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Nicotine addiction and vaping in post-pandemic adolescents: Investigating the prevalence, neurodevelopmental impact, and pulmonary pathogenesis in relation to Covid-19-driven behavioral shifts

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ABSTRACT

The post-pandemic era has witnessed a concerning global surge in adolescent nicotine consumption, primarily through the use of electronic cigarettes and vapes. This study investigates the epigenetic mechanisms underlying nicotine dependence in adolescents, the pathogenesis of vaping related pulmonary conditions, and the comparative health risks between vape use and traditional cigarette smoking. Adolescence is a critical neurodevelopmental stage marked by a heightened susceptibility to drugs and alcohol abuse and addiction due to significant plasticity in dopamine signaling and neurotransmitter activity.

This literature review explores the rise in adolescent vaping and nicotine addiction in the post-COVID-19 era, emphasizing the interplay between neurobiological vulnerability, pandemic-driven mental health disruptions, and sociocultural influences. It investigates the developmental consequences of nicotine exposure on the adolescent brain, the pathogenesis of vaping-related pulmonary and systemic health conditions, and compares the health risks of vaping to traditional tobacco use. The review also examines the pandemic's impact on adolescent mental health and how it intersected with substance use behaviors. It highlights the alarming resurgence of vaping among adolescents, driven by neurological sensitivity to nicotine, reinforcement learning, digital media influence, and inadequate regulatory enforcement. Finally, it addresses adolescent misperceptions surrounding nicotine addiction and underscores the need for effective prevention strategies, targeted education, and policy reform to mitigate the growing public health threat posed by adolescent e-cigarette use.

The COVID-19 pandemic dramatically reshaped adolescent behavior across the globe, contributing to a surge in nicotine use, particularly through electronic cigarettes (e-cigarettes) or vaping. As educational institutions closed, social interaction diminished, and prolonged uncertainty prevailed, adolescents increasingly turned to vaping as a coping mechanism for boredom, isolation, and psychological distress—often without fully understanding the health risks involved. This trend has drawn significant attention from public health researchers, given the widespread adoption of vaping among youth and its potential to catalyze long-term nicotine dependence and adverse developmental outcomes.

Adolescence is a critical developmental period marked by heightened neuroplasticity, hormonal changes, and behavioral shifts such as increased risk-taking and peer susceptibility. These biological and psychosocial dynamics make adolescents particularly vulnerable to the reinforcing properties of nicotine. Emerging evidence suggests that nicotine exposure during this sensitive developmental window can disrupt neurochemical pathways, impair cognitive function, and induce long-term alterations in emotional regulation and brain architecture. Furthermore, the pandemic exacerbated underlying mental health challenges among adolescents, creating fertile ground for the initiation and reinforcement of substance use behaviors, including vaping.

Compounding this issue is the rapid proliferation of vape-related content on social media, the glamorization of nicotine in popular culture, and the ease of access to vaping products among underage users—factors that continue to normalize and amplify e-cigarette use. Despite these growing concerns, adolescent misperceptions about nicotine addiction remain prevalent, often leading to an underestimation of dependency risks and a false sense of control over cessation.

This research paper critically examines the prevalence of vaping and nicotine addiction in post-pandemic adolescents, explores the developmental and physiological consequences of nicotine exposure, evaluates the pathogenesis of vaping-related pulmonary conditions, and highlights the broader public health implications of increased adolescent vulnerability to nicotine use. By synthesizing current evidence across biological, psychological, and sociocultural domains, the paper aims to provide a comprehensive understanding of the multifaceted threat vaping poses to adolescent health and development, while emphasizing the urgent need for targeted interventions and policy reform.

NICOTINE ADDICTION AND DEPENDENCE IN ADOLESCENTS

Biologically, adolescence begins with the onset of puberty, a hormonal cascade primarily regulated by the hypothalamic-pituitary-gonadal (HPG) axis. This initiates rapid changes in the body, such as growth spurts, development of secondary sexual characteristics, and changes in reproductive organs. Simultaneously, hormonal shifts—particularly increases in testosterone, estrogen, and dopamine—influence mood, behavior, and emotional reactivity.

