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E-cigarettes and systemic effects: updated mini-review 2026

Cigarrillo electrónico y alteraciones sistémicas: minireview actualizada 2026

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Abstract

Tobacco use remains one of the leading causes of morbidity and mortality worldwide. In Ecuador, although traditional tobacco use is on the decline, with a prevalence of 10.1%, the use of electronic nicotine delivery systems (ENDS) has gained ground, with more than 290,000 vapers. Objective: To synthesize the current scientific evidence on the pathophysiological mechanisms and systemic alterations associated with the use of e-cigarettes. An updated narrative review of the literature was conducted to analyze the biological, respiratory, cardiovascular, and neurological impacts of vaping. The evidence shows that the aerosol emitted by these devices contains nicotine, heavy metals, carbonyl compounds, and solvents such as propylene glycol and glycerin, all of which

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induce oxidative stress, cytotoxicity, and systemic inflammation. At the respiratory level, vaping reduces spirometric parameters (FEV1 and FeNO) and disrupts mucociliary and phagocytic defense mechanisms, contributing to acute and chronic conditions such as vaping-associated lung injury (EVALI), pneumonitis, asthma, and bronchiectasis. In the cardiovascular system, nicotine-mediated sympathetic stimulation and endothelial dysfunction lead to hypertension, arterial stiffness, hypercoagulability, and accelerated atherogenesis, increasing the risk of heart attack and arrhythmias. With regard to the nervous system, nicotine acts on the mesolimbic dopaminergic pathway, reinforcing addiction; furthermore, animal models show that gestational and postnatal exposure to aerosols—with or without nicotine—causes neuroimmunological imbalance, epigenetic modifications (DNA methylation), and behavioral or cognitive disorders in offspring. E-cigarettes are not a harmless alternative and cause significant multisystemic damage. It is essential to further clinical research and implement stricter public health policies and regulatory measures to limit their use, protect vulnerable groups, and reduce the economic impact on healthcare systems.

Keywords: cigarette, public health, vapers.

Resumen

El tabaquismo continúa siendo una de las principales causas de morbimortalidad a nivel mundial. En Ecuador, aunque el consumo del tabaco tradicional muestra una tendencia a la baja alcanzando una prevalencia del 10,1%, el uso de Sistemas Electrónicos de Administración de Nicotina (SEAN) ha ganado terreno, registrándose más de 290.000 vapeadores. Objetivo: Sintetizar la evidencia científica actual sobre los mecanismos fisiopatológicos y las alteraciones sistémicas asociadas al

consumo de cigarrillos electrónicos. Se llevó a cabo una revisión narrativa actualizada de la literatura orientada a analizar los impactos biológicos, respiratorios, cardiovasculares y neurológicos del vapeo. La evidencia demuestra que el aerosol emitido por estos dispositivos contiene nicotina, metales pesados, compuestos carbonílicos y solventes como propilenglicol y glicerina, los cuales inducen estrés oxidativo, citotoxicidad e inflamación sistémica. A nivel respiratorio, el vapeo reduce parámetros espirométricos (FEV1 y FeNO) y altera los mecanismos de defensa mucociliar y fagocítica, favoreciendo patologías agudas y crónicas como la lesión pulmonar asociada al vapeo (EVALI), neumonitis, asma y bronquiectasias. En el sistema cardiovascular, la estimulación simpática mediada por nicotina y la disfunción endotelial generan hipertensión, rigidez arterial, hipercoagulabilidad y aceleración de la aterogénesis, incrementando el riesgo de infarto y arritmias. Respecto al sistema nervioso, la nicotina actúa sobre la vía dopaminérgica mesolímbica reforzando la adicción; asimismo, modelos animales evidencian que la exposición gestacional y postnatal a aerosoles con o sin nicotina provoca desequilibrio neuroinmunológico, modificaciones epigenéticas (metilación del ADN) y trastornos conductuales o cognitivos en la descendencia. El cigarrillo electrónico no constituye una alternativa inocua y desencadena daños multisistémicos significativos. Resulta imprescindible profundizar en la investigación clínica e implementar políticas de salud pública y regulaciones normativas más estrictas para limitar su consumo, proteger a los grupos vulnerables y reducir el impacto económico en los sistemas sanitarios.

