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The Impact of E-Cigarettes on Endothelial Function and Cardiovascular Disease Risk

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ABSTRACT

Background: Electronic cigarettes (e-cigarettes) are now the most widely used inhalational nicotine product among adolescents and young adults. Although marketed as safer than combustible tobacco, accumulating evidence indicates that vaping injures the vascular endothelium and elevates cardiovascular disease (CVD) risk.

Aim: This review synthesizes current evidence on how e-cigarette aerosol affects endothelial function and the resulting cardiovascular consequences, with attention to mechanism, population outcomes, and special populations.

Material and methods: PubMed, MEDLINE, Google Scholar, JEHS, and Quality in Sport were searched for peer-reviewed publications between 2015 and 2026, combining "e-cigarette" or "vaping" with terms for endothelial function, flow-mediated dilation, and cardiovascular outcomes.

Results: E-cigarette aerosol carries nicotine, propylene glycol and vegetable glycerin thermolysis products, flavorings, and metals leached from heating coils. Acute use raises pulse-wave velocity by approximately 0.26 m/s and heart rate by about 5 beats/min. Chronic users show flow-mediated dilation reduced to roughly 5% versus 11% in non-users. Mechanisms include eNOS uncoupling, NADPH-oxidase activation, formaldehyde-driven TRPA1 signaling, flavoring-induced inflammation, and increased platelet reactivity. Daily vaping was associated with self-reported myocardial infarction in NHIS data (OR 1.79, 95% CI 1.20–2.66); dual use carried higher CVD odds than exclusive smoking (OR 2.56, 95% CI 2.11–3.11).

Conclusions: E-cigarettes are not cardiovascularly neutral. They impair endothelial function through multiple, partially nicotine-independent pathways and are associated with adverse cardiovascular outcomes, particularly in dual users and youth.

Key words: e-cigarettes, vaping, endothelial dysfunction, cardiovascular disease, nicotine, flow-mediated dilation, oxidative stress.

Introduction

Electronic cigarettes have, in less than two decades, evolved from an obscure inhalational nicotine device into a globally distributed consumer product whose epidemiological footprint now overlaps every major cardiovascular risk demographic. The contemporary device class, ranging from cig-a-likes through refillable tank systems to modern pod-based and disposable products, delivers an aerosol generated by heating a liquid composed principally of propylene glycol (PG), vegetable glycerin (VG), nicotine, and flavorings. While initial public messaging framed these devices as a substantially safer substitute for combustible tobacco, the trajectory of the past decade has produced two outcomes that have reframed the clinical debate: a sustained rise in adolescent uptake among individuals with no prior history of smoking, and a growing volume of mechanistic and clinical evidence that vaping itself is biologically active in the cardiovascular system.

Adolescent uptake is the most visible part of this transition. A Polish cross-sectional survey of 323 high-school students aged 15 to 19 in Krosno found that 59% of respondents had tried e-cigarettes, that 89% described access to the products as easy, and that more than 62% reported regular passive vapor exposure, with most adolescents initiating tobacco-product use through e-cigarettes rather than combustible cigarettes [1]. United States surveillance data from the 2024 National Youth Tobacco Survey reported current e-cigarette use among 5.9% of all middle and high school students, with a year-on-year decline in high-school current use from 10.0% in 2023 to 7.8% in 2024, equivalent to roughly 350,000 fewer high-school users [2]. Despite the recent decline, e-cigarettes remain the single most

commonly used tobacco product in this age group, and disposable, high-nicotine pod devices have largely replaced cig-a-likes as the dominant format.

Concurrently, the cardiovascular research community has issued increasingly explicit warnings. The American Heart Association's 2023 Scientific Statement on the cardiopulmonary impact of electronic cigarettes synthesized acute hemodynamic, autonomic, endothelial, and population-level data and concluded that e-cigarette use is associated with elevated cardiopulmonary risk and that nicotine-driven sympathetic activation, flavoring toxicity, heating-coil metal leaching, and vaping-associated lung injury together constitute a multi-component vascular hazard [3]. This stands against a long-established understanding that combustible cigarettes are among the most potent modifiable cardiovascular toxins, accelerating endothelial dysfunction, atherosclerosis, and thrombosis through nicotine, carbon monoxide, oxidants, and particulate matter [4]. The early position that e-cigarettes were "much less harmful" than combustible cigarettes was already flagged as premature in 2020, and contemporary review data have confirmed that the absence of combustion does not eliminate cardiovascular toxicity [5, 6].

A complex clinical paradox arises when the harm-reduction case for adult smokers attempting cessation is weighed against the cardiovascular signal in adolescents and never-smokers who initiate vaping. This review aims to synthesize the current state of knowledge on the effects of e-cigarettes on endothelial function and on the cardiovascular system more broadly. After a description of device classes and aerosol composition, the manuscript surveys the pharmacology of inhaled nicotine and acute hemodynamic responses, the cellular and molecular mechanisms of endothelial injury, the human and population-level evidence linking vaping to cardiovascular outcomes, e-cigarette or vaping product use-associated lung injury (EVALI) as a systemic cardiovascular stressor, and the implications for special populations including adolescents, young adults, pregnant women, and combustible smokers attempting to switch. The review closes with an integrated discussion and a structured set of conclusions oriented toward clinical and public-health practice.

Materials and methods

The literature search supporting this narrative review was conducted across major scientific databases including PubMed, MEDLINE, Google Scholar, the Cochrane Library, the Journal of Education, Health and Sport (JEHS), and Quality in Sport (QS). The search strategy combined Medical Subject Headings and free-text terms describing the exposure with terms describing the cardiovascular and endothelial outcomes of interest, using the Boolean string ("e-cigarette" OR "electronic cigarette" OR "vaping" OR "ENDS" OR "electronic nicotine delivery system") AND ("endothelial function" OR "endothelial dysfunction" OR "flow-mediated dilation" OR "FMD" OR "nitric oxide" OR "oxidative

stress" OR "arterial stiffness" OR "pulse wave velocity" OR "cardiovascular disease" OR "myocardial infarction" OR "atherosclerosis" OR "platelet" OR "EVALI").

The inclusion criteria were defined as:

1. Peer-reviewed original research, meta-analyses, systematic reviews, and authoritative scientific society statements.
2. Studies published between 2015 and 2026, with priority given to publications from the last six years and seminal earlier works retained where they remain canonical.
3. Articles addressing human populations across age strata, including adolescents, young adults, pregnant women, and patients with established cardiovascular disease, complemented by mechanistic evidence from human cell models, induced pluripotent stem cell–derived endothelial cells, and animal exposure studies.
4. Reports from international health organizations and learned societies, including the American Heart Association, the European Society of Cardiology, the World Health Organization, and the United States Centers for Disease Control and Prevention.