From a neurological standpoint, the adolescent brain undergoes significant restructuring, especially in the prefrontal cortex, the region responsible for executive functioning, including impulse control, planning, judgment, and emotional regulation. However, the prefrontal cortex matures slower than the limbic system, which governs reward, motivation, and emotional responses. This developmental mismatch contributes to heightened risk-taking behaviors, increased sensation-seeking, and susceptibility to peer influence—a phenomenon often referred to as an "imbalance in neurodevelopment."

This developmental stage also involves increased vulnerability, both psychologically and socially. Adolescents often struggle with identity formation, self-esteem, and autonomy, while also facing external pressures related to social

acceptance, academic performance, and future uncertainty. These vulnerabilities can lead to increased emotional volatility, anxiety, or depressive symptoms, especially in environments lacking adequate emotional support and guidance.

One of the most critical challenges during adolescence is the urge to explore boundaries, including those involving sexuality, independence, and psychoactive substances. During this developmental window, the brain is sensitive to novel experiences with major experience-dependent plasticity occurring in executive control and decision-making regions, particularly in the prefrontal cortex (Bernheim et al. 2013). It is also, however, a time of increased vulnerability to drug abuse (Chambers et al. 2003; Crews et al. 2007). Initiation of substance abuse typically occurs during this period, with progression from use of alcohol and tobacco in early teens to illegal substances at later ages (Lai et al. 2000; Hanna et al. 2001; Biederman et al. 2006). Almost 90% of adult smokers started before the age of 18 (Substance Abuse and Mental Health Services Administration, 2011; Yuan et al., 2015)

Nicotine interacts with epigenetic mechanisms to enable the maintenance of nicotine-seeking across time. Research across species suggests that nicotine increases permissive histone acetylation, decreases repressive histone methylation, and modulates levels of DNA methylation and noncoding RNA expression throughout the brain. These changes are linked to the promoter regions of genes critical for learning and memory, reward processing and addiction. Pharmacological manipulation of enzymes that catalyze core epigenetic modifications regulate nicotine reward and associative learning, demonstrating a functional role of epigenetic modifications in nicotine dependence. These findings are consistent with nicotine promoting an overall permissive chromatin state at genes important for learning, memory and reward. (Muenstermann & Clemens., 2024)

Since the adolescent brain is still undergoing synaptic pruning and myelination, exposure to drugs during this period can disrupt normal neural development, increase the risk of addiction, and impair memory, attention, learning capacity, and emotional health in the long term.

Adolescence represents a pivotal phase of biological, neurological, and psychological transformation, during which individuals are particularly susceptible to environmental influences, including substance use. The asynchronous maturation of the prefrontal cortex and the limbic system predisposes adolescents to heightened impulsivity, risk-taking behavior, and peer-driven decision-making, all of which can significantly compromise their long-term health and cognitive development. Given the brain's heightened plasticity during this critical window, exposure to psychoactive substances—including nicotine and other harmful chemicals found in e-cigarettes—can lead to lasting neural alterations, increased vulnerability to addiction, and detrimental impacts on memory, emotional regulation, and learning. The alarming prevalence of adolescent vaping, compounded by aggressive marketing tactics and easy accessibility, poses a direct threat to this vulnerable demographic. Therefore, it is imperative to implement stringent measures to restrict youth access to vaping products, enhance public awareness, and enforce educational and policy-based interventions. Protecting adolescents from early exposure to harmful substances is not merely a public health priority—it is a necessary investment in the cognitive, emotional, and physical well-being of future generations.

