Waiting for better things

and other animals are driven to act by rewards, be they primary biological rewards like food, shelter and sex, or more complex social or personal goals. When animals act, they are sometimes rewarded (or reinforced) immediately, but often this is not the case. A foraging animal must choose an area in which to search for food, and a predator must choose which prey to stalk; in both cases, the meal may be some time in arriving. Humans regularly make financial and career decisions based on outcomes that are years or even decades away. So to be successful, animals must learn to act on the basis of delayed reinforcement. If the reinforcers are the same, this won’t be an issue: offered the choice between two identical rewards, one available now and one available some time off, animals consistently and sensibly prefer not to hang about. But if the delayed reward is bigger than what’s being put on a plate, it may be wise to wait for the larger bounty.

Playing the waiting game isn’t for everyone: individuals differ in their ability to choose delayed rewards. Self-controlled individuals are strongly influenced by delayed reinforcement, and choose large, delayed rewards in preference to small, immediate rewards. In contrast, those who are relatively insensitive to delayed reinforcement choose impulsively, preferring the immediate, smaller reward in this situation (Ainslie, 1975). Impulsivity has long been recognised as a normal human characteristic, and in some circumstances it may be beneficial – for example, someone with impulsive personality traits may be well placed to take advantage of unexpected opportunities (Evenden, 1999). However, impulsive choice contributes to deleterious states, such as drug addiction (e.g. Bickel et al., 1999), in which addicts may forgo long-term good health for the immediate reward of their drug. Children with ADHD also exhibit impulsive choice (see Sagvolden & Sergeant, 1998). In this article I will discuss briefly the neurobiological systems that play a part in determining the effects of delayed reinforcement, which may therefore contribute to pathological impulsivity.

Neurochemistry of delayed reinforcement

One major avenue of research into impulsivity has concerned the brain’s neuromodulator systems. These systems do not convey vast amounts of highly specific information (in the way that, say, the optic nerve conveys visual information); instead, they comprise small groups of neurons that project to wide areas of the brain, releasing chemicals that influence the behaviour of these other brain regions. Two such systems are the serotonin (5HT) and dopamine neurotransmitter systems; both of these have been implicated in the ability to choose delayed rewards.

The suggestion that 5HT is involved in impulse control stemmed from the observations that drugs that suppress 5HT function appeared to reduce animals’ ability to inhibit inappropriate behaviour (motor acts). Animals with suppressed 5HT function continued to respond even if their responding was punished, or it they had to

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animals to make impulsive choices in a variety of tasks (e.g. Wogar et al., 1993). It is normal that delayed rewards are valued somewhat less than immediate rewards, all other things being equal (known as "temporal discounting" of future rewards), but 5HT depletion has been suggested to steepen the temporal discounting function—which means that delayed rewards lose their capacity to motivate or reinforce behaviour. The 5HT-depleted animal becomes hypersensitive to delays, or hyposensitive to delayed reward. As delayed rewards have unusually low value, the animal consistently chooses small immediate rewards over large delayed rewards, like an impulsive person. Conversely, increasing 5HT function with the 5HT indirect agonist fenfluramine has the opposite effect, decreasing impulsive choice (Poulos et al., 1996).

However, it should be noted that the effects of 5HT manipulations have not always followed this general pattern (see Cardinal et al., 2004). For example, feeding humans a diet low in the amino acid tryptophan, which reduces central 5HT levels, may increase ‘motor’ impulsivity (Walderhaug et al., 2002), but it has not been shown to increase impulsive choice in humans (Crean et al., 2002). There is good evidence that not all types of impulsivity are promoted by low 5HT function in a simple way (Dalley et al., 2002; Evenden, 1999).

Of course, altered 5HT function has also been strongly implicated in depression (e.g. Caspi et al., 2003; Feldman et al., 1997), but the relationship between depression, impulsivity, and 5HT is complex. The precise neurochemical abnormality or set of abnormalities in depression is far from clear: there is no clear-cut relationship between depression itself and levels of 5HIAA in the CSF (Feldman et al., 1997), although antidepressant drugs themselves tend to lower CSF 5HIAA (Böckman et al., 2000). However, there is a consistent association between low CSF 5HIAA and suicidal behaviour—not only in depression, but also in schizophrenia and other disorders (e.g. Cooper et al., 1992; Cremmier et al., 1999). Patients who are prone to suicide (many of whom are depressed) show high impulsivity (e.g. Apter et al., 1993; Corruble et al., 2003). Thus, low 5HT function has been linked with impulsive behaviour, which is a risk factor for suicide, and abnormalities of the 5HT system are also associated with depression, also a strong risk factor for suicide.

The dopamine neuromodulator system also plays a role in animals’ ability to choose delayed rewards; specifically, dopamine appears to promote the choice of delayed reinforcement via D2-type dopamine receptors (Wade et al., 2000). This is in keeping with the observation that psychostimulant drugs such as amphetamine and methylphenidate (Ritalin) can be an effective therapy for ADHD (Bradley, 1937). These drugs release monoamine neurotransmitters such as dopamine from neurons, and prevent their subsequent reuptake from the synapse back into the neuron. However, these are complex drugs and their mechanism of action is not wholly clear. For example, it is thought that psychostimulants promote or reduce impulsive choice depends on the environmental conditions, such as whether the animal is given an explicit signal indicating that the delayed reward is on the way or must simply wait for the delayed reward with no overt environmental cue (Cardinal et al., 2000). Furthermore, some of the actions of psychostimulants in this regard may be through their effects on 5HT as well as dopamine neurotransmission (Winstanley et al., 2003).

