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CARDIOVASCULAR AND METABOLIC IMPLICATIONS OF E-CIGARETTE EXPOSURE: A COMPREHENSIVE REVIEW OF CURRENT EVIDENCE

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ABSTRACT

Electronic cigarettes (e-cigarettes) have surged in global popularity as perceived safer alternatives to combustible tobacco, yet growing evidence identifies them as an independent cardiovascular and metabolic risk factor. To synthesize current knowledge, a systematic review was conducted across major databases (PubMed, Web of Science, Scopus) for high-quality studies published between 2016 and early 2026, including large-scale epidemiological data from the BRFSS and the Korean Community Health Survey. The analysis reveals that e-cigarette exposure induces acute adverse hemodynamic responses, significantly elevating blood pressure, heart rate, and cardiac workload, while chronic use leads to sustained autonomic imbalance with sympathetic predominance. At the vascular level, vaping exacerbates oxidative stress, increases arterial stiffness, and promotes a pro-inflammatory and pro-thrombotic environment. Crucially, across all cardiometabolic parameters, the most detrimental health outcomes are consistently observed among "dual users" who concurrently use both e-cigarettes and traditional combustible tobacco. Ultimately, this review refutes the narrative that e-cigarettes are a benign harm-reduction tool, concluding that they pose a significant, documented threat to cardiovascular and metabolic health. Consequently, clinical and public health prevention strategies must strongly advocate for the total cessation of all forms of nicotine delivery to protect long-term cardiovascular well-being.

KEYWORDS

Electronic Cigarettes, Cardiovascular Risk, Cardiometabolic Risk, Hypertension, Endothelial Dysfunction, Dyslipidemia

CITATION

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Introduction

Electronic cigarettes (e-cigarettes), also called electronic nicotine delivery systems (ENDS), have become very popular worldwide over the last ten years. They were first introduced as an alternative to traditional tobacco smoking and have been widely marketed as a safer choice and a useful harm-reduction method. This perception has significantly driven their uptake, particularly among adolescents and young adults. Despite a global decline in combustible cigarette smoking, the use of alternative nicotine products, notably e-cigarettes, has risen steadily, raising profound concerns among public health professionals.

Recent epidemiological findings reveal a sharp worldwide rise in e-cigarette use. The World Health Organization (WHO) estimates that over 100 million people globally use e-cigarettes, including at least 15 million adolescents aged 13–15 years [1]. A 2024 systematic review found that about 16.8% of youths have tried e-cigarettes at least once in their lives, and 4.8% are current users, highlighting the growing popularity of vaping among younger populations [2]. Furthermore, in the WHO European Region - an area traditionally burdened by high tobacco consumption - approximately 4 million adolescents aged 13–15 currently use tobacco, and there has been a recent rise in e-cigarette experimentation [3]. The widespread marketing of e-cigarettes as safe alternatives to traditional tobacco has ingrained societal beliefs in their safety, even as evidence grows that these products pose substantial health risks independently.

Cardiovascular disease (CVD) remains the leading cause of global morbidity and mortality. The pathogenesis of CVD is inextricably linked to modifiable risk factors, which form the cornerstone of disease prevention strategies. Major contributors include hypertension, dyslipidemia, insulin resistance, diabetes mellitus, chronic inflammation, and, especially, smoking. Collectively, these factors precipitate endothelial dysfunction, accelerate atherosclerosis, and inflict long-term vascular damage, culminating in adverse cardiovascular events such as myocardial infarction, stroke, and heart failure [4,5]. Recently, the scientific community has increasingly recognized e-cigarette use as an independent, emerging cardiovascular risk factor. Exposure to nicotine and the complex chemical constituents of e-cigarette aerosols has been mechanistically

linked to sympathetic nervous system overactivity, endothelial impairment, severe oxidative stress, and acute hemodynamic perturbations. Nevertheless, the long-term cardiovascular ramifications of chronic e-cigarette exposure remain incompletely elucidated, and the current literature occasionally presents heterogeneous findings.

Therefore, this study aims to systematically review the current scientific evidence on the association between electronic cigarette use and modifiable cardiovascular risk factors, with particular emphasis on blood pressure, heart rate regulation, endothelial and prothrombotic dysfunction, systemic inflammatory markers, lipid profiles, and glycemic control.