IMPACT OF COVID-19 ON ADOLESCENT MENTAL HEALTH AND VAPING BEHAVIOR

The COVID-19 pandemic has significantly impacted adolescent mental health and vaping behaviors in several ways: Mental health impacts on adolescents during the pandemic have been substantial. Studies found increases in symptoms of anxiety, depression, psychological distress, and other mental health issues among teenagers (Bera et al., 2022; Windarwati et al., 2022). One study reported the number of

adolescents with poor mental health nearly doubled from 12.3% to 24.2% during the pandemic (Qin et al., 2021). Factors like family confinement, infection exposure risk, and disruptions to daily routines contributed to these mental health challenges (Qu et al., 2022).

Interestingly, some research found mixed or even positive effects of pandemic restrictions on adolescent mental health. One study reported decreases in rates of depressive and anxiety symptoms after two months of home confinement in China (Qu et al., 2022). The long-term home confinement was found to have minimal psychological impact on some adolescents. Regarding vaping tendencies, the pandemic had varied effects. Some studies found increases in e-cigarette use among youth during the pandemic (Clendennen et al., 2022), while others reported reduced access and opportunities to use nicotine products as a potential facilitator for quitting (Sharma et al., 2023). The pandemic created both barriers and facilitators to addressing adolescent vaping in schools (Liu et al., 2023).

Overall, poor mental health was associated with increased likelihood of substance use, including vaping, as a coping mechanism for pandemic-related changes (Romano et al., 2021). This highlights the interconnected nature of mental health challenges and vaping behaviors among adolescents during the COVID-19 pandemic.

In a recent review, Pfefferbaum highlighted the negative psychological effects of the pandemic on children and youth, including the significant increase in the prevalence of clinical depression, suicidal ideation, and anxiety, all of which have the potential to contribute to an increase in substance use behaviors (Pfefferbaum B., 2021).

Youth substance use most often takes place outside the home environment and usually within the context of the peer group. Moreover, youth substance use is highly dependent on availability and access to drugs and other substances. The public health restrictions that were necessary during the COVID-19 pandemic limited the time most adolescents spent in-person with their peers, and it follows that availability and access to alcohol, tobacco, and other substances was effectively limited during community lockdowns. In short, young people confined to their homes with parents had fewer opportunities for accessing and using substances. Thus, limited peer-group gatherings, decreased availability and access to substances, and increased time spent in the home with parents—all well-established factors shown to be effective in prevention efforts aimed at decreasing substance use (Kristjansson et al., 2021)—are likely to have conferred important protection against substance use during COVID-19 (Layman et al., 2022).

Although the overall decline in substance use is encouraging, these findings must be interpreted with caution. Specific subgroups of adolescents may have been at elevated risk for substance use during the pandemic due to pre-existing vulnerabilities. Notably, emerging evidence suggests that mental health concerns among adolescents were already increasing prior to the pandemic and continued to intensify throughout its duration. Additionally, for older adolescents and young adults experiencing increased stress and mental health problems, there is evidence that alcohol, drugs, and other substances may have offered a coping mechanism during the pandemic (Prowse et al., 2021).

Increased time spent within the household does not uniformly serve as a protective factor against substance use. One study observed that, shortly after the implementation of social distancing measures, some adolescents engaged in alcohol and other substance use in the presence of their parents. This finding implies that permissive parental attitudes and behaviors may contribute to the normalization and facilitation of youth substance use. These permissive attitudes and modelling of health compromising behavior can influence the perceived norms towards substance use, resulting in increased use after the pandemic. Moreover, adolescents living with family conflict or dysfunction are more likely to engage

in substance use (Smith et al., 2021; Layman et al., 2022).

Finally, in addition, youth living under the stress of parental substance use, family dysfunction, and domestic violence could predispose the later onset of substance use and violent behavior. Youth who missed out on "normal teenage years" or important rites of passage that were interrupted by the pandemic may also have difficulties with substance use later in life when restrictions are removed, and social gatherings allowed. What this means for the prevalence of substance use in the post-pandemic years will require monitoring and further surveillance (Layman et al., 2022).

