



From Contents to Consequences: Toxicological Profile of e-Cigarette Aerosols

Dr. Suyog Savant¹, Dr. Mrunmayee Soman², Dr. Nilescha Kadam³, Dr. Pallavi Kale⁴, Dr. Farheen Tafti^{5*}, Dr. Ankita Kadam⁶

¹Associate Professor, Department of Public Health Dentistry, Bharati Vidyapeeth (Deemed to be University) Dental College and Hospital, Navi Mumbai, Maharashtra, India Orcid id: 0000-0001-6706-9684

²PG Student, Department of Pediatric and Preventive Dentistry, Bharati Vidyapeeth (Deemed to be University) Dental College and Hospital, Navi Mumbai, Maharashtra, India Orcid id: 0000-0002-8890-8130

³Assistant Professor, Department of Prosthodontics, Crown & Bridge and Implantology, Bharati Vidyapeeth (Deemed to be University) Dental College and Hospital, Navi Mumbai, Maharashtra, India Orcid id: 0009-0001-4666-5636

⁴Tutor, Department of Oral Pathology and Microbiology, Bharati Vidyapeeth (Deemed to be University) Dental College and Hospital, Navi Mumbai, Maharashtra, India Orcid id: 0009-0003-2110-5119

^{5*}Assistant Professor, Department of Pediatric and Preventive Dentistry, Bharati Vidyapeeth (Deemed to be University) Dental College and Hospital, Navi Mumbai, Maharashtra, India Orcid id: 0009-0007-6975-319X

⁶Assistant Professor, Department of Prosthodontics, Crown & Bridge and Implantology, Yerala Dental College and Hospital, Navi Mumbai, Maharashtra, India Orcid id: 0009-0001-3023-935X

*Corresponding Author:

Dr. Farheen Tafti, Assistant Professor, Department of Pediatric and Preventive Dentistry, Bharati Vidyapeeth (Deemed to be University) Dental College and Hospital, Navi Mumbai, Maharashtra, India

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ABSTRACT:

Background: Promotion of e-cigarette use among adolescents as safer alternatives to conventionally used cigarettes has emerged over the last two years. This narrative review aims to synthesize the current evidence on the chemical composition of e-cigarette liquids and aerosols, their toxicity mechanisms and organ specific harms along with implications for special populations.

Methods: Relevant literature was examined to summarise the constituents, the mechanisms of toxicity and organ system harms with a specific attention to youth, pregnancy and pre-existing comorbidities.

Results: E-cigarette aerosols contain humectants, nicotine, flavouring agents and thermal degradation product, volatile organic compounds and metals that generate reactive oxygen species inducing oxidative stress, inflammation, endothelial dysfunction and DNA damage. The mechanisms observed cardiopulmonary and metabolic risks along with neurocognitive developmental defects in adolescents which were seen to be amplified in youth, pregnancy and those with pre-existing comorbidities. Also, complete switching from smoking to vaping may reduce exposure to a few toxicants, widespread dual use, limited long-term data along with increased uptake among adolescents has seen to create a significant public health impact.

Introduction:

The early 2000s saw the emergence of a new non-combustible nicotine delivery device initially intended to

help quit or reduction of the use of conventional cigarettes [1]. Early products imitated the appearance of conventional cigarettes which delivered relatively lower



doses of nicotine; however with commercialization further generations were developed with comparatively larger batteries; adjustable power and refillable tanks [2]. Today, the most popularly used are single use and high-nicotine delivery pods that deliver nicotine at levels comparable or exceeding conventional cigarettes [3,4]. Thus, commercialization has led to an exceedingly heterogenous class of products that present with differing nicotine delivery levels and toxic emissions complicating the assessment of both regulation and health risk.

The use of tobacco has shown a significant global decline which has dropped from 1.38 billion users in the early 2000 to 1.2 billion in 2024 thus showing a cumulative reduction of 27% since the later part of 2010. Notably women's prevalence has fallen faster from 11% to 6.6%. Between this decline in the use of combustible cigarettes, the use of vaping and alternative nicotine products has shown a sudden surge. Recent estimates given by the WHO in their Tobacco Trends Report published on 6th October 2025 state that e-cigarette use exceed more than 100 million people world-wide with approximately 86 million adults especially in high-income countries. Current surveys estimate that around 15 million adolescents between the ages of 13-15 years have already

started vaping [5]. This report thus highlights the position of vape as an emerging public health issue.

