

The Hidden Effects of Adolescence Nicotine Exposure

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Abstract

Nicotine exposure during the adolescent stage has become a growing global concern, with many adolescents perceiving the use of combustible cigarettes and e-cigarettes/vapes as a stress reliever, a way to be rebellious, or a way to fit into social groups. However, nicotine exposure during the adolescent period can alter both neurological and emotional processes. The adolescent brain is still maturing, particularly in the prefrontal cortex, which supports decision making and impulse control, as well as in the limbic system, which regulates the reward system and emotional processes (“Brain Development During Adolescence”). Nicotine activates the mesolimbic dopamine system, which reinforces addictive behaviors (Tiwari et al.) as well as modifies neural circuits that are involved with learning and memory. These changes can interact with psychological factors, such as perception of risk, peer pressure, and stress, shaping initiation and continued intake of nicotine. This paper puts forth how interactions between brain development and behavioral processes contribute to vulnerability, emphasizing the importance of awareness of the drawbacks of adolescent nicotine usage.

Introduction

Nicotine is a highly addictive chemical compound that is found in the tobacco plant (“Nicotine Is Why Tobacco Products Are Addictive”). Nicotine is commonly delivered through combustible cigarettes, e-cigarettes, zyns, etc., and it functions as a stimulant, releasing neurotransmitters (such as dopamine) that affect the central nervous system by increasing alertness, memory, and offering a sense of pleasure (Davis). Due to nicotine rapidly reaching the brain after inhalation or oral absorption, nicotine produces reinforcing effects that contribute to dependence. In recent years, e-cigarettes, commonly known as vapes, have been the most accessible source of nicotine for teenagers, often due to eye-catching designs, flavoring of the product, and widespread normalization. This increased accessibility has led to an upsurge in adolescent exposure to nicotine during a critical phase of brain development. At the biological level, nicotine alters neural communication by binding to specific receptors and stimulating dopamine release within the brain’s reward circuit. In **Figure 1**, when nicotine binds to the nicotine receptor on the sending neuron, it triggers a release of dopamine into the synaptic gap. Dopamine then attaches to the receptors on the receiving neuron, sending a signal that activates the brain’s reward system. Since dopamine is linked to pleasure, motivation, and alertness, this pathway being repeated can strengthen habits and increase the risk of nicotine addiction, especially to the developing adolescent brain (Feldhausen).

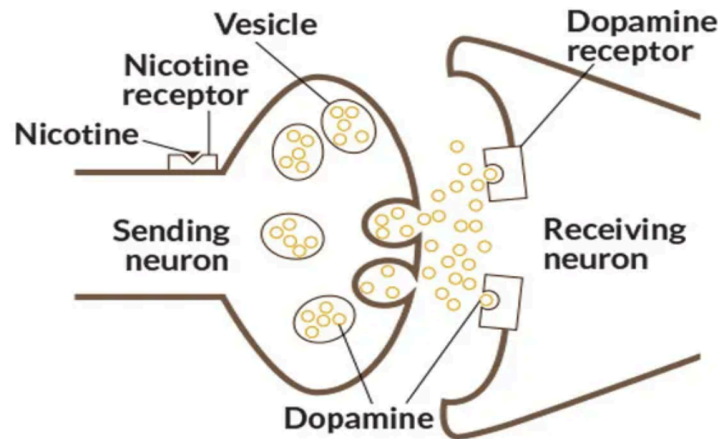


Fig 1: Nicotine (black triangle towards center left) tricks the nerve cell (neuron) into sending a message to release more dopamine (yellow dots). Those molecules enter the space (synapse) between one nerve cell and the next. When they get picked up by neighboring cells, this gives users a feel-good high. It also creates the risk of addiction and other health problems.

Neuroadaptations to Nicotine in the Adolescent Brain

To better understand how nicotine alters the developing brain, researchers have used neuroimaging through a PET Scan. A PET Scan (Positron Emission Tomography Scan) shows the amount of specific proteins in brain cells to which the drugs can attach. In this case, the proteins are nicotine receptors (“Can Nicotine Really Change My Brain?”). In a case study, two high school students, Corey (a smoker) and James (a non-smoker), underwent PET scans to measure the density of receptors in both of their brains (“Can Nicotine Really Change My Brain?”). In **Figure 2**, the brain scans of both boys are shown. The blue areas represent the

lowest amount of nicotine receptors, while the red areas represent the highest amount of nicotine receptors in the brain (“Can Nicotine Really Change My Brain?”).

