

Theories of craving and urges

Professor David Clark describes craving and urges for drugs and alcohol, and briefly outlines some of the underlying theories.

The terms **craving and urges** have enjoyed wide popularity in both subjective reports of people misusing substances and in the clinical literature of addiction.

Craving occurs for substances that cause problematic behaviour and addiction, including opiates, stimulants, alcohol and nicotine. Moreover, craving has been suggested as a prominent feature maintaining drug and alcohol use and precipitating relapse after a period of abstinence.

Craving is regarded as a subjective motivational state in which an individual experiences an intense or overpowering desire to engage in drug-taking or alcohol consumption. It can vary in intensity, sometimes reaching a level that can overwhelm the individual, dominating their thoughts, feelings and actions to the exclusion of all else.

There has been some confusion in the field related to the terms craving and urges. While some people use the terms interchangeably, others consider a distinction between the two phenomena.

For example, Tom Horvarth describes craving as a desire to achieve the psychological state induced by the substance that one has given up – the ‘I want it badly’ feeling. Cravings serve as a cue for urges.

An urge is considered to be the impulse or intention to get the substance and use it – it is the ‘I have to do it now’ feeling. Some therapeutic strategies focus on managing the response to urges because they cue the addictive behaviour.

Cravings and urges have been intimately linked to classical conditioning processes. Over a long history of drinking or drug-taking, stimuli that have been repeatedly associated with consumption of alcohol or drugs (eg sight of the pub, or the syringe) become conditioned stimuli.

These conditioned stimuli become capable of eliciting the same responses that are produced by alcohol or drugs themselves. They activate conditioned motivational states that produce craving and urges, physiological reactions, and drug or alcohol seeking behaviour.

Craving and urges can be linked to both positive and negative reinforcement systems. One model proposes that these states arise from the anticipation of the positive reinforcing or pleasurable effects of drugs or alcohol. Watching someone smoke a cigarette and the smell of the tobacco can remind an ex-smoker of the relaxing effect of smoking and trigger an intense desire to experience this again.

Another model proposes that craving and urges arise from the need to relieve withdrawal or



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conditioned withdrawal symptoms. Thus, a person returning to an area where they have experienced withdrawal on many occasions in the past may experience conditioned withdrawal symptoms, which in turn can generate craving.

In contrast, Terry Robinson and Kent Berridge argue that drug or alcohol craving is a psychological process that is distinct from conditioned withdrawal and the anticipation of pleasurable drug or alcohol effects.

They propose that repeated use of these substances can lead to neuroadaptations (increased sensitivity) in brain dopamine systems that are involved in attributing incentive salience to stimuli. Incentive salience is a psychological process that ‘transforms the perception of stimuli, imbuing them

with salience, making them attractive, “wanted”, incentive stimuli’.

The sensitisation of these brain dopamine systems causes excessive incentive salience to be attributed to the act of drug-taking and to stimuli associated with drug-taking, transforming ordinary wanting into excessive drug craving.

Importantly, these researchers make a distinction between ‘wanting’ and ‘liking’; although a person may want a drug, they may not necessarily like it.

While some models assume a tight link between drug or alcohol misuse and craving, the cognitive model of Steve Tiffany proposes that drug and alcohol use in addicts can function independently of the processes that control craving.

Tiffany argues that over a long history of drinking (or drug use), many of the actions involved in acquiring and consuming alcohol become automatic for people with an alcohol problem. Stimulus triggers (eg clock reaching 17.00) activate automatic cognitive processes that result in automatic drinking, with craving playing no controlling role.

However, when the automated alcohol use sequences in a drinker are blocked by an environmental obstacle, eg favourite pub is closed for renovation, the person must activate non-automatic processes to cope with the problem. These non-automated processes, when activated simultaneously with automated alcohol use sequences, generate craving.

People with a drink problem who are abstaining from alcohol face a continuous barrage of cues and situations that trigger their automated alcohol use sequences. By trying to abstain they are using non-automated cognitive processes, which in turn generate craving.

This model can account for the fact that addicts often do not identify craving as a major, immediate cause of their relapse.

Tiffany points out that relapses that occur where craving is not identified as a major cause might be the result of automatic alcohol or drug use sequences being activated without concurrent mobilisation of non-automatic processes directed towards impeding these automated sequences.

Craving and urges for drugs and alcohol cause a repeated, short-term discomfort to a person, which they need to learn to deal with if they are to overcome their substance use problem. While they are a natural part of addiction, they do disappear over time. Strategies have been developed that help people deal with craving and urges; use of these can facilitate behavioural change and the path to recovery.