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IMPACT OF ELECTRONIC CIGARETTES ON ENDOTHELIAL FUNCTION AND CARDIOPULMONARY HEALTH IN YOUNG ADULTS

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The global use of electronic cigarettes has increased rapidly, particularly among adolescents and young adults, driven by aggressive marketing, flavored products and misconceptions about relative safety. Electronic cigarette aerosols contain nicotine, propylene glycol, glycerol, flavoring chemicals, carbonyl compounds, volatile organic compounds, heavy metals and other toxicants. Current evidence indicates that vaping induces oxidative stress, endothelial dysfunction, immune dysregulation and structural injury of the respiratory tract. Clinically, electronic cigarette use is associated with electronic cigarette or vaping product use-associated lung injury, worsening of asthma, decreased lung function and adverse cardiovascular effects, including acute blood pressure and heart rate elevation, impaired flow-mediated dilation and activation of pro-thrombotic pathways. In adolescents, additional concerns relate to neurocognitive development and nicotine dependence. This narrative review summarizes contemporary data on the pulmonary, cardiovascular and immunological effects of electronic cigarettes, compares them with combustible tobacco, highlights gaps in long-term evidence and identifies the need for prospective studies of endothelial and lung injury biomarkers in young users.

Key words: electronic cigarettes, vaping, endothelial dysfunction, cardiovascular disease, lung injury, adolescents.

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ВПЛИВ ЕЛЕКТРОННИХ СИГАРЕТ НА ЕНДОТЕЛІАЛЬНУ ФУНКЦІЮ ТА КАРДІОПУЛЬМОНАЛЬНЕ ЗДОРОВ'Я У МОЛОДИХ ДОРОСЛИХ

Поширеність використання електронних сигарет швидко зростає, особливо серед підлітків та молодих дорослих, що зумовлено активним маркетингом, ароматизованими продуктами та уявленням про їхню відносну безпеку. Аерозолі електронних сигарет містять нікотин, пропіленгліколь, гліцерин, ароматизатори, карбонільні сполуки, леткі органічні речовини, важкі метали та інші токсичні речовини. Сучасні дані свідчать, що вейпінг викликає окислювальний стрес, ендотеліальну дисфункцію, імунну дисрегуляцію та ураження дихальних шляхів. Клінічно застосування електронних сигарет пов'язане з ураженням легенів, асоційованим із використанням електронних сигарет або продуктів для вейпінгу, погіршенням перебігу астми, зниженням функції легенів та несприятливими серцево-судинними ефектами. Особливе значення для підлітків мають ризики для нейрокогнітивного розвитку та формування нікотинової залежності. У огляді узагальнено сучасні дані щодо легеневих, серцево-судинних та імунологічних ефектів електронних сигарет, а також зазначено необхідність подальших проспективних досліджень.

Ключові слова: електронні сигарети, вейпінг, ендотеліальна дисфункція, серцево-судинні захворювання, ураження легенів, підлітки.

The global prevalence of electronic cigarette use has risen sharply over the past decade. The increase is especially evident among adolescents and young adults, and in several countries the prevalence of vaping among school-aged individuals exceeds that among adults [4, 12, 15].

Several factors have contributed to this expansion. Electronic cigarettes are available in disposable and refillable formats, are promoted through social media and influencer marketing, and are offered in a wide range of flavor varieties that increase their appeal to younger users [4, 12, 34]. Although these products were initially introduced as potential harm-reduction tools for adult smokers, accumulating evidence raises substantial concerns regarding their independent health effects [3, 6, 22].

Chemical analyses and experimental studies demonstrate that electronic cigarette aerosols contain nicotine, carbonyl compounds, volatile organic compounds, metals and flavoring chemicals capable of inducing oxidative stress, inflammatory responses and structural injury [5, 10, 32]. Clinical and experimental evidence associates vaping with adverse respiratory outcomes, including electronic cigarette or vaping product use-associated lung injury, airway inflammation and impaired lung function, as well as cardiovascular effects such as acute hemodynamic stress, endothelial dysfunction and pro-thrombotic activation [1, 6, 7].