The exclusion criteria included non-peer-reviewed content, opinion pieces without primary evidence synthesis, animal-only studies without translational relevance to human vascular biology, and case reports with fewer than five participants. A total of 28 sources were selected for the final synthesis, comprising 5 mandatory publications from JEHS specified by the brief and 23 additional sources identified through database searching and citation tracking. Quantitative findings were extracted with attention to effect direction, magnitude, and reported confidence intervals, and the evidence was organized thematically into sub-chapters reflecting the major dimensions of the e-cigarette–cardiovascular relationship.

Devices and aerosol composition

Electronic nicotine delivery systems (ENDS) constitute a heterogeneous device class whose only common feature is the production of an inhalable aerosol from a liquid by an electrically driven heating element. The first-generation cig-a-like products that defined the early market in the 2010s have been largely displaced by tank-style devices with refillable reservoirs and adjustable wattage, by closed pod systems that deliver nicotine in protonated salt form, and, since 2020, by single-use disposable devices that are typically prefilled with several milliliters of high-concentration nicotine salt liquid and discarded once the battery or coil is exhausted [6]. An understanding of which compounds the user actually inhales is the necessary starting point for any cardiovascular risk assessment, because the cardiovascular signal is driven by interactions among nicotine, the aerosol carrier, thermal degradation products of that carrier, flavoring chemicals, and trace metals that leach from the heating element.

The principal carriers in commercial e-liquids are PG and VG, mixed in proportions ranging from approximately 30/70 to 70/30, supplemented with nicotine and flavorings. PG and VG are inhaled directly, but at coil temperatures that exceed 200 °C, both molecules undergo partial pyrolysis to a defined set of carbonyl byproducts including formaldehyde, acetaldehyde, and acrolein. A systematic review of carbonyl emissions analyzing data from 32 studies reported that under realistic non-dry-puff conditions, total carbonyl output was 7- to 450-fold lower than from a reference combustible cigarette, but rose sharply with increasing wattage; at 20 W coil power and corresponding temperatures above 300 °C, formaldehyde output ranged from 24.2 to 1599.9 ng per puff, with acrolein and acetaldehyde dominating at the highest temperatures [7]. The dependence of carbonyl emissions on power setting is clinically relevant, because user behavior, coil age, and device design all shift output across this range without any signal to the consumer.

Nicotine in modern devices is increasingly delivered as a salt rather than as freebase. Protonation with an organic acid such as benzoic acid lowers aerosol harshness and allows delivery of much higher nicotine concentrations without the throat irritation associated with freebase nicotine. A randomized crossover pharmacokinetic study comparing nicotine salt at 20 mg/mL and 40 mg/mL with freebase nicotine at 20 mg/mL under standardized inhalation showed that 20 mg/mL salt produced 1.8-fold higher peak serum nicotine and 46% higher area under the curve than equivalent freebase nicotine, and that the 40 mg/mL salt formulation matched the pharmacokinetic profile of a combustible cigarette [8]. The implication is that contemporary pod and disposable devices deliver nicotine doses comparable to combustion at exposure intensities high enough to drive sympathetic activation and sustain dependence, and that pharmacokinetic claims of "lower-dose" nicotine exposure do not generalize across the device class.

Flavorings are the third compositional pillar. Several thousand individual flavor chemicals are in commercial use, with cinnamaldehyde, vanillin, menthol, eugenol, diacetyl, and acetylpyrazine among the most extensively studied. Flavoring chemicals are not pharmacologically inert. In human aortic endothelial cells, low concentrations of these compounds have been shown to induce inflammation and impair stimulated nitric oxide production at exposure levels that are achievable with realistic flavored-product use [9]. The relevance of flavoring biology for vascular function is discussed in detail in section 3.3.4.

Heating-coil metals constitute the fourth and least-controlled component of aerosol composition. A Johns Hopkins study comparing metal concentrations in unopened refill liquid, in tank liquid in contact with the coil, and in inhaled aerosol across 56 e-cigarette users found that the heating coil was the predominant source of metal contamination [10]. Median metal concentrations rose from refill dispenser to tank by 65-fold for nickel, 70-fold for chromium, and 116-fold for lead, and approximately half of aerosol samples exceeded United States Environmental Protection Agency health-based limits for at

least one of nickel, chromium, or lead. Concurrent surveillance has confirmed that the metal-leaching problem has not resolved with newer device generations and may be exacerbated in disposable products.

Table 1 summarizes the dominant chemical classes in e-cigarette aerosol with their cardiovascular relevance and combustible-cigarette comparison where data are available.

Table 1. Principal chemical classes in e-cigarette aerosol, sources, and cardiovascular relevance, drawn from sources cited in the body of this review

Class	**Origin**	**Cardiovascular relevance**	**Comparison to combustible smoke**
Nicotine (freebase or salt)	E-liquid	Sympathetic activation, vasoconstriction, increased heart rate and blood pressure, platelet activation, atherogenesis in animal models	Comparable plasma levels achievable with high-strength salt formulations
Propylene glycol and vegetable glycerin	E-liquid carrier	Direct airway irritation; substrate for thermal carbonyl generation	Absent in combustible smoke
Carbonyls (formaldehyde, acetaldehyde, acrolein)	Pyrolysis of PG/VG, dependent on coil power	Endothelial injury via TRPA1 signaling, oxidative stress	7- to 450-fold lower than combustible smoke under realistic use, rising sharply at high wattage
Flavorings (cinnamaldehyde, vanillin, menthol, diacetyl, eugenol)	E-liquid additives	Induce endothelial inflammation, suppress nitric oxide production, inhibit soluble guanylate cyclase	Largely unique to e-cigarettes
Trace metals (nickel, chromium, lead)	Heating coil and bronze components	Direct vascular toxicity, oxidative stress, atherogenesis	Lead in some disposable devices reportedly exceeds combustible cigarette emissions

Sources: [3,6,7,8,9,10]

Inhaled nicotine pharmacology and acute hemodynamic effects

Nicotine is the central pharmacologically active constituent of e-cigarette aerosol and the dominant proximate driver of acute cardiovascular responses to vaping. Inhaled nicotine binds neuronal-type nicotinic acetylcholine receptors at adrenal chromaffin cells and at sympathetic ganglia, producing rapid release of norepinephrine and epinephrine and a stereotyped sympathetic response characterized by an increase in heart rate, an increase in systemic vascular resistance, an elevation in blood pressure, and an increase in myocardial oxygen demand [11]. In animal models, chronic nicotine exposure independent of combustion accelerates atherosclerotic plaque formation in apolipoprotein E knockout mice, induces endothelial dysfunction, promotes platelet activation, and contributes to arterial stiffness through structural remodeling of the arterial wall [11].