Palabras clave: cigarrillo, salud pública, vapeadores.

Introduction

Tobacco use is one of the leading causes of death worldwide and accounts for a significant percentage of public health issues. According to data reported in 2017 by the WHO, the prevalence of tobacco use in Ecuador is approximately 23% among males and 6% among females; this percentage is higher among individuals aged 13–15 (31.2% and 26.1%, respectively) (Revista Latinoamericana de Hipertensión, 2017).

The most recent estimates from the WHO show a downward trend in national prevalence. According to the WHO Report on the Global Tobacco Epidemic, 2023, the estimated prevalence of current tobacco use among people aged 15 and older in Ecuador was 10.3% for the year 2021, with a notable difference by sex (18.0% for men versus 2.7% for women) (World Health Organization [WHO], 2023).

Similarly, the WHO Global Report on Trends in Prevalence of Tobacco Use 2000–2024 and Projections 2025–2030 states that the prevalence stands at approximately 10.1%, which translates to around 1.3 million smokers in the country, with a clear gender gap in tobacco use also evident between men (17.6%) and women (2.5%) (WHO, 2024).

On the other hand, mortality figures remain significant. In 2021, an estimated 3,600 deaths were attributable to tobacco use, of which approximately 2,800 were among men and 808 among women. In proportional terms, smoking accounted for 2.9% of all deaths in the country, representing 3.94% among men and 1.52% among women (Global Burden of Disease [GBD], 2021).

The complex chemical composition of tobacco smoke and its systemic toxic effects largely explain this impact on mortality. Nearly 5,000 components have been detected in tobacco smoke, and it is estimated that another 10,000 may exist that have not yet been identified, although most of them are present in very low concentrations. Typically, these components are

categorized as carbon monoxide (CO), tar, nicotine, and other vaporized substances, such as carbon dioxide (CO_2), benzene, toluene, formaldehyde, acetone, cyanide, pyridine, and acrolein (Fernández González & Figueroa Oliva, 2018).

In this context, nicotine is a nitrogen-containing alkaloid and the primary psychoactive compound in tobacco. It acts as an agonist of nicotinic acetylcholine receptors in the central nervous system, where it activates the mesolimbic dopaminergic pathway and generates neurochemical changes that, through the reward system, promote the development of addiction to tobacco and e-cigarettes (Fernández González & Figueroa Oliva, 2018; Le Foll et al., 2022).

Furthermore, its rapid pulmonary absorption allows it to reach the brain in approximately 9–10 seconds after inhalation, reinforcing its high addictive potential. Furthermore, nicotine has a significant impact on the cardiovascular system, as each inhalation results in the absorption of between 50 and 150 micrograms of this substance, which stimulates the release of norepinephrine and raises adrenaline levels in the bloodstream. This leads to an increase in blood pressure and peripheral vascular resistance, thereby increasing cardiac output (Fernández González & Figueroa Oliva, 2018; Le Foll et al., 2022).

Nicotine use not only increases the risk of cardiovascular and respiratory diseases; furthermore, exposure to tobacco smoke or any other tobacco byproduct, such as tobacco residue, produces harmful health effects, including a decrease in the blood's oxygen-carrying capacity, which can lead to myocardial ischemia, especially in people with preexisting coronary artery disease or those exposed to high levels of CO in the environment (Fernández González & Figueroa Oliva, 2018).

Electronic cigarettes were designed by the Chinese pharmacist Hon Lik in the early 2000s and were introduced to the market in 2003 by a Chinese company, which by 2007 had already

registered and patented its design in a considerable number of countries (Restrepo et al., 2019).

E-cigarettes, also known as e-pens, e-cigars, or vaping devices, are electronic nicotine delivery systems that generate an aerosol mixture containing flavored liquids and nicotine, which the user inhales (Qasim et al., 2017).

These devices do not burn tobacco leaves; rather, through a heating mechanism, they release an aerosol containing residues of solutions such as propylene glycol, ethylene glycol, glycerol, vitamin E acetate, and other substances, which may or may not contain nicotine (Restrepo et al., 2019; Qasim et al., 2017).

