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ELECTRONIC CIGARETTES AND CARDIOVASCULAR HEALTH: CURRENT EVIDENCE AND POLICY IN POLAND

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ABSTRACT

The increasing prevalence of electronic cigarette (e-cigarette) use during the past decade has generated significant concern among public health professionals. Although e-cigarettes are often marketed as safer alternatives to conventional tobacco products, emerging evidence demonstrates that aerosolized compounds produced during vaping may exert adverse effects on cardiovascular health. This review aims to summarize the current understanding of how substances released during vaping affect the cardiovascular system, with an emphasis on molecular mechanisms, clinical findings, and epidemiological data. Additionally, the paper examines the regulatory environment in Poland and compares it to broader European Union (EU) legislation. Despite the absence of combustion-related toxins, e-cigarette aerosols contain nicotine, aldehydes, volatile organic compounds, and heavy metals, all of which can induce oxidative stress, inflammation, and endothelial dysfunction. The available evidence indicates that long-term e-cigarette use is associated with increased cardiovascular risk. Therefore, comprehensive regulation, public education, and ongoing research are essential to mitigate the potential health burden associated with vaping.

KEYWORDS

Electronic Cigarettes, Cardiovascular Risk, Vaping, Nicotine

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Introduction

Over the past decade, electronic cigarettes have emerged as widely used nicotine delivery systems. Initially introduced as tools to support smoking cessation, e-cigarettes have rapidly gained popularity across all age groups, particularly among adolescents and young adults. The absence of tobacco combustion has led to the perception that vaping is a safe or low-risk behavior. However, recent studies have demonstrated that e-cigarette aerosols contain numerous harmful substances capable of inducing cardiovascular, pulmonary, and systemic damage (Benowitz & Fraiman, 2017; Skotsimara et al., 2019).

Cardiovascular diseases (CVDs) remain the leading cause of morbidity and mortality worldwide and are a major public health concern in Poland (World Health Organization [WHO], 2023). Any factor capable of impairing vascular integrity or cardiac function warrants attention. While e-cigarettes produce fewer combustion byproducts compared to traditional cigarettes, they introduce different classes of toxicants - such as aldehydes, reactive oxygen species, and metal nanoparticles - that contribute to oxidative stress and vascular inflammation (Ambrose & Barua, 2004; Fetterman et al., 2018).

The prevalence of e-cigarette use in Poland has grown substantially since the early 2010s. Young users, drawn by flavored e-liquids and the perception of reduced harm, represent the fastest-growing demographic. Early nicotine exposure not only promotes addiction but may also interfere with cardiovascular and neurological development. Although Poland has implemented laws prohibiting e-cigarette sales to minors and banning advertising, enforcement remains inconsistent, and regulatory gaps persist (Ministry of Health of the Republic of Poland, 2025).

This review presents a synthesis of current evidence regarding the cardiovascular effects of e-cigarette use and evaluates the adequacy of Polish regulations in the context of EU policy. The findings underscore the necessity of stronger preventive measures and evidence-based policy interventions.

Methodology: This paper reviewing clinical data from trials conducted in 1999-2024 linked to nicotine use, electronic and conventional cigarettes evaluation in cardiovascular risk among users. Another source of dates are law regulations in Poland and other European Union countries according to the use of tobacco and e-cigarettes market.

Aim of the Study

The objectives of this review are to:

1. Examine the composition and chemical properties of e-cigarette aerosols and their relevance to cardiovascular toxicity.
2. Summarize molecular and physiological mechanisms through which e-cigarette exposure affects cardiovascular function.
3. Evaluate clinical and epidemiological evidence linking e-cigarette use with cardiovascular outcomes.
4. Analyze Polish regulations related to e-cigarettes and compare them with EU directives.
5. Propose recommendations to reduce the cardiovascular risks associated with vaping.

1.1 Mechanism of Action and Composition of E-Cigarette Aerosols

Electronic cigarettes are battery-powered devices designed to heat an e-liquid composed primarily of propylene glycol, vegetable glycerin, nicotine, and flavoring agents (Strongin, 2019). When heated, these components undergo thermal degradation, producing a complex aerosol of ultrafine particles that can reach deep into the respiratory tract and enter systemic circulation.

The heating process generates aldehydes such as formaldehyde, acetaldehyde, and acrolein - compounds known for their cytotoxic and pro-inflammatory effects. The amount of these chemicals depends on device voltage, coil material, and puff duration (Sleiman et al., 2016). Moreover, metal elements from the heating coil, including nickel, chromium, and lead, may leach into the vapor, contributing to heavy metal exposure (Williams et al., 2013; Lee et al., 2019).

