Conditioning models of addiction: Part 3

In this Background Briefing, Professor David Clark describes how stimuli associated with the pleasurable effects of drugs can strongly influence behaviour.

In my last two Briefings, I looked at two ways that classical conditioning may be involved in problematic substance use or addiction. I described the conditioned withdrawal model, as well the concepts of conditioned drug-opposite responses and conditioned tolerance.

In the conditioned incentive model of addiction, proposed by Jane Stewart and colleagues in the mid-1980s, environmental stimuli previously associated with the pleasurable effects of drugs become conditioned stimuli (CS) via classical conditioning processes.

These CS are considered to activate the same neuronal pathways in the brain that mediate the direct pleasurable effects of drugs, albeit weakly, and they thereby elicit a motivational state that directly primes drug-taking behaviour. The CS are positive incentives that drive drug use.

Thus, when a heroin user sees the paraphernalia that they usually use for administering the drug, the paraphernalia act as a CS that elicits feelings somewhat similar to that triggered by the drug itself, which result in the person wanting to use the drug.

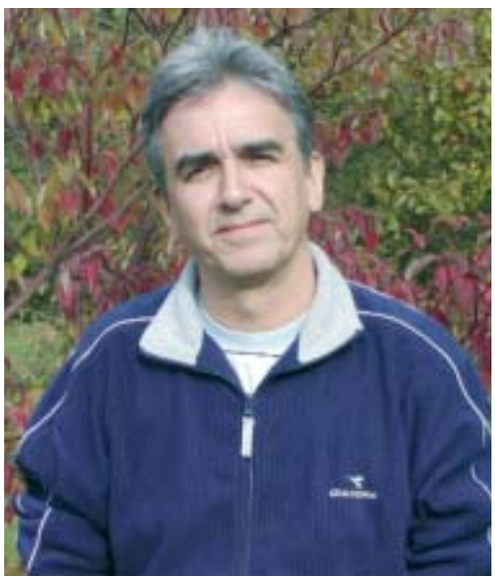
The present model is derived primarily from incentive motivation theory, which was developed on the basis of work with laboratory rats using natural reinforcers such as food. This theory asserts that organisms are motivated by incentives, stimuli that predict a primary reward. The motivation is the expectancy of the primary reward, be it food or drug.

Thus, one person may want to eat a doughnut when they see the bakery assistant who regularly sells them their favourite vice, while another person may want to inject heroin when they see their regular dealer.

There is considerable evidence from animal research that positive incentive effects of drugs motivate drug-seeking behaviour.

In the place conditioning paradigm, rats are introduced to a three-compartment box, containing two end compartments with distinctly different environments (light walls, grid floor *vs* dark walls, smooth floor), and a smaller 'neutral' central area. The time spent in each of the end compartments is measured over a 15-minute period, and one side is assigned as the original least-preferred side.

In subsequent sessions (days one, three and five), animals are administered a drug of abuse such as amphetamine and restricted to their original least-preferred side for 30 minutes. On days two, four and six, they are administered an inert substance (saline) and restricted to the original preferred side.



'Wanting' is not "liking" – a person may strongly want a drug without actually liking the experiences that it produces.'

On the following day, the rats are given free access to all parts of the box, with the time spent in each end compartment measured. When given this free choice, rats show a shift in preference towards the side in which they had received the drug – even though no drug was administered in this test session.

These studies demonstrate that a wide variety of drugs of abuse (*eg* amphetamine, cocaine, heroin), as well as natural reinforcers such as food, can induce place conditioning. Thus, environments associated with the pleasurable effects of drugs, or natural reinforcers, become positive incentives that motivate approach behaviours.

We can safely assume that animals find the effects of drugs of abuse to be pleasurable in that they will learn to perform specific tasks (*eg* pressing a lever in a Skinner box) to obtain intravenous injections of drugs of abuse such as amphetamine, cocaine and heroin. They also learn to respond to a stimulus (*eg* a light) that was previously associated with their lever presses for drug.

Brain dopamine neurons, in particular those projecting from a midbrain region known as the ventral tegmental area to forebrain regions such as the nucleus accumbens (mesolimbic dopamine neurons), are thought to play a major role in mediating drug self-administration.

Terry Robinson and Kent Berridge, two leading researchers from the States, propose that the primary role of mesolimbic dopamine neurons is to mediate what is called incentive salience.

Incentive salience is a characteristic of the mental representation of a stimulus that allows it to become attractive and wanted, thereby eliciting approach behaviours towards a specific goal. (A juicy piece of apple pie possesses a high degree of incentive salience – at least to me!)

In their incentive sensitisation model, Robinson and Berridge propose that drugs of abuse produce a long-lasting sensitisation of the neural system mediating incentive salience (mesolimbic dopamine system), so that the incentive salience attributed to drug-taking and to drug-associated stimuli become pathologically amplified, leading to compulsive drug-seeking and drug-taking.

The sensitisation of incentive salience can occur at the same time that the pleasurable effects of the drugs are diminished, due to the repeated drug administration producing tolerance to this effect.

In fact, these researchers emphasise that the neuronal systems responsible for excessive incentive salience are dissociable from the systems mediating the pleasurable effects of drugs. 'Wanting' is not 'liking' – a person may strongly want a drug without actually liking the experiences that it produces.

Moreover, it is also proposed that the wanting system can be activated and influence behaviour without a person having conscious awareness of ongoing processes.

A considerable degree of animal research has been focused on drug-induced sensitisation, and the incentive salience model is very popular among neuroscientists. While it has been argued that there is little evidence in humans supporting the model, this is in part due to a difficulty in testing the ideas.

Recommended reading:
Robert West (2006) Theory of Addiction. Blackwell Publishing. (Available at discounted rate from the DDN bookshop at www.drinkanddrugs.net.)