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Brain Mechanisms and Clinical Aspects Involved in Nicotine Addiction

Megan Jason*

Department of Food Science, College of Agriculture and Life Sciences, Cornell University, United states

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***Corresponding author:**
Megan Jason

Department of Food Science, College of Agriculture and Life Sciences, Cornell University, United states

meganJ@cornell.edu

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Introduction

Tobacco smoke contains nicotine and other compounds that are easily taken into the bloodstream through the lungs. Nicotine rapidly travels throughout the body from there. Nicotine produces pleasant sensations and distracts the user from negative feelings when used in modest doses. This incentivizes the tobacco user to consume more. It affects mood through altering the chemistry of the brain and central nervous system. Nicotine, like other addictive substances, operates by flooding the reward circuits of the brain with a neurotransmitter called dopamine. Nicotine also produces a little amount of adrenaline, which causes the heart to beat faster and blood pressure to rise [1].

Nicotine reaches the brain in a matter of seconds after a puff, and its effects begin to fade within a few minutes. The user may become annoyed and agitated. It usually does not progress to the point of severe withdrawal symptoms, but the user becomes increasingly uncomfortable over time. This is usually what causes the person to light up again [2,3]. The person consumes tobacco at some time, the bad sensations go away, and the cycle resumes. Withdrawal symptoms worsen over time if the user does not smoke again shortly. People who use nicotine tend to increase the amount of tobacco they use as their bodies adapt to it. This increases the quantity of nicotine in their blood, necessitating the use of more cigarettes to have the same effect. This is referred to as tolerance. Over time, a specific nicotine level is achieved, and the user must continue to use the product to keep the nicotine level within a safe range.

Mechanism in the Brain

Nicotinic Acetylcholine Receptors

Inhaling cigarette smoke extracts nicotine from the tobacco in the cigarette. Nicotine is carried into the lungs by smoke particles, where it is quickly absorbed into the pulmonary venous circulation. Nicotine then enters the bloodstream and travels swiftly from the lungs to the brain, where it attaches to nicotinic cholinergic receptors (ligand-gated ion channels that normally bind acetylcholine). Nicotine binding at the receptor's contact between two subunits opens the channel, enabling sodium or calcium to pass through [4]. As these cations enter the cell, voltage-dependent calcium channels are activated, enabling additional calcium to enter.

Nicotine and Neurotransmitter Release

The brain produces a number of neurotransmitters when nicotinic cholinergic receptors are stimulated. Dopamine, for example, indicates a positive experience and is required for the reinforcing effects (effects that encourage self-administration) of nicotine and other addictive substances, as well as compelling urges like hunger. In rats, experimentally induced lesions in dopamine-releasing neurons inhibit nicotine self-administration [5]. Nicotine causes the mesolimbic region, corpus striatum, and frontal cortex to produce dopamine. In drug-induced reward, dopaminergic neurons in the ventral tegmental area of the midbrain and the shell of the nucleus accumbens are crucial (both regions have a role in perceptions of pleasure and reward).

Nicotine increases both glutamate and aminobutyric acid (GABA) release, which promotes dopamine release and inhibits dopamine release. Some nicotinic cholinergic receptors become desensitised after long-term nicotine use, whereas others do not. As a result, GABA-mediated inhibitory tone decreases while glutamate-mediated excitation increases, boosting dopaminergic neuron excitation and improving nicotine responsiveness [6].

Monoamine oxidase

Nicotine addiction is aided by cigarette smoke constituents other than nicotine. Monoamine oxidases are enzymes that catalyse the metabolism of dopamine, norepinephrine, and serotonin in catecholaminergic and other neurons. The activity of monoamine oxidase type A and type B is inhibited by the condensation products of acetaldehyde in cigarette smoke with biogenic amines, and there is evidence that inhibition of monoamine oxidase contributes to the addictiveness of smoking by decreasing dopamine metabolism.

Clinical Aspects Involved In Nicotine Addiction

Psychoactive effects of nicotine

Nicotine provides a pleasurable experience while also reducing tension and anxiety. Smokers utilise it to manage their mood and alter their

arousal levels. Smoking boosts your focus, response time, and ability to do specific activities. This improved performance and improved mood are most likely due to relief from withdrawal symptoms. When you stop smoking, you'll experience withdrawal symptoms including irritation, depression, restlessness, and anxiety. These mood abnormalities are similar in intensity to those seen in psychiatric outpatients [7]. Withdrawal from nicotine, as well as other substances of abuse, can cause anhedonia, or the sense that there is little joy in life.

Conditioned behaviour

When a nicotine addict quits smoking, the desire to restart is strong and lasts long after withdrawal symptoms have passed. Smokers learn to link certain moods, events, or environmental variables — smoking-related signals with the pleasurable effects of nicotine as they continue to smoke. Relapse is usually triggered by these factors.

The link between these signals and the expected effects of nicotine, as well as the consequent desire to use nicotine, is referred to as conditioning. Nicotine exposure induces changes in the protein expression of brain cells and their synaptic connections, a process known as neuronal plasticity, which underpins conditioning, according to animal studies. Nicotine also improves behavioural reactions to conditioned stimuli, which may contribute to smoking addiction. Furthermore, research in nicotine-dependent rats demonstrates that conditioned cues linked to nicotine withdrawal increase the severity of withdrawal by raising the reward threshold in the brain. Such training helps to keep the desire to smoke alive. Smokers generally light up after a meal, with a cup of coffee or an alcoholic beverage, or with smoking buddies. When these events are repeated often enough, they constitute a potent trigger for the desire to smoke. Drug-associated signals activate cortical areas of the brain, including the insula, according to functional imaging studies (a structure in the cortex associated with certain basic emotions).Smokers who have damage to the insula (e.g., brain trauma) are more likely to quit smoking shortly after the injury and stay abstinent, as well as having less conscious impulses to smoke, than smokers who do not have insula damage [8].

Nicotine addiction is therefore a pharmacological combination of positive rewards, such as improved mood and mental or physical functioning, and avoidance of withdrawal symptoms when nicotine is not available.

References

1. Dajas-Bailador F, Wonnacott S (2004) Nicotinic acetylcholine receptors and the regulation of neuronal signalling. Trends Pharmacol Sci 25(6):317–324.
2. Mansvelder HD, McGehee DS (2000) Long-term potentiation of excitatory inputs to brain reward areas by nicotine. Neuron 27(2):349–357.
3. Nestler EJ (2005) Is there a common molecular pathway for addiction? Nat Neurosci 8:1445–1449.
4. Fowler JS, Logan J, Wang GJ, Volkow ND (2003) Monoamine oxidase and cigarette smoking. Neurotoxicol 24(1):75–82.
5. Lewis A, Miller JH, Lea RA. Monoamine oxidase and tobacco dependence (2007) Neurotoxicol 28(1):182–195.
6. Hughes JR (2006) Clinical significance of tobacco withdrawal. Nicotine Tob Res 8(2):153–156.
7. Kauer JA, Malenka RC (2007) Synaptic plasticity and addiction. Nat Rev Neurosci 8:844–858.
8. Kenny PJ, Markou A (2005) Conditioned nicotine withdrawal profoundly decreases the activity of brain reward systems. J Neurosci 25(26):6208–12.