Nicotine, the primary pharmacologically active compound in most e-liquids, exerts sympathomimetic effects through stimulation of nicotinic acetylcholine receptors. This results in the release of adrenaline and noradrenaline, leading to transient increases in heart rate, blood pressure, and myocardial contractility (Benowitz & Fraiman, 2017). Repeated exposure to nicotine promotes vascular remodeling, increases arterial stiffness, and contributes to hypertension (Carnevale et al., 2016).

Flavoring chemicals present an additional source of toxicity. Substances such as diacetyl and cinnamaldehyde, commonly used in flavored e-liquids, have been shown to disrupt endothelial integrity and impair nitric oxide production (Fetterman et al., 2018). Although these compounds are considered safe for ingestion, their inhalation at high temperatures produces distinct metabolic and toxicological effects.

Taken together, these data indicate that even though e-cigarettes eliminate tar and carbon monoxide exposure, they introduce novel chemical hazards that may adversely affect cardiovascular health.

1.2 Molecular and Physiological Effects on the Cardiovascular System

1.2.1. Nicotine

Nicotine functions as a potent sympathomimetic compound that triggers activation of nicotinic acetylcholine receptors in the central and peripheral nervous systems. This activation stimulates catecholamine release from the adrenal medulla and sympathetic nerve terminals, producing transient tachycardia, increased myocardial contractility, and elevated systemic blood pressure (Benowitz, 2009). Recurrent nicotine exposure sustains this adrenergic stimulation, ultimately leading to structural and functional remodeling of the cardiovascular system. Chronic sympathetic overactivity has been implicated in endothelial dysfunction, arterial stiffening, and increased left ventricular afterload, predisposing individuals to hypertension and ischemic heart disease (Boas et al., 2020).

On the molecular level, nicotine promotes the expression of genes responsible for extracellular matrix deposition, including those encoding collagen types I and III, while downregulating elastin synthesis (Lee et al., 2019). The resulting imbalance reduces arterial compliance and promotes fibrotic remodeling of the vessel wall. These structural changes are further amplified by oxidative stress induced through mitochondrial dysfunction and activation of NADPH oxidase pathways. Nicotine-mediated production of reactive oxygen species (ROS) diminishes nitric oxide (NO) bioavailability by directly reacting with NO to form peroxynitrite, an oxidant that damages endothelial proteins and lipids (Uchida, 1999). Consequently, endothelium-dependent vasodilation is impaired, a first step in early atherogenesis.

1.2.2. Aldehydes and their proinflammatory potent

Aldehydes produced during the heating of e-liquids particularly acrolein, formaldehyde, and acetaldehyde exert additional toxicological effects on vascular tissue. Acrolein reacts with cysteine residues on cellular proteins, leading to adduct formation and loss of enzymatic function. This process disrupts mitochondrial respiration and augments lipid peroxidation, perpetuating oxidative injury within endothelial cells (Carnevale et al., 2016). Moreover, aldehydes modulate signaling through nuclear factor κB (NF-κB) and activator protein-1 (AP-1), promoting transcription of pro-inflammatory cytokines such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6). The resulting chronic low-grade inflammation favors monocyte adhesion and migration into the subendothelial space, where lipid accumulation and foam-cell formation occur - key stages in the development of atherosclerotic plaques.

1.2.3 Heavy metals induce oxidative stress

Heavy metals, including nickel, cadmium, lead, and chromium, have been consistently detected in e-cigarette aerosols and condensates (Simons et al., 2022). These elements catalyze Fenton-type reactions that generate hydroxyl radicals, one of the most damaging species of ROS. Even at low concentrations, cadmium interferes with calcium signaling and endothelial nitric oxide synthase (eNOS) expression, thereby impairing vascular relaxation. Nickel exposure activates the NLRP3 inflammasome complex, triggering release of IL-1β and IL-18 and amplifying systemic inflammatory responses. Lead, in turn, disrupts heme synthesis and mitochondrial energy metabolism, leading to endothelial apoptosis and microvascular rarefaction. Collectively, these interactions establish a molecular environment conducive to vascular injury, thrombogenesis, and progressive atherosclerosis.

2.1 Patogenesis of endothelial dysfunction due to vaporization

2.1.1 Protrombotic activity

Animal studies have corroborated these cellular mechanisms. Rodent models chronically exposed to e-cigarette vapor demonstrate endothelial denudation, increased arterial wall thickness, and impaired baroreflex sensitivity (Lee et al., 2019). Electron microscopy reveals mitochondrial swelling and disruption of cristae architecture in cardiac myocytes, consistent with oxidative stress-induced injury. Furthermore, both nicotine and aldehydes potentiate platelet activation by enhancing expression of P-selectin and glycoprotein IIb/IIIa on platelet surfaces, increasing the risk of thrombus formation (Carnevale et al., 2016). The resulting pro-thrombotic mechanisms activation contributes to acute cardiovascular events such as myocardial infarction and ischemic stroke.