The COVID-19 pandemic had a multifaceted impact on adolescent mental health and vaping behaviors. While some public health restrictions offered temporary protective effects—such as reduced peer interaction and limited substance access—adolescents simultaneously experienced heightened psychological distress, anxiety, and depression, all of which are closely linked to increased substance use as a coping mechanism. The pandemic's effects were not uniform, with certain vulnerable groups at greater risk due to pre-existing mental health issues, family dysfunction, or permissive environments. Although declines in substance use were reported in some cases, these trends must be interpreted with caution. As social restrictions ease, it remains critical to closely monitor adolescent substance use patterns and prioritize mental health support to prevent a potential resurgence in vaping and other harmful behaviors.

PREVALENCE OF VAPING AMONG POST-PANDEMIC ADOLESCENTS

The prevalence of vaping among adolescents has shown notable changes in the post-pandemic period. After initial declines during the early stages of the COVID-19 pandemic, vaping rates have rebounded to near pre-pandemic levels in the USA and Canada, while reaching record highs in England by 2022 (Hammond & Reid, 2023). This resurgence is particularly concerning given the high levels of nicotine exposure observed among adolescent e-cigarette users, with frequent users showing significantly elevated cotinine concentrations compared to non-users (Dai et al., 2024).

Interestingly, the pandemic's impact on vaping behaviors varied by gender. Both males and females reported reduced vaping frequency during the pandemic, but females were found to take more puffs per vaping episode compared to males in the pandemic period (Hopkins & Al-Hamdani, 2021). This gender difference highlights the need for targeted interventions.

Adolescence encompasses a sensitive developmental period of enhanced clinical vulnerability to nicotine, tobacco, and e-cigarettes. While there are sociocultural influences, data at preclinical and clinical levels indicate that this adolescent sensitivity has strong neurobiological underpinnings. Although definitions of adolescence vary, the hallmark of this period is a profound reorganization of brain regions necessary for mature cognitive and executive function, working memory, reward processing, emotional regulation, and motivated behavior. Regulating critical facets of brain maturation are nicotinic acetylcholine receptors (nAChRs). However, perturbations of cholinergic systems during this time with nicotine, via tobacco or e-cigarettes, have unique consequences on adolescent development (Yuan et al., 2015).

A major contributing factor to nicotine dependence is the persistence of cravings across abstinence. Through associative learning processes, places, people, or paraphernalia previously associated with smoking, become predictive of nicotine intake (Janes et al., 2010; Cynthia et al., 2015). In the absence of nicotine, these stimuli come to elicit strong cravings, prompt approach and procurement of nicotine/cigarettes, leading to a lapse of smoking behavior often weeks, months or even years after cessation (Betts et al., 2020; Carter & Tiffany, 1999; Cynthia et al., 2015). Increasing evidence suggests that the associations between nicotine and the stimuli that predict it are more robust and longer lasting than other memories

(Puma et al., 1999; Rezvani and Levin, 2001) and that this is reflected in changes in brain regions involved in reward learning, including the hippocampus (Kenney and Gould 2008; Fujii et al., 1999), ventral tegmental area (VTA) (Jin et al., 2011) and prefrontal cortex (PFC) (Couey et al., 2007). Among other changes, nicotine leads to increased hippocampal activity (Due et al., 2002) and synaptic plasticity (Fujii et al., 1999), affects excitability and N-methyl-D-aspartate (NMDA) receptor expression in the VTA (Jin et al., 2011) and enhances inhibitory transmission and memory in the PFC (Couey et al., 2007), thus showing widespread regulation of connectivity and plasticity in brain regions responsible for memory storage and consolidation (Muenstermann & Clemens, 2024).

The evolving landscape of adolescent vaping reflects a complex intersection of neurobiological vulnerability, behavioral conditioning, and sociocultural influence. During this critical stage of development, the adolescent brain undergoes profound restructuring, making it particularly sensitive to external stimuli such as nicotine. Disruptions to neurochemical systems during this period can have lasting implications on cognitive function, emotional regulation, and behavioral patterns. Craving persistence, triggered by environmental cues and associative learning, only intensifies the challenge of cessation, often embedding substance use deeper into adolescent behavior.

