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Review Article

E-cigarettes and cardiovascular health: a review of components, mechanisms, and clinical risk

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Abstract: Electronic cigarettes (e-cigarettes) have become one of the most urgent public health issues of the twenty-first century. Originally marketed as less harmful than burning cigarettes, e-cigarettes have gained enormous popularity, especially among young adults, thus leading to nicotine addiction and new trends of cardiovascular damage. Even though e-cigarettes limit exposure to products of combustion, they supply similar doses of nicotine and other elements such as aldehydes, metals, and flavoring by-products, whose cardiovascular effects are incompletely characterized. This narrative review addresses a comprehensive analysis of the cardiovascular effects of e-cigarettes, particularly the contribution to the development of arrhythmias and cardiomyopathy. Evidence was synthesized from in vitro, animal models, and clinical research that covers e-cigarette constituents, delivery mechanisms of toxins, and both acute and chronic cardiovascular effects. E-liquid constituents can result in an autonomic imbalance, increasing heart rate and pressure, conductivity, and the predisposition of arrhythmias and cardiomyopathies. Recent research has shown oxidative stress, endothelial dysfunction, and autonomic imbalance instigated by nicotine and non-nicotinic additives, which destabilize cardiac electrophysiology. However, most human data remain short-term, with limited sample sizes and heterogeneous exposure assessment, underscoring the lack of longitudinal evidence on long-term outcomes such as myocardial infarction, heart failure, or sudden cardiac death. The clinical and public health implications are significant, and it is imperative to shift the focus to vaping as a separate cardiovascular risk factor, particularly among young adults. To reduce the increasing burden of e-cigarette-related cardiovascular disease, preventive actions and enhanced monitoring must be prioritized. They become cardiovascular toxic through nicotine and other chemical exposures, and there is growing evidence of arrhythmogenic and cardiomyopathic effects. Strong prospective studies are urgently needed to define dose-response relationships, long-term outcomes, and cessation strategies.

Keywords: Electronic cigarettes, cardiovascular disease, nicotine, arrhythmias, cardiomyopathy

Introduction

Electronic cigarettes (e-cigarettes or vapes) have rapidly emerged as one of the most debated public health issues in the 21st century. Their impact extends beyond recreational use, increasingly influencing clinical practice in ways that are not fully recognized by many clinicians. The most affected group remains that of adolescents and young adults, which raises urgent concerns regarding prevention and management strategies. Electronic cigarettes were first introduced in 2003 as a less harmful alternative to combustible cigarettes. Consequently, adults who struggled to quit conventional cigarette smoking began reaching out to e-cigarettes, considering them a safer alternative. There were only 7 million users (about twice the population of Oklahoma) in 2011. However, the situation changed rapidly when the prevalence

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of e-cigarettes rose to 68 million in 2020 and 82 million in 2021 [1].

A combustible cigarette delivers ~1-2 mg of absorbed nicotine, with peak levels in 5-7 minutes. Therefore, a full pack (20 cigarettes) provides approximately 20-40 mg of nicotine. By comparison, a single pod in modern electronic nicotine delivery systems contains 40-59 mg of nicotine, equivalent to or exceeding two packs of cigarettes. Thus, one pod delivers more nicotine than an entire pack of combustible cigarettes [2]. Notably, the introduction of e-cigarettes has reduced cigarette use while maintaining or even increasing nicotine intake, while also introducing multiple harmful compounds [3]. For U.S. adults in 2021, approximately 30.3% of e-cigarette users had never smoked combustible cigarettes [4]. This emphasizes e-cigarettes' role as an addiction and nicotine delivery source with a complete individualistic presence, separate from combustible cigarettes. E-cigarettes heat a liquid that generally includes propylene glycol, vegetable glycerin, nicotine, and flavorings into an inhalable aerosol. During this process, toxic aldehydes and trace metals are released from the heating elements of the device. These components, along with nicotine, are known to have cardiovascular effects. The exact "recipe" of exposure varies with the device generation, power, liquid composition, and user behavior (length and intensity of a puff). In short, there is no single e-cigarette exposure; there are many. Studies in mice and humans have shown that nicotine, flavoring compounds, and propylene glycol can cause acute increases in heart rate and blood pressure, transient endothelial dysfunction, and changes in vascular biomarker levels [5].

In a murine model, chronic exposure to nicotine-containing e-cigarette aerosol impaired cardiac function, promoted cardiomyocyte structural abnormalities, increased oxidative stress, and accelerated atherosclerosis, highlighting the potential of e-cigarettes to cause serious cardiovascular harm [6]. In an in vitro study, cardiomyocytes exposed to e-cigarette and cigarette smoke extracts showed markedly reduced viability. Exposure to these compounds caused disruptions in all metabolic endpoints, pointing toward potential structural and genetic changes in cells [7, 8]. The biggest factor responsible for the rapid rise in e-cigarette use and popularity is misinformation or lack of information. They have been marketed as 'safer alternatives' to combustible cigarettes, 'less harmful', or even to fight smoking addiction. Emergency departments across the world have reported presentations of adolescents with arrhythmias, high blood pressure, and heart rate and function changes, which only track back to heavy e-cigarette use. Researching the components, mechanisms, and effects of e-cigarette use has become more important. A recent study found that e-cigarette users had significantly longer hospital stays for cardiac issues. On average, it was 2.45 days longer for ever-users and 3.24 days longer for current users than for non-users [9].

These findings underscore a critical gap between public perception and clinical reality. E-cigarettes are frequently dismissed as a benign habit in clinical encounters, yet the cardiovascular burden they impose, even in short-term users, demands that they be treated with the same clinical urgency as conventional tobacco. Although the current evidence based on e-cigarette use remains limited and largely short-term, emerging data suggest potential cardiovascular effects, including arrhythmogenic risk and myocardial dysfunction. In this narrative review, we summarize the current evidence on e-cigarette components, mechanisms of substance delivery, and their potential cardiovascular impact, with a focus on arrhythmias and cardiomyopathy.

Electronic cigarettes

Design of E-cigarettes

Electronic nicotine delivery systems (e-cigarettes) are portable electronic devices designed to deliver aerosolized substances, including nicotine, without causing combustion [10-12]. Evolution of e-cigarette design (**Figure 1**): (1) Since their introduction, e-cigarette design has evolved considerably across four distinct generations, each reflecting advances in battery capacity, customization, and nicotine delivery efficiency (**Figure 1**). The earliest devices, known as "cigalikes", were designed to resemble conventional cigarettes closely and were either disposable or used pre-filled cartridges [13]. The second generation, commonly

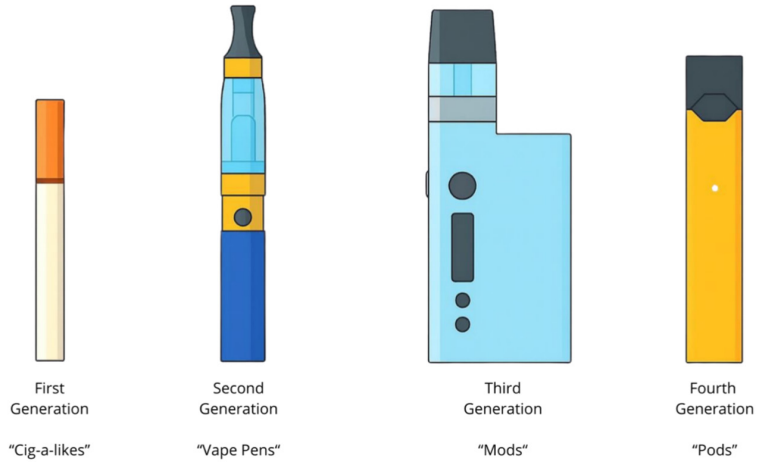

Figure 1. Types of e-cigarette devices. From left to right, this figure shows the first-generation "cigalike", second-generation or "vape pen", third-generation "box mod", and fourth-generation "pod system".

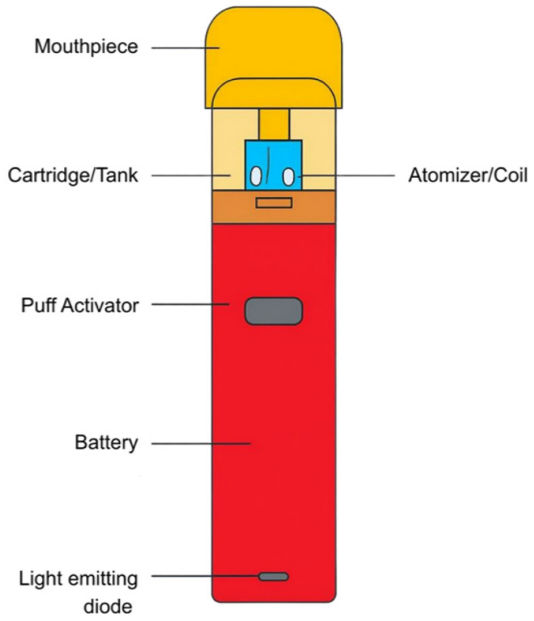

Figure 2. E-cigarette parts. The illustration identifies key elements of an e-cigarette, including a mouthpiece, cartridge with e-liquid, atomizer [heating element], battery, and LED indicator.

referred to as "vape pens", introduced larger battery capacities, refillable tank systems, and adjustable voltage settings that allowed users greater control over vapor production [13]. (2) Third-generation devices, or "mods", expanded on this further with highly customizable designs, larger e-liquid capacity, and the incorporation of sub-ohm coils that enabled significantly increased aerosol output [12, 13]. (3) The most recent fourth-generation devices, known as "pod systems", represent a shift toward compactness, using closed or refillable pods that typically deliver higher concentrations of nicotine salts - JUUL products being among the most widely recognized examples [12, 13].

The vaporization process begins when the e-cigarette is activated, in most models, by either pressing a button or automatic breath activation, which triggers the flow of electrical current from the battery to the heating coil [14]. The heating coil vaporizes the e-liquid stored on the wick, and the resulting aerosol produced from the coil is inhaled through the mouthpiece of the device [10, 15].