2.1.2 Impaired lipids metabolism

Emerging evidence also implicates vaping in dysregulation of endothelial progenitor cells (EPCs), which play a critical role in vascular repair. Reduced EPC counts and functional impairment have been observed in habitual e-cigarette users, mirroring changes seen in traditional smokers. This reduction compromises vascular regenerative capacity and may accelerate the progression of atherosclerotic changes(Skotsimara et al., 2019). At the biochemical level, prolonged exposure to e-cigarette aerosols alters lipid metabolism, increasing circulating low-density lipoprotein (LDL) oxidation and decreasing high-density lipoprotein (HDL) functionality, thereby enhancing atherogenic potential (Benowitz & Fraiman, 2017).

These findings collectively illustrate that e-cigarette aerosols induce multifactorial cardiovascular toxicity through oxidative, inflammatory, and fibrotic pathways. Although the absence of tar and carbon monoxide distinguishes vaping from traditional smoking, the alternative toxicants it introduces may evoke comparable, if not subtler, long-term cardiovascular risks. Continued molecular investigations are needed to delineate dose response relationships, synergistic interactions between aerosol constituents, and potential genetic susceptibilities that modulate individual risk profiles.

3. Clinical and Epidemiological Evidence

Clinical studies consistently report acute cardiovascular responses following e-cigarette use. Short-term exposure increases heart rate, systolic blood pressure, and arterial stiffness, comparable to effects observed with traditional cigarette smoking (Boas et al., 2020; Skotsimara et al., 2019). Endothelial function, measured by flow-mediated dilation (FMD), is significantly reduced following vaping, suggesting transient vascular impairment (Carnevale et al., 2016).

The Population Assessment of Tobacco and Health (PATH) study in the United States revealed that e-cigarette users had a 1.8-fold higher risk of myocardial infarction compared to non-users, independent of other risk factors (Alzahrani et al., 2018). Furthermore, dual users - those who smoke both traditional and electronic cigarettes exhibited the greatest risk of cardiovascular events, including myocardial infarction and stroke (Ndunda & Muutu, 2019).

While certain studies report temporary cardiovascular improvements among smokers who fully transition to e-cigarettes, these benefits are often short-lived and offset by prolonged nicotine exposure (Benowitz & Fraiman, 2017). Long-term epidemiological data remain limited, but existing evidence indicates that e-cigarette use contributes to elevated cardiovascular morbidity, particularly among dual users and chronic vapers.

4. Regulatory Framework and Public Health Policy in Poland and the European Union

Poland regulates e-cigarettes under the Act on Protection of Health Against the Consequences of Tobacco Use (2016), which aligns with the EU Tobacco Products Directive (2014/40/EU). This legislation prohibits the sale of e-cigarettes to individuals under 18 years of age, restricts advertising and sponsorship, and mandates clear health warnings on packaging (Ministry of Health of the Republic of Poland, 2025).

Despite these provisions, enforcement remains inconsistent. Online sales and marketing frequently circumvent age verification requirements. Additionally, non-nicotine liquids often escape regulatory scrutiny, despite containing potentially hazardous chemicals.

Comparatively, several EU member states enforce stricter measures. Finland, Hungary, and Denmark restrict flavored e-liquids, permitting only tobacco-flavored variants. Germany and France impose comprehensive advertising bans and levy taxes on e-cigarettes proportional to nicotine concentration (European Commission, 2024). In contrast, Poland's regulatory system lacks uniform monitoring mechanisms and taxation policies, limiting the effectiveness of its public health efforts.To strengthen control and minimize harm, future policy initiatives should emphasize stricter regulation of flavorings, consistent enforcement of age restrictions, public education campaigns, and systematic monitoring of market trends.

5. Discussion and Public Health Implications

The convergence of experimental, clinical, and epidemiological data demonstrates that e-cigarette use is not a benign alternative to tobacco smoking. While vaping eliminates combustion-related carcinogens, its aerosols deliver a complex chemical mixture that exerts measurable adverse cardiovascular effects. Nicotine, aldehydes, volatile organic compounds, and heavy metals act synergistically to induce sympathetic activation, endothelial dysfunction, and chronic inflammation - three central mechanisms in the pathogenesis of cardiovascular disease (Benowitz & Fraiman, 2017; Boas et al., 2020).