From my academic standpoint as a teenager, it is increasingly clear that beyond mental health factors and coping mechanisms, the surge in vape culture is heavily driven by its glorification on social media, the influence of music that romanticizes nicotine use, and the concerning ease with which underage users can access these products without facing real consequences. Addressing adolescent vaping today requires not only a scientific understanding of brain development but also a critical awareness of the digital and cultural ecosystems that shape youth behavior.

VAPING RELATED DEVELOPMENTAL DETRIMENT IN ADOLESCENTS

When a person inhales smoke from a cigarette, nicotine is distilled from the tobacco and is carried in smoke particles into the lungs, where it is absorbed rapidly into the pulmonary venous circulation. It then enters the arterial circulation and moves quickly to the brain. Nicotine diffuses readily into brain tissue, where it binds to nAChRs, which are ligand-gated ion channels. When a cholinergic agonist binds to the outside of the channel, the channel opens, allowing the entry of cations, including sodium and calcium. These cations further activate voltage-dependent calcium channels, allowing further calcium entry (Benowitz., 2009).

Brain imaging studies demonstrate that nicotine acutely increases activity in the prefrontal cortex, thalamus, and visual system, consistent with activation of corticobasal ganglia-thalamic brain circuits (Brody, 2006). Stimulation of central nAChRs by nicotine results in the release of a variety of neurotransmitters in the brain, most importantly dopamine. Nicotine causes the release of dopamine in the mesolimbic area, the corpus striatum, and the frontal cortex. Of particular importance are the dopaminergic neurons in the ventral tegmental area of the midbrain, and the release of dopamine in the shell of the nucleus accumbens, as this pathway appears to be critical in drug-induced reward (Dani & De Biasi, 2001; Nestler, 2005). Other neurotransmitters, including norepinephrine, acetylcholine, serotonin, γ-aminobutyric acid (GABA), glutamate, and endorphins, are released as well, mediating various behaviors of nicotine (Benowitz, 2009).

Dopamine release signals a pleasurable experience, and is critical to the reinforcing effects of nicotine and other drugs of abuse (Nestler, 2005). With repeated exposure to nicotine, tolerance (neuroadaptation) develops to some, but not all, of the effects of nicotine. Concurrent with this neuroadaptation is an increase in the number of nAChR binding sites in the brain. This increase is believed to represent upregulation in response to nicotine-mediated desensitization of receptors.

This desensitization may play a role in nicotine tolerance and dependence (Benowitz, 2009). Smokers may continue to smoke throughout the day to maintain plasma nicotine levels that prevent the occurrence of withdrawal symptoms, and may also continue to derive some rewarding effects from the conditioned reinforcers associated with smoking such as the taste and feel of the smoke (Balfour, 2004; Benowitz, 2009).

Nicotine withdrawal is associated with a negative emotional state, including anxiety and the perception of increased stress, which may represent powerful stimuli to relapse to tobacco use (Benowitz, 2009).

In humans, nicotine from tobacco induces stimulation and pleasure, and reduces stress and anxiety. Smokers come to use nicotine to modulate their level of arousal and for mood control in daily life. Smoking may improve concentration, reaction time, and performance of certain tasks. When a person stops smoking, nicotine withdrawal symptoms emerge. These include irritability, depressed mood, restlessness, anxiety, problems getting along with friends and family, difficulty concentrating, increased hunger and eating, insomnia, and craving for tobacco (Hughes & Hatsukami, 1986). Nicotine withdrawal in untreated smokers produces mood disturbances comparable in intensity to those seen in psychiatric outpatients (Hughes, 2006). Hedonic dysregulation, the feeling that there is little pleasure in life and that activities that were once rewarding are no longer enjoyable, is seen with withdrawal from nicotine and from other drugs of abuse (Koob & LeMoal, 1997). It is hypothesized that a relative deficiency in dopamine release following long-standing nicotine exposure accounts for many of the mood disorders and the anhedonia, as well as the tobacco craving, that may persist in smokers for a long time after they have quit (Benowitz, 2009).