A 2024 systematic review and meta-analysis of nine randomized controlled trials including 370 participants quantified the acute hemodynamic effects of vaping by isolating nicotine-containing aerosol from nicotine-free aerosol [12]. Nicotine-containing e-cigarettes acutely increased pulse wave velocity, the gold-standard measure of arterial stiffness, by a weighted mean difference of 0.26 m/s (95% CI 0.14–0.38, $p < 0.001$) compared with nicotine-free vaping. Augmentation index normalized to a heart rate of 75 beats per minute (AIx75) rose by 4.29 units (95% CI 2.07–6.51), and heart rate increased by 5.06 beats per minute (95% CI 2.13–7.98). The acute reductions in flow-mediated dilation (FMD) and increases in central blood pressure observed in the same trials did not differ significantly between nicotine-containing and nicotine-free aerosol, indicating that some of the immediate vascular effects of vaping are driven by carrier and flavoring components rather than by nicotine alone. The American Heart Association's pooled analysis converged on a similar quantitative range, with acute systolic and diastolic blood pressure increases of approximately 2 mmHg and heart rate increases of approximately 2 beats per minute attributable to nicotine-containing devices [3].

Beyond minute-to-minute hemodynamics, chronic vaping shifts cardiac autonomic balance toward sympathetic predominance. A systematic review of heart rate variability (HRV) and autonomic outcomes synthesized chronic and acute studies and reported that chronic e-cigarette users showed reduced parasympathetic tone, with the high-frequency (vagal) HRV component at 46.5 normalized units versus 57.8 in non-users ($p = 0.04$), an increased low-frequency (sympathetic) component at 52.7 versus 39.9 normalized units ($p = 0.03$), and an elevated low-frequency to high-frequency ratio of 1.37 versus 0.85 ($p = 0.05$) [13]. Notably, this autonomic imbalance was present despite normal resting blood pressure and heart rate, consistent with subclinical sympathetic stress that may translate over time into established hypertension, ventricular arrhythmia susceptibility, and accelerated atherogenesis.

Mechanisms of endothelial injury

The vascular endothelium is the primary interface between circulating blood and the vessel wall and the principal regulator of vascular tone, anticoagulant balance, and leukocyte adhesion. Endothelial dysfunction, defined by impaired endothelium-dependent vasodilation and a shift from a quiescent to a pro-inflammatory and pro-thrombotic phenotype, is the earliest detectable vascular abnormality preceding clinical atherosclerosis and predicts incident cardiovascular events independent of conventional risk factors. The evidence reviewed below indicates that e-cigarette aerosol injures the endothelium through multiple, partially overlapping pathways involving nitric oxide synthase uncoupling, oxidative stress, thermal degradation products, flavoring biology, and platelet activation.

eNOS uncoupling and loss of nitric oxide bioavailability

Endothelial nitric oxide synthase (eNOS) is the dominant source of vascular nitric oxide (NO) in healthy adults and the linchpin of endothelium-dependent vasodilation. In a long-duration murine inhalation study, El-Mahdy and colleagues exposed C57BL/6 mice to e-cigarette vapor with or without nicotine for 16 and 60 weeks and characterized the time course of vascular dysfunction [14]. NADPH oxidase subunit NOX2 expression rose in a time- and nicotine-dependent fashion, while eNOS expression and Akt-dependent eNOS phosphorylation were progressively downregulated. Tetrahydrobiopterin (BH4), an essential eNOS cofactor, was depleted along with its salvage enzyme dihydrofolate reductase, producing the biochemical hallmark of eNOS uncoupling, in which the enzyme generates superoxide rather than NO. Acetylcholine-induced aortic relaxation was impaired and systolic and mean arterial blood pressure rose progressively over the exposure period. Importantly, partial endothelial dysfunction was detectable in animals exposed to nicotine-free aerosol, indicating that carrier and aerosol components contribute independently of nicotine.

Human evidence parallels these animal findings. Mohammadi and colleagues compared chronic e-cigarette users with combustible smokers and never-users and reported brachial artery FMD of 5.3 ± 2.3% in vapers and 6.5 ± 2.8% in smokers versus 10.7 ± 5.2% in non-users, an effect of similar magnitude in the two exposure groups [15]. When sera from these participants were applied to cultured endothelial cells, both groups suppressed VEGF-induced NO release, but only e-cigarette user sera increased microvascular endothelial permeability and elevated circulating ligands of the receptor for advanced glycation end products (S100A8 and HMGB1), pointing to partly distinct mechanisms of endothelial injury between vaping and combustion.

Reactive oxygen species and oxidative stress

Oxidative stress is the principal mechanistic theme connecting the disparate compositional elements of e-cigarette aerosol. Lee and colleagues used human induced pluripotent stem cell–derived endothelial cells (iPSC-ECs) to characterize the cellular response to e-liquids and to vaper sera [16]. Six of six tested flavored e-liquids induced cytotoxicity in iPSC-ECs, with cinnamon-flavored product the most potent, producing reduced cell viability, increased intracellular ROS, increased caspase-3/7 activity, and impaired tube formation and migration. Sera from chronic e-cigarette users elevated ROS in iPSC-ECs, impaired pro-angiogenic properties, and increased inflammatory cytokine expression, with a trend toward macrophage activation. The convergence of in vitro flavoring toxicity and in vivo serum effects supports oxidative stress as the dominant proximate driver of endothelial injury.

A mechanistic human study by Halstead and colleagues focused on young adult vapers and demonstrated that microvascular endothelial dysfunction in this population is mediated by superoxide and shows clear sex differences [17]. Among young adult women who vaped, endothelium-dependent dilation was reduced to 71 ± 13% of maximum cutaneous vascular conductance versus 89 ± 7% in non-vapers (p = 0.002), and NO-dependent dilation was reduced to 50 ± 6% versus 69 ± 11% (p = 0.005). Co-perfusion with the antioxidant tempol restored endothelium-dependent dilation in female vapers, isolating superoxide as the proximate mediator. No significant differences in endothelium-dependent or NO-dependent dilation were observed in male young adult vapers, raising the possibility that female sex confers heightened vascular vulnerability to chronic vaping.