The e-cigarette is distinguished by being a constantly evolving product, with a wide range of brands and various mechanisms that can alter the bioavailability of the inhaled products and their potential toxicity. Although many of the flavors banned in traditional cigarettes—such as coffee, fruit, caramel, and cola—are now attractive selling points for e-cigarettes, especially in marketing campaigns targeting young people (Restrepo et al., 2019; Qasim et al., 2017).

E-cigarettes typically consist of several components, including a nicotine cartridge containing e-liquid, a vaporization chamber, a heating coil to heat the liquid, an atomizer to generate vapor, a rechargeable battery, a voltage controller that regulates the amount of nicotine released during vaping, a microcompressor, and, in some cases, an LED indicator that activates the battery and visually simulates a conventional cigarette. This LED indicator, also known as a light-emitting diode, may not be present in all types of e-cigarettes (Qasim et al., 2017).

In Ecuador, the sale and use of electronic cigarettes—also known as Electronic Nicotine Delivery Systems (ENDS)—are permitted, but under specific regulations. These devices are regulated by Decree No. 1047 and the 2011 Law on Tobacco Regulation and Control, which means they cannot be used freely anywhere. Like traditional cigarettes, their use is

prohibited in enclosed public spaces or in areas where smoking restrictions already exist (Ministry of Public Health [MSP], 2018; Global State of Tobacco Harm Reduction [GSTHR], n.d.).

In terms of prevalence, it is estimated that there are currently around 292,836 e-cigarette users in the country. According to data reported in 2018, the prevalence of e-cigarette use among the adult population was 2.2%. When analyzed by sex, a higher prevalence is observed among women (4.8%) compared to men (1.7%), which highlights differences in consumption patterns by gender (WHO, 2023; GSTHR, n.d.).

Globally, e-cigarette use accounts for 1.9% of the population aged 15 and older, with the highest concentration in the Region of the Americas and in high-income countries. Within this context, the increase among young people is attributed primarily to their curiosity and the appealing flavors of e-liquids. It is concerning that this segment of the population is experiencing the greatest increase in the adoption of this practice, as this situation may have fatal consequences for their health in the future (WHO, 2023; Restrepo et al., 2019).

Despite prevention campaigns, testimonials, advertising, etc., which encourage awareness of the adverse effects of smoking, many pregnant women choose to use e-cigarettes based on the perception that they are safer than traditional tobacco. However, according to the scientific article "Impact of E-Cigarettes on Fetal and Neonatal Lung Development: The Influence of Oxidative Stress and Inflammation" published in the journal *Antioxidants*, it has been shown that nicotine from e-cigarettes crosses the placenta and reaches high concentrations in the fetus and amniotic fluid, in addition to being found at elevated levels in breast milk during lactation (Gambadauro et al., 2025).

Studies, mostly conducted in animal models, have shown that prenatal and neonatal exposure to vaping can disrupt critical stages of lung development, such as the sacular and alveolar

stages, leading to reduced lung size, delayed thinning of the lung epithelium (delayed lung maturation), and a phenotype similar to emphysema. Furthermore, evidence has been found of a potentiating effect that promotes the proliferation of oxidative stress-related genes (Gambadauro et al., 2025).

In research published in the journal PubMed, a study conducted on mice revealed that the offspring of female mice exposed to e-cigarettes experience a higher incidence of short-term memory deficits and hyperactivity during adulthood. These studies also identified significant changes in DNA, such as methylation and alterations in chromatin-modifying enzymes, which are present at all stages of the offspring's development. In the brains of these mice, a widespread increase in DNA methylation was observed, as well as abnormalities in the aforementioned enzymes (Nguyen et al., 2018).

Furthermore, other research has shown that epigenetic modifications resulting from e-cigarette use are linked to clinical conditions, such as an earlier onset of Alzheimer's disease. It has also been suggested that e-cigarette use may be associated with the development of mood disorders, anxiety, an increased propensity for substance abuse, and attention disorders (Ferrer, 2022).