All drug-taking behavior is learned, a result of conditioning. Drug-taking behavior is made more probable, or reinforced, by the consequences of the pharmacologic actions of the drug, as discussed for nicotine above. At the same time, the user begins to associate specific moods, situations, or environmental factors with the rewarding effects of the drug (Benowitz., 2009).

The mechanisms by which nicotine interacts with the adolescent brain highlight the profound developmental risks associated with early exposure to tobacco and e-cigarettes. The heightened sensitivity of adolescent neural circuits to nicotine, combined with stronger reward responses and reduced aversion, contributes to greater susceptibility to dependence. These neurobiological vulnerabilities, reinforced by behavioral conditioning and emotional regulation mechanisms, increase the likelihood of long-term use and relapse. Understanding these pathways underscores the importance of preventative strategies aimed at reducing adolescent access to nicotine products and mitigating the lasting developmental consequences of early nicotine exposure.

PATHOGENESIS OF VAPING RELATED PULMONARY AND PHYSICAL HEALTH CONDITIONS

Use of nicotine sustains tobacco addiction, which in turn causes devastating health problems, including heart disease, lung disease, and cancer, and increased susceptibility to a variety of infectious diseases. Smoking harms almost every organ of the body (U.S. Dept. of Health, 2004). Quitting smoking at any age leads to significant reductions in the risks associated with it.

An understanding of how nicotine produces addiction and influences smoking behavior provides a necessary basis for optimal smoking cessation intervention.

Nicotine is a tertiary amine consisting of a pyridine and a pyrrolidine ring. (S)-nicotine, found in tobacco, binds stereoselectively to nicotinic cholinergic receptors (nAChRs). (R)-nicotine, found in small quantities in cigarette smoke owing to racemization during the pyrolysis process, is a weak agonist at nAChRs (Benowitz, 2009).

Nicotine is a sympathomimetic drug that releases catecholamines, increases heart rate and cardiac contractility, constricts cutaneous and coronary blood vessels, and transiently increases blood pressure (Benowitz, 2003). Nicotine also reduces sensitivity to insulin and may aggravate or precipitate diabetes, and nicotine may contribute to endothelial dysfunction (Eliasson, 2003; Puranik & Celermajer, 2003). These various effects of nicotine on the cardiovascular system could, in theory, promote atherogenesis and precipitate acute ischemic events in people who have coronary artery disease (Benowitz, 2009).

Nicotine is not a direct carcinogen, but there are concerns that it may be a tumor promoter. In animal studies, nicotine can inhibit apoptosis, resulting in impaired killing of cancer cells (Zeidler et al., 2007). Nicotine also promotes angiogenesis in animals, an effect which could lead to greater tumor invasion and metastasis (Cooke & Bitterman, 2004). Whether nicotine is a cancer promoter in people has not been established, but one report that suggests that smokers who switch to smokeless tobacco may have an increased risk of lung cancer compared with smokers who quit entirely raises concern about this possibility (Henley et al. 2007; Benowitz, 2009).

Chronic nicotine exposure during adolescence produces alterations in neurochemistry and behavior that differ markedly from those in adulthood. Adults are more responsive than adolescents to nicotine-induced upregulation of $\alpha4\beta2*$ and $\alpha7$ nAChR binding sites; in contrast, adolescents exhibit greater downregulation of $\alpha6*$ nAChRs than adults following chronic nicotine treatment (Collins et al. 2004; Doura et al. 2008). Chronic nicotine treatment also produces age-specific effects on monoamine systems, with serotonin systems particularly vulnerable during adolescence. Biochemical studies have shown that chronic, high-dose nicotine exposure during adolescence results in altered indices of serotonin receptor function, with decreased 5-HT2 receptor binding in terminal regions and a switch in signalling of 5-HT1A receptors from stimulation to inhibition of adenylyl cyclase activity (Xu et al., 2002). Chronic nicotine treatment during adolescence also alters subsequent response of the serotonin system to nicotine later in life, suggesting that nicotine may elicit lifelong detriments in serotonergic signalling (Slotkin & Seidler, 2009; Slotkin et al. 2014; Yuan et al., 2015).