Carrier thermolysis products and TRPA1 signaling

Thermal degradation of PG and VG produces formaldehyde, acetaldehyde, and acrolein, all of which are airway and vascular irritants. Jin and colleagues exposed mice to PG/VG aerosol or to direct formaldehyde or acetaldehyde and characterized the resulting respiratory and vascular injury [18]. PG/VG aerosol caused a 50% drop in respiratory rate and impaired endothelium-dependent aortic relaxation by –61.8 ± 4.2% versus filtered air. Direct formaldehyde exposure in female mice over four days reproduced the aortic endothelial dysfunction caused by PG/VG aerosol, while acetaldehyde at equivalent concentrations did not. Mechanistic follow-up established that this formaldehyde-driven vascular injury requires the transient receptor potential ankyrin-1 (TRPA1) channel, providing a defined molecular target for the carrier-thermolysis component of the cardiovascular response. The relevance of these data is that they isolate a nicotine-independent and flavoring-independent pathway by which e-cigarette aerosol injures the endothelium, undermining the position that a nicotine-free, flavor-free e-liquid would be vascularly safe.

Flavoring chemicals as direct endothelial toxins

Flavorings have emerged as biologically active components of e-cigarette aerosol with the potential to injure endothelial cells at concentrations achievable in users. In the human aortic endothelial

cell study by Fetterman and colleagues, low concentrations (0.001–0.01 mmol/L) of vanillin, menthol, cinnamaldehyde, eugenol, and acetylpyrazine increased interleukin-6 expression and impaired A23187-stimulated NO production, and human menthol smokers showed impaired NO responses compared with non-smokers [9]. The combination of in vitro and ex vivo human evidence raises specific concerns about flavored disposable devices, in which cinnamaldehyde, menthol, and fruit-based flavor blends are heavily marketed to younger users.

Platelet activation and thrombotic risk

Endothelial dysfunction does not act in isolation; it is embedded in a network of pro-thrombotic shifts that include altered platelet biology. Metzen and colleagues compared platelet aggregation by whole-blood aggregometry in vapers, combustible smokers, and non-smokers [19]. Collagen-induced aggregation was significantly higher in vapers (66.6 ± 19.0 area under the curve units) than in non-smokers (52.6 ± 24.0; $p = 0.002$) and than in smokers (49.5 ± 26.1; $p < 0.0001$). Adenosine diphosphate–induced aggregation in vapers (45.3 ± 18.7) also exceeded that in non-smokers (33.2 ± 16.6; $p = 0.001$). The finding that vaping produces enhanced platelet reactivity beyond conventional smoking is striking and points to a pro-thrombotic phenotype that compounds the primary endothelial defect.

Table 2. Mechanisms of e-cigarette–induced endothelial injury, with representative model systems and key findings, drawn from sources cited in the body of this review

Mechanism	Model system	Representative finding
eNOS uncoupling, BH4 depletion	Mouse 16- and 60-week aerosol exposure	Progressive eNOS downregulation, NOX2 upregulation, impaired aortic relaxation [14]
Reduced NO bioavailability and FMD in humans	Cross-sectional human study	Brachial FMD 5.3% in vapers vs 10.7% in non-users [15]
Cytotoxicity and ROS in cellular models	iPSC-derived endothelial cells	Six of six flavored e-liquids increase ROS and impair tube formation [16]

Sex-specific microvascular dysfunction	Young adult human vapers	Endothelium-dependent dilation 71% vs 89%, reversed by tempol in females [17]
Carrier thermolysis–driven vascular injury	Mouse aerosol exposure plus aldehyde challenge	Formaldehyde reproduces PG/VG vascular injury via TRPA1 [18]
Flavoring-induced endothelial inflammation	Human aortic endothelial cells	Cinnamaldehyde, menthol, vanillin increase IL-6 and impair NO production [9]
Enhanced platelet reactivity	Whole-blood aggregometry in human vapers	Collagen-induced aggregation higher in vapers than smokers and non-users [19]

Sources:[9,14,15,16,17,18,19]

Clinical and population-level cardiovascular evidence

Vascular function in young adult vapers

The mechanistic data above predict that chronic vaping should produce detectable arterial dysfunction in young, otherwise-healthy users. Matheson and colleagues tested this prediction in a case-control study comparing 21 young adult vapers aged 21 to 31 years with 21 matched non-users [20]. Regular vapers showed reduced macrovascular FMD and impaired cutaneous microvascular endothelium-dependent and endothelium-independent dilation. Vapers with more than three years of use had significantly worse macro- and microvascular function than shorter-term users, providing the only published dose-response signal in young chronic vapers and confirming that vascular injury accumulates with exposure duration even in the absence of established cardiovascular risk factors.

Population cohort and case-control evidence

Population-level data have steadily moved from cross-sectional signals toward prospective evidence. Berlowitz and colleagues analyzed the Population Assessment of Tobacco and Health (PATH) study, a nationally representative United States cohort of approximately 24,000 adults followed across waves 1 through 5 (2013–2019), and examined the relationship between e-cigarette use and incident self-reported cardiovascular disease defined as myocardial infarction, stroke, heart failure, or other heart conditions [21]. Exclusive e-cigarette use was associated with elevated incident self-reported CVD

versus never-tobacco use, although associations attenuated under full adjustment for combustible-tobacco history; dual users carried the highest risk. Earlier cross-sectional data from the National Health Interview Survey (NHIS) had reported that daily e-cigarette use was independently associated with self-reported myocardial infarction with an odds ratio of 1.79 (95% CI 1.20–2.66, $p = 0.004$), compared with daily combustible smoking at an odds ratio of 2.72 (95% CI 2.29–3.24) [22]. While cross-sectional designs cannot establish temporal precedence and self-report has known limitations, the convergence of the PATH and NHIS data is consistent with a clinically meaningful elevation in cardiovascular events among chronic exclusive vapers and an additive risk profile among dual users.

A 2024 meta-analysis of 20 observational studies including approximately 8.5 million participants directly addressed the additivity question [23]. Dual users (concurrent e-cigarette and combustible cigarette use) carried odds of CVD of 2.56 (95% CI 2.11–3.11) versus never-users, while exclusive e-cigarette users showed odds of 1.24 (95% CI 0.93–1.67), the latter not reaching statistical significance. The asymmetry between dual users and exclusive vapers is the most clinically actionable finding in the population evidence: it indicates that adding vaping to ongoing combustible-cigarette use does not mitigate cardiovascular risk and may worsen it, whereas exclusive switching, while not risk-free, may carry a lower magnitude of harm than continued combustion.