The carcinogenic potential of e-cigarettes, which manifests primarily in the lungs, mouth, and throat, constitutes another critical aspect of the associated negative health profile. This phenomenon may be linked to the presence of nitrosamines, propylene glycol (the primary carrier used in e-liquids), and possibly certain flavoring agents. In particular, one study has suggested that, after being heated and vaporized, propylene glycol can transform into propylene oxide, which is classified as a Class 2B carcinogen (Ferrer, 2022).

According to clinical evidence, prolonged exposure to e-cigarettes has been found to cause significant alterations in respiratory function. A significant decrease in fractional exhaled

nitric oxide (FeNO)—which is related to pulmonary homeostasis and airway inflammation—was observed, suggesting an imbalance in pulmonary oxidative and inflammatory processes. A reduction in the FeNO biomarker has previously been associated with impaired respiratory function and increased severity of diseases such as COPD (Ferrer, 2022).

Similarly, a decrease in forced expiratory volume in the first second (FEV1) has been reported for both nicotine-containing and nicotine-free devices, suggesting that the deterioration in lung function does not depend exclusively on nicotine but also on other components present in the aerosol. It is believed that the vaporization process can induce oxidative stress and release toxic substances resulting from the heating of the e-liquid, promoting bronchoconstriction and alterations in spirometric parameters (Ferrer, 2022).

Furthermore, frequent episodes of wheezing have been observed, suggesting a link between e-cigarette use and the induction or exacerbation of airway reactivity. Specific clinical cases have linked e-cigarette use to the onset of bronchiectasis (Larue et al., 2021).

Cross-sectional studies have shown that e-cigarette use among young people may be associated with a higher prevalence and worsening of asthma. Furthermore, the use of these devices has been linked to a higher incidence of respiratory diseases and a deterioration in lung health among individuals with chronic obstructive pulmonary disease (Larue et al., 2021).

Furthermore, significant alterations in gene expression—indicative of immunosuppression—have been identified in bronchial biopsies from e-cigarette users. Proteomic studies have revealed that exposure to e-cigarettes produces notable changes in the bronchial epithelium and airway secretions (Larue et al., 2021; Martínez-Larenas et al., 2022).

At the same time, alterations in pulmonary defense mechanisms have been documented, including impaired mucociliary

clearance, disruption of the epithelial barrier, and decreased phagocytic function of alveolar macrophages, all of which increase susceptibility to respiratory infections (Park et al., 2022).

In this context, changes at the genetic and protein levels in the airways have been observed, indicating that vaping compromises the body's defenses and increases susceptibility to viral and bacterial pathogens. In humans, upregulation of the virulence factor and the platelet-activating factor receptor has been observed in vapers, suggesting a connection or pathophysiological mechanism between vaping and an increased risk of bacterial infections (Larue et al., 2021).

Clinically, multiple forms of acute lung disease associated with e-cigarette use have been described, including acute eosinophilic pneumonia, hypersensitivity pneumonitis, and interstitial lung disease associated with respiratory bronchiolitis (Park et al., 2022). Among these pulmonary conditions, e-cigarette or vaping-associated lung injury (EVALI) stands out. Patients with this condition show bilateral pulmonary infiltrates on imaging studies and may clinically experience dyspnea, cough, chest pain, and other respiratory symptoms. Chest X-rays and CT scans reveal ground-glass opacity patterns, particularly in the lower lobes of the lungs (Martínez-Larenas et al., 2022).

From a histopathological perspective, findings indicative of diffuse alveolar damage, organized pneumonia, and fibrinous pneumonia have been reported, frequently presenting with foamy macrophages and vacuolated pneumocytes, which indicate lipid accumulation and an inflammatory response, as well as direct damage to the alveolar epithelium, completely compromising the integrity of the alveolo- -capillary membrane and impairing gas exchange (Martínez-Larenas et al., 2022).

Regarding the pathophysiology of this condition, vitamin E acetate has been identified as a potential agent implicated in

EVALI, as it has been detected in a high percentage of bronchoalveolar lavage samples from affected patients. It is believed that this compound may interfere with the proper function of pulmonary surfactant, altering alveolar surface tension and promoting intraalveolar lipid accumulation, which leads to respiratory dysfunction and even respiratory failure in severe cases (Martínez-Larenas et al., 2022).