Additionally, chronic adolescent nicotine exposure induces unique and persistent dendritic remodeling in the prelimbic cortex (Bergstrom et al., 2008) and in the nucleus accumbens shell (McDonald et al., 2007), where the effects are mediated by D1 receptors (Ehlinger et al., 2015). Chronic nicotine exposure during adolescence also has long-term consequences on cognitive behavior. Adolescent, but not post-adolescent, treatment with nicotine has been shown to result in diminished cognitive function as adults with reduced attention span and enhanced impulsivity (Trauth et al., 2000; Counotte et al., 2009; Counotte et al., 2011). These cognitive disturbances are associated with reduced presynaptic mGluR2 protein and function on excitatory synapses in the prefrontal cortex, which alters the rules for spike timing-dependent plasticity in prefrontal networks (Counotte et al., 2011; Goriounova & Mansvelder, 2012). Attentional deficits in adults that received adolescent nicotine can be rescued by local infusion of a group II mGluR agonist (Counotte et al., 2011). Emotional responses also exhibit long-term alterations following adolescent nicotine treatment, with enhanced anxiety and fear (Slaweckiet al., 2003; Smith et al., 2006). Furthermore, adolescent but not adult nicotine treatment can result in a depression-like state in adulthood that is normalized by treatment with nicotine or antidepressants (Iniguez et al., 2009; Yuan, 2015).

The alarming rise in adolescent e-cigarette use prompted the U.S. Surgeon General to declare youth vaping an “epidemic” on December 18th, 2018 (U.S. Surgeon General, 2018). Albeit prior to the Covid-19 pandemic, the emergence of vaping was on a steady rise even before the introduction of confinement vaping. One of the most significant health risks

associated with vaping among adolescents is electronic cigarette or vaping-associated lung injury (EVALI), which gained attention during a 2019 outbreak in the United States (CDC, 2019). This condition is characterized by severe pulmonary inflammation and respiratory distress, often requiring hospitalization. During the Covid-19 pandemic, symptoms of EVALI and Covid-19 often presented themselves similarly, befuddling medical professionals and disrupting the availability of statistics to pinpoint the harmful effects of electronic cigarettes on youth (CDC, 2019).
One of the primary culprits in EVALI cases is vitamin E acetate (VEA), a diluent used in tetrahydrocannabinol vape cartridges. Although safe for ingestion, pulmonary tissue lacks the ability to metabolize or absorb VEA when inhaled, leading to its accumulation and subsequent lung injury (Blount et al., 2019).

Furthermore, propylene glycol and glycerin, common e-liquid humectants, can degrade into carcinogenic carbonyl compounds such as formaldehyde, acetaldehyde, and acrolein when heated within the metal coils of the vapes (Jensen et al., 2015). In addition, the metal components used in heating coils and cartridge casings pose an additional risk. Studies indicate that these materials pose the threat of leaching toxic metals—including aluminum, chromium, iron, lead, manganese, nickel, and tin—directly into the inhaled vapor (Olmedo et al., 2018). Prolonged exposure to the aforementioned heavy metals is linked to neurotoxicity, cardiovascular disease, and pulmonary inflammation, thereby subjecting youth and young adults to life-threatening injuries without sufficient warning (Olmedo et al., 2018).

