

Conditioning models of addiction: Part 2

In this Background Briefing, Professor David Clark describes two of the three models pertaining to the involvement of classical conditioning in problematic substance use and addiction.

In my last Briefing, I described classical conditioning as a process that involves a neutral unconditioned stimulus, such as a coloured light, becoming rewarding and influencing behaviour because it has reliably preceded a reward such as food.

During a history of drug use, certain stimuli, such as environmental contexts or drug paraphernalia, reliably accompany drug administration. These stimuli, by virtue of their pairing with the drug effects, become conditioned stimuli capable of eliciting conditioned responses, *eg* drug-seeking behaviour.

There are three ways that classical conditioning may be involved in problematic substance use or addiction.

In the conditioned withdrawal model, proposed by Abraham Wikler in the late 1940s, environmental stimuli paired with drug withdrawal become conditioned stimuli capable of eliciting conditioned withdrawal reactions.

For example, in people dependent on heroin, withdrawal symptoms can occur and be paired repeatedly with environmental stimuli. At a later time, when the individual is no longer dependent, the environmental cues alone can be enough to elicit the symptoms of withdrawal.

The cues that trigger conditioned withdrawal can be both external (places or situations) or internal (moods). Conditioned withdrawal can play a role in relapse. In fact, the conditioned withdrawal model of addiction involves both classical and operant (or instrumental) conditioning. Repeated pairing of environmental stimuli with withdrawal results in these stimuli are capable of inducing conditioned withdrawal (classical conditioning).

The operant conditioning component involves the person taking the drug to alleviate an aversive state, the withdrawal symptoms, which can be regarded as a negative reinforcer. The second classical conditioning model involves the concepts of conditioned drug-opposite responses and conditioned tolerance.

Whenever a disturbance occurs in the body, such as produced by a drug, a physiological process known as homeostasis, in which the body tries to counteract the disturbance, comes into play.

For example, amphetamine enhances release of the neurotransmitter dopamine in the brain, but at the same time regulatory mechanisms reduce dopaminergic function in order to try and maintain the status quo – although the amphetamine still increases dopamine function overall.

Researchers believe that these compensatory



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mechanisms can eventually be triggered by stimuli and cues previously associated with drug administration, and this can happen even before the drug is taken.

In situations where the predictive stimuli appear but no drug is taken, the body's compensatory mechanisms come into play and go unopposed because there is no drug effect. This can be expressed as overt physiological reactions and/or form the basis for the subjective experience of withdrawal sickness and craving.

Take for example a person who is drinking alcohol every evening to reduce the anxiety they have experienced from working in a stressful job. The clock at work approaching 17.00 acts as a conditioned stimulus to the anxiety-alleviating effects of alcohol.

If the person were to attend a school play one evening, without going to the pub, their body's compensatory mechanisms would come into play but not be diminished by the physiological effects of consumed alcohol. The person would experience the opposite subjective effects to those produced by alcohol, *ie* anxiety. According to this model, tolerance

and withdrawal symptoms are intimately linked.

Tolerance – the gradual diminution of effect following repeated administration of the same dose of drug – is thought to occur because of the homeostatic processes that occur in the body to counteract the action of a drug. The homeostatic (or opponent) responses are thought to be strengthened by repeated drug administration, and the net effect of the drug is therefore reduced.

These processes are explained in more detail by the Opponent Process Theory of Solomon and Corbit (1973), summarised in Robert West's book *Theory of Addiction*. Shepard Siegel (1975) first proposed that a complete account of tolerance requires an appreciation of the role of environmental influences or cues. There is now abundant evidence showing that animals that are pre-administered a drug repeatedly in one environment and tested behaviourally in another environment, will not show as much tolerance as those animals given chronic drug and behavioural testing in the same environment.

An important consequence of this idea in relation to heroin overdose was illustrated by Shepard Siegel in the early 1980s. Tolerance develops to the effects of heroin, so that users face the possibility of overdose (and death) if they take much larger amounts of drug than normal.

Siegel reasoned that if tolerance to heroin was partially conditioned to the environment where the drug was usually administered, if the drug was administered in a new setting, much of the conditioned tolerance would disappear, and the person would be more likely to overdose.

In his study, many heroin users admitted to hospital suffering from a heroin overdose reported that they had taken this near-fatal overdose in an unusual environment, or that their normal pattern of use was different on that day.

The next Background Briefing will consider the third model involving classical conditioning, involving conditioned drug-like responses.



Recommended reading:

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

Nick Heather and Ian Robertson (2001)

Problem Drinking. Oxford Medical Publications.