Furthermore, e-cigarette liquids may contain nicotine-derived nitrosamines, heavy metals, and carbonyl compounds formed by the heating of propylene glycol and glycerin. These substances cause cytotoxicity in the respiratory epithelium, increase oxidative stress, and activate pro-inflammatory pathways, thereby prolonging a chronic pulmonary inflammatory state (Martínez-Larenas et al., 2022).

This, in turn, causes severe cellular damage, including an increase in reactive oxygen species, DNA alterations, mitochondrial dysfunction, and cell death via apoptosis or necrosis—even in the absence of nicotine—which exacerbates pulmonary inflammation and contributes to the development of conditions such as EVALI associated with vaping (Park et al., 2022).

Similarly, passive exposure to e-cigarette aerosol is not without risk. It has been documented that bystanders can inhale nicotine, propylene glycol, carbonyl compounds, and heavy metals present in the ambient air, which can cause irritation of the respiratory tract and systemic effects such as palpitations and elevated blood pressure (Park et al., 2022).

Methodology

A descriptive, documentary, and narrative review (mini-review) was conducted, focusing on the search, synthesis, and critical analysis of the most recent scientific evidence regarding the biomedical repercussions and pathophysiological mechanisms of the use of electronic cigarettes or Electronic Nicotine Delivery

Systems (ENDS). The qualitative research design allowed for the integration of findings from various methodological approaches, including population-based epidemiological studies, in vitro laboratory experiments, animal model studies, and clinical trials in humans, with the aim of providing a holistic and rigorous overview of the systemic changes caused by the inhalation of vaping aerosol.

The literature search was conducted systematically during the period from 2017 to 2026, using the most internationally relevant search engines and databases in health sciences and biomedicine: PubMed/MEDLINE, ScienceDirect, Scopus, Google Scholar, and SciELO. Additionally, the search was supplemented by direct consultation of institutional repositories and official epidemiological bulletins from international and national public health organizations, such as the World Health Organization (WHO), the Global Burden of Disease (GBD) project, the American Heart Association (AHA), and the Ministry of Public Health of Ecuador (MSP).

Standardized MeSH (Medical Subject Headings) and DeCS (Descriptors in Health Sciences) terms were used to structure the search queries, which were combined using the Boolean operators AND and OR. The search strings included combinations of the following descriptors: ("Electronic Cigarettes" OR "E-cigarettes" OR "Vape" OR "Vaping" OR "SEAN") AND ("Systemic Alterations" OR "Toxicity" OR "Pathophysiology" OR "Cardiovascular System" OR "Respiratory System" OR "Neurotoxicity" OR "Epigenetics") AND ('Epidemiology' OR "Ecuador").

To ensure high scientific rigor, methodological validity, and maximum clinical relevance in conducting the study, the selection of documents was based on the strict application of the following criteria:

Inclusion criteria:

- Original scientific articles, systematic reviews, meta-analyses, mini-reviews, and scientific consensus statements published in indexed international and national peer-reviewed journals.

- Experimental and clinical studies that evaluated the chemical composition of e-liquids and aerosols, or that analyzed the direct pathophysiological effects on respiratory, cardiovascular, and neurological health, as well as pulmonary or brain development during the prenatal and neonatal periods.

- Official reports from health agencies on data regarding consumption prevalence, smoking-related mortality rates, and current legal and health regulations in the Ecuadorian context.

- Articles published in Spanish and English, with a publication window focused primarily on the period 2017–2026.

Exclusion criteria:

- Publications in non-indexed journals, editorials lacking empirical basis, opinion pieces, errata, conference abstracts, or informational press releases.

- Studies with serious methodological flaws in their design or whose data reports were incomplete, inconsistent, or ambiguous.

- Studies focused solely on traditional combustible tobacco use that did not include an explicit evaluation or comparison with electronic vaping devices.