E-cigarettes share a handful of core components that determine their functioning and safety characteristics while in use (**Figure 2**) [11, 14]. The primary power source is typically a lithium-ion battery. Most advanced devices offer adjustable voltage and wattage options, which influence the aerosol temperature and the composition of the aerosol [10, 15].

The heating coil is commonly made of kanthal, nichrome, or stainless steel, with its resistance [in ohms] defining the temperature and volume of the vapor produced. Studies have indicated that higher temperatures increase the formation of undesirable carbonyl compounds, including formaldehyde and acrolein [10, 15].

The wick, made of cotton, silica, or ceramic, is a pathway for the e-liquid to move from the reservoir to the heating coil [14]. The wick material can change the amount of thermal degradation byproducts. E-liquids are stored in various types of reservoirs, including cartridges, tanks, or pods [14, 15].

Constituents in E-cigarette liquids and emissions

The e-liquid typically contains nicotine in varying concentrations, dissolved in carrier solvents such as vegetable glycerin (VG) and pro-

pylene glycol (PG), blended in various PG/VG ratios [16]. Additional constituents may include flavoring agents, N-nitrosamines, volatile organic compounds, and trace metals (e.g., cadmium, chromium, lead, nickel, silver, tin, and silicates) that can leach from device components such as coils, wicks, and solder joints [17, 18].

Upon heating, the e-liquid is aerosolized into an inhalable mixture of nicotine, solvents, and flavoring compounds [19]. Nicotine and toxicant delivery are influenced by device settings (e.g., voltage/power), e-liquid composition (including PG/VG ratio and pH), and user puffing behavior (puff duration, volume, and frequency) [20]. The PG/VG ratio affects aerosol particle size and nicotine yield, while e-liquid pH determines the proportion of free-base versus protonated nicotine, influencing absorption characteristics [21]. Commercial formulations range from 0 to >36 mg/mL nicotine, with some capable of delivering nicotine doses comparable to or exceeding those of conventional cigarettes [20].

Nicotine: Nicotine is the primary psychoactive and addictive component responsible for many of the cardiovascular effects of e-cigarettes. It is rapidly absorbed through the oral mucosa and pulmonary epithelium and readily crosses the blood-brain barrier, where it binds to nicotinic acetylcholine receptors (nAChRs). This interaction stimulates the release of neurotransmitters, including dopamine, norepinephrine, serotonin, γ-aminobutyric acid (GABA), glutamate, and endorphins. Dopamine release, in particular, activates central reward pathways, contributing to nicotine dependence [22, 23].

Nicotine also activates the sympathetic nervous system, leading to increased heart rate, blood pressure, and cardiac output. Sustained sympathetic stimulation contributes to arrhythmogenesis, including atrial fibrillation and ventricular tachycardia, and may increase the risk of sudden cardiac death. It also promotes myocardial remodeling, thereby increasing the risk of heart failure [22, 24]. Nicotine-induced catecholamine release accelerates atherosclerosis and exacerbates dyslipidemia by promoting lipolysis, increasing plasma free fatty acids, reducing high-density lipoprotein cholesterol, and elevating low-density lipoprotein cholesterol [25]. In addition, upregulation of vascular growth factors, including platelet-derived and vascular endothelial growth factors, contributes to endothelial dysfunction, oxidative stress, fibrosis, and arterial stiffness [24]. Although nicotine plays a central role in these effects, overall cardiovascular risk is also influenced by variability in nicotine delivery, as well as potential contributions from other e-cigarette constituents.

Nicotine delivery and device variability: Nicotine delivery from e-cigarettes is governed by interactions between device characteristics, e-liquid formulation, and user behavior. Newer-generation devices, particularly pod-based systems, are capable of achieving higher plasma nicotine concentrations than earlier models [26]. Early e-cigarettes primarily use free-base nicotine, an unprotonated, more lipophilic form (pH~7-9) that facilitates membrane diffusion but produces greater inhalation harshness, thereby limiting tolerable concentrations [27]. In contrast, modern devices commonly use protonated nicotine salts, which are more hydrophilic and have a lower pH. This reduces sensory irritation, enabling higher nicotine concentrations and deeper inhalation [27].

For example, studies of JUUL have shown that a single 5% nicotine pod can deliver nicotine amounts comparable to up to ~30 cigarettes, depending on user puffing patterns and formulation characteristics [2, 11]. Lower pH aerosols favor smoother inhalation and deeper lung deposition, enhancing systemic absorption, whereas higher pH promotes the gaseous phase, increasing upper airway absorption but also harshness [22, 27]. In contemporary nicotine salt-based systems, nicotine exposure is primarily driven by puff topography and device power output, with longer puff duration, greater puff volume, and higher power significantly increasing systemic nicotine delivery [28].

Solvents: The main ingredients in e-liquid are propylene glycol and glycerol. These act as solvents that dilute nicotine to the right concentration and give it the viscosity needed for vaporization (varying PG/VG ratios). When heated, these substances break down and release various harmful aldehydes and related byprod-

ucts. Glycerol can produce acrolein, formaldehyde, and dehydrated glycerol, while propylene glycol can create acetaldehyde, formaldehyde, propylene oxide, acetol, allyl alcohol, glyoxal, and methylglyoxal [29, 30]. Formaldehyde is particularly dangerous for myocytes. It has been linked to cardiovascular issues such as atrioventricular block, different arrhythmias, ventricular tachycardia, ventricular fibrillation, and atrial fibrillation. The reactive aldehyde acrolein can increase thrombosis risk by worsening dyslipidemia, speeding up atherosclerosis, altering apolipoprotein A-I in high-density lipoprotein, promoting inflammation from neutrophils, and increasing platelet activation [31, 32]. Both acrolein and formaldehyde cause toxicity partly by forming adducts with DNA and proteins [32].

In animal studies, acetaldehyde has been tied to mitochondrial issues that lead to alcoholic cardiomyopathy [33]. Device features influence the generation of these aldehydes. A higher battery voltage significantly raises aldehyde emissions, sometimes matching or exceeding those of combustible cigarettes [34]. Reusing devices can further increase aldehyde production, likely due to the breakdown of built-up polymerization products [35].

Besides aldehydes, aerosolized vegetable glycerin has been associated with irritation in the esophagus, lungs, and eyes. Propylene glycol exposure has been linked to symptoms affecting the upper respiratory tract [36]. The ratio of propylene glycol to vegetable glycerin in e-liquids also impacts the production of reactive oxygen species. These species are connected to atherosclerosis, hypertension, restenosis, and ischemia [37]. The amount of propylene glycol also affects nicotine delivery; larger amounts are linked to greater nicotine yields. However, establishing a clear quantitative link is made difficult by the variety among brands and the differences between labeled and actual solvent ratios [26, 29].

Flavorings: E-liquid flavorings commonly include chemicals such as cinnamaldehyde, vanillin, benzaldehyde, ethyl maltol, menthol, and dimethylpyrazine [38]. These appealing flavors are key drivers initiation of e-cigarette use among youth [39]. Flavoring agents are not biologically inert; even in the absence of nicotine, they have been shown to induce inflammation, oxidative stress, and DNA damage, along with endothelial dysfunction and epithelial barrier disruption. Additionally, they may cause electrophysiological alterations, immunomodulatory effects, and adverse cardiovascular outcomes, including impaired nitric oxide signaling, altered vasoreactivity, arrhythmias, and reduced cell viability.

Notably, many flavoring compounds carry 'Generally Recognized as Safe' status for oral ingestion, yet this designation was never intended to cover inhalation. This regulatory blind spot represents a fundamental failure of premarket safety evaluation for e-cigarette constituents, and highlights that the absence of regulation should not be conflated with the absence of harm.

Nitrosamines: Tobacco-specific nitrosamines [TSNAs] are potent carcinogens found in tobacco products, including some e-cigarette liquids and aerosols. They can damage DNA and cause mutations, leading to lungs, mouth, and several other cancers [40]. The formation of TSNAs increases with time and at higher temperatures, which can provide important information for evaluating and ensuring the quality of e-cigarette products [41].

Metals: Studies have shown that e-cigarette vapor can contain toxic metals such as lead [Pb], nickel [Ni], chromium [Cr], and cadmium [Cd] [42]. E-liquids that touch the heating coils often have higher amounts of metals than e-liquids straight from the bottle. This occurs because parts inside the device, such as wires and clamps made of metals such as copper, silver, and nickel, can release these metals into the liquid. For example, copper wires with silver coatings and brass clamps are linked to more metals, such as zinc and aluminum, which appear in the vapor that people inhale. Inhalation of these metals is a health concern because they are poisonous; lead and Cd are known carcinogens, and Ni or Cr can harm the lungs, heart, kidneys, and nervous system [43].

Mechanisms of cardiovascular injury

The increased presence of ECs is one of the troubling emerging world trends at present, as it lacks longitudinal data on its safety and health impacts, and they are being suggested

as alternatives that are less damaging [44, 45]. Multiple flavored e-liquids and typical flavoring agents were found to cause deterioration of endothelial cell functionality in vitro [6, 46]. Endothelial cells generated from pluripotent stem cells show reduced viability, tube formation, and wound healing (e.g., a scratch test) upon exposure to cinnamaldehyde and menthol, as observed in flavored e-liquids, and upregulate the expression of inflammatory cytokines [6]. Cinnamaldehyde and vanillin promote inflammatory cytokine and chemokine production by endothelial cells exposed to flavored e-liquid or RY-4 e-liquid. Vanillin, menthol, cinnamaldehyde, eugenol (clove flavoring), and acetylpyridine (burnt flavoring) accelerate the production of interleukin-6 and inhibit A23187-mediated nitric oxide production in human aortic endothelial cells [6]. In standing e-liquid, the reaction between flavor aldehydes and propylene glycol or glycerin may produce toxic acetals. These acetals, in turn, cause more respiratory irritation than the original aldehyde [47, 48].