Despite growing research, significant uncertainties persist regarding early vascular injury, subclinical lung damage and long-term outcomes in young individuals who vape but do not smoke combustible tobacco [35]. The aim of this review is to synthesize current evidence on pulmonary, cardiovascular and immunological effects of electronic cigarettes, compare these findings with those associated with conventional tobacco smoking, outline key public health considerations and justify the need for targeted clinical research on endothelial dysfunction and early lung injury in young electronic cigarette users [20, 25, 26].

The purpose of the study was to establish the impact of electronic cigarettes on endothelial function and cardiopulmonary health in young adults.

Materials and methods. A narrative literature review was performed according to the principles of transparent evidence selection used in systematic reviews. The search was conducted in PubMed, Scopus, Web of Science and Google Scholar. The last search was performed on 28 June 2026. Publications from 2021 to 2026 were prioritized for inclusion; older publications were excluded from the final reference list unless they were required for historical context, and such sources were not retained in the final list.

The following verbatim search queries were used:

– PubMed: ("electronic cigarette" OR "e-cigarette" OR vaping OR "electronic nicotine delivery systems") AND (endothelial OR cardiovascular OR cardiopulmonary OR respiratory OR pulmonary OR immune OR adolescent) AND (review OR meta-analysis OR cohort OR clinical study).

– Scopus: TITLE-ABS-KEY ("electronic cigarette" OR "e-cigarette" OR vaping) AND TITLE-ABS-KEY (endothelial OR cardiovascular OR respiratory OR pulmonary OR immunological OR adolescent) AND PUBYEAR > 2020 AND PUBYEAR < 2027.

– Web of Science: TS=("electronic cigarette" OR "e-cigarette" OR vaping OR "electronic nicotine delivery systems") AND TS=(endothelial OR cardiovascular OR respiratory OR pulmonary OR adolescent OR immune) AND PY=(2021-2026).

– Google Scholar: "electronic cigarettes" "endothelial dysfunction" "respiratory" "young adults" "doi" 2021 2026.

Inclusion criteria were as follows: publications in English; original studies, systematic reviews, meta-analyses, narrative reviews or consensus statements addressing electronic cigarettes and respiratory, cardiovascular, endothelial, immunological or adolescent health outcomes; studies involving human participants, experimental models or toxicological analyses relevant to human exposure; availability of a digital object identifier; and publication in peer-reviewed journals indexed in Scopus or Web of Science.

Exclusion criteria were as follows: publications before 2021; articles without a digital object identifier; non-peer-reviewed materials; conference abstracts without full text; studies focused exclusively on combustible tobacco without electronic cigarette data; duplicate publications; and papers in which the outcomes were not relevant to cardiopulmonary, endothelial, immunological or adolescent health (Table 1).

Table 1

Simplified PRISMA Flow

Stage	Description	Number of Records/Studies
1. Identified	Total number of records identified through database searching and other sources	142
2. Duplicates Removed	Records removed before screening as duplicates	38
3. Screened (Title/Abstract)	Records screened after duplicates were removed	104
4. Assessed for Eligibility (Full-text)	Full-text articles assessed against inclusion and exclusion criteria	56
5. Included in Review	Primary and review studies finally included in the review	35

Results of the study and their discussion. Chemical Composition of Electronic Cigarette Aerosols. Electronic cigarette devices function by heating a liquid formulation to produce an inhalable aerosol. These formulations typically contain nicotine, propylene glycol, vegetable glycerin and a broad spectrum of flavoring agents, including sweet, fruit-based and confectionery-type additives [5, 10, 35]. Many products also incorporate auxiliary solvents and chemical additives whose toxicological properties remain incompletely characterized [3, 23].

Analytical and toxicological studies demonstrate that aerosols generated by contemporary devices contain fine and ultrafine particulate matter, volatile organic compounds, carbonyl compounds such as formaldehyde, acetaldehyde and acrolein, tobacco-specific nitrosamines, flavoring-related diketones and metals originating from heating coils or

device degradation [5, 10, 32]. Thermal decomposition during aerosol generation may increase the production of toxic carbonyl species, particularly at higher power settings [32, 35].