Epidemiology and popularity:

A recent meta-analysis published in 2024 has estimated a pooled prevalence of the lifetime use of e-cigarettes among youth at 16.8% (10.6 – 25.6) and a past 30 day (current) use with a pooled prevalence at 4.8% (3-7.6). Boys showed a higher pooled prevalence of lifetime and current use - 18.8% (8.4 – 36.8) and 4.9% (3-8) respectively than girls – 9.9% (5-18.6) and 1.6% (1-3.1) respectively [6]. Another meta-analysis found that the current use of e-cigarettes was approximately 22% in school students and 10% in college students [7]. Use of e-cigarettes among adolescents may be due to a myriad of factors such as peer pressure and influence, seeking acceptance and a yearning for validation [8]. Additionally, curiosity, the appealing flavours of fruit and candy, perceived health benefits, conventional smoking quitting attempts along with influence of family members using the product also contribute towards use [9,10]. Few other contributing factors also include relative lower costs, extensive availability, judicious and discreet design helping the ease of hiding and using in school and college environments [9-12].

Constituents of e-cigarettes:

Table 1: Key Constituents of E-cigarettes

Constituent	Typical amount/ percentage	Function
Propylene Glycol (PG)	>65% of e-liquid	Humectant and solvent; produces aerosol simulating smoke when vaporized
Vegetable Glycerine (VG)	10–50% (balance with PG)	Thickener and solvent; enhances smoothness and cloud production
Nicotine	0-50 mg/ml	Primary addictive agent; provides pharmacological satisfaction
Flavouring Agents	1–20%	Imparts taste (e.g., fruit, candy); includes diacetyl, vanillin, ethyl maltol
Sweeteners	<1–5% (e.g., ethyl maltol, sucralose)	Enhances flavour perception; improves palatability



Formaldehyde	0.022–22.5 µg/breath (due to glycerol degradation)	Thermal degradation by-product; no intended function
Acetaldehyde	1.1-13.6 µg/150 puffs	Thermal degradation by-product; irritant, no function
Acrolein	0.7-41.9 µg/150 puffs	Thermal degradation from overheating glycerol; respiratory irritant
Volatile Organic Compounds (VOCs)	Variable (e.g., benzene, toluene µg/puff levels)	Degradation products or impurities; no function
Metals (e.g., Nickel, Chromium, Lead)	ng–µg/puff (coil leaching)	Device contaminants; no intended function

Table 1 summarizes the key constituents of e-cigarettes and their aerosols. Base ingredients that is propylene glycol, vegetable glycol and nicotine enable the formation of aerosol sensory appeal from thermal degradation products and leached metals. PG and VG act as primary solvents and humectants, and nicotine is the primary cause of addiction helping mask the hardness thus promoting use. Whereas degradation by-products such as formaldehyde and acrolein arising from overheating of solvents along with leached metals contribute towards unintended toxicity despite lower levels than traditional cigarette smoke [14].

Mechanism of Action:

Vape aerosols trigger a reactionary cascade of cellular stress responses that begins in the respiratory tract that extends to other systems via circulation. Oxidants like carbonyls, reactive flavoring aldehydes and metals together form reactive oxygen species (ROS) either directly or due to impaired mitochondrial function. The formed ROS then overloads the antioxidant defences such as glutathione, superoxide dismutase and catalase thus leading to lipid peroxidation, oxidation of proteins

and redox-sensitive signalling pathway disruption. Activation of pattern recognition receptors such as TLRs and transcription factors such as NF-κB and AP-1 in epithelial cells and macrophages follows causing upregulation of pro-inflammatory cytokines, chemokines, adhesion molecules promoting the recruitment of leukocytes into the airway and vascular wall. Alongside this, impairment of endothelial nitric oxide synthase (eNOS) activity and nitric oxide bioavailability reduction causes vasoconstriction and loss of normal vasodilatory responses. The exposed endothelial cells show an increased expression of adhesion molecules, increased permeability favouring activation of platelets and microvascular injury. The reactive metabolites and aldehydes form adducts and crosslinks of DNA, simultaneously ROS induces breaks in single and double strands and oxidative base lesions. This can thus lead to mutations, chromosomal instability and dysregulated control of the cell-cycle leading to carcinogenic potential with long-term exposure [13] (refer Fig 1).