Large systemic doses of propylene glycol (more than the suggested maximum dose of 25 mg/kg-1·d-1) may induce severe metabolic acidosis, acute kidney injury, and sepsis-like conditions [49]. Theatrical fog and smoke are generated via glycol mixtures, and occupational exposure to glycol mixtures at work is linked to increased reports of glycol wheezing and tightness in the chest [50, 51]. Acute effects of glycol mixture exposure include acute dry cough, throat irritation, and lung impairment in heavily exposed individuals [51]. In vitro exposure of pulmonary epithelial cells and monocytes to aerosols generated with each of the flavored JUUL pods raised levels of inflammatory cytokines, generated mitochondrial oxidants, increased DNA damage, and disrupted pulmonary epithelial barrier integrity [52]. Exposure of human bronchial epithelial cells to the flavoring agents acetoin (presenting a buttery taste), diacetyl, ortho-vanillin, and maltol (malt taste) after 24 hours showed an increase in interleukin-8 production, indicating that the flavoring agents act independently to induce toxicity.

The long-term cardiovascular and cardiopulmonary consequences remain largely undefined due to the absence of longitudinal human data. This gap has contributed to the widespread perception of ECs as a safer alternative to combustible cigarettes. However, as discussed above, accumulating experimental and short-term clinical evidence suggests that this perception may be premature. Many of the chemical constituents used in EC liquids have well-established toxic, pro-inflammatory, and pro-atherogenic effects when studied individually. Their repeated inhalational exposure, often at high concentrations and over prolonged periods, raises concern for delayed but clinically significant cardiovascular pathology that may only become apparent after years or decades of use.

Before examining how these mechanisms translate into clinical outcomes, it is useful to clarify how electronic cigarettes differ from combustible cigarettes, as the two are often grouped together despite delivering nicotine through fundamentally different processes. A combustible cigarette burns tobacco at elevated temperature, and this combustion produces smoke carrying tar, carbon monoxide, and thousands of byproducts that drive much of the cardiovascular harm linked to conventional smoking. An electronic cigarette instead heats a liquid to generate an inhalable aerosol without igniting tobacco, thereby removing tar and greatly reducing carbon monoxide exposure. This difference, however, is not the same as safety. Heating introduces a distinct toxicological burden, including reactive aldehydes formed as solvents degrade, flavoring chemicals that were never assessed for inhalation, and trace metals shed from the heating coil and other device parts. Nicotine delivery complicates the comparison further, since modern pod systems using nicotine salts can reach plasma nicotine levels that match or exceed those of combustible cigarettes, meaning the move from smoking to vaping does not reliably reduce, and may even raise, nicotine intake. A final and often overlooked difference is predictability: combustible cigarettes are uniform products, whereas the chemical output of an electronic cigarette shifts with device generation, power setting, solvent ratio, flavoring, and individual puffing behavior.

Taking together, these differences suggest that e-cigarettes and combustible cigarettes share a broadly overlapping cardiovascular mechanism, rooted in nicotine-mediated sympathetic activation, oxidative stress, and endothelial injury, while diverging substantially in chemical

Table 1. Comparative cardiovascular effects of e-cigarettes and combustible cigarettes

Domain	E-cigarettes	Combustible cigarettes
Particle Profile	Lower total aerosol particle mass, but aerosol contains ultrafine particles, thermally degraded aldehydes (e.g., formaldehyde, acrolein), flavoring-related toxicants, and trace metals leached from heating components [18]	High levels of fine particulate matter, tar, oxidants, and established carcinogens generated through tobacco combustion [19]
Nicotine Delivery	Variable; modern pod-based systems using nicotine salts can match or exceed combustible cigarette levels; highly dependent on device generation, power setting, and puffing behavior [2, 30]	Relatively standardized; approximately 1-2 mg absorbed per cigarette; peak plasma levels reached within 5-7 minutes [2]
Processing	Heating of e-liquid produces an inhalable aerosol without combustion; carbon monoxide exposure is minimal [10, 15]	Combustion of tobacco at high temperatures produces tar, carbon monoxide, and thousands of toxic byproducts [19]
Arterial stiffness	Increased pulse wave velocity and augmentation index; effects observed even in the absence of nicotine, suggesting a contribution from non-nicotine aerosol constituents [101, 107]	Significant and well-established increase in arterial stiffness and vascular resistance with chronic exposure [101, 107]
Oxidative Stress and Inflammation	Acute ROS generation from heated PG/VG and flavoring chemicals; mitochondrial oxidative stress and DNA damage; early endothelial dysfunction without established atherosclerosis [90, 91]	Persistent ROS exposure from combustion products; oxidative LDL modification and foam cell formation; chronic endothelial injury leading to progressive atherosclerosis [37]
Hemodynamic effects	Acute increases in heart rate, blood pressure, and sympathetic activation; magnitude generally less sustained than combustible cigarettes [63, 64]	Sustained increases in heart rate and blood pressure with chronic autonomic imbalance and persistent sympathetic overdrive [24]
Carbon monoxide exposure	Minimal, due to the absence of combustion; does not contribute meaningfully to hypoxic cardiovascular injury [10, 15, 19]	High exposure from tobacco combustion; contributes to tissue hypoxia, impaired oxygen delivery, and vascular injury [19]
Clinical cardiovascular risk	Limited long-term data; emerging associations with cardiovascular dysfunction, arrhythmias, and endothelial injury; causal relationships not yet established [101-103]	Strong and well-established association with myocardial infarction, stroke, heart failure, and cardiovascular mortality across decades of prospective cohort data [100]
Overall interpretation	Not without risk; causes measurable acute vascular and electrophysiological dysfunction; long-term cardiovascular harm plausible but incompletely characterized	A major, well-characterized cardiovascular risk factor with robust epidemiological evidence supporting causal harm across multiple endpoints

Abbreviations: ROS, reactive oxygen species; PG, propylene glycol; VG, vegetable glycerin; LDL, low-density lipoprotein; CO, carbon monoxide.

composition, nicotine delivery dynamics, and the degree to which any given exposure can be predicted or standardized. To organize these points of overlap and divergence systematically across the principal domains of cardiovascular injury, and to establish the comparative framework that informs the mechanistic and clinical discussion that follows, the key characteristics of both product types are summarized in **Table 1**.

Table 1 places the two products side by side across domains including particle profile, oxidative stress, hemodynamic effects, and clinical cardiovascular risk. However, one distinction the table cannot fully capture deserves emphasis. The evidence base for combustible cigarettes spans decades of large prospective cohort studies with hard clinical endpoints such as myocardial infarction and cardiovascular mortality. For e-cigarettes, the equivalent evidence simply does not exist yet. Drawing direct comparisons, therefore, risks either falsely reassuring users that e-cigarettes are safer, or overstating risk in ways that undermine cessation efforts for conventional smokers.

Cardiovascular effects of E-cigarettes

As discussed earlier, exposure to nicotine, aldehydes, metals, and other aerosolized toxicants has been shown to disrupt autonomic balance, impair vascular function, and promote electrophysiological instability [47, 53]. However, toxicological plausibility alone does not establish clinical relevance. Therefore, beyond the experimental and mechanistic data on individual e-liquid constituents, it is critical to examine whether these effects translate into documented cardiovascular pathology in humans. This section reviews the available clinical and ob-

servational evidence linking e-cigarette use to arrhythmias and cardiomyopathy, with a particular focus on reported cases, physiological alterations, and early signals of myocardial dysfunction that may substantiate the mechanistic concerns raised by constituent-level studies.

Arrhythmias

The rhythmic contraction of the heart is highly dependent on coordinated electrical activity, which begins in the sinoatrial (SA) node and propagates through the atria, reaching the atrioventricular (AV) node, the His-Purkinje cell system, and the rest of the ventricular myocardium [54, 55]. At a cellular level, electrical conductivity begins with the activation of an action potential, regulated by fluxes of sodium (Na^+), calcium (Ca^{2+}), and potassium (K^+) across ion channels and transporters [56]. This orchestration ensures depolarization and repolarization in a regular sequence, allowing effective excitation-contraction coupling to occur. This process can be viewed through an electrocardiogram (ECG), appearing as the P wave (atrial depolarization), PR interval (AV-nodal conduction), QRS complex (ventricular depolarization via fast Na^+ influx), and T wave [ventricular repolarization, reflecting K+ currents and late Na^+/Ca^{2+} dynamics]. Disruption of any of these parts can result in abnormal heartbeats, formally known as arrhythmias or dysrhythmias [57, 58].

Recent studies have indicated that e-cigarettes can induce multiple perturbations in cardiovascular activity that closely resemble those observed with conventional tobacco smoking. Clinical findings have shown that e-liquid constituents induce cellular distress and autonomic imbalance, destabilizing the electrophysiological current that sustains the normal rhythm [59-62]. These disturbances translate clinically into irregularities in heart rate (HR), blood pressure (BP), and heart rate variability (HRV) [45, 63, 64]. Experimental data suggest that e-cigarette aerosols can depolarize the resting membrane potential, increase heterogeneity of repolarization, and furthermore, the susceptibility to arrhythmias (view **Figure 3**). When combined with common triggers, such as premature ventricular beats (PVBs), re-entry circuits, or abnormal automaticity can occur, leading to tachyarrhythmias, fibrillations, and other conduction disturbances. In this way, e-cigarette exposure may mimic and amplify the arrhythmogenic potential originally only attributed to tobacco smoking.

Tachyarrhythmias induced by E-cigarettes: Rapid heart rhythms over 100 bpm are known as tachycardias or tachyarrhythmias, which can originate from disruptions in the impulse generation [automaticity], conduction, or myocardial repolarization [57, 58]. This group contains the most common types of arrhythmias and can be subdivided into supraventricular arrhythmias (SVT) and ventricular arrhythmias (VT). Where SVT typically presents a narrow QRS complex on ECGs [65], while VT is represented by a wide QRS complex [66]. Emerging studies suggest that e-cigarettes can lead to cellular ion exchange abnormalities [67], increased heart rate variability (HRV) [68, 69], modification in repolarization [70], and boosting ventricular premature beats (VPB) [71]. These findings suggest that e-cigarette exposure can generate the electrophysiological setting that favors both SVT and VT.