Because electronic cigarette liquids and device components remain heterogeneous and are often subject to incomplete regulatory oversight, their chemical composition varies substantially between manufacturers, batches and device types. This variability complicates exposure assessment and makes the long-term health consequences difficult to define precisely [3, 22, 23].

Immunological and Cellular Effects. Experimental studies show that electronic cigarette aerosol constituents elicit a pro-inflammatory response in immune and structural cells of the respiratory tract. Exposure of airway epithelial cells and immune cells to aerosol extracts is associated with increased cytokine release, oxidative stress, impaired barrier integrity and altered host defense signaling [5, 14, 18].

These molecular changes may translate into impairment of innate immune function. Studies report altered macrophage and neutrophil activity, changes in antimicrobial defense and increased susceptibility to bacterial respiratory infection. Human biomarker studies also demonstrate increases in markers of oxidative stress and endothelial activation after electronic cigarette exposure [14, 18, 20].

Collectively, available evidence supports the concept that vaping induces a systemic pro-inflammatory and immunomodulatory state. Although the long-term clinical implications of these immune alterations are incompletely defined, the findings provide a plausible mechanistic link between electronic cigarette use and respiratory and cardiovascular morbidity [14, 20, 22].

Effects on the Respiratory System. The most severe pulmonary consequence of electronic cigarette use is electronic cigarette or vaping product use-associated lung injury. It was first recognized during a large outbreak in the United States and was linked particularly to tetrahydrocannabinol-containing products and vitamin E acetate; nevertheless, the outbreak emphasized the general danger of inhaling poorly regulated mixtures of solvents, oils and chemical additives [1, 8, 29].

Beyond acute lung injury, habitual electronic cigarette use is associated with a spectrum of subclinical and clinical respiratory alterations. Systematic reviews and cohort studies report increased respiratory symptoms, airway inflammation, asthma-related outcomes, chronic obstructive pulmonary disease-related outcomes and changes in lung function among current users, although the certainty of evidence varies across outcomes [3, 17, 25].

Young habitual users may demonstrate early obstructive ventilatory changes and inflammatory remodeling phenotypes that differ from those observed in combustible cigarette smokers. Such findings are clinically important because many young users have no prior smoking history and may underestimate the possibility of progressive respiratory injury [3, 17, 31].

Evidence regarding individuals with asthma remains heterogeneous. Some studies describe worsening symptoms, increased airway resistance and heightened bronchial reactivity, whereas others show no short-term deterioration. This inconsistency probably reflects differences in exposure duration, nicotine concentration, device type, flavoring composition and individual susceptibility [1, 17, 25].

Cardiovascular Effects. Nicotine is a potent sympathomimetic compound that exerts immediate cardiovascular effects through activation of nicotinic acetylcholine receptors and stimulation of the sympathetic nervous system. Acute exposure to nicotine-containing aerosols increases heart rate, blood pressure and myocardial oxygen demand, while repeated exposure may contribute to autonomic imbalance and arterial stiffness [6, 13, 22].

Endothelial dysfunction is a central initiating event in atherosclerosis. Recent studies and meta-analyses indicate that chronic electronic cigarette use may impair flow-mediated dilation and may be associated with biomarkers of vascular injury, inflammation and reduced nitric oxide bioavailability [7, 24, 26]. However, findings are not fully uniform; one study in relatively young and healthy adults did not find a significant association with endothelial dysfunction, emphasizing the need for larger and longer prospective cohorts [7, 29].

Mechanistic studies suggest that electronic cigarette exposure can induce oxidative stress, reduce nitric oxide signaling, increase endothelial permeability and activate inflammatory pathways. These effects may occur even when exposure is lower than that produced by conventional cigarettes, suggesting that vaping should not be considered cardiovascularly neutral [6, 10, 26].