Scientific society synthesis

The American Heart Association's 2023 Scientific Statement integrates the mechanistic and population data and constitutes the current consensus reference for clinicians [3]. The statement reports systematic-review-level estimates of acute systolic and diastolic blood pressure increases of approximately 2 mmHg and heart rate increases of approximately 2 beats per minute with nicotine-containing ENDS use, impaired endothelium-dependent dilation in chronic users, and elevated self-reported coronary heart disease, arrhythmia, and chest pain in cross-sectional cohorts including the Health eHeart cohort of 39,747 participants. The accompanying recommendation framework calls for clinicians to screen for vaping, to avoid endorsing e-cigarettes as a primary cessation strategy in patients with established CVD, and to remain alert to the EVALI presentation reviewed in section 3.5.

Table 3. Selected clinical and population studies of e-cigarettes and cardiovascular outcomes

Study	**Design**	**Headline cardiovascular finding**
Mohammadi et al. 2022 [15]	Three-arm human comparison	FMD 5.3% in vapers vs 10.7% in non-users; serum effects on cultured ECs

Matheson et al. 2024 [20]	Case-control young adults	Reduced macro- and microvascular dilation with >3-year vaping duration
Halstead et al. 2023 [17]	Young adult mechanistic study	Female-predominant superoxide-mediated microvascular dysfunction
Cheraghi et al. 2024 [12]	Meta-analysis of 9 RCTs	Acute PWV +0.26 m/s, AIx75 +4.29, HR +5.06 bpm with nicotine vaping
Garcia et al. 2020 [13]	HRV systematic review	Sympathetic predominance in chronic vapers despite normal resting HR/BP
Metzen et al. 2021 [19]	Aggregometry in vapers, smokers, non-users	Collagen-induced aggregation higher in vapers than smokers and non-users
Berlowitz et al. 2022 [21]	PATH cohort, prospective	Exclusive vaping associated with incident CVD; dual use highest risk
Alzahrani et al. 2018 [22]	NHIS pooled cross-section	Daily vaping OR 1.79 for self-reported MI
Chen et al. 2024 [23]	Meta-analysis 20 studies	Dual use OR 2.56 for CVD; exclusive vaping OR 1.24 (NS)
Rose et al. 2023 [3]	AHA Scientific Statement	Synthesizes mechanism, acute hemodynamics, and population evidence

Sources:[3, 12, 13, 15, 17, 19,20,21,22,23]

EVALI as a systemic cardiovascular stressor

E-cigarette or vaping product use-associated lung injury (EVALI) is the acute pulmonary syndrome that emerged into clinical visibility in the United States during 2019 and that produced more than 2,800 hospitalizations and 68 deaths during the index outbreak [3]. The clinical manifestations described by Hądzlik and colleagues in adolescents include progressive dyspnea, cough, fever, gastrointestinal symptoms, hypoxemia, and bilateral diffuse alveolar opacities on chest imaging,

frequently with respiratory failure requiring mechanical ventilation, and in survivors, a substantial risk of persistent fibrotic pulmonary scarring [24]. Although EVALI is properly classified as a respiratory illness, its cardiovascular relevance derives from three considerations: severe hypoxemia and the acute respiratory distress phenotype place an immediate strain on the right ventricle and the systemic circulation, the systemic inflammatory response associated with EVALI elevates pro-inflammatory cytokines that promote endothelial activation and thrombosis, and the dominant epidemiology of EVALI in adolescents and young adults coincides with the cohort least likely to be screened for cardiovascular risk.

The etiologic agent of the 2019 outbreak was identified by the Centers for Disease Control and Prevention Lung Injury Response Working Group through a case-control analysis of bronchoalveolar lavage (BAL) fluid from 51 EVALI patients across 16 states and 99 healthy controls [25]. Vitamin E acetate, used as a thickening diluent in illicit tetrahydrocannabinol (THC) vape cartridges, was detected in BAL fluid of 48 of 51 (94%) EVALI patients and in none of 18 healthy exclusive nicotine e-cigarette users. THC or its metabolites were detected or reported in 47 of 50 (94%) cases. The mechanistic explanation proposed in subsequent work is that vitamin E acetate undergoes pyrolysis during vaping to release pulmonary-toxic ketene, an electrophile that injures airway epithelium and provokes an alveolar–capillary inflammatory response [24]. The clinical implication is that EVALI represents the acute end of the cardiopulmonary spectrum of e-cigarette injury and that history-taking in any patient presenting with otherwise-unexplained respiratory or cardiovascular decompensation should specifically inquire about THC- or non-nicotine-vaping products.

Special populations

Adolescents and young adults

The cardiovascular implications of vaping are most consequential in users with the longest expected exposure horizon. Polish data from the Sejnowska survey establish a 59% lifetime prevalence among high-school students with high passive-vapor exposure, while United States surveillance reports current use among 7.8% of high-school students in 2024 with disposable, high-nicotine pod devices the dominant format [1, 2]. Cardiovascular evidence specific to this age group includes the dose-dependent vascular dysfunction in young adult vapers reported by Matheson and colleagues, and the female-predominant superoxide-mediated microvascular dysfunction documented by Halstead and colleagues, both of which indicate that subclinical cardiovascular injury is detectable in healthy users in their twenties [17, 20]. The convergence of high prevalence, early subclinical injury, and decades of expected continued exposure underscores the need for effective primary prevention.

Pregnancy and fetal cardiovascular development

Vaping during pregnancy presents a distinct biological scenario in which inhaled nicotine and aerosol components cross the placenta and may influence fetal cardiovascular development. A 2026 scoping review using Joanna Briggs Institute methodology synthesized eight studies on perinatal e-cigarette exposure and offspring outcomes through age three [26]. Animal data demonstrate that maternal e-cigarette exposure during gestation produces abnormal vascular development in three- and seven-month-old rat offspring, with impaired aortic relaxation, histologic vascular changes, and transcriptional shifts mirroring the impaired distal lung development seen in maternally vaped embryos. Direct human cardiovascular outcome data in offspring of vaping mothers remain sparse, but the absence of evidence for safety should not be interpreted as evidence for absence of harm; the precautionary recommendation across major societies is to advise against any nicotine product use during pregnancy.