Recent evidence places nicotine in the central role for the arrhythmogenic effects of e-cigarettes. Upon inhalation, nicotine is rapidly absorbed into the bloodstream, binding with nicotinic acetylcholine receptors (nAChRs), and activating catecholamine release in peripheral sympathetic nerve terminals and adrenal chromaffin cells [60, 71]. This commences the shift to a dominance of sympathetic activity that reduces parasympathetic action [71, 72]. Further studies demonstrated that in humans, the binding of nicotine to nAChRs decreased acetylcholine release and M2 receptor activation, blocking inward rectifying K^+ channels coupled with G-protein, thus obstructing the dromotropic modulation in cardiomyocytes by the parasympathetic system [67]. The reduced modulation shortens the PR interval, which results in a briefer refractory time in the AV-node and therefore a rapid AV conduction. This last mechanism especially favors reentrant supraventricular tachycardias (AVNRT).

The β1-adrenergic dominance due to nicotine leads to the excretion of catecholamines such as dopamine and norepinephrine, which increases heart rate and contractility [67, 73]. However, human studies have shown that dopamine is the primary mediator in accelerated AV conduction by shortening both the PR inter-

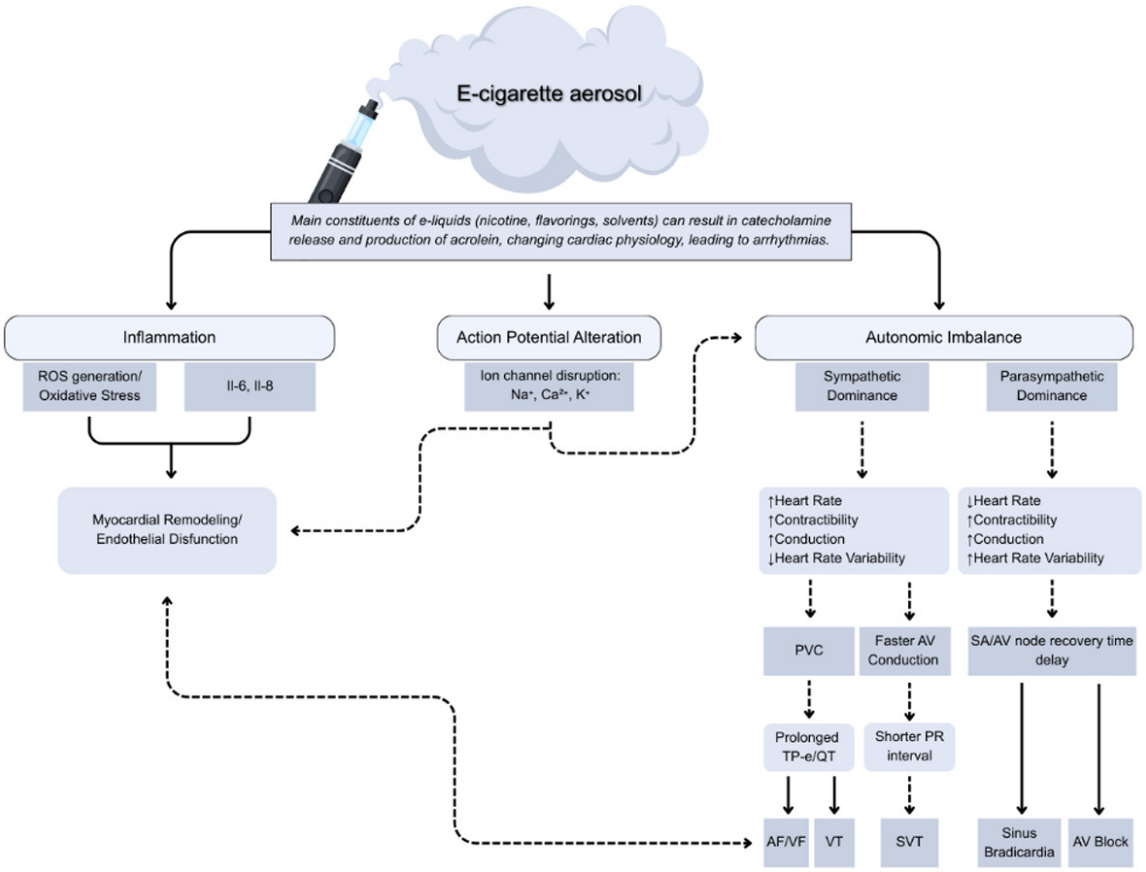

Figure 3. Main triggers in e-cigarette aerosol lead to cardiac arrhythmia. The main constituents in e-cigarette aerosol (nicotine, flavorings, solvents) provoke inflammation, autonomic imbalance, and alterations in cardiac action potential through ion channel disruption. These mechanisms lead to oxidative stress, cytokine release [interleukin-6 (IL-6), interleukin-8 (IL-8)], myocardial remodeling, and conduction abnormalities, which increase the risk of several types of arrhythmias.

val and the segment, leading to supraventricular tachyarrhythmias [67]. This effect on the augmentation in conduction velocity by catecholamines is also prolonged by nicotine through the partial suppression of catecholamine breakdown via inhibition of monoamine oxidase [74]. Following a similar mechanism, the nicotine-induced sympathetic overdrive is also involved in ventricular tachyarrhythmias and sudden death risk [63, 75]. A study in healthy e-cigarette users and non-users showed an increased Tp-e/QT ratio on ECGs after e-cigarettes with nicotine exposure [63]. This prolongation of the Tp-e/QT ratio suggests a dispersion of ventricular repolarization that could be used as a marker for ventricular tachycardia, fibrillation, and sudden death [63, 76, 77].

It is important to note that the formulation of nicotine appears to influence the arrhythmogenic risk [59, 77]. Current research has demonstrated that systems with nicotine salts deliver nicotine more efficiently and with less irritation, increasing plasma concentration compared to free-base nicotine [78, 79]. In mice, nicotine salts, especially at 5%, increased HR and decreased HRV at a greater level than those with 1% and 2.5%, though all increased the risk for VPB compared to air [59]. Nonetheless, randomized clinical studies have shown that 5% vs 2.4% nicotine delivery systems have no clinically relevant difference in HR or BP, likely due to real-life puff volume variation [80]. Both studies corroborate the tilt toward the β1-adrenergic pathway through nicotine-induced ion-channel inhibition and heightened risk of ventricular tachyarrhythmias.

Separately, non-nicotine constituents such as flavor aldehydes, solvent vehicles, and byprod-

ucts also affect cardiac conduction [71, 72]. Evidence shows e-cigarette liquid containing both menthol and nicotine (as in JUUL-type pods) provoked higher VPBs than those with nicotine or menthol alone [71]. This is relevant because accelerated atrial conduction, but prolonged AV conduction indicates a synergistic and pro-arrhythmic interaction. Other products, such as vegetable glycerin (VG) and propylene glycol (PG), which are solvent vehicles, when observed separately from nicotine, studies showed an initial parasympathetic dominance, measurement post-exposure exhibited tachycardia and lower HRV, probably due to a sympathetic rebound shift [71, 81].

Fibrillation induced by E-cigarettes: Cardiac fibrillations are the most common type of arrhythmias, with complications like strokes and life-threatening risks [81, 82]. Both forms, atrial and ventricular, distinguish themselves from tachycardias by their chaotic or irregular rhythm, which results in critically impaired cardiac output and, on many occasions, sudden death [75]. On the ECG, atrial fibrillation (AF) is characterized by the absence of P waves and irregular R-R intervals [83], while ventricular fibrillation (VF) is characterized by low-amplitude oscillations without abnormal QRS complexes [84]. Both forms are commonly associated with cellular damage and structural remodeling [fibrotic], creating a medium with poor conduction sustaining abnormal reentry and automaticity [57, 85]. The latest data show that e-cigarette exposure harms the cardiovascular musculature by intra- and extracellular mechanisms [6].

Nicotine itself drives a feed-forward injury loop by upregulating the phosphatase PHLPP1 via an ERK1/2-4E-BP1 signaling axis, which in turn amplifies NADPH-oxidase-derived reactive oxygen species (ROS) and mitochondrial dysfunction, leading to cardiomyocyte death [61]. The resulting oxidative stress destabilizes calcium-handling proteins (RyR2, SERCA, L-type Ca^{2+} channels) and gap-junction connectivity, shortens action-potential duration, and promotes both atrial and ventricular fibrillation [86-88]. Moreover, oxidative stress-induced ROS can further activate pro-fibrotic pathways, contributing to structural remodeling that predisposes to atrial fibrillation. Several studies implicate nicotine-mediated suppression of microRNAs (e.g., miR-133, miR-590) in fibrotic remodeling by causing a greater deposition of collagen; further investigations must be conducted in humans [89]. Together, excessive ROS production, PHLPP1, mitochondrial injury, and consequent electrophysiological derangements create a potent substrate for fibrillatory arrhythmias in e-cigarette users.

Although widely used compounds such as vanillin and cinnamaldehyde are growing in popularity, there is a lack of safety regulation for inhalation use, despite being recognized as safe for ingestion [98, 99]. This is thought to be due to the generation of reactive aldehydes after the aerosolization process, which new evidence indicates to have direct cardiotoxic and pro-arrhythmic contribution [6, 99, 100]. Studies show that exposure to these aldehydes increases the ROS production, impairs mitochondrial membrane potential, and amplifies electrical instability [6, 101]. The cinnamon-flavored e-liquid has been identified as the most cytotoxic among the rest [100]. In acute exposure, cinnamaldehyde has elevated ROS, reduced endothelial cell viability, activates apoptotic pathways, and impairs angiogenesis [6, 102].

The heterogeneity of flavoring compounds across e-cigarette products makes population-level risk assessment particularly difficult. Unlike nicotine, whose concentration can be measured and regulated, the flavoring landscape is vast, largely unregulated, and rapidly evolving. A user switching brands or flavors may unknowingly alter their arrhythmogenic risk profile in ways that neither they nor their clinician can anticipate. This represents a unique challenge that has no direct parallel in conventional tobacco regulation.

Bradyarrhythmia induced by E-cigarettes: As opposed to the rapid and chaotic heartbeats of tachyarrhythmia and fibrillation, bradyarrhythmia or bradycardia are atypical slow conduction beats, with HR lower than 60 bpm [58]. Commonly due to a suppression of nodal activity or overcompensation of the parasympathetic tone. The two categories are sinus bradycardia and atrioventricular conduction blocks (first, second, third degree) [81]. Kucera et al. [59] demonstrate that free-base nicotine at 5% increased HRV without raising the HR, which

suggests a parasympathetic reaction. Similarly, Carll et al. [71] describe how VG and PG aerosols separately and without nicotine cause bradycardia, bradyarrhythmia, and increased HRV during inhalation.