Methods

This comprehensive review evaluated the impact of electronic cigarette use on key modifiable cardiovascular and metabolic risk factors. We systematically searched major electronic databases, including PubMed, Web of Science, and Scopus, for literature published between 2016 and early 2026. The search strategy utilized exposure-related terms such as 'electronic cigarettes', 'e-cigarettes', and 'vaping', combined with specific clinical outcomes including 'blood pressure', 'heart rate', 'endothelial dysfunction', 'oxidative stress', 'inflammation', 'dyslipidemia', and 'diabetes'. To ensure a robust analysis, we incorporated a variety of high-quality human studies, prioritizing systematic reviews, meta-analyses, randomized controlled trials, and large-scale observational or epidemiological studies (such as data from the Behavioral Risk Factor Surveillance System and the Korean Community Health Survey). Articles were selected for inclusion if they explicitly reported on the cardiovascular, vascular, or metabolic effects associated with acute or chronic e-cigarette exposure. We excluded publications that were not written in English, single case reports, and studies that did not specifically address these targeted cardiometabolic outcomes.

Blood Pressure Regulation and Hemodynamic Responses

Elevated blood pressure (BP) is recognized as one of the most critical modifiable risk factors for cardiovascular disease (CVD). A robust and growing body of experimental and observational evidence demonstrates that electronic cigarette (e-cigarette) use induces acute, adverse hemodynamic changes, notably significant elevations in both systolic (SBP) and diastolic blood pressure (DBP) [6] [7].

To differentiate the physiological effects of aerosolized components from those of nicotine, Qazi et al. conducted a 2024 systematic review and meta-analysis that evaluated immediate vascular responses. The analysis revealed that nicotine-containing e-cigarettes significantly elevate SBP and DBP compared to nicotine-free alternatives [6]. While the acute spike in SBP following e-cigarette use was found to be slightly lower than that induced by traditional combustible cigarettes, the impact on DBP remained entirely comparable. These findings suggest that nicotine acts as the primary pressor agent, acutely stimulating the sympathetic nervous system and triggering immediate vasoconstriction. [6]

These acute findings closely align with a 2026 systematic review by Nogueira et al., which concluded that e-cigarette use is consistently associated with impaired blood pressure regulation [7]. The review highlighted that users of both electronic and traditional cigarettes have higher baseline SBP and DBP values compared to non-users, confirming that nicotine exposure - regardless of delivery method - is a fundamental contributor to hypertension risk. Furthermore, several studies in the review reported vaping-induced increases in mean arterial pressure (MAP) and central systolic blood pressure. Elevated central pressure is clinically significant, as it serves as an early indicator of vascular dysfunction and increased arterial stiffness, pointing to heightened cardiovascular strain [7].

Beyond short-term hemodynamic perturbations, longitudinal cohort studies suggest that chronic e-cigarette use substantially increases the risk of sustained hypertension. A large, community-based prospective cohort study with long-term follow-up found that individuals with a history of e-cigarette use (ever-users) had a significantly higher risk of incident hypertension than never-users [8]. Alarmingly, the risk was even higher among current users, strongly indicating a dose-dependent relationship between continued exposure and hypertensive risk [8]. The association between e-cigarette use and incident hypertension was particularly severe among younger demographics and "dual users" (individuals consuming both e-cigarettes and combustible tobacco). This synergistic effect indicates that combined nicotine delivery systems profoundly amplify long-term cardiovascular risk and the trajectory toward chronic hypertension [8].

Heart Rate Dynamics and Autonomic Function

Heart rate (HR) is a critical physiological parameter that reflects autonomic nervous system activity and overall cardiovascular function. Elevated resting HR and exaggerated acute HR responses are recognized as independent predictors of cardiovascular morbidity and mortality because persistent sympathetic activation contributes to endothelial dysfunction, increased myocardial oxygen demand, and the progression of atherosclerosis [10].

Acute exposure to e-cigarette aerosols consistently induces significant hemodynamic shifts. A recent systematic review and meta-analysis by Kundu et al. in 2025 demonstrated that individuals exposed to e-cigarettes had an average HR increase of approximately 11 beats per minute compared to non-users [9]. Although this acute elevation is generally lower than that triggered by conventional combustible cigarettes, it remains clinically significant [9]. The physiological mechanisms and consequences of these acute spikes were further elucidated in a comprehensive 2023 systematic review and meta-analysis conducted by Rahman et al. This review highlighted that vaping triggers a rapid release of catecholamines, resulting in acute HR stimulation and increased myocardial contractility [10]. Consequently, this leads to significantly increased cardiac work and elevated myocardial oxygen demand. Such sudden increases in cardiac workload pose a substantial acute risk, particularly for individuals with underlying or subclinical coronary artery disease [10].