Given the diversity and heterogeneity of the methodological designs reported in the selected scientific literature (which ranged from epidemiological studies to in vitro molecular assays and murine models), the information was processed using a descriptive thematic analysis. The extracted evidence was organized and categorized into four key analytical areas:

Epidemiological and Legal Area: Compilation and comparison of consumption patterns and the prevalence of use among adult and youth populations at the global and national levels, as well as a review of the regulatory legal framework in Ecuador (Decree No. 1047 and the Law on Tobacco Regulation and Control).

Respiratory Axis: Evaluation of cytotoxic and inflammatory effects on the pulmonary epithelium, alterations in spirometric markers (decreased FEV1 and FeNO), impairment of mucociliary clearance, and clinical and histopathological characteristics of acute lung injury (EVALI) and chronic diseases.

Cardiovascular Focus: Analysis of hemodynamic alterations caused by nicotine-mediated sympathetic stimulation, endothelial dysfunction, increased oxidative stress, myocardial remodeling, atherogenesis, and hypercoagulable states.

Neurological and Epigenetic Axis: Explanation of the neurobiological mechanisms of addiction (mesolimbic dopaminergic pathway), neuroimmunological alterations (cytokine imbalance), behavioral changes, and epigenetic modifications (DNA methylation and histone modifications) induced by prenatal and postnatal exposure.

Results

The action of nicotine is associated with the effects of e-cigarettes on the cardiovascular system, as this compound activates nicotinic acetylcholine receptors in the autonomic nervous system and the adrenal medulla, leading to the release of epinephrine and norepinephrine (Ferrer, 2022; Espinoza-Derout et al., 2022). This sympathetic stimulation leads to an increase in heart rate, cardiac inotropism, cardiac output, and blood pressure, accompanied by peripheral vasoconstriction. Although these effects may initially be transient, repeated exposure can cause cardiac overload, inflammation, cardiac remodeling, and an increased risk of arrhythmias (Espinoza-Derout et al., 2022).

According to various sources, several acute hemodynamic changes have been observed, such as an increase in arterial stiffness and impaired endothelial function. In the short term, a reduction in the increase in blood flow to the heart muscle during exercise has also been noted, with no changes in ventricular relaxation (Ferrer, 2022; Martínez-Larenas et al., 2022).

As mentioned earlier, nicotine directly impairs endothelial function. It reduces the expression of the enzyme endothelial nitric oxide synthase and decreases the availability of nitric oxide, a compound essential for vasodilation. At the same time, it increases the release of endothelin-1, a potent vasoconstrictor, and stimulates the production of reactive oxygen species, which cause oxidative stress, inactivate nitric oxide, and cause cellular damage, thereby promoting endothelial dysfunction and increased arterial stiffness (Espinoza-Derout et al., 2022).

Furthermore, nicotine also plays a pro-inflammatory and pro-atherogenic role. It activates macrophages and monocytes, stimulates cytokines such as TNF-**α** and IL-1**β**, and promotes the expression of endothelial adhesion molecules such as ICAM-1 and VCAM-1, thereby facilitating the infiltration of inflammatory cells into the arterial wall and the formation of foam cells from low-density lipoproteins (LDL), accelerating the development of atherosclerotic plaques. Prolonged oxidative stress also contributes to mitochondrial damage and the progression of atherosclerosis (Espinoza-Derout et al., 2022).

In this regard, nicotine also exerts a prothrombotic effect by increasing platelet activation and aggregation, promoting thrombus formation and raising the risk of acute cardiovascular events such as heart attack and stroke. Furthermore, it induces insulin resistance and alters lipid metabolism by increasing triglycerides, LDL, and VLDL and decreasing HDL, thereby promoting atherogenesis, systemic inflammation, and myocardial dysfunction, which contributes to metabolic

syndrome and increases the risk of hypertension, coronary artery disease, and heart failure (Espinoza-Derout et al., 2022; Hamann et al., 2023).

In experimental models, chronic exposure to nicotine-containing e-cigarettes has been linked to cardiac fibrosis, decreased ejection fraction, ventricular hypertrophy, increased vascular stiffness, and persistent activation of nicotinic receptors—which promotes fibroblast proliferation, collagen production, and adverse cardiac remodeling (Espinoza-Derout et al., 2022). Furthermore, in studies conducted among young people, short-term use of nicotine-free e-cigarettes has been associated with elevated C-reactive protein and other inflammatory markers, suggesting a possible increase in long-term cardiovascular risk (Ferrer, 2022).

