneau et al. (1996) showed that medicated patients are more impaired on an incentive learning task than an associative learning task without incentives. However, patients with Parkinson's disease have not been found to be impaired on the IGT (Stout et al., 2001). Thus, it appears that, in spite of extensive damage to dopamine transmission, the reward-related function of patients with Parkinson's disease is not completely compromised.

4.4. Depression

Aspects of the symptomatology of primary depression can be characterised in terms of reinforcement deficits (Lewinsohn, 1979; Murphy et al., 1998). Lewinsohn (1979) suggests that depressed patients may show either, or both, a reduced capacity to experience reward or a reduction in reward-seeking behaviour. There is some evidence that this group has deficits in gambling-type situations (Pacini et al., 1998; Must, 2007). Elliott et al., (1997) have argued that depressed patients may fail to use normal motivational cues to modify performance on complex tasks.

4.5. Substance abuse

Another class of psychiatric disorder that can be theoretically related to reward processing deficits is substance abuse (Grant et al., 1996; Jentsch and Taylor, 1999). Many abused substances directly or indirectly stimulate dopaminergic reward systems.

Several studies have shown reward-processing abnormalities in heroin addicts (Madden et al., 1997), problem drinkers (Vuchinich and Simpson, 1998), and mixed substance abusers (Petry and Casarella, 1999). This last study also reported a significant interaction with co-morbid gambling behaviour.

Performance on the IGT has been shown to be impaired in people dependent on cocaine (Grant et al., 1997), opiates (Petry et al., 1998), and alcohol (Mazas et al., 2000). Rogers et al. (1999) also reported deficits on their gambling task in chronic amphetamine and opiate abusers.

5. Neuroimaging of reward processing

Functional imaging has provided us with the means to study reward processes without needing objective measures. In a simple experiment, one could image a subject as they experience a reward. We have no measure of what the subject is doing, but the neuroimaging results will tell us whether areas of their brain are significantly more activated when a reward is experienced.

5.1. *Imaging primary rewards: taste and smell*

In much of the animal electrophysiological literature studying reward processing, the rewards used are either appetising food or drink. Imaging people while eating or drinking introduces significant and problematic head movement, so an alternative approach has been to use tastes and smells strongly associated with food.

An fMRI study of taste and smell stimuli (Rolls et al., 1997) demonstrated neuronal responses in the medial orbitofrontal cortex (OFC). In a follow-up study, O'Doherty et al. (2000) scanned subjects before and after a large meal and then presented smells of food that were part of the meal and foods that were not. Their OFC response was significantly reduced for foods that were part of the meal: evidence that the OFC codes motivational as well as sensory properties. Similarly, Small et al. (2001) scanned subjects while eating large amounts of chocolate. As subjects' appetite for chocolate decreased (due to satiation), activation in medial OFC diminished.

5.2. *Imaging studies of financial reward and loss*

In the neuroimaging context, most studies of reward have used money as the reinforcer. Although money has no intrinsic physiological value, it has enormous social value and is thus a strong behavioural motivator to most people. In an early PET study (Thut et al., 1997), subjects performed a simple cognitive task under the two conditions: one where they were simply told 'OK'; and one where they received money for accurate performance. Financial reward was associated with activation in regions of an extended reward system, including midbrain, thalamus, dorsolateral prefrontal cortex (PFC) and OFC.

In a more sophisticated study, Delgado et al. (2000) examined neuronal responses to winning and losing money in a guessing game. When subjects received a reward, increased neuronal responses were seen in the striatum. Similarly, Elliott et al. (2000) reported increased striatal responses to winning money. Imaging has also been used to look at key variables affecting motivation. One obvious variable is reward value, and studies have shown that medial OFC responses are dependent on this (O'Doherty et al., 2001; Elliott et al., 2003).

The most recent imaging studies of financial reward have used increasingly complex mathematical modelling to look at reward prediction (Knutson and Cooper, 2005; O'Doherty et al., 2006). The experience of reward depends critically on the extent to which outcomes match expectations. If a reward is fully expected, the response to it will not be the same as if it were at least partly unexpected. In fact, if you do not receive an expected reward, the experience feels more like a punishment.

Thus, reward responses in the brain are extensively modulated by expectations, and new modelling and analysis techniques in functional imaging are allowing these relationships to be explored.