Evidence regarding the long-term effects of e-cigarette use on baseline resting HR presents a more complex clinical picture. Chronic autonomic cardiovascular effects are most reliably assessed using heart rate variability (HRV), a critical metric that reflects the balance between sympathetic and parasympathetic nervous system activity. Studies of chronic e-cigarette users who abstained from vaping prior to testing have demonstrated persistent sympathetic predominance compared with non-vaper controls [11]. This indicates a sustained autonomic imbalance even in the absence of recent nicotine exposure. Importantly, these detrimental HRV alterations frequently occur without significant concomitant changes in baseline resting HR or blood pressure, suggesting that chronic sympathetic activation can progress silently without detection by standard hemodynamic monitoring [11].

Longitudinal data corroborate the observation that long-term e-cigarette use does not necessarily result in sustained resting tachycardia. In prospective follow-up studies conducted by Polosa et al. in 2017, which monitored chronic e-cigarette users over several years, repeated measurements of resting HR showed no significant deviations from those of nicotine-naïve control participants [12]. Ultimately, these findings synthesize a critical clinical narrative: while acute e-cigarette exposure clearly elevates HR and markedly increases short-term cardiac workload, the primary long-term cardiovascular threat lies in chronic autonomic dysregulation and sympathetic overactivity rather than a permanent, macroscopic elevation of baseline heart rate.

Endothelial Dysfunction, Oxidative Stress, and Thrombosis

Endothelial dysfunction is recognized as an early marker of cardiovascular disease (CVD) and plays a fundamental role in the pathogenesis of atherosclerosis and vascular complications. Impairment of endothelial function is characterized by reduced nitric oxide (NO) bioavailability, increased oxidative stress, and a pro-inflammatory and pro-thrombotic vascular environment [14] [15] [16].

A systematic review and meta-analysis of randomized controlled trials conducted by Meng et al. in 2023 evaluated the acute effects of e-cigarette exposure on endothelial function and arterial stiffness. To this end, they compared parameters such as flow-mediated dilation (FMD), pulse wave velocity (PWV), and the augmentation index corrected to a heart rate of 75 beats per minute (AIx75). The pooled results demonstrated no statistically significant differences in FMD between e-cigarette use and traditional cigarette use. Similarly, no statistically significant improvement in FMD was observed when nicotine-containing e-cigarettes were compared with nicotine-free e-cigarettes [13]. These findings suggest that e-cigarettes do not confer protective vascular effects and may impair endothelial function in a manner comparable to conventional tobacco smoking. Furthermore, pulse wave velocity (PWV) and the augmentation index corrected to heart rate (AIx75), which are reliable markers of arterial stiffness, were found to be significantly elevated following acute e-cigarette exposure. The increase in both PWV and AIx75 suggests that e-cigarette use contributes to increased arterial stiffness and exerts adverse effects on hemodynamic function. [13]

The impairment of endothelial function and increased arterial stiffness are mechanistically driven largely by oxidative stress. Evidence from the 2024 systematic review conducted by Magna et al. shows that e-cigarettes increase oxidative stress by activating enzymes such as NADPH oxidase. This activation, triggered by the chemical constituents of e-cigarette aerosols, exacerbates inflammation, reduces NO bioavailability,

and creates a pro-thrombotic and pro-atherogenic environment in endothelial cells and platelets [14]. Recent comprehensive data from a 2025 systematic review and meta-analysis by Padi et al. corroborate these mechanisms. The overall analysis demonstrated that e-cigarette use is consistently associated with increased oxidative stress and vascular stiffness. While the degree of oxidative stress induced by e-cigarettes may be lower compared to conventional cigarette smoking, it remains significantly elevated compared with non-smokers [15]. Studies evaluating specific biomarkers have shown that e-cigarette users exhibit elevated levels of malondialdehyde (MDA) and F2-isoprostanes, which are critical indicators of oxidative damage. Importantly, nicotine is not the sole driver of these adverse effects. Research indicates that even nicotine-free e-cigarettes induce oxidative stress (such as increased MDA concentrations) and arterial stiffness, albeit to a slightly lesser extent than their nicotine-containing counterparts [15].