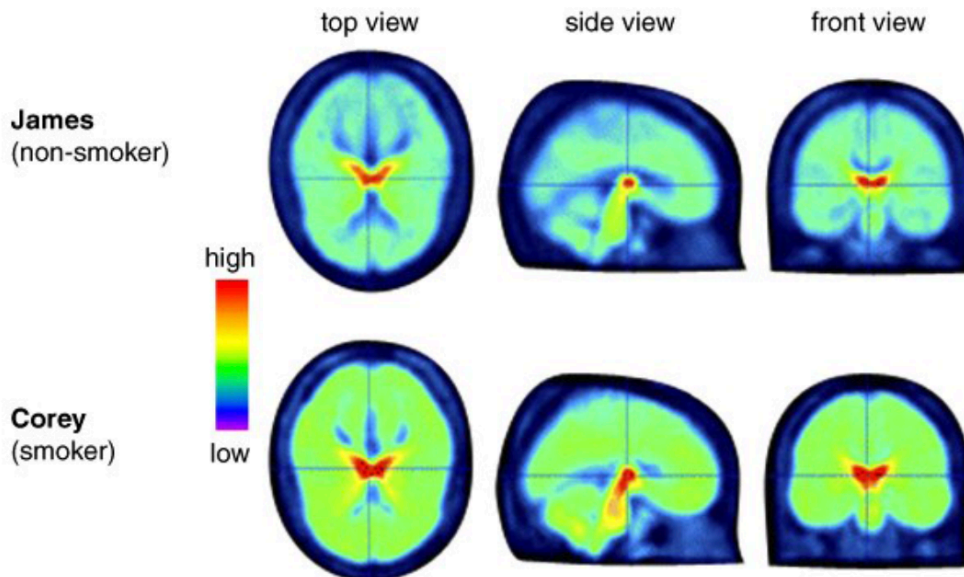


Fig 2: Corey and James' Brain Scans

According to the scans, Corey exhibited a significantly greater density of nicotine receptors in his brain than James. Additionally, Corey had higher amounts of green and red regions, a sign of repeated nicotine usage, than James, who had high amounts of blue regions, illustrating lower receptor density (“Can Nicotine Really Change My Brain?”). Corey’s increase displays receptor upregulation, which is an increase in the number of receptors on the neuronal surface (“Receptor Upregulation”). Specific to nicotine, sustained activation of nicotinic acetylcholine receptors (nAChRs) causes the brain to compensate by producing more receptor sites. During adolescence, when neural circuits are undergoing synaptic pruning, the process by which the brain removes

weak and unused connections between cells, called synapses (“Synaptic Pruning”), and structural refinement. This upregulation may interfere with normal developmental processes, leading to increased long-term vulnerability to addiction. In addition, the repeated intake of nicotine coincides with a decrease in grey matter, specifically in the prefrontal cortex, parietal cortex, and basal ganglia, which is critical for functioning, sensory processing, and behavior (Giedd et al.; Sowell et al.). Decrease in grey matter is linked to impulsive, rule-breaking behavior without recognizing the consequences (“Reduced Grey Matter Linked to Teenage Smoking and Nicotine Addiction”).

Psychological and Emotional Effects of Nicotine Usage

According to a 2023 survey of 2,505 adolescents conducted by the American Heart Association, those who vaped were associated with a higher risk of anxiety, depression, and suicidal thoughts compared to non-vapers (“Teen Vaping and Mental Health”). In addition to these statistics, 60% of vapers self-reported anxiety symptoms, compared to 40% of non-vapers. For depression, over 50% of vapers experienced depression symptoms compared to 25% of non-vapers. This was also linked to a significantly higher rate of suicide intentions, which was reported by half of vapers compared to a third of non-vapers (“Teen Vaping and Mental Health”). While these findings do not prove that vaping and nicotine intake directly cause mental health disorders, they show a strong association between nicotine use and the emotional well-being of adolescents. Since the adolescent brain is still developing, repeated stimulation of the brain’s reward and decision-making system may disrupt mood imbalance. Over time, this disruption can make teens more vulnerable to anxiety, depressive symptoms, and difficulty coping with stress.

Conclusion

Adolescent nicotine exposure is a global public health concern due to its high-impact effects on the developing brain and emotional well-being of youth. During adolescence, regions in the brain such as the prefrontal cortex and the limbic system are still in development, making the brain extremely vulnerable to nicotine. Through neuroimaging, such as PET scans and receptor upregulation findings, repeated nicotine use alters neural circuitry, strengthening addictive pathways and interfering with synaptic pruning and structural refinement. These neurological adaptations increase the chances of long-term dependence but also impair behavioral traits and regulations.

Beyond neurological effects, nicotine is strongly associated with increased rates of anxiety and depression among adolescents. Although mental health concerns linked to nicotine usage are still being researched, a consistent association between vaping and poor mental health suggests that nicotine exposure may lead to psychological vulnerabilities during a developmental period. Understanding the hidden biological and psychological consequences of adolescent nicotine exposure is vital for prevention efforts. Overall, recognizing adolescence as a sensitive period of human development strengthens the importance of addressing nicotine exposure not as just a behavioral choice, but as a developmental risk with long term complications.

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