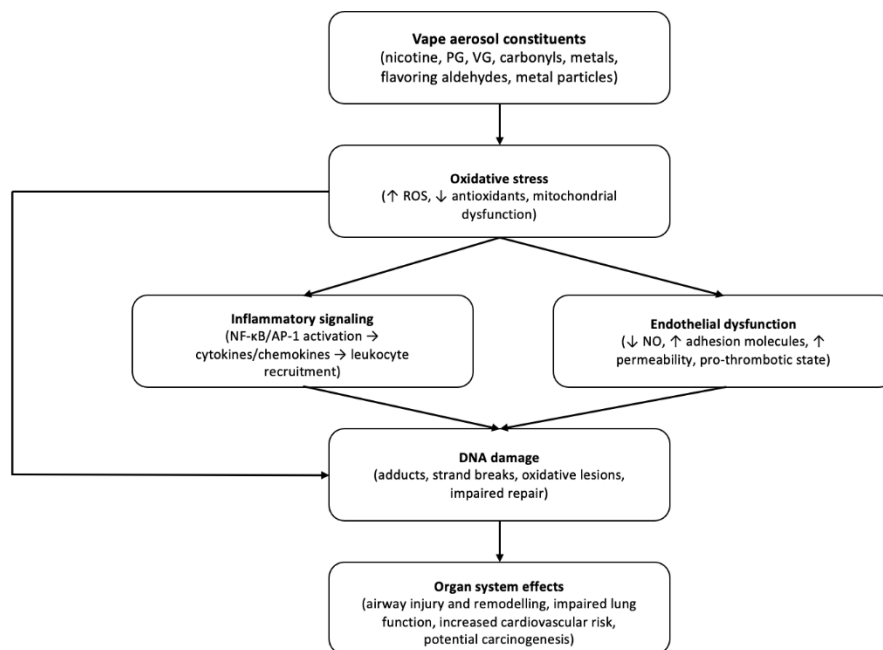


Fig 1: Mechanism of action of e-cigarette constituents and aerosols

Organ specific harms:

Vaping exposes multiple organs to a spectrum of acute and chronic health effects due to inhaled aerosols. Although toxicity levels are generally lower than conventional cigarettes, evidence shows that e-cigarettes contribute to respiratory symptoms and lung injuries, hemodynamic changes and changes in vasculature, inflammation and neurodevelopmental risks in users [15].

Oral and Respiratory system toxicity:

E-cigarettes aerosols are delivered into the airway epithelium directly inducing oxidative stress and inflammatory responses leading to coughing, wheezing and worsening of asthma and bronchitis-like symptoms. However the oral cavity is usually the first involved with various studies stating alterations in the oral microbial structures, inflammation of periodontium, higher risk of dental caries along with cellular changes elevating premalignancy or malignancy [16,17]. Nasal toxicity due to aerosols has been seen to be associated with immune-related gene expression decrease [18]. E-cigarette or Vaping Use-Associated Lung Injury (EVALI) that resulted in approximately 3,000 hospitalizations is an acute and often times severe vaping

associated injury of the lung that is characterized by diffuse inflammation of the lungs, radiographic infiltrates, hypoxemia and pathological patterns similar to organizing pneumonia and diffuse damage of the alveoli [19]. Many cases required intensive care along with mechanical ventilation resulting in death [19,20]. Repeated exposures have also been associated with airway hyper-responsiveness, impaired mucociliary clearance along with acceleration of chronic obstructive airway disease especially seen in people with any underlying lung vulnerabilities [15, 21].

Cardiovascular system toxicity:

Acute sympathetic activation may lead to transient increase in both heart rate and blood pressure due to the nicotine, while due to aerosol particles and oxidants lead to endothelial dysfunction [15]. Both experimental and clinical studies have shown impaired flow-mediated vasodilation, increased arterial stiffness and pro-thrombotic changes after vaping may lead to reduction in bioavailability of nitric oxide and vascular adhesion molecules upregulation. Over repeated use, may lead to the enhancement of atherosclerotic risks that further contribute to ischemia, arrhythmia and other cardiovascular events especially in those with pre-



existing cardiovascular disease or conjunctive use of conventional cigarettes [19,22].