This parasympathetic tilt, as opposed to the ones seen with nicotine, could be due to acrolein generation in the VG and PG smoke. Acrolein inhalation activates airway sensory nerves (TRPA-1), triggering a vagal reflex acetylcholine release at the SA and AV nodes, hence more M_2 receptor activation. Particularly, VG aerosols cause even more pronounced bradycardias and bradyarrhythmia -as high-grade supraventricular blocks-, which could be explained because it has higher acrolein levels as opposed to PG aerosols [71]. There is also evidence that PG aerosols alone can cause aortic endothelial dysfunction, by making the smooth muscle in the vessel wall less responsive, increasing the risk factor for arrhythmia and other CVDs [95]. On the other hand, vegetable glycerin (VG) and propylene glycol (PG), which are solvent vehicles, when observed separately and without nicotine, showed an initial parasympathetic dominance, measurement post-exposure reversed tachycardia and lower HRV, probably due to a sympathetic rebound [71, 96].

The biphasic autonomic response observed with solvent exposure, initial parasympathetic dominance followed by sympathetic rebound, is particularly clinically relevant. It suggests that the cardiovascular risk of e-cigarettes is not static but dynamic, shifting across the exposure timeline. Standard acute measurement protocols likely capture only one phase of this response, which may explain some of the contradictory findings across studies and warrant investigation through continuous ambulatory monitoring designs.

Cardiomyopathy

Cardiomyopathy is an anatomical and pathological diagnosis associated with muscular or electrical dysfunction of the heart [97]. There are three major types of cardiomyopathies: restrictive, dilated, and hypertrophic. It often leads to heart failure, arrhythmias, ischemic heart disease, and sudden cardiac death. Though nicotine is an established risk for cardiomyopathy, data are still limited to e-Cigarettes as the cause. This is predominantly because of the recent introduction (2004 in China and 2006 in the US) and limited longitudinal studies conducted on vapers. Recent evidence, however, uncovers a link between toxic chemical exposure from e-cigarettes and long-term effects on cardiovascular function. There are a range of interconnected pathophysiological mechanisms proposed. The primary ones are endothelial cell dysfunction and oxidative stress. There are also considerations of vascular injury and inflammation. Short-term e-cigarette use in non-smokers can lead to reduced myocardial perfusion, ischemia, and fibrotic remodeling over time, leading to cardiomyopathy.

Preliminary studies in mice have been conducted by developing a model that delivers chemicals in mice like those of human e-cigarette users. E-cigarettes of concentrations of 2.4% [A] and 0% [B] were used. Echocardiography studies at 12 weeks showed a decreased left ventricular ejection fraction of 60±2% in the 2.4% group [A] against 70±7% of the 0% group [B]. LV fractional Shortening in the 2.4% [A] group was 27±1% compared to 34±4% in the 0% group [B]. Ventricular Transcriptomic analysis revealed changes in genes associated with metabolism, circadian rhythm, and inflammation in the 2.4% mice group [A]. Transcription electron microscopy also uncovered that cardiomyocytes of mice in the 2.4% group [A] indicated microstructural abnormalities suggestive of systolic dysfunction without hypertrophy or diastolic impairment; suggestive of early changes of cardiomyopathy [7].

One in vitro study conducted on Human Induced Pluripotent Stem Cell-derived Cardiomyocytes (hiPSC-CM) demonstrates that e-cigarette extract (ECE) and cigarette smoke extract (CSE) both prolong calcium decay time and increase time to peak calcium. This indicates slower clearance and impaired handling of calcium within heart muscles. This has eventually demonstrated a reduction in contraction amplitude. A 48-hour exposure to smoke extracts raised cardiomyocyte apoptosis and cell death rates. These findings suggest that e-cigarette-derived toxicants impair cardiomyocyte calcium cycling, contractility, and viability, thereby giving evidence of progression to cardiomyopathy [9].

All the human data we have is a few case reports. These cases are especially concerning because they have happened in otherwise healthy people after acute e-cigarette use. A healthy 35-year-old bodybuilder developed severe dilated cardiomyopathy with an ejection fraction of 32%. He presented with heart failure and EVALI. All other causes were ruled out, supporting e-cigarettes as the most probable etiology of his condition [98]. Another case series showed 2 patients (42-year-old and 48-year-old), developing non-ischemic cardiomyopathy after 1 and 3 months of use, respectively [99].

The existence of cardiomyopathy cases in otherwise healthy young individuals following e-cigarette use is striking, but the evidence base remains too sparse to establish causality. The fundamental challenge is that cardiomyopathy develops over months to years, while the longitudinal studies needed to track this progression in e-cigarette users do not yet exist. Clinicians are therefore left in the uncomfortable position of managing a plausible but unquantified risk. In this context, a high index of suspicion is warranted - particularly in young patients with unexplained cardiac dysfunction and a vaping history - even in the absence of definitive population-level proof.

Limitations of current evidence

Despite increased interest in the cardiovascular consequences of e-cigarette use, current evidence is restricted by methodological challenges. Recent studies have accurately recompiled acute or short-term effects (HR, BP, ECG intervals) in both animals and humans; long-term follow-up for clinical outcomes such as arrhythmias, myocardial infarction, or cardiomyopathy is still scarce [92, 100]. The follow-up periods are often short, given the recent introduction of e-cigarettes, making multi-decade cohort data unavailable [101]. Another limiting factor is the variability of dosing protocols, which results in inaccurate real-world exposure recreation, obscured cumulative dose-response effects or progressive cardiac remodeling, and poor generalization of outcomes. Another significant limitation is variation in device characteristics and e-liquid formulations. The variability in nicotine concentration, solvent ratios, flavor additives, and heating coil composition affects the delivery of toxicants such as aldehydes and metals, making cross-study comparability and generalizability difficult.

Much of the research available now is on preclinical in vitro or animal studies, such as isolated cardiomyocytes and rodent exposures, which can only mimic human autonomic regulation or the long-term remodeling to a certain point [89]. Most clinical studies are often small, limiting generalizability. The PATH longitudinal study found no association between e-cigarette exclusive use and cardiovascular disease; this could have been due to the low number of e-cigarette exclusive users and poor reliance on self-reported myocardial infarction and stroke [102]. Perhaps the most fundamental limitation is the mismatch between the pace of e-cigarette market evolution and the pace of scientific investigation. New device generations, nicotine salt formulations, and flavoring compounds reach consumers years before their cardiovascular effects can be meaningfully studied. This means the evidence base will perpetually lag real-world exposure, placing clinicians and regulators in a position of making consequential decisions under chronic uncertainty. Acknowledging this structural limitation is essential to interpreting the current literature honestly.

Public health and clinical implications

E-cigarette use has been associated with rapid heart rate and an increase in blood pressure, oxidative stress, and endothelial damage and inflammation, which are well-established as evident from recent research and reviews [93, 94, 103-106]. The scientific statements of the American Heart Association reported that youth who use E-cigarettes show rapid hemodynamic changes, increased arterial stiffness, and arterial dysfunction, with concerns that these effects can accumulate and add to their long-term cardiovascular risk [101, 107]. These side effects are associated with nicotine but also with other compounds such as flavorings and aldehydes [104, 106, 108].

Although some studies indicate that the transition from traditional combustible cigarettes to e-cigarettes can yield modest benefits in select cardiovascular measurements, such as circulation and blood pressure, particularly in the younger population, e-cigarettes are not devoid

of risk, and they may still produce a pro-atherosclerotic, pro-arrhythmogenic, and other cardiovascular effects [89, 103, 105, 108]. The lack of long-term safety data on e-cigarettes, as well as the high rates of continued use, are given as reasons why the American College of Cardiology, in its joint guideline, does not recommend e-cigarettes as first-line therapy to assist with smoking cessation, and calls for further research into quitting strategies and CVD effects [109]. Consensus statements from the American Heart Association and other societies have clarified that e-cigarettes are not a safe alternative to conventional tobacco use, particularly for youth and non-smokers, and have called for immediate long-term epidemiological studies [101, 107, 109]. Key controversies and gaps include paucity of long-term data, differences in device compositions, and uncertain effects of chronic exposure, especially in adolescents and dual users [101, 103, 106, 110].

E-cigarettes are not beneficial to cardiovascular health and are associated with an increased risk of hypertension, arrhythmias, and cardiovascular disease, particularly in young people. Complete cessation of all forms of tobacco products is best for smokers, with FDA-approved cessation medications safer than e-cigarettes and having the most current, best evidence base for safety and efficacy. Products must not be used concurrently with combustible tobacco product use, and e-cigarettes must not be used by non-smokers or by adolescents. Uncertainty exists on the long-term cardiovascular impacts, requiring further studies.

Conclusion

E-cigarettes pose a real but incompletely defined cardiovascular risk. They are not the safe alternative to combustible cigarettes that early marketing claimed. Experimental and clinical studies consistently link their use to autonomic imbalance, oxidative stress, endothelial dysfunction, and disrupted cardiac electrophysiology.

What this body of evidence shows, taken together, is a technology that changes the cardiovascular risk equation rather than removing it - trading one set of hazards for another rather than eliminating harm. The field still lacks the long-term data needed to size that risk precisely: most studies remain short-term, samples stay small, and device generations and e-liquid formulations vary too wide for clean comparison. No large prospective cohort has tracked e-cigarette users the way decades of research have tracked combustible smokers, so clinicians and regulators are left weighing a credible signal of harm without the hard endpoints to quantify it.

Clinicians should treat vaping as a genuine cardiovascular risk factor, not a benign habit, particularly in adolescents and young adults. Future research should prioritize longitudinal studies with standardized exposure measures and direct comparisons between exclusive e-cigarette use and dual use, to establish the dose-response data this scenario now demands.

Disclosure of conflict of interest

None.

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References

[1] Jerzyński T, Stimson GV, Shapiro H and Król G. Estimation of the global number of e-cigarette users in 2020. Harm Reduct J 2021; 18: 109.