**Neuroanatomy of delayed reinforcement**

Little is known anatomically about how the brain learns from or chooses delayed reinforcement. We studied three brain regions previously implicated in other kinds of reinforcement learning: the nucleus accumbens core (AcbC), anterior cingulate cortex (ACC), and medial prefrontal cortex (mPFC) (Cardinal et al., 2001). The AcbC is a critical site where signals that predict reward have their motivational impact (see Cardinal et al., 2002). All three structures are abnormal in humans with ADHD or in animal models of ADHD (see Cardinal et al., 2003), and all three receive projections from both the dopamine and serotonin neuromodulator systems (Tallon & Loughlin, 1993, Halliday et al., 1995; Pickel & Chan, 1999). All, therefore, were candidate structures that might mediate choice involving delayed rewards.

To establish whether abnormalities in these regions might cause impulsive choice, we used a task in which hungry rats regularly had to choose between two levers. Responding on one lever led to the immediate delivery of a small food reward; responding on the other led to a much larger food reward, but this reward was delayed by anything up to 60 seconds (Evenden & Ryan, 1996). Once they had been trained on this task, we selectively destroyed neurons of the AcbC, ACC or mPFC, and retested the rats.

Rats with AcbC lesions became and remained impulsive; they began to choose the immediate small reward much more often than sham-operated controls. They persisted in choosing impulsively, even though they were made to experience the larger, delayed alternative at regular intervals.

Why did they do this? In theory, impulsive choice might arise for a variety of reasons. Take abstaining smokers who were offered a cigarette. The choice is between a small immediate reward (a cigarette) and a large delayed reward (better health in the future). If some act impulsively where others do not, it could be because they do not perceive the larger reward to be as worthwhile as their self-controlled counterparts do, or because they are simply less influenced by outcomes that are delayed considerably. Which is true of rats whose AcbC has been destroyed? Our findings (Cardinal et al., 2001, 2003) and others’ (e.g. Brown & Bowman, 1995) suggest the latter. Even those AcbC-lesioned rats who showed an extreme preference for the larger reward when it was not delayed were incapable of choosing it as often as normal rats when...
it was delayed. In fact, accumbens-lesioned rats appear just as sensitive to the magnitude of reward as normal rats (Brown & Bowman, 1995), suggesting that their impulsive choice arises not because the large reward is subjectively too small to compensate for the normal effects of the delay, but because they would have to wait too long for it.

In contrast, we found that damage to the ACC or mPFC did not produce impulsive choice. So although the ACC and mPFC have been shown to be abnormal in disorders of impulsivity, our findings suggest that dysfunction of these regions is not an important contributor to impulsive choice. The abnormalities observed in these regions in the brains of people with ADHD may therefore be responsible for other features of the disorder (such as inattention or an inability to suppress motor acts), or these regions may have altered as a consequence of a disease process beginning elsewhere.

However, there are other regions of cortex whose dysfunction might cause impulsive choice. Recent evidence has indicated that rats' propensity to choose delayed rewards is altered by damage to the basolateral amygdala or orbitofrontal cortex (Mobini et al., 2002; Winstanley et al., 2004), two other structures that send information to the AChC. A clearer understanding of the neurochemical and neuroanatomical abnormalities that underlie the symptoms and signs of clinical disorders of impulsivity may lead to more effective therapy.

**Dripping the delay**

While our study (Cardinal et al., 2001) examined the role of the AChC in choosing delayed rewards, it did not address whether the AChC is also a critical structure for learning from delayed reinforcement. It is one thing to choose between rewards that differ in the amount of time you must wait to obtain them – like a hungry connoisseur who is also an expert chef choosing either to cook boeuf bourguignon and eat in two hours (a delayed but considerable reward), or to cook a frozen pizza and eat in 15 minutes (an early but lesser reward). Though the rewards differ in their delay, the cook is certain to achieve either goal and is in no doubt as to the relationship between the actions and the final result; there is merely a choice of preferences. It is another thing to work out which of your actions are the ones actually leading to particular outcomes. Consider
someone manoeuvring a Venetian gondola for the first time—a long, heavy vessel, powered and steered by a single small oar, pressed against (but not attached to) a complicated rowlock at one side of the boat. The novice gondolier must determine which oar handle to use in a given situation, yet the gondola’s inertia means that it takes several seconds for each action to have a perceptible effect. The hapless novice must wait to see if his action was the correct one and, of course, must learn the task while being free to change tactics at any time.

How do animals accomplish this difficult task of learning to act with delayed outcomes, and does the AcbC contribute to this process too? In order to learn which actions are the correct ones that eventually lead to reward, and which are not, some mechanism must ‘bridge’ the delay between action and outcome. We recently took two groups of hungry rats, one with Acb lesions and one without, and presented them with two levers; one did nothing, while every press on the other lever delivered a single food pellet. For some rats, this pellet was delivered immediately; for others, it was delayed. Normal rats took longer to learn to press the lever when the reinforcement was delayed, which is not surprising (Dickinson et al., 1992), but they learned successfully with delayed reinforcement. Rats with Acb lesions were perfectly able to learn this task when there was no delay, but were profoundly impaired when there was a delay between action and outcome (Cardinal & Cheung, 2004).

Taken together, these results suggest that the AcbC is a reinforcement learning structure specialised for the difficult task of learning with, and choosing, delayed reinforcement. Further understanding of the mechanism by which it does so, or fails to do so, might provide insight into the pathology of a number of neuropsychiatric disorders. Behavioural neuroscience techniques may make it possible to distinguish the brain regions that underlie different types of impulsivity (Evenden, 1990) and to segregate the neural abnormalities that contribute to complex disorders such as ADHD and drug addiction.

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**References**