Neurological and developmental effects:

The blood-brain barrier is readily crossed by nicotine, and exposure mainly occurs in critical periods of maturation that involve regulation of emotions, executive functions, and involve refinement of reward circuits [23]. Repeated vaping also causes enhancement of mesolimbic dopamine signalling, while simultaneously reinforcing use and dependence. Pre-clinical and epidemiologic studies associate with impaired attention, mood dysregulation and an increased vulnerability for use of other substances such as cocaine and methamphetamine [24]. Adolescent users also can experience increased persistent alterations of cognitive control, regulation of impulses and processing of rewards due to increased sensitivity on synapse plasticity [24]. A study published in 2021 projected a total loss of approximately 29 billion years of life in men and 22 billion years in women between the years of 2022 and 2050 [25].

At-risk populations:

While the above section outlines the major organs affected by vaping, the magnitude as well as the implications of the harms have been seen to be amplified in specific vulnerable groups, particularly adolescents, pregnant women along with their fetuses and individuals with pre-existing cardiopulmonary or metabolic diseases.

Adolescents:

As adolescents are in critical phases of brain, lung and behavioural development, they are particularly sensitive to the neurodevelopmental effects of nicotine and airway injuries. Those who vape also are at a higher risk of presenting with respiratory symptoms, nicotine dependence and transitioning to conventional forms of tobacco along with poly-substance use as compared with non-users [23,24]. Misconceptions of vaping being harmless have been promoted strongly by social media along with the stronger flavour appeal thus leading to early initiation and increased use; thereby potentially establishing a lifelong addiction of nicotine [24].

Pregnant women and fetus:

Nicotine and vape aerosol contents have been seen to readily cross the placental barrier thus impairing

placental function, reducing the uteroplacental blood flow and interfering with organ development. Studies have shown linkage of e-cigarette use with low birth weight, preterm labour and abnormal fetal growth [26].

People with existing comorbidities:

Individuals suffering with existing comorbidities such as cardiovascular diseases, chronic lung diseases, diabetes may show exacerbated symptoms on the use of vape. Increased cough, wheezing, exacerbations alongside airway inflammation and irritant effects have been seen in people with pre-existing pulmonary conditions. Whereas, nicotine-induced sympathetic activation, endothelial dysfunction and pro-thrombotic changes precipitating to ischemic events have been seen in those with pre-existing cardiovascular or metabolic changes [24].

Current controversies:

Although evidence highlights the increased risks for vulnerable groups, the overall place of e-cigarettes in control of tobacco is yet contested. These uncertainties affect their potential role in harm reduction versus contribution to nicotine dependence alongside dual use along with important gaps in long-term safety data.

Harm reduction vs continued nicotine dependence:

A lower level of carcinogens and toxicants is generally emitted by e-cigarettes, thus has led to experts argue that a complete switch from conventional smoking to vaping can reduce risk for heavily dependent adult smokers who are unable to quit by other means. However, studies have shown that majority may become dual users – maintaining both vaping and smoking rather than a full substitution [27, 28].

Long-term unknowns:

As the prolific use of e-cigarettes has barely been for the last decade, long term data for chronic outcomes such as COPD progression, incidence of cancer and cardiac events along with neurocognitive trajectories is still limited; especially among exclusive vape users. Also many constituents have not yet been evaluated for inhalation toxicity; thus still leading to uncertainty about the cumulative risks as well as later emerging diseases [29].



Conclusion:

Initial introduction of e-cigarettes was as alternative nicotine delivery systems; however rapidly they have evolved into heterogenous products delivering high nicotine doses and aerosols with toxic potential. Current evidence states that aerosols contain a myriad of harmful contents such as carbonyls, volatile organic compounds, metals and flavouring agents which contribute towards oxidative stress, inflammation, endothelial dysfunction and DNA damage to multiple organ systems. Although the emissions are of lower toxicity as compared to conventional cigarettes, vaping shows links with cardiopulmonary changes, oral and periodontal pathology and neurocognitive risks especially in adolescents, pregnant women and those with pre-existing comorbidities. However, at the same time uncertainties persist regarding long term consequences and public health concerns as dual use is widespread and a persistent ongoing initiation is present among youths. Thus to conclude, e-cigarettes should not be considered harmless or as cessation tools even if the long-term longitudinal data is absent.

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