[2] Prochaska JJ, Vogel EA and Benowitz N. Nicotine delivery and cigarette equivalents from vaping a JUULpod. Tob Control 2022; 31: e88-e93.

[3] Espinoza-Derout J, Shao XM, Lao CJ, Hasan KM, Rivera JC, Jordan MC, Echeverria V, Roos KP, Sinha-Hikim AP and Friedman TC. Electronic cigarette use and the risk of cardiovascular diseases. Front Cardiovasc Med 2022; 9: 879726.

[4] Centers for Disease Control and Prevention. QuickStats: percentage distribution of cigarette smoking status among current adult e-cigarette users, by age group - national health interview survey, United States, 2021. MMWR Morb Mortal Wkly Rep 2023; 72: 270.

[5] Savko C, Esquer C AS, Molinaro C, Rokaw S, Shain AG, Jaafar F, Wright MK, Phillips JA, Hop-

kins T, Mikhail S, Rieder A, Mardani A, Bailey B and Sussman MA. Myocardial infarction injury is exacerbated by nicotine in vape aerosol exposure. J Am Heart Assoc 2025; 14: e038012.
[6] Lee WH, Ong SG, Zhou Y, Tian L, Bae HR, Baker N, Whitlatch A, Mohammadi L, Guo H, Nadeau KC, Springer ML, Schick SF, Bhatnagar A and Wu JC. Modeling cardiovascular risks of e-cigarettes with human-induced pluripotent stem cell-derived endothelial cells. J Am Coll Cardiol 2019; 73: 2722-37.
[7] Espinoza-Derout J, Hasan KM, Shao XM, Jordan MC, Sims C, Lee DL, Sinha S, Simmons Z, Mtume N, Liu Y, Roos KP, Sinha-Hikim AP and Friedman TC. Chronic intermittent electronic cigarette exposure induces cardiac dysfunction and atherosclerosis in apolipoprotein-E knockout mice. Am J Physiol Heart Circ Physiol 2019; 317: H445-H59.
[8] Simms L, Yu F, Palmer J, Rudd K, Sticken ET, Wieczorek R, Chapman F, Czekala L, Stevenson M and O'Connell G. Use of human induced pluripotent stem cell-derived cardiomyocytes to predict the cardiotoxicity potential of next generation nicotine products. Front Toxicol 2022; 4: 747508.
[9] Tatekoshi Y, Chen C, Shapiro JS, Chang HC, Blancard M, Lyra-Leite DM, Burridge PW, Feinstein M, D'Aquila R, Hsue P and Ardehali H. Human induced pluripotent stem cell-derived cardiomyocytes to study inflammation-induced aberrant calcium transient. ELife 2024; 13: RP95867.
[10] Marques P, Piqueras L and Sanz MJ. An updated overview of e-cigarette impact on human health. Respir Res 2021; 22: 151.
[11] Herbst RS, Hatsukami D, Acton D, Giuliani M, Moushey A, Phillips J, Sherwood S, Toll BA, Viswanath K, Warren NJH, Warren GW and Alberg AJ. Electronic nicotine delivery systems: an updated policy statement from the American association for cancer research and the American society of clinical oncology. Clin Cancer Res 2022; 28: 4861-4870.
[12] Sala M and Gotti C. Electronic nicotine delivery systems (ENDS): a convenient means of smoking? Pharmacol Res 2023; 195: 106885.
[13] Ozga JE, Felicione NJ, Douglas A, Childers M and Blank MD. Electronic cigarette terminology: where does one generation end and the next begin? Nicotine Tob Res 2022; 24: 421-424.
[14] Strongin RM, Sharma E, Erythropel HC, El-Hellani A, Kassem NOF, Mikheev VB, Noël A, Peyton DH and Springer ML. Emerging ENDS products and challenges in tobacco control toxicity research. Tob Control 2023; 33: 110-115.
[15] Reilly SM, Cheng T, Feng C and Walters MJ. Harmful and potentially harmful constituents in e-liquids and aerosols from electronic nicotine delivery systems (ENDS). Chem Res Toxicol 2024; 37: 1155-1170.
[16] Shehata SA, Toraih EA, Ismail EA, Hagras AM, Elmorsy E and Fawzy MS. Vaping, environmental toxicants exposure, and lung cancer risk. Cancers (Basel) 2023; 15: 4525.
[17] Wang C, Li W, Zeng Y, Wu Y, Chen D, Meng H, Zhang K, Zhang X, Li G and Liu K. Determination of four tobacco-specific nitrosamines in electronic cigarette liquids and aerosols by UPLC-QTOF-HRMS. Chin J Anal Chem 2024; 52: 100430.
[18] Lechasseur A, Altmejd S, Turgeon N, Buonanno G, Morawska L, Brunet D, Duchaine C and Morissette MC. Variations in coil temperature/power and e-liquid constituents change size and lung deposition of particles emitted by an electronic cigarette. Physiol Rep 2019; 7: e14093.
[19] Margham J, McAdam K, Cunningham A, Porter A, Fiebelkorn S, Mariner D, Digard H and Proctor C. The chemical complexity of e-cigarette aerosols compared with the smoke from a tobacco burning cigarette. Front Chem 2021; 9: 743060.
[20] Talih S, Balhas Z, Eissenberg T, Salman R, Karaoghlanian N, El Hellani A, Baalbaki R, Saliba N and Shihadeh A. Effects of user puff topography, device voltage, and liquid nicotine concentration on electronic cigarette nicotine yield: measurements and model predictions. Nicotine Tob Res 2015; 17: 150-7.
[21] Harris T. Physical and chemical characterization of aerosols produced from commercial nicotine salt-based e-liquids. Chem Res Toxicol 2025; 38: 115-128.
[22] Benowitz NL. Pharmacology of nicotine: addiction, smoking-induced disease, and therapeutics. Annu Rev Pharmacol Toxicol 2009; 49: 57-71.
[23] Zhang L, Dong Y, Doyon WM and Dani JA. Withdrawal from chronic nicotine exposure alters dopamine signaling dynamics in the nucleus accumbens. Biol Psychiatry 2012; 71: 184-91.
[24] D'Alessandro A, Boeckelmann I, Hammwhöner M and Goette A. Nicotine, cigarette smoking and cardiac arrhythmia: an overview. Eur J Prev Cardiol 2012; 19: 297-305.
[25] Lee J and Cooke JP. The role of nicotine in the pathogenesis of atherosclerosis. Atherosclerosis 2011; 215: 281-3.
[26] Voos N, Goniewicz ML and Eissenberg T. What is the nicotine delivery profile of electronic cigarettes? Expert Opin Drug Deliv 2019; 16: 1193-1203.
[27] Gholap VV, Kosmider L, Golshahi L and Halquist MS. Nicotine forms: why and how do they matter in nicotine delivery from electronic

cigarettes? Expert Opin Drug Deliv 2020; 17: 1727-1736.
[28] Eddingsaas NC, Hensel EC, O'Dea S, Kunselman P, DiFrancesco AG and Robinson RJ. Effect of user puffing topography on total particulate matter, nicotine and volatile carbonyl emissions from narghile waterpipes. Tob Control 2020; 29 Suppl 2: s117-s122.
[29] Li L, Lee ES, Nguyen C and Zhu Y. Effects of propylene glycol, vegetable glycerin, and nicotine on emissions and dynamics of electronic cigarette aerosols. Aerosol Sci Technol 2020; 54: 1270-1281.
[30] Jaegers NR, Hu W, Weber TJ and Hu JZ. Low-temperature (< 200 degrees) degradation of electronic nicotine delivery system liquids generates toxic aldehydes. Sci Rep 2021; 11: 7800.
[31] Zhang Y, Yang Y, He X, Yang P, Zong T, Sun P, Sun RC, Yu T and Jiang Z. The cellular function and molecular mechanism of formaldehyde in cardiovascular disease and heart development. J Cell Mol Med 2021; 25: 5358-5371.
[32] DeJarnett N, Conklin DJ, Riggs DW, Myers JA, O'Toole TE, Hamzeh I, Wagner S, Chugh A, Ramos KS, Srivastava S, Higdon D, Tollerud DJ, DeFilippis A, Becher C, Wyatt B, McCracken J, Abplanalp W, Rai SN, Ciszewski T, Xie Z, Yeager R, Prabhu SD and Bhatnagar A. Acrolein exposure is associated with increased cardiovascular disease risk. J Am Heart Assoc 2014; 3: e000934.
[33] Brandt M, Garlapati V, Oelze M, Sotiriou E, Knorr M, Kröller-Schön S, Kossmann S, Schönfelder T, Morawietz H, Schulz E, Schultheiss HP, Daiber A, Münzel T and Wenzel P. NOX2 amplifies acetaldehyde-mediated cardiomyocyte mitochondrial dysfunction in alcoholic cardiomyopathy. Sci Rep 2016; 6: 32554.
[34] Kosmider L, Sobczak A, Fik M, Knysak J, Zaciera M, Kurek J and Goniewicz ML. Carbonyl compounds in electronic cigarette vapors: effects of nicotine solvent and battery output voltage. Nicotine Tob Res 2014; 16: 1319-26.
[35] Ogunwale MA, Li M, Ramakrishnam Raju MV, Chen Y, Nantz MH, Conklin DJ and Fu XA. Aldehyde detection in electronic cigarette aerosols. ACS Omega 2017; 2: 1207-1214.
[36] Kim MD, Chung S, Dennis JS, Yoshida M, Aguiar C, Aller SP, Mendes ES, Schmid A, Sabater J, Baumlin N and Salathe M. Vegetable glycerin e-cigarette aerosols cause airway inflammation and ion channel dysfunction. Front Pharmacol 2022; 13: 1012723.
[37] Batty M, Bennett MR and Yu E. The role of oxidative stress in atherosclerosis. Cells 2022; 11: 3843.
[38] Sachdeva J, Karunananthan A, Shi J, Dai W, Kleinman MT, Herman D and Kloner RA. Flavoring agents in e-cigarette liquids: a comprehensive analysis of multiple health risks. Cureus 2023; 15: e48995.
[39] Stefaniak AB, LeBouf RF, Ranpara AC and Leonard SS. Toxicology of flavoring- and cannabis-containing e-liquids used in electronic delivery systems. Pharmacol Ther 2021; 224: 107838.
[40] Farsalinos KE, Gillman G, Poulas K and Voudris V. Tobacco-specific nitrosamines in electronic cigarettes: comparison between liquid and aerosol levels. Int J Environ Res Public Health 2015; 12: 9046-53.
[41] Jin XC, Wagner KA, Melvin MS, Smith DC, Pithawalla YB, Gardner WP, Avery KC and Karles GD. Influence of nitrite on formation of tobacco-specific nitrosamines in electronic cigarette liquids and aerosols. Chem Res Toxicol 2022; 35: 782-791.
[42] Olmedo P, Goessler W, Tanda S, Grau-Perez M, Jarmul S, Aherrera A, Chen R, Hilpert M, Cohen JE, Navas-Acien A and Rule AM. Metal concentrations in e-cigarette liquid and aerosol samples: the contribution of metallic coils. Environ Health Perspect 2018; 126: 027010.
[43] Zhao D, Aravindakshan A, Hilpert M, Olmedo P, Rule AM, Navas-Acien A and Aherrera A. Metal/metalloid levels in electronic cigarette liquids, aerosols, and human biosamples: a systematic review. Environ Health Perspect 2020; 128: 36001.
[44] Middlekauff HR, Park J and Moheimani RS. Adverse effects of cigarette and noncigarette smoke exposure on the autonomic nervous system: mechanisms and implications for cardiovascular risk. J Am Coll Cardiol 2014; 64: 1740-50.
[45] Dimitriadis K, Narkiewicz K, Leontsinis I, Konstantinidis D, Mihas C, Andrikou I, Thomopoulos C, Tousoulis D and Tsioufis K. Acute effects of electronic and tobacco cigarette smoking on sympathetic nerve activity and blood pressure in humans. Int J Environ Res Public Health 2022; 19: 3237.
[46] Fetterman JL, Weisbrod RM, Feng B, Bastin R, Tuttle ST, Holbrook M, Baker G, Robertson RM, Conklin DJ, Bhatnagar A and Hamburg NM. Flavorings in tobacco products induce endothelial cell dysfunction. Arterioscler Thromb Vasc Biol 2018; 38: 1607-1615.
[47] Erythropel HC, Jabba SV, DeWinter TM, Mendizabal M, Anastas PT, Jordt SE and Zimmerman JB. Formation of flavorant-propylene glycol adducts with novel toxicological properties in chemically unstable e-cigarette liquids. Nicotine Tob Res 2019; 21: 1248-1258.

