Drug Withdrawal Effects on Cognition

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Introduction

Considerable neurological evidence indicates that the prefrontal cortex mediates complex decision-making functions, including behavioral autonomy and self-control (Lyvers, 2000). When drugs such as oxycodone are used as pain relievers, they may cause mild cognitive deficits in attention and complex working memory, and episodic memory (Rapeli, Kivisaari, Autti, Kahkonen, Puuskari, Jokela and Kalska, 2006). There is repeated evidence to support that chronic drug abuse will affect intellect and attention (Rapeli, et al., 2006). Patients who have abused different drugs in the past, have reported to have fatigue and poor concentration (Rapeli, 2006). For patients to exhibit withdrawal signs, tolerance or in some cases, addiction must be in play. The urge and craving that patients go through, while having withdrawals, is a key concept in cognitive functioning (Tiffany, 1990).

Over the past 10 years, there has been a resurgence of interest in the role of urges and cravings in addictive behavior (Tiffany, 1990). Several factors have promoted this revival. One has been the decline in the influence of radical behavioral theories in the study of addictive behavior with a corresponding ascendancy of social cognitive models (Tiffany, 1990). Drug urges are assumed to be subjective, emotional-motivational states (Tiffany, 1990). They are viewed as subjective in the sense that they refer to the philosophical system experience of the individual (Tiffany, 1990). The experience of urge has some technical quality, and motivational in the sense that the subjective urge state presumably activates drug-seeking behavior (Tiffany, 1990).
Most drugs that support compulsive self-administration will produce a physiological dependence syndrome when the drug is withdrawn (Tiffany, 1990). By the early 1990s, converging evidence suggested that many drugs of abuse act through mechanisms involving the brain neurotransmitter dopamine and different neural systems (Robbins, Everitt, 1999). Although drugs, including stimulants such as amphetamine and cocaine, opiates such as heroin, and even legal drugs such as alcohol and nicotine, can influence several different chemical neurotransmitter systems in the brain, many of these primary responses lead to secondary effects involving dopamine (Robbins, Everitt, 1999). The evidence for this information came from an experiment done on rats. The rats will self-administer injections of a drug to the nucleus accumbens by pressing a lever to activate a micro syringe connected to a stainless-steel tube, or cannula (Robbins, Everitt, 1999). The rats give themselves more of the amphetamine when their dopamine receptors are partly blocked pharmacologically, suggesting a drive to self-regulate the level of dopamine activity (Robbins, Everitt, 1999).

The rewarding effects of drugs other than stimulants may also depend on the mesolimbic dopamine system (Robbins, Everitt, 1999). For example, rats will also self-administer morphine to the area of the midbrain from which the dopamine neurons project (Robbins, Everitt, 1999). Using a micro dialysis technique, which allows extracellular dopamine concentrations to be sampled directly from living brain tissue, withdrawal from several drugs, including alcohol and nicotine, as well as opiates and stimulants, has been shown to be associated with reduced levels of dopamine in the nucleus accumbens (Robbins, Everitt, 1999). From these observations comes the modal hypothesis’, which states that the reinforcing effects
of all drugs of abuse partly depend on the mesolimbic dopamine system (Robbins, Everitt, 1999).

Another big focus of interest has been the dopamine transporter (Robbins, Everitt, 1999). During transmission of a nerve impulse, dopamine neurons release this neurotransmitter into the synapse, which is a tiny gap between two neurons (Robbins, Everitt, 1999). Dopamine diffuses across the synapse and binds to receptors on the other side. Having done its job, dopamine is then recycled by the transporter, which facilitates re-uptake by the presynaptic neuron (Robbins, Everitt, 1999). All these discoveries support the consensus that drug dependence and addiction can be partly understood as gradual adaptations of the brain to chronic drug exposure (Robbins, Everitt, 1999). These adaptations may be triggered by a drive to regulate activity of the various brain systems within certain defined activity limits and they are the underlying processes for both the decreasing and increasing effects of repeatedly administered drugs (Robbins, Everitt, 1999).

The drugs are consistent with the rebound consequences, following drug withdrawal, of chronic drug administration, which may further modulate the addiction process (Robbins, Everitt, 1999). It is important to understand how addiction works at the behavioral, cognitive and neuropsychological levels (Robbins, Everitt, 1999). Drug-seeking behavior can become powerfully associated with environmental cues, which, as conditioned stimuli, predict not only the availability of drugs and their associated hedonic effects, but also aversive withdrawal states (Robbins, Everitt, 1999).
Looking back specifically to opioids, opiates also have a specific role in cognitive, behavioral and physiological processing. At the receptor site, opioids have endogenous and exogenous inhibitory post synaptic effects (Lyvers, 2000). It has also been proposed that endogenous opioid mechanisms are involved in stimulus processing at early stages of learning in conditioning tasks (Hebb, Poulin, Roach, Zacharko, Drolet, 2005). One study looked at exploring cognitive function of individuals with opioid dependence during early abstinence in comparison to normal controls (Rapeli, et al., 2006). The results of this study supported the main hypothesis of reduced cognitive performance in specific areas during early opioid abstinence (Rapeli, et al., 2006).

Repeated practice of a cognitive or motor task under fixed stimulus conditions typically leads to the development of skilled behavior that is qualitatively different from the performance level observed when the task was originally undertaken (Tiffany, 1990). A person learning to drive a car initially pays a great deal of attention to each action, and his or her performance is slow, hesitant, and filled with error. With extended practice, this task can become virtually effortless, and what once demanded considerable attention and concentration can be carried out rapidly and accurately, with little or no awareness of the component actions (Tiffany, 1990). This transformation of performance with practice has been described by many cognitive psychologists as the development of automaticity, and considerable research has been conducted over the past decade to explain essential features and cognitive processes underlying automatic and nonautomatic functioning (Tiffany, 1990).

When it comes to addiction, it mirrors the same concept of automaticity. After repeated behavior of a certain stimuli, it becomes a habit and autonomic (Tiffany, 1990). When patients
start undergoing withdrawal, these behaviors of automaticity start to become that urge and craving (Tiffany, 1990). It may be useful to conceptualize urges and cravings as responses supported by nonautomatic cognitive processes that are activated in parallel with drug-use action schemata (Tiffany, 1990). There are two situations under which this nonautomatic processing might be invoked (Tiffany, 1990). Some environmental condition impedes or blocks the drug-use action plan in an individual not attempting to avoid drug use, or nonautomatic cognitive processes are invoked in an explicit attempt to impede or block a drug use action plan (Tiffany, 1990).

The observed correlations between drug taking and neuropsychological changes raise several points. First, the cognitive consequences of drug abuse and addiction may lead to problems of rehabilitation that far outreach just reducing drug-seeking behavior (Robbins, Everitt, 1999). Second, the additional induced behavioral changes may accelerate the progression to addiction, for example, by impairing self-control (Robbins, Everitt, 1999). Third, the causal relationships between drug taking and neural, as well as neuropsychological, impairments are not clear (Robbins, Everitt, 1999). There may be some concurrent drug-taking behavior with other impulsive or risk-taking behavioral traits, because of genetic or developmental factors (Robbins, Everitt, 1999). Research regarding withdrawals on cognition is still occurring and is a growing field. What the research shows, helps bring new ideas and information, treatment facilities could use to treat patients with a variety of symptoms.
References