The effect of e-cigarettes on the nervous system has been little studied; however, the study "Neurotoxicity of e-cigarettes" describes how vaping liquid—also known as e-liquid—whether in aerosol form or in liquid form, can seriously compromise our neurological health (Ruszkiewicz et al., 2020).

From a neurobiological perspective, nicotine binds to nicotinic acetylcholine receptors, which are distributed throughout the brain. When these receptors are activated, they allow ions such as sodium, potassium, and calcium to enter the neuron, causing its activation and increasing neuronal excitability (Herman & Tarran, 2020).

In addition, nicotine acts on the reward system by stimulating dopaminergic neurons in the ventral tegmental area, promoting the release of dopamine in the nucleus accumbens. This increase in dopamine is associated with feelings of pleasure, which reinforces use, facilitates the development of dependence, and explains the onset of withdrawal symptoms following prolonged exposure (Herman & Tarran, 2020).

In another experimental study, rats were exposed to e-cigarette aerosol during the pre- and postpartum periods, resulting in

short-term memory deficits, hyperactivity, and reduced anxiety. These mitigating effects are assumed to result from nicotine's stimulation of the central nervous system, although reduced anxiety was also observed in offspring exposed to nicotine-free aerosols (Nguyen et al., 2018).

It is worth noting that exposure to these nicotine-free aerosols was shown to promote gene methylation processes (reduction in gene expression without altering the original sequence) and to alter the function of histone acetyltransferases, leading to changes in the expression of genes linked to neurological activity (Nguyen et al., 2018; Ruszkiewicz et al., 2020). More specifically, changes in the expression of the genes Aurka, Aurkb, Aurkc, Kdm5c, Kdm6b, Dnmt3a, Dnmt3b, and Atf2—all associated with the modulation of neurological activity—were detected via RT-qPCR (Nguyen et al., 2018).

Complementarily, another study in mice demonstrated that prenatal exposure to aerosols containing propylene glycol and vegetable glycerol, with or without nicotine, during gestation causes behavioral alterations in adult offspring, such as hyperactivity, difficulties coping with stress, and memory impairment (Church et al., 2020).

Neuroimmunological changes were also observed, including reduced levels of IL-4 and IFN-**γ** in certain brain regions and increased levels of IL-6 in the cerebellum, suggesting a neuroimmunological imbalance in which the loss of anti-inflammatory control and the increase in pro-inflammatory mediators (IL-6) could promote a persistent inflammatory environment in the brain. This persistent inflammatory state may be the cause of the behavioral, cognitive, and neurological alterations described above (Church et al., 2020).

On the other hand, current evidence suggests that nicotine may influence the risk or severity of stroke in e-cigarette users. Although the evidence is still insufficient, these findings suggest a possible role for vaping in the pathophysiological mechanisms

of cerebral ischemia (Siegel et al., 2022). However, most of these findings come from animal models, so direct generalization to humans should be done with caution. Further clinical research is needed to determine more precisely the effects of e-cigarettes on the human nervous system.

Conclusions

To date, research on this topic has been limited, and the information presented in this article is based on the available findings provided by researchers. The limited number of studies in this area highlights the need for further exploration and understanding of the effects of e-cigarettes—both with and without nicotine—on various aspects of health, including potential cardiovascular risks. The current body of knowledge is based on existing findings, underscoring the importance of conducting additional research to shed light on the possible health impacts associated with e-cigarette use. Given that the various health conditions and consequences associated with the use of e-cigarettes represent a significant financial burden on the public health system, the implementation of specific regulations for these devices is anticipated. However, this measure seeks not only to protect the health system's finances but also to address the potentially irreversible consequences that may result from the continued use of these devices on people's health.

The need for stricter, prevention-oriented policies is evident, given the emerging evidence on the adverse health impacts associated with these devices. These regulations should focus on limiting access—particularly among the most vulnerable groups—and on providing clear information about the associated risks, with the goal of preventing long-term harm to the population's health.

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