[48] Jabba SV, Diaz AN, Erythropel HC, Zimmerman JB and Jordt SE. Chemical adducts of reactive flavor aldehydes formed in e-cigarette liquids are cytotoxic and inhibit mitochondrial function in respiratory epithelial cells. Nicotine Tob Res 2020; 22 Suppl 1: S25-S34.
[49] Ji EH, Sun B, Zhao T, Shu S, Chang CH, Messadi D, Xia T, Zhu Y and Hu S. Characterization of electronic cigarette aerosol and its induction of oxidative stress response in oral keratinocytes. PLoS One 2016; 11: e0154447.
[50] Putzhammer R, Doppler C, Jakschitz T, Heinz K, Forste J, Danzl K, Messner B and Bernhard D. Vapours of US and EU market leader electronic cigarette brands and liquids are cytotoxic for human vascular endothelial cells. PLoS One 2016; 11: e0157337.
[51] Connor Gorber S, Schofield-Hurwitz S, Hardt J, Levasseur G and Tremblay M. The accuracy of self-reported smoking: a systematic review of the relationship between self-reported and cotinine-assessed smoking status. Nicotine Tob Res 2009; 11: 12-24.
[52] Moheimani RS, Bhetraratana M, Yin F, Peters KM, Gornbein J, Araujo JA and Middlekauff HR. Increased cardiac sympathetic activity and oxidative stress in habitual electronic cigarette users: implications for cardiovascular risk. JAMA Cardiol 2017; 2: 278-284.
[53] D'Amario D, Migliaro S, Borovac JA, Vergallo R, Galli M, Restivo A, Bonini M, Romagnoli E, Leone AM and Crea F. Electronic cigarettes and cardiovascular risk: caution waiting for evidence. Eur Cardiol 2019; 14: 151-158.
[54] Zheng M, Erhardt S, Cao Y and Wang J. Emerging signaling regulation of sinoatrial node dysfunction. Curr Cardiol Rep 2023; 25: 621-630.
[55] Kalyanasundaram A, Li N, Augostini RS, Weiss R, Hummel JD and Fedorov VV. Three-dimensional functional anatomy of the human sinoatrial node for epicardial and endocardial mapping and ablation. Heart Rhythm 2023; 20: 122-133.
[56] Perez-Riera AR, Barbosa-Barros R, Daminello-Raimundo R, de Abreu LC and Nikus K. Current aspects of the basic concepts of the electrophysiology of the sinoatrial node. J Electrocardiol 2019; 57: 112-118.
[57] Kingma J, Simard C and Drolet B. Overview of cardiac arrhythmias and treatment strategies. Pharmaceuticals (Basel) 2023; 16: 844.
[58] Witczyńska A, Alaburda A, Grześk G, Nowaczyk J and Nowaczyk A. Unveiling the multifaceted problems associated with dysrhythmia. Int J Mol Sci 2023; 25: 263.
[59] Kucera C, Ramalingam A, Srivastava S, Bhatnagar A and Carll AP. Nicotine formulation influences the autonomic and arrhythmogenic effects of electronic cigarettes. Nicotine Tob Res 2024; 26: 536-544.
[60] Abou-Assali O, Ebner M, Chams J, Chang M, Zhang Y, Calcul L, Chidipi B and Noujaim S. Inhalation exposure to flavored electronic nicotine delivery systems (vaping) negatively impacts cardiac electrophysiology. EP Europace 2024; 26.
[61] Mohammed Abdul KS, Han K, Guerrero AB, Wilson CN, Kulkarni A and Purcell NH. Increased PHLPP1 expression through ERK-4E-BP1 signaling axis drives nicotine induced oxidative stress related damage of cardiomyocytes. J Mol Cell Cardiol 2024; 193: 100-112.
[62] El-Mahdy MA, Ewees MG, Eid MS, Mahgoup EM, Khaleel SA and Zweier JL. Electronic cigarette exposure causes vascular endothelial dysfunction due to NADPH oxidase activation and eNOS uncoupling. Am J Physiol Heart Circ Physiol 2022; 322: H549-H567.
[63] Ip M, Diamantakos E, Haptonstall K, Choroomi Y, Moheimani RS, Nguyen KH, Tran E, Gornbein J and Middlekauff HR. Tobacco and electronic cigarettes adversely impact ECG indexes of ventricular repolarization: implication for sudden death risk. Am J Physiol Heart Circ Physiol 2020; 318: H1176-H1184.
[64] Nguyen R, Ruedisueli I, Lakhani K, Ma J and Middlekauff HR. Acute cardiovascular effects of 4th generation electronic cigarettes and combusted cigarettes: implications for harm reduction. J Appl Physiol (1985) 2024; 136: 440-449.
[65] Whitaker J, Wright MJ and Tedrow U. Diagnosis and management of ventricular tachycardia. Clin Med (Lond) 2023; 23: 442-8.
[66] Abedin Z. Differential diagnosis of wide QRS tachycardia: a review. J Arrhythm 2021; 37: 1162-1172.
[67] Irfan AB, Arab C, DeFilippis AP, Lorkiewicz P, Keith RJ, Xie Z, Bhatnagar A and Carll AP. Smoking accelerates atrioventricular conduction in humans concordant with increased dopamine release. Cardiovasc Toxicol 2021; 21: 169-178.
[68] Ruedisueli I, Shi K, Lopez S, Gornbein J and Middlekauff HR. Arrhythmogenic effects of acute electronic cigarette compared to tobacco cigarette smoking in people living with HIV. Physiol Rep 2024; 12: e16158.
[69] Moheimani RS, Bhetraratana M, Peters KM, Yang BK, Yin F, Gornbein J, Araujo JA and Middlekauff HR. Sympathomimetic Effects of acute e-cigarette use: role of nicotine and non-nicotine constituents. J Am Heart Assoc 2017; 6: e006579.
[70] Ammar H, Hashish R, Ali SM, Salem AS and Hagras AM. A comparison of electrocardiographic parameters in e-cigarette users, conventional cigarette smokers, and non-smokers. Egypt J Intern Med 2024; 36.