Combustible smokers attempting to switch

The harm-reduction case for adult smokers is the strongest argument advanced for e-cigarettes and rests primarily on cessation efficacy rather than on vascular safety. The 2024 Cochrane Living Systematic Review of 88 studies including 47 randomized controlled trials and 27,235 participants reported that nicotine-containing e-cigarettes outperform nicotine replacement therapy for smoking cessation, with a relative risk of 1.59 (95% CI 1.29–1.93) and high-certainty evidence of approximately four additional quitters per 100 (95% CI 2–6) [27]. The Cochrane authors are explicit that quit-rate data do not equal cardiovascular safety data and that the long-term cardiopulmonary risk profile of exclusive vaping remains incompletely characterized. The clinical implication is that for an adult smoker with established combustible-tobacco dependence and contraindications or failures with first-line cessation therapies, exclusive switching to a regulated e-cigarette may produce a net reduction in measured biomarkers of harm exposure, while the long-term cardiovascular trajectory of that switch remains under active study.

Toward a synthesized cardiovascular comparison with combustible smoking

The defining challenge in synthesizing the e-cigarette evidence is that the comparator question is asymmetric across populations. For an adolescent or never-smoker, the relevant comparator is no inhalational nicotine exposure at all, and the evidence reviewed above indicates a substantial cardiovascular signal that did not exist in the unexposed state. For an adult exclusive combustible smoker considering switching, the relevant comparator is continued combustion, against which vaping carries a different and probably lower toxicant load while still producing endothelial dysfunction, sympathetic activation, and platelet reactivity that approach those seen in active smokers [4–6]. For dual

users, the population evidence indicates that adding vaping to combustible-cigarette use does not mitigate cardiovascular risk and may worsen it, the most internally consistent inference from the meta-analytic data [23]. The Tomaszewski systematic review of e-cigarette health effects across cardiovascular, respiratory, and metabolic systems converges on this multi-pronged risk picture, emphasizing both acute and chronic cardiovascular impacts and special vulnerability among individuals with pre-existing conditions [6]. The combination of these positions defines the contours of the contemporary clinical message: e-cigarettes are not cardiovascularly neutral, dual use is the worst trajectory, exclusive switching is preferable to continued combustion, and exclusive vaping in never-smokers is a net new cardiovascular exposure that should not be normalized.

Discussion

The integrated evidence from the past decade of e-cigarette research has dismantled the early claim that vaping is cardiovascularly inert. Mechanistic studies in human aortic endothelial cells, induced pluripotent stem cell–derived endothelial cells, murine aerosol exposure models, and direct human FMD measurements describe a coherent injury pathway in which e-cigarette aerosol drives reactive oxygen species generation, eNOS uncoupling and loss of NO bioavailability, formaldehyde-mediated TRPA1 activation, flavoring-induced endothelial inflammation, and enhanced platelet reactivity. This mechanistic coherence is reinforced by the convergence of acute hemodynamic data showing nicotine-driven elevations in heart rate, arterial stiffness, and central hemodynamics; chronic functional data showing FMD reductions in young adult vapers comparable in magnitude to those in combustible smokers; and population-level data linking exclusive and dual vaping to incident cardiovascular events. The vascular injury is therefore neither hypothetical nor confined to extreme exposure conditions; it is detectable at typical use intensities in young, otherwise-healthy individuals.

A second integrative theme is that nicotine, while central, is not solely responsible for the cardiovascular signal. The animal and human evidence that nicotine-free e-cigarette aerosol still produces endothelial dysfunction, that PG/VG carrier thermolysis releases formaldehyde with documented aortic effects, and that flavoring chemicals injure endothelial cells at realistic exposure concentrations together undermine the position that a hypothetical nicotine-free, flavor-free product would be a cardiovascularly safe consumer good [3, 14, 18]. This finding is clinically consequential because it implies that regulatory measures aimed at reducing nicotine concentration in e-liquids, while addressing the addictive and sympathetic-activation dimension, would not by themselves eliminate the vascular hazard. The European Society of Cardiology consensus that frames nicotine as a direct cardiovascular toxin in its own right and that addresses the limits of harm-reduction framing under the revised European Tobacco Taxation Directive represents the most explicit recognition of this position to date [28].

A third theme is the asymmetry of risk across user populations. Dual users, exclusive adolescent vapers, vapers with pre-existing CVD, and pregnant women constitute the populations of greatest concern. Dual

use carries the highest measured CVD odds, and the meta-analytic evidence indicates that adding vaping to combustible-cigarette use does not produce a net cardiovascular benefit [23]. Adolescents and young adults face a long expected exposure horizon over which subclinical vascular injury is likely to compound into established disease, an effect already detectable in users with three or more years of vaping [20]. The harm-reduction argument retains validity for the narrow population of adult exclusive smokers who have failed conventional cessation and who switch completely to a regulated e-cigarette, with corresponding reductions in biomarkers of harm exposure documented by Cochrane secondary analyses [27]. The clinical message therefore varies by population, and the integration of pharmacological cessation aids with behavioral and policy interventions remains the cornerstone of practice.

A fourth theme is the inadequacy of current regulatory approaches given the speed of product evolution. The dominant device class on the contemporary market, the high-concentration nicotine-salt disposable, did not exist when the first major regulatory frameworks for e-cigarettes were drafted, and current evidence indicates that some of these products produce per-day lead emissions exceeding combustible cigarettes and deliver pharmacokinetic peaks comparable to combustion [8, 10]. The flavoring landscape, with thousands of compounds in commercial use, exceeds the throughput of any feasible product-by-product safety review, and EVALI demonstrated that supply-chain anomalies in unregulated cartridges can produce mass-casualty pulmonary injury. The integration of pharmacological interventions with lifestyle modifications and effective regulatory oversight is therefore non-negotiable for any meaningful reduction in the population-level cardiovascular footprint of vaping.

The principal limitations of the current evidence base reflect the recency of widespread e-cigarette use. Long-term prospective data on hard cardiovascular endpoints in exclusive vapers remain limited, the heterogeneity of devices and e-liquids complicates cross-study comparison, and the most-affected adolescent populations entered chronic vaping less than a decade ago, leaving substantial uncertainty about decade- and lifetime-scale outcomes. Nonetheless, the convergence of mechanistic, hemodynamic, vascular, and population evidence is sufficient to justify a clinical posture that treats e-cigarettes as a non-trivial cardiovascular exposure rather than as a neutral consumer product, and to support continued investment in primary prevention, screening for vaping at routine clinical encounters, and longitudinal surveillance of users.