6. The importance of social factors

The studies discussed above have provided evidence for distributed neural circuitry underpinning human responses to reward. However, these studies have focused on individual subjects in isolation, whereas much of our real-life motivation depends on social factors. Social approval, acceptance and inclusion, for example, are powerful motivators of behaviour. Functional imaging is providing insight into how basic reward systems also mediate more complex aspects of social reinforcement.

One example of this comes from studies of social cooperation. Rilling et al. (2002; 2006) studied people playing an interactive game in which they can choose whether to cooperate with one another or not. Mutual cooperation was associated with enhanced neuronal response in reward areas (medial OFC and striatum), suggesting that social cooperation is intrinsically rewarding.

Another example comes from studies of so-called 'altruistic punishment'. This is the concept that in situations where someone else has broken a social or moral rule, punishment is felt to be appropriate and perhaps even desirable. De Quervain et al. (2004) found that reward regions of the striatum were activated when subjects administered an altruistic punishment, suggesting that punishing transgression is rewarding.

These two examples suggest that understanding human reward mechanisms may have profound implications for understanding complex social behaviours.

7. Conclusions and future directions

The neuroscience of human reward is a rapidly growing area of study.

Traditionally, human reward has been difficult to study in a laboratory setting because paradigms from the animal literature have been difficult to adapt to the human context. However, paradigms based on gambling have allowed neuroscientists to identify possible brain mechanisms of reward processing in patient groups.

Patients with focal damage to ventral prefrontal regions and the amygdala show gambling impairments. An involvement of the dopamine neurotransmitter system is suggested by deficits in patients with Parkinson's disease.

In the psychiatric domain, depression may involve reward-processing disturbances, while substance abuse may fundamentally depend on abnormalities of reward responsivity.

Functional neuroimaging has led to huge advances in our understanding of human reward processing, essentially providing a human analogue of the electrophysiological techniques used in animals. Ventral and medial frontal regions, striatal structures, and the amygdala have been identified as core components of human reward circuitry. Researchers are now exploring how this human reward network responds under different conditions and how the different regions interact in different aspects of reward processing.

Further, the last few years have seen the development of social neuroscience. It is clear that understanding the brain basis of social behaviour is intimately linked with understanding reward mechanisms.

There are various important avenues for future research in this field. The area of social neuroscience is developing rapidly and, over the next few years, it may be possible to gain a fuller understanding of the complex interactions between different factors in human motivation. This has huge implications for psychiatric research, since so many disorders are characterised by disturbances of social function.

Another developing direction is to ascertain the factors determining normal variation in reward function. It seems intuitively plausible that there are individual differences in motivated behaviours; for example, some people are more driven to thrill-seeking, while others are more cautious. Tom et al. (2007) found that individual differences in loss aversion on a gambling task were predicted by neural responses in ventral striatum and prefrontal cortex. Studies like this hint at how fMRI can potentially be used to characterise normal variation in motivational behaviour.

It is possible that genotype plays a role in these differences. Recent research combining genotyping with fMRI suggests that different genotypes may be associated with differential brain response to rewarding information (Chakrabarti et al., 2006).

New developments in imaging techniques have allowed researchers to study the effects of psychoactive drugs on brain responses. Pharmacology-MRI (abbreviated to pMRI or phMRI) has enormous potential in the area of reward processing. The animal literature, as well as addiction research, have provided many clues about the psychopharmacology of reward processing, generating hypotheses that can be tested with pMRI.

In a recent study, Abler et al. (2007) found that the dopaminergic agent olanzapine significantly modulated brain response to a financial incentive paradigm in healthy subjects. Further studies of the modulation of reward responses by dopamine and other neurotransmitters will allow direct exploration of the psychopharmacology of human reward, both in normal subjects and psychiatric patients. The mechanisms of drug addiction are a particular challenge in this field of research.

Finally, the neurobiology of reward has potential commercial applications. Recent studies have recognised the potential of neuroscience methodologies to explore the mechanisms of advertising and marketing. For example, Schaefer and Rotte (2007) have shown that favoured brands elicit neuronal responses in reward circuitry. Such studies have spawned the concept of neuromarketing – the idea that understanding human reward mechanisms will inform the development of better methods to sell products. These concepts also have arguably more important implications for health promotion and other public education campaigns.

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