How a Habit Becomes an Addiction

January 14, 2015

Research suggests that only 20–30% of drug users actually descend into addiction — defined as the persistent seeking and taking of drugs even in the face of dire personal consequences. Why are some people who use drugs able to do so without turning into addicts, while others continue to abuse, even when the repercussions range from jail time to serious health problems?

In a comprehensive review in the *European Journal of Neuroscience*, Barry Everitt outlines the neural correlates and learning-based processes associated with the transition from drug use, to abuse, to addiction.

Drug seeking begins as a goal-directed behavior, with an action (finding and taking drugs) leading to a particular outcome (the drug high). This type of associative learning is mediated by the dorsomedial region of the striatum, the area of the brain that is associated with reward processing, which functions primarily through the neurotransmitter dopamine.

In this kind of learning, devaluing the outcome (by decreasing the potency of the drug, for example) tends to decrease the pursuit of the action. When the high is not what it used to be, the motivation to continue seeking it out decreases.

However, in long-term abusers, this devalued outcome does *not* reduce the action — indeed, researchers have found that in cases of chronic drug use, a parallel associative learning process eventually comes to the fore. This process is one of stimulus–response; the conditioned stimuli in this case are the various environmental cues — the sight of the powdery white stuff, the smell of burning aluminum foil — that users associate with getting high and that compel them to seek out drugs.

As Everitt puts it, the “must have” of the goal-directed behavior eventually develops into a habitual “must do” response. This second kind of learning is mediated in the brain by a separate section of the striatum — the dorsolateral region, which is connected to areas of the cortex that control sensorimotor function. Everitt outlines the neural mechanisms underlying this shift in learning behaviors, which seems to occur due to changes in two different dopamine signaling pathways that involve the striatum.

Although impulsivity is often seen as an effect of stimulant drug use, it may also be a causal factor in the loss of control that occurs when a drug abuser descends into addiction. The author presents evidence suggesting there is an intrinsic element that can make certain people more vulnerable to impulsivity and, consequently, drug addiction — potentially explaining why not all habitual users go on to become addicted.

With these discoveries about the neural and psychological activity of addiction in mind, Everitt also reviews some potential treatment options. These include drugs that block specific dopamine receptors to disrupt the reward-processing circuits in the brain and those that induce plasticity in the brain regions associated with habitual drug-seeking behavior.

Everitt also discusses existing drugs that could be repurposed to treat drug addiction — such as SSRIs, common antidepressant medications that raise serotonin levels in the brain, or atomoxetine, a pharmaceutical treatment for ADHD that tends to reduce impulsivity.

One of the more intriguing prospects is a class of drugs that targets memory reconsolidation, designed specifically for preventing relapse. If memories of previous drug experiences can be disconnected from the environmental cues that normally induce craving, the theory goes, those stimuli will cease to bring about drug-seeking behavior.

As this review shows, a more complete understanding of the neural and learning processes associated with the transition from drug use to addiction can help researchers better identify and treat those most at risk for descending into compulsive drug abuse.

Reference

Everitt, B. J. (2014). Neural and psychological mechanisms underlying compulsive drug seeking habit and drub memories: Indications for novel treatments of addiction. *European Journal of Neuroscience, 40*, 2163–2168.