Conclusions

Electronic cigarette aerosol injures the vascular endothelium through multiple mechanisms involving eNOS uncoupling, reactive oxygen species generation, formaldehyde-mediated TRPA1

activation, flavoring-induced inflammation, and enhanced platelet reactivity, producing measurable endothelial dysfunction in users with no other cardiovascular risk factors. Acute vaping with nicotine-containing aerosol raises pulse wave velocity, arterial augmentation index, heart rate, and central blood pressure, with the magnitude of these responses approaching that of combustible cigarettes for some endpoints. Chronic vaping is associated with reduced flow-mediated dilation in young adult users comparable to that observed in conventional smokers, with a dose-response signal apparent after three or more years of regular use. Population-level evidence from PATH, NHIS, and meta-analyses links e-cigarette use to incident self-reported cardiovascular disease, with dual users carrying the highest measured risk and exclusive vapers showing a smaller but non-trivial signal. The harm-reduction case for e-cigarettes is supported for adult exclusive combustible smokers attempting cessation under regulated conditions, but it does not extend to adolescents, never-smokers, dual users, pregnant women, or patients with established cardiovascular disease, in whom vaping should be discouraged. Effective policy responses require a recognition that nicotine itself is a cardiovascular toxin, that flavor and carrier components produce nicotine-independent vascular injury, and that the integration of clinical screening, regulatory oversight, and structured primary-prevention messaging is the cornerstone of future risk reduction.

Disclosure

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References:

1. Sejnowska, N., Dobko, K., Frącz, G., Glac, A., Kuliga, K., Musz, K., Socha, A., Surowiec, A., Śnieżek, K., & Zając, A. (2023). Evaluation of the frequency of e-cigarette usage among high school students and factors that influence vaping: Adolescents' knowledge of chemical composition of e-cigarettes and side effects. *Journal of Education, Health and Sport, 16*(1), 88–105. https://doi.org/10.12775/JEHS.2023.16.01.010
2. Jamal, A., Park-Lee, E., Birdsey, J., West, A., Cornelius, M., Cooper, M. R., Cowan, H., Wang, J., Sawdey, M. D., Cullen, K. A., & Navon, L. (2024). Tobacco product use among middle and high school students — National Youth Tobacco Survey, United States, 2024. *MMWR Morbidity and Mortality Weekly Report*, *73*(41), 917–924. https://doi.org/10.15585/mmwr.mm7341a2
3. Rose, J. J., Krishnan-Sarin, S., Exil, V. J., Hamburg, N. M., Fetterman, J. L., Ichinose, F., Perez-Pinzon, M. A., Rezk-Hanna, M., Williamson, E., et al. (2023). Cardiopulmonary impact of electronic cigarettes and vaping products: A scientific statement from the American Heart Association. *Circulation, 148*(8), 703–728. https://doi.org/10.1161/CIR.0000000000001160
4. Janowska, K., Szydziak, J., Hrapkowicz, A., Dąbkowska, D., Szeidl, O., Rehan, D., & Mioskowska, A. (2025). The effects of cigarette smoking on the cardiovascular system: Mechanisms of damage, risk factors, and prevention strategies. *Journal of Education, Health and Sport, 77*, 56630. https://doi.org/10.12775/JEHS.2025.77.56630
5. Romaniuk, M., Suswał, K., Komisarczuk, M., Tuszyńska, W., Krupa, K., Zygmunt, W., & Milanowski, J. (2020). E-cigarettes, is this alternative safe? *Journal of Education, Health and Sport, 10*(8), 313–321. https://doi.org/10.12775/JEHS.2020.10.08.037
6. Tomaszewski, J., Borowska-Łygan, M., Rejmak, R. W., Karamus, K., Biłogras, J., Strużek, K., & Urban, W. (2025). E-cigarette health effects on major body systems: A comprehensive review of cardiovascular, respiratory, and diabetic implications. *Journal of Education, Health and Sport, 80*, 59621. https://doi.org/10.12775/JEHS.2025.80.59621

7. Farsalinos, K. E., & Gillman, G. (2018). Carbonyl emissions in e-cigarette aerosol: A systematic review and methodological considerations. *Frontiers in Physiology, 8*, 1119. https://doi.org/10.3389/fphys.2017.01119
8. Christen, S. E., Hermann, L., Bekka, E., Vonwyl, C., Hammann, F., van der Velpen, V., et al. (2024). Pharmacokinetics and pharmacodynamics of inhaled nicotine salt and free-base using an e-cigarette: A randomized crossover study. *Nicotine & Tobacco Research, 26*(10), 1313–1321. https://doi.org/10.1093/ntr/ntae074
9. Fetterman, J. L., Weisbrod, R. M., Feng, B., Bastin, R., Tuttle, S. T., Holbrook, M., et al. (2018). Flavorings in tobacco products induce endothelial cell dysfunction. *Arteriosclerosis, Thrombosis, and Vascular Biology*, *38*(7), 1607–1615. https://doi.org/10.1161/ATVBAHA.118.311156
10. Olmedo, P., Goessler, W., Tanda, S., Grau-Perez, M., Jarmul, S., Aherrera, A., et al. (2018). Metal concentrations in e-cigarette liquid and aerosol samples: The contribution of metallic coils. *Environmental Health Perspectives, 126*(2), 027010. https://doi.org/10.1289/EHP2175
11. Espinoza-Derout, J., Shao, X. M., Lao, C. J., Hasan, K. M., Rivera, J. C., Jordan, M. C., et al. (2022). Electronic cigarette use and the risk of cardiovascular diseases. *Frontiers in Cardiovascular Medicine, 9*, 879726. https://doi.org/10.3389/fcvm.2022.879726
12. Cheraghi, M., Amiri, M., Omidi, F., Shahidi Bonjar, A. H., Bakhshi, H., Vaezi, A., Nasiri, M. J., & Mirsaeidi, M. (2024). Acute cardiovascular effects of electronic cigarettes: A systematic review and meta-analysis. *European Heart Journal Open, 4*(6), oeae098. https://doi.org/10.1093/ehjopen/oeae098
13. Garcia, P. D., Gornbein, J. A., & Middlekauff, H. R. (2020). Cardiovascular autonomic effects of electronic cigarette use: A systematic review. *Clinical Autonomic Research, 30*(6), 507–519. https://doi.org/10.1007/s10286-020-00683-4
14. El-Mahdy, M. A., Ewees, M. G., Eid, M. S., Mahgoup, E. M., Khaleel, S. A., & Zweier, J. L. (2022). Electronic cigarette exposure causes vascular endothelial dysfunction due to NADPH oxidase activation and eNOS uncoupling. *American Journal of Physiology–Heart and Circulatory Physiology*, *322*(4), H549–H567. https://doi.org/10.1152/ajpheart.00460.2021
15. Mohammadi, L., Han, D. D., Xu, F., Huang, A., Derakhshandeh, R., Rao, P., et al. (2022). Chronic e-cigarette use impairs endothelial function on the physiological and cellular levels. *Arteriosclerosis, Thrombosis, and Vascular Biology*, *42*(11), 1333–1350. https://doi.org/10.1161/ATVBAHA.121.317749
16. Lee, W. H., Ong, S. G., Zhou, Y., Tian, L., Bae, H. R., Baker, N., et al. (2019). Modeling cardiovascular risks of e-cigarettes with human-induced pluripotent stem cell–derived endothelial cells. *Journal of the American College of Cardiology*, *73*(21), 2722–2737. https://doi.org/10.1016/j.jacc.2019.03.476
17. Halstead, K. M., Wetzel, E. M., Cho, J. L., & Stanhewicz, A. E. (2023). Sex differences in oxidative stress–mediated reductions in microvascular endothelial function in young adult e-cigarette users. *Hypertension, 80*(12), 2641–2649. https://doi.org/10.1161/HYPERTENSIONAHA.123.21684
18. Jin, L., Lynch, J., Richardson, A., Sadu Singh, B. K., Lorkiewicz, P., Theis, W., et al. (2021). Electronic cigarette solvents, pulmonary irritation, and endothelial dysfunction: Role of acetaldehyde and formaldehyde. *American Journal of Physiology–Heart and Circulatory Physiology*, *320*(4), H1510–H1525. https://doi.org/10.1152/ajpheart.00878.2020
19. Metzen, D., M'Pembele, R., Zako, S., Brockmeyer, M., Jung, C., Bönner, F., et al. (2021). Platelet reactivity is higher in e-cigarette vaping as compared to traditional smoking. *International Journal of Cardiology, 343*, 146–148. https://doi.org/10.1016/j.ijcard.2021.09.005