One of the most pressing concerns is the widespread adoption of nicotine salt formulations, which increase nicotine bioavailability while minimizing throat irritation. These formulations enable higher plasma nicotine levels than those achievable with conventional free-base nicotine solutions. Sustained exposure maintains constant adrenergic stimulation, prolongs oxidative stress, and exacerbates endothelial injury. Over time, such physiological perturbations accumulate to produce clinically significant hypertension and atherosclerotic vascular disease (Carnevale et al., 2016).

From a population-health perspective, e-cigarette marketing and accessibility have shifted nicotine use toward younger demographics. Adolescents and young adults, whose cardiovascular and neurological systems are still developing, face unique vulnerabilities. Early initiation increases the probability of long-term dependence and magnifies lifetime exposure to nicotine and its cardiovascular effects. Moreover, the normalization of vaping behavior undermines the social stigma previously associated with smoking, reversing decades of successful tobacco-control progress (World Health Organization [WHO], 2023).

The concept of harm reduction, frequently invoked by the vaping industry, remains controversial. Although some evidence suggests that smokers who completely switch to e-cigarettes may experience short-term respiratory or vascular improvements, these benefits are partial and transient. Dual use - simultaneous consumption of traditional and electronic cigarettes eliminates potential advantages and in some cases heightens risk because of additive exposure to toxins from both sources (Alzahrani et al., 2018; Ndunda & Muutu, 2019). Consequently, public health messaging must emphasize that e-cigarettes are less harmful only relative to combustible tobacco, not inherently safe.

In Poland, where the prevalence of vaping among adolescents has risen sharply, existing legal restrictions appear insufficient. The enforcement of age-limit regulations and advertising bans remains

inconsistent, particularly in online markets. Additionally, non-nicotine liquids often marketed as "safe" alternatives may still contain hazardous chemicals that fall outside the current regulatory framework. National strategies should therefore expand regulatory oversight to include all aerosol-producing nicotine and non-nicotine devices.

Effective prevention will require a multi-pronged approach combining legislation, education, and healthcare intervention. Educational campaigns targeted at schools and universities can help dispel misconceptions about vaping safety. Integrating e-cigarette screening questions into routine medical assessments would allow healthcare professionals to identify at-risk individuals and provide cessation counseling early. Partnerships between public-health institutions and social-media platforms could reduce exposure of minors to targeted marketing, a proven driver of initiation.

Internationally, lessons can be drawn from EU member states that have adopted stringent regulatory frameworks. For instance, Finland and Denmark have successfully reduced youth vaping through comprehensive flavor bans and increased taxation (European Commission, 2024). Poland could adapt similar strategies while maintaining alignment with EU directives. Furthermore, investment in independent, longitudinal research is essential to provide policymakers with reliable data on the chronic cardiovascular effects of vaping. Without such evidence, regulatory efforts risk lagging behind technological innovation in the e-cigarette industry.

Future studies should also explore sex-specific and genetic differences in susceptibility to vaping-related cardiovascular injury. Preliminary evidence indicates that estrogen status, oxidative enzyme polymorphisms, and pre-existing metabolic disorders may modulate the severity of vascular responses to e-cigarette aerosols. Understanding these interactions will enable precision-based prevention and therapeutic strategies.

Finally, from a global health standpoint, e-cigarettes exemplify the challenges of regulating emerging nicotine technologies that evolve faster than legislation. Effective control must strike a balance between supporting harm reduction for established smokers and preventing initiation among youth and non-smokers. A robust surveillance system, transparent product reporting, and continuous risk assessment should become integral components of national tobacco-control programs. The ultimate public-health goal remains unchanged: to minimize population exposure to nicotine and its associated cardiovascular harms.

To strengthen control and minimize harm, future policy initiatives should emphasize stricter regulation of flavorings, consistent enforcement of age restrictions, public education campaigns, and systematic monitoring of market trends.

6. Conclusions

E-cigarettes have introduced a new dimension to nicotine consumption, offering perceived harm reduction but introducing unique health risks. The evidence summarized in this review demonstrates that e-cigarette aerosols contain numerous substances such as nicotine, aldehydes, and heavy metals that negatively influence cardiovascular health through oxidative stress, endothelial dysfunction, and inflammation.

Clinical and epidemiological findings confirm that e-cigarette use is associated with increased cardiovascular risk, particularly among dual users. In Poland, existing regulations provide a framework for control, yet enforcement gaps and limited oversight reduce their effectiveness. Stronger regulatory policies, targeted education, and sustained research are required to mitigate these emerging health threats.

Although e-cigarettes may play a role in smoking cessation under medical supervision, their unrestricted availability and appeal to youth create substantial long-term risks. Continued vigilance, scientific inquiry, and evidence-based policymaking are essential to safeguard cardiovascular health in the face of evolving nicotine technologies.

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