

Causes of addiction and how to counteract it



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What causes addiction, and what is the best approach to counteract it?

Due to its ambiguity, addiction is intentionally omitted from the official Diagnostic and Statistical Manual of Mental Disorders (DSM-V) (American Psychiatric, American Psychiatric, & Force, 2013). Similarly, with the exception of gambling disorders, all other groups of repetitive behaviours (e.g. sex addiction) are omitted from the DSM-V due to insufficient peer-reviewed evidence needed to categorize these responses as mental disorders (American Psychiatric et al., 2013). Thus, the following argument will take addiction defined as compulsive substance seeking and use (Erickson & Wilcox, 2001). A prevailing hypothesis is that addiction is a brain disease (Leshner, 1997), leading to the proposition that the transition from substance use to addiction is motivated by changes at the neural level (Everitt & Robbins, 2005; Goodman, 2008; Hyman, Malenka, & Nestler, 2006; Leshner, 1997; Nestler, 2005).

Transition from substance use to addiction has traditionally thought to only be driven by the higher level theories of positive (substance induce euphoria) and negative reinforcement (withdrawal avoidance) cycles (Wise & Koob, 2014). However, an emerging premise that substance addiction is driven by multiple neurobiological processes and factors compliments and underlies this reinforcement theory (Goodman, 2008; Hyman et al., 2006; Leshner, 1997; Nestler, 2005; Terry E. Robinson & Berridge, 2000). From this, it can be posited that neurobiological predisposition indicators (i. e. genes) and neural adaptations are the major precursors or causes of the transition from substance use to substance addiction (Erickson & Wilcox, 2001; Terry E. Robinson & Berridge, 2000). Causes including genetics

(Cloninger, 1999), alterations in brain chemistry (Hyman & Malenka, 2001), and incentive-sensitisation (Terry E. Robinson & Berridge, 2000; Terry E Robinson & Berridge, 2008), among others, are often considered key. Genetic factors contribution to an individual's risk for the development of substance addictions has been reported to be as high as 30-60% (Kreek, Nielsen, Butelman, & LaForge, 2005). Studies spanning two decades suggest strongly that some humans suffer a genetic predisposition to alcohol addiction, with animal, twin, and adoption studies confirming this (Crabbe, McSwigan, & Belknap, 1985; Uhl, 1999). Specific vulnerability genes have been suggested (Crabbe et al., 1985; Uhl, 1999). Alterations in brain chemistry are forced by a substance's effect on several neurotransmitter systems (e. g. Cocaine matching to dopamine) (Erickson & Wilcox, 2001). Synapses and circuits within the mesolimbic dopamine pathway (Medial forebrain bundle) are thought to be moulded by molecular processes due to substance use (Hyman & Malenka, 2001). The Medial forebrain bundle effects, most importantly, pleasure and decision-making ability, leading to permanent dopamine activity dysregulation within a substance user. This dysregulation of the reward circuitry, combined with an inability of the brain to distinguish between natural (e. g. food) and unnatural stimuli (e. g. addictive substances), gives addictive substance immense control over compulsive substance use behaviour (Hyman & Malenka, 2001). Repeated substance exposure is thought to cause Incentive-sensitisation, the sensitisation of the mesocorticolimbic system by imposing incentive salience on stimuli linked with reward circuitry firing (Terry E Robinson & Berridge, 2008). A study of 202 subjects examined between the ages of 16-40 has been shown to confirm incentive-sensitisation theory by showing those

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exposed to cocaine reported more 'wanting' for the substance (Lambert, McLeod, & Schenk, 2006). Thus, there is clearly overwhelming evidence for the given hypothesis of the cause of substance addiction being interconnected with genetics and neural adaptations.

Having linked the cause of substance addiction to neural adaptations, it is logical to centre the methods of counteraction on neural circuitry changes. Pharmacotherapies centre the treatment of substance addiction with the aid of pharmaceutical drugs eliciting a neuromodulatory effect (i. e restoration of normal neurobiological functions due to changes from substance use). Two approaches have shown aptitude to target and counteract the neurobiological adaptations due to substance use; the agonist approach (e. g. gamma-aminobutyric acid (GABA) agonists), and the antagonist approach (e. g. glutamate transmission inhibitors) (Kalivas, 2007; Potenza, Sofuoglu, Carroll, & Rounsaville, 2011). The use of baclofen, a GABA B-type agonist, has been shown to reduce the fortifying effects of cocaine addiction in rats (Roberts, 2005). Preclinical human trials with patients given baclofen displayed reduced limbic activation just 7-10 days after administration, indicating encouraging results for GABA agonists for the treatment of cocaine addiction (Kalivas, 2007; Roberts, 2005). Agonist approaches to therapy for other substance addictions have shown strong promise (Potenza et al., 2011). The administration a glutamate antagonist (N-methyl-D-aspartate, NMDA) has shown an ability to reduce tolerance to opiates, while also reducing sensitisation to other addictive substances. However, the complex reaction between these agonists and substances of abuse are difficult to characterise and could have powerful reinforcing effects of their

own (Nestler & Aghajanian, 1997). A combination of both an agonist and antagonist approach using the drug Acamprosate has demonstrated its effectiveness in curbing alcohol addiction (Mason & Heyser, 2010), indicating a mixture of approaches is ideal for effective treatment. While varying specific genes is not currently feasible, there have been strong indications that the inclusion of genetics in therapy development will aid in the understanding of natural variations in therapy efficacy (Edenberg & Kranzler, 2005). Consequently, there is strong evidence for a mixture of agonist and antagonist approaches coupled with targeted genetic therapies for effective and counteraction of addiction.

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