Experimental and clinical evidence also suggests that vaping may promote platelet activation and pro-coagulant signaling. Systematic reviews of cardiovascular outcomes indicate increased risk estimates for composite cardiovascular disease among current electronic cigarette users, particularly in dual users and those with previous or current combustible tobacco exposure. Residual confounding remains an important limitation [13, 22, 34].

Neurocognitive and Developmental Considerations. Adolescence is a critical neurodevelopmental period characterized by synaptic pruning, refinement of neural circuitry and maturation of dopaminergic pathways. Nicotine exposure during this period may alter reward processing, attention, impulse control and vulnerability to dependence [4, 9, 11].

Observational data link adolescent electronic cigarette use with subsequent combustible cigarette initiation, mental health symptoms, risk-taking behavior and poorer cognitive outcomes. A recent cohort study reported that adolescent users had lower neurocognitive performance after one year of follow-up, although causality and long-term persistence require further investigation [9, 11, 28].

The high prevalence of flavored and disposable products among adolescents increases the public health importance of these findings. Prevention of youth initiation remains a central priority because early nicotine exposure may establish sustained dependence and increase the probability of dual tobacco exposure [4, 12, 15].

Public Health and Regulatory Aspects. International public health organizations emphasize that electronic cigarettes are not harmless consumer products. Their rapid uptake among adolescents has occurred in parallel with aggressive marketing, attractive flavor profiles, online sales and inconsistent national regulation [4, 12, 23].

Core public health risks include the following:

– nicotine-containing electronic cigarettes are highly addictive;

– device emissions contain toxic and potentially carcinogenic substances that contribute to respiratory and cardiovascular harm;

– adolescent nicotine exposure may impair brain development and learning capacity;

– aerosol emissions may pose risks not only to users but also to bystanders.

Recommended regulatory measures include bans or strict controls on flavorings, limitations on nicotine concentration, product design oversight, taxation, restrictions on advertising and sponsorship, and explicit prohibition of promotional activities targeting children, adolescents and non-smokers [4, 12, 23].

Regarding tobacco cessation, population-level evidence is mixed. Electronic cigarettes may support cessation in some adult smokers under controlled circumstances, but in real-world settings they often supplement rather than replace combustible cigarettes [21]. Therefore, their use should be clinically supervised and should not be promoted to adolescents, pregnant women or non-smokers [2, 16, 19].

Conclusions

1. Electronic cigarette aerosols contain a complex mixture of potentially harmful substances, including nicotine, solvents, flavoring chemicals, carbonyl compounds, volatile organic compounds and heavy metals. Thermal degradation during device operation further increases toxic exposure.

2. Experimental and clinical evidence demonstrates that vaping induces oxidative stress, promotes pro-inflammatory signaling, disrupts immune homeostasis and impairs epithelial and endothelial barrier integrity.

3. On the respiratory level, electronic cigarette use is associated with vaping-related lung injury, airway inflammation, increased respiratory symptoms, early decline in lung function and potential exacerbation of asthma and other chronic pulmonary conditions.

4. Cardiovascular studies indicate that vaping can trigger acute hemodynamic stress, endothelial dysfunction, reduced nitric oxide bioavailability, increased arterial stiffness and activation of inflammatory and pro-thrombotic pathways.

5. In adolescents and young adults, electronic cigarette use presents additional concerns related to neurocognitive development, mental health vulnerability and sustained nicotine dependence.

6. From a public health perspective, aggressive marketing, attractive flavor profiles and regulatory gaps have facilitated widespread uptake among children and adolescents, supporting the need for comprehensive control measures.

7. Current evidence does not support the perception of electronic cigarettes as harmless or universally safe smoking-cessation tools. Their use should be limited, particularly among youth, pregnant women and individuals with existing cardiopulmonary disease.

Prospects for further research. Further prospective research is required to clarify long-term cardiopulmonary risks, especially early endothelial dysfunction, subclinical lung injury and biomarker-defined susceptibility in young exclusive electronic cigarette users. Future studies should combine non-invasive vascular testing, spirometry and circulating biomarkers of endothelial and lung injury.

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