20. Matheson, C., Simovic, T., Heefner, A., et al. (2024). Evidence of premature vascular dysfunction in young adults who regularly use e-cigarettes and the impact of usage length. *Angiogenesis, 27*(2), 229–243. https://doi.org/10.1007/s10456-023-09903-7
21. Berlowitz, J. B., Xie, W., Harlow, A. F., Hamburg, N. M., Blaha, M. J., Bhatnagar, A., Benjamin, E. J., & Stokes, A. C. (2022). E-cigarette use and risk of cardiovascular disease: A longitudinal analysis of the PATH Study (2013–2019). *Circulation, 145*(20), 1557–1559. https://doi.org/10.1161/CIRCULATIONAHA.121.057369
22. Alzahrani, T., Pena, I., Temesgen, N., & Glantz, S. A. (2018). Association between electronic cigarette use and myocardial infarction. *American Journal of Preventive Medicine, 55*(4), 455–461. https://doi.org/10.1016/j.amepre.2018.05.004
23. Chen, C., Huo, C., Mattey-Mora, P. P., Bidulescu, A., & Parker, M. A. (2024). Assessing the association between e-cigarette use and cardiovascular disease: A meta-analysis of exclusive and dual use with combustible cigarettes. *Addictive Behaviors, 157*, 108086. https://doi.org/10.1016/j.addbeh.2024.108086
24. Hądzlik, I., Piotrowski, J., Durowicz, M., Paśnik, J., Sendecka, G., Kistela, N., Skrzypczak, K. O., Krala-Szkaradowska, M., Stencel, N. J., & Stuczyński, S. K. (2024). Clinical manifestations of EVALI in adolescents and the impact of e-cigarettes on the respiratory disease. *Journal of Education, Health and Sport, 67*, 50879. https://doi.org/10.12775/JEHS.2024.67.50879
25. Blount, B. C., Karwowski, M. P., Shields, P. G., Morel-Espinosa, M., Valentin-Blasini, L., Gardner, M., et al. (2020). Vitamin E acetate in bronchoalveolar-lavage fluid associated with EVALI. *New England Journal of Medicine, 382*(8), 697–705. https://doi.org/10.1056/NEJMoa1916433
26. Ramesh Babu, Y. S., Kapil, A. M., Gunthner, A. R., Rajan, R. V., & Triyono, A. (2026). Adverse physical consequences of perinatal e-cigarette use by pregnant mothers on their offspring: A scoping review. *Cureus, 18*(1), e102109. https://doi.org/10.7759/cureus.102109
27. Lindson, N., Butler, A. R., McRobbie, H., Bullen, C., Hajek, P., Begh, R., Theodoulou, A., Notley, C., Rigotti, N. A., Turner, T., Livingstone-Banks, J., Morris, T., & Hartmann-Boyce, J. (2024). Electronic cigarettes for smoking cessation. *Cochrane Database of Systematic Reviews, 2024*(1), CD010216. https://doi.org/10.1002/14651858.CD010216.pub8
28. Münzel, T., Crea, F., Rajagopalan, S., & Lüscher, T. F. (2025). Nicotine and the cardiovascular system: Unmasking a global public health threat. *European Heart Journal*. Advance online publication. https://doi.org/10.1093/eurheartj/ehaf1010
29. George, J., Hussain, M., Vadiveloo, T., Ireland, S., Kennedy, P., Harrison, D., Struthers, A. D., & Lang, C. C. (2019). Cardiovascular effects of switching from tobacco cigarettes to electronic cigarettes. *Journal of the American College of Cardiology, 74*(25), 3112–3120. https://doi.org/10.1016/j.jacc.2019.09.067
30. Daiber, A., Kuntic, M., Oelze, M., & Hahad, O. (2023). E-cigarette effects on vascular function in animals and humans. *Pflügers Archiv – European Journal of Physiology, 475*, 783–796. https://doi.org/10.1007/s00424-023-02813-z
31. Plizga, J., Głuszczyk, A., Makłowicz, A., Kopczyńska, E., Szpulak, A., Frańczuk, A., Grzelka, M., Głoskowska, J., Kuleta, K., & Słychan, K. (2024). The impact of e-cigarettes and tobacco heating systems on health: A review of current research. *Quality in Sport, 20*, 53680. https://doi.org/10.12775/QS.2024.20.53680