[71] Carll AP, Arab C, Salatini R, Miles MD, Nystoriak MA, Fulghum KL, Riggs DW, Shirk GA, Theis WS, Talebi N, Bhatnagar A and Conklin DJ. E-cigarettes and their lone constituents induce cardiac arrhythmia and conduction defects in mice. Nat Commun 2022; 13: 6088.
[72] Abouassali O, Chang M, Chidipi B, Martinez JL, Reiser M, Kanithi M, Soni R, McDonald TV, Herweg B, Saiz J, Calcul L and Noujaim SF. In vitro and in vivo cardiac toxicity of flavored electronic nicotine delivery systems. Am J Physiol Heart Circ Physiol 2021; 320: H133-H143.
[73] Motiejunaite J, Amar L and Vidal-Petiot E. Adrenergic receptors and cardiovascular effects of catecholamines. Ann Endocrinol (Paris) 2021; 82: 193-197.
[74] Sved AF, Weeks JJ, Grace AA, Smith TT and Donny EC. Monoamine oxidase inhibition in cigarette smokers: from preclinical studies to tobacco product regulation. Front Neurosci 2022; 16: 886496.
[75] Abd S, Al, Phil O, Cardiologst and Omran S. Nicotine adds risk factors to the cardiovascular system and increases mortality and sudden cardiac arrest. Medico Legal Update 2021; 20.
[76] Cekirdekci EI and Bugan B. Can abnormal dispersion of ventricular repolarization be a predictor of mortality in arrhythmogenic right ventricular cardiomyopathy: the importance of Tp-e interval. Ann Noninvasive Electrocardiol 2019; 24: e12619.
[77] Menshov VA, Trofimov AV, Zagurskaya AV, Berdnikova NG, Yablonskaya OI and Platonova AG. Influence of nicotine from diverse delivery tools on the autonomic nervous and hormonal systems. Biomedicines 2022; 10: 121.
[78] Christen SE, Hermann L, Bekka E, Vonwyl C, Hammann F, van der Velpen V, Eap CB, Benowitz NL, Haschke M and Liakoni E. Pharmacokinetics and pharmacodynamics of inhaled nicotine salt and free-base using an e-cigarette: a randomized crossover study. Nicotine Tob Res 2024; 26: 1313-1321.
[79] Han S, Liu C, Chen H, Fu Y, Zhang Y, Miao R, Ren P, Yu P, Shi Z, Tian Y, Wang H, Liu T, Hou H and Hu Q. Pharmacokinetics of freebase nicotine and nicotine salts following subcutaneous administration in male rats. Drug Test Anal 2023; 15: 1099-1106.
[80] Prevette K, Jordan KG, Keyser BM, Kanobe MN, Jin T, Ayoku S, Darnell J, Makena P and Baxter SA. Randomized clinical studies assessing the pharmacokinetics and subjective effects of vuse alto electronic nicotine delivery systems. Sci Rep 2025; 15: 21711.
[81] Nagpal AK, Pundkar A, Singh A and Gadkari C. Cardiac arrhythmias and their management: an in-depth review of current practices and emerging therapies. Cureus 2024; 16: e66549.
[82] Fatih Gündüz A and Fatih Talu M. Atrial fibrillation classification and detection from ECG recordings. Biomed Signal Process Control 2023; 82: 104531.
[83] Kistler PM, Chieng D, Tonchev IR, Sugumar H, Voskoboinik A, Schwartz LA, McLellan AJ, Prabhu S, Ling LH, Al-Kaisey A, Parameswaran R, Anderson RD, Lee G and Kalman JM. P-wave morphology in focal atrial tachycardia: an updated algorithm to predict site of origin. JACC Clin Electrophysiol 2021; 7: 1547-1556.
[84] Candia-Rivera D and Machado C. Reduced heartbeat-evoked responses in a near-death case report. J Clin Neurol 2023; 19: 581-588.
[85] Vera-Sarmiento HL, Tanriverdi T, Hurtado-de-Mendoza D, Sivalokanathan S, Damera RR, Ketty D, Lu D, Zimmerman S, Sinha S, Scheinman M and Abraham MR. QRS prolongation is associated with associated with adverse cardiac remodeling in hypertrophic cardiomyopathy. J Electrocardiol 2024; 87: 153818.
[86] Adameova A, Shah AK and Dhalla NS. Role of oxidative stress in the genesis of ventricular arrhythmias. Int J Mol Sci 2020; 21: 4200.
[87] Rababa'h AM, Guillory AN, Mustafa R and Hijjawi T. Oxidative stress and cardiac remodeling: an updated edge. Curr Cardiol Rev 2018; 14: 53-59.
[88] Mongirdienė A, Skrodenis L, Varoneckaitė L, Mierkytė G and Gerulis J. Reactive oxygen species induced pathways in heart failure pathogenesis and potential therapeutic strategies. Biomedicines 2022; 10: 602.
[89] Jones CA, Wallace MJ, Bandaru P, Woodbury ED, Mohler PJ and Wold LE. E-cigarettes and arrhythmogenesis: a comprehensive review of pre-clinical studies and their clinical implications. Cardiovasc Res 2023; 119: 2157-2164.
[90] Clapp PW, Lavrich KS, van Heusden CA, Lazarowski ER, Carson JL and Jaspers I. Cinnamaldehyde in flavored e-cigarette liquids temporarily suppresses bronchial epithelial cell ciliary motility by dysregulation of mitochondrial function. Am J Physiol Lung Cell Mol Physiol 2019; 316: L470-L486.
[91] Lerner CA, Sundar IK, Yao H, Gerloff J, Ossip DJ, McIntosh S, Robinson R and Rahman I. Vapors produced by electronic cigarettes and e-juices with flavorings induce toxicity, oxidative stress, and inflammatory response in lung epithelial cells and in mouse lung. PLoS One 2015; 10: e0116732.
[92] Bitar M, Mercier C, Bertoletti L, Pourchez J and Forest V. Flavor-induced inflammation and cytotoxicity in human aortic smooth muscle cells: potential implications for e-cigarette safety. Toxicol Appl Pharmacol 2025; 500: 117388.
[93] Mears MJ, Hookfin HL, Bandaru P, Vidal P, Stanford KI and Wold LE. Electronic nicotine deliv-

ery systems and cardiovascular/cardiometabolic health. Circ Res 2023; 132: 1168-1180.
[94] Emma R, Caruso M, Campagna D, Pulvirenti R and Li Volti G. The impact of tobacco cigarettes, vaping products and tobacco heating products on oxidative stress. Antioxidants (Basel) 2022; 11: 1829.
[95] Frazier J, Coblentz T, Bruce J, Nassabeh S, Plants R, Burrage E, Mills A, Chantler P and Olfert I. Effect of e-liquid base solution (Vegetable Glycerin or Propylene Glycol) on aortic function in mice. FASEB J 2021; 35.
[96] Kim MD, Chung S, Baumlin N, Qian J, Montgomery RN, Sabater J, Berkland C and Salathe M. The combination of propylene glycol and vegetable glycerin e-cigarette aerosols induces airway inflammation and mucus hyperconcentration. Sci Rep 2024; 14: 1942.
[97] Sinagra G, Carriere C, Clemenza F, Mina C, Bandera F, Zaffalon D, Gugliandolo P, Merlo M, Guazzi M and Agostoni P. Risk stratification in cardiomyopathy. Eur J Prev Cardiol 2020; 27: 52-58.
[98] Brennan E, Kahn A, Kopyt M, Khan A and Castillo R. Don't go vaping my heart: a case of vaping-associated cardiomyopathy and lung injury. Cureus 2023; 15: e42723.
[99] Benji LIM, Sheldon LEE and Siang Chew C. Vaping induced cardiac complications - a smokescreen coming to engulf the heart? Int J Clin Cardiol 2020; 7.
[100] Yacoub MI, Aslanoğlu A, Khraim F, Alsharawneh A, Abdelkader R, Almagharbeh WT, Alhejaili AA, Alshahrani BD, Sobeh DE, Eltayeb MM, Elshatarat RA and Saleh ZT. Comparing e-cigarettes and traditional cigarettes in relation to myocardial infarction, arrhythmias, and sudden cardiac death: a systematic review and meta-analysis. Biol Res Nurs 2025; 27: 168-185.
[101] Rose JJ, Krishnan-Sarin S, Exil VJ, Hamburg NM, Fetterman JL, Ichinose F, Perez-Pinzon MA, Rezk-Hanna M and Williamson E; American Heart Association Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation; Council on Epidemiology and Prevention; Council on Cardiovascular Radiology and Intervention; Council on Lifestyle and Cardiometabolic Health; Council on Peripheral Vascular Disease; Stroke Council; Council on Arteriosclerosis, Thrombosis and Vascular Biology. Cardiopulmonary impact of electronic cigarettes and vaping products: a scientific statement from the American heart association. Circulation 2023; 148: 703-728.
[102] Berlowitz JB, Xie W, Harlow AF, Hamburg NM, Blaha MJ, Bhatnagar A, Benjamin EJ and Stokes AC. E-cigarette use and risk of cardiovascular disease: a longitudinal analysis of the PATH study (2013-2019). Circulation 2022; 145: 1557-1559.
[103] Kundu A, Feore A, Sanchez S, Abu-Zarour N, Sutton M, Sachdeva K, Seth S, Schwartz R and Chaiton M. Cardiovascular health effects of vaping e-cigarettes: a systematic review and meta-analysis. Heart 2025; 111: 599-608.
[104] Zong H, Hu Z, Li W, Wang M, Zhou Q, Li X and Liu H. Electronic cigarettes and cardiovascular disease: epidemiological and biological links. Pflugers Arch 2024; 476: 875-888.
[105] Critselis E and Panagiotakos D. Impact of electronic cigarette use on cardiovascular health: current evidence, causal pathways, and public health implications. Angiology 2024; 75: 417-424.
[106] Daiber A, Kuntic M, Oelze M, Hahad O and Münzel T. E-cigarette effects on vascular function in animals and humans. Pflugers Arch 2023; 475: 783-796.
[107] Wold LE, Tarran R, Crotty Alexander LE, Hamburg NM, Kheradmand F, St Helen G and Wu JC; American Heart Association Council on Basic Cardiovascular Sciences; Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Hypertension; and Stroke Council. Cardiopulmonary consequences of vaping in adolescents: a scientific statement from the American heart association. Circ Res 2022; 131: e70-e82.
[108] Magna A, Polisena N, Polisena L, Bagnato C, Pacella E, Carnevale R, Nocella C and Loffredo L. The hidden dangers: e-cigarettes, heated tobacco, and their impact on oxidative stress and atherosclerosis-a systematic review and narrative synthesis of the evidence. Antioxidants (Basel) 2024; 13: 1395.
[109] Virani SS, Newby LK, Arnold SV, Bittner V, Brewer LC, Demeter SH, Dixon DL, Fearon WF, Hess B, Johnson HM, Kazi DS, Kolte D, Kumbhani DJ, LoFaso J, Mahtta D, Mark DB, Minissian M, Navar AM, Patel AR, Piano MR, Rodriguez F, Talbot AW, Taqueti VR, Thomas RJ, van Diepen S, Wiggins B and Williams MS; Peer Review Committee Members. 2023 AHA/ACC/ACCP/ASPC/NLA/PCNA Guideline for the management of patients with chronic coronary disease: a report of the American heart association/American college of cardiology joint committee on clinical practice guidelines. Circulation 2023; 148: e9-e119.
[110] Neczypor EW, Mears MJ, Ghosh A, Sassano MF, Gumina RJ, Wold LE and Tarran R. E-cigarettes and cardiopulmonary health: review for clinicians. Circulation 2022; 145: 219-232.