Additionally, many flavoring agents, the primary culprits of adolescent interest in vaping, while deemed safe for ingestion, lack safety data for inhalational exposure. Diacetyl, a commonly used flavoring agent, has been linked to bronchiolitis obliterans (popcorn lung), a severe and irreversible lung disease (Allen et al., 2016). Beyond pulmonary risks, emerging studies suggest extrapulmonary effects of e-cigarette use, including immunologic, cardiovascular, and neurodevelopmental damage to adolescents (National Academies of Sciences, Engineering, and Medicine, 2018). Moreover, reports of physical vape malfunctions and explosions have risen, with a growing body of evidence documenting severe injuries such as burns, facial and oral trauma, hand injuries, and even limb damage resulting from battery failures, typically during device use, charging, or while being carried in pockets (Hua & Talbot, 2016). These incidents underscore the dangers of unregulated and poorly manufactured vaping devices, with lithium-ion battery malfunctions identified as a primary cause. In some cases, the injuries required surgical intervention, skin grafting, or long-term medical care, highlighting the need for enhanced safety regulations and product standards (Hua & Talbot, 2016).

Specifically, adolescents and youth may be particularly vulnerable to such injuries due to a higher likelihood of engaging in risky behaviors, purchasing unregulated or low-cost devices, and lacking knowledge of proper device maintenance and safety practices. Furthermore, the appeal of customization, social trends, and thrill-seeking behavior among teens can increase the chance of unsafe handling or modification of vape devices, exacerbating the risk of injury (Hua & Talbot, 2016).

ADOLESCENT MISPERCEPTIONS ABOUT NICOTINE ADDICTION

Adolescents often have misconceptions about nicotine addiction, underestimating its risks and overestimating their ability to quit smoking. Despite evidence that adolescents can become addicted to nicotine even after limited use, many believe they can experiment with or smoke cigarettes for a few years and easily quit (Roditis et al., 2015). This misperception is particularly concerning given that symptoms of physical nicotine dependence can appear when adolescents are smoking only a few cigarettes each month (Difranza, 2015;Roditis et al., 2015).

Interestingly, adolescents' understanding of addiction is complex and sometimes contradictory. While they have received the

message that cigarettes are addictive, they remain uncertain about the definition of addiction and fail to recognize that it means experiencing difficulty quitting and continuing to smoke longer than expected (Roditis et al., 2015). This disconnect is further illustrated by the fact that adolescents rate their perceived risk for addiction, likelihood of still being a smoker in 5 years, and ability to quit as significantly different from one another (Roditis et al., 2015).

The misconceptions held by adolescents about nicotine addiction highlight the need for comprehensive messaging regarding nicotine addiction in educational, clinical, and intervention settings. Additionally, there is a need for product warning messages aimed at reducing and preventing tobacco use among youth (Roditis et al., 2015). Addressing these misperceptions is crucial, especially considering the rapid onset of nicotine dependence in adolescents and its potential long-term consequences (Difranza, 2015; Mahajan et al., 2021).

CONCLUSION

The body of literature reviewed in this paper illustrates that adolescent vaping is not merely a behavioral trend, but a public health crisis with deep-rooted developmental, neurological, and sociocultural implications. The neurochemical susceptibility of the adolescent brain to nicotine, combined with the psychological stressors intensified by the COVID-19 pandemic, has contributed to an environment in which vaping behaviors flourish. While some public health restrictions temporarily reduced substance use opportunities, the post-pandemic rebound in vaping rates—fueled by digital glorification, product accessibility, and peer normalization—signals an urgent need for comprehensive intervention. Moreover, the pathophysiological impact of vaping, including EVALI, neurodevelopmental disruption, cardiovascular strain, and cognitive impairment, poses serious long-term health risks. Compounding this crisis are adolescents' misperceptions of nicotine addiction, which diminish their perceived vulnerability and hinder cessation efforts. Effective prevention must therefore extend beyond neurobiological awareness to address misinformation, strengthen health education, and enforce stricter access control. Protecting adolescents from the enduring harm of vaping demands coordinated efforts in research, policy, and public engagement to foster resilience, informed decision-making, and healthier developmental trajectories.

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