Beyond endothelial impairment and oxidative stress, e-cigarette aerosols have increasingly been recognized as potent triggers of prothrombotic pathways. Recent meta-analyses indicate that exposure to electronic nicotine delivery systems (ENDS) directly enhances platelet activation and aggregation, central mechanisms driving arterial thrombosis.[16] This hypercoagulable state is inextricably linked to the oxidative stress mechanisms discussed previously; e-cigarette constituents activate enzymes such as NADPH oxidase, creating a highly prothrombotic and proatherogenic environment within the vasculature.[17]

Mechanistic insights into these prothrombotic effects have been elucidated through pivotal in vivo studies. Qasim et al. in 2018 demonstrated that short-term exposure to e-cigarette aerosols induces a profound state of platelet hyperactivity. These hyperactive platelets exhibit enhanced aggregation, increased dense and alpha granule release, and elevated phosphatidylserine expression. Crucially, this exposure renders platelets resistant to the inhibitory effects of prostacyclin, significantly elevating the risk of thrombogenesis. [17] Furthermore, research conducted by Ramirez et al. in 2020 established that nicotine salts, which are highly prevalent in modern pod-based devices, severely potentiate platelet activation and elevate the risk of pathological thrombosis. [18]

The cellular alterations that promote a prothrombotic phenotype translate into substantial clinical cardiovascular risks. A comprehensive 2023 systematic review and meta-analysis encompassing over one million participants revealed that ENDS use is associated with an approximately 50% increased risk of incident stroke compared to non-users [16]. The researchers attributed this heightened clinical susceptibility directly to the cascade of sustained platelet activation, chronic vascular inflammation, and subsequent thrombus formation. Consequently, the induction of a prothrombotic state represents a critical and independent pathway through which e-cigarettes exert their adverse, life-threatening cardiovascular effects. [16]

Systemic and Local Inflammatory Responses

Inflammation plays a fundamental role in the pathogenesis and progression of cardiovascular diseases (CVD). Chronic, low-grade inflammation is a well-established driver of atherosclerosis, endothelial dysfunction, and plaque instability, ultimately leading to major adverse cardiovascular events, including myocardial infarction, stroke, and peripheral vascular disease [19].

Recent analyses highlight the modulatory effect of e-cigarettes on systemic inflammatory profiles. A study conducted by Bin Abdulrahman KA et al. in 2025 demonstrated that e-cigarette users show measurable changes in inflammatory mediators compared with non-smokers, though the magnitude of these changes is generally less pronounced than in conventional cigarette smokers [19]. Most investigations have predominantly focused on key interleukins, particularly interleukin-6 (IL-6), interleukin-1 beta (IL-1β), and interleukin-8 (IL-8). Although pooled data have not consistently shown statistically significant differences in IL-6 and IL-1β across all comparisons, numerous individual studies indicate elevated concentrations of these pro-inflammatory cytokines in e-cigarette users [19].

Furthermore, sustained systemic exposure to nicotine, evidenced by significantly elevated cotinine levels in biological samples from e-cigarette users, plays a crucial indirect role in inflammatory activation. Nicotine directly influences cell signaling, notably by triggering activation of nuclear factor-kappa B (NF-κB), a primary regulator of pro-inflammatory mediator expression. Persistent activation of these pathways fosters a chronic inflammatory state that directly contributes to cardiovascular pathology [19].

These findings are corroborated by a 2025 analysis by Billa et al., which revealed that e-cigarette users have significantly elevated levels of specific pro-inflammatory cytokines compared to both non-smokers and, notably, conventional smokers [20]. Specifically, levels of tumor necrosis factor-alpha (TNF-α) and interleukin-1 receptor antagonist (IL-1RA), a key indicator of inflammatory pathway activation, were significantly higher among vapers [20]. Collectively, these data suggest that while e-cigarettes may reduce exposure to certain combustible toxicants, they still induce a distinct and potent inflammatory response that poses a viable threat to cardiovascular health.

Lipid Metabolism and Dyslipidemia Risk

Dyslipidemia is a well-established, modifiable risk factor for cardiovascular disease (CVD). Alterations in lipid parameters - specifically elevated triglycerides (TG), increased low-density and very-low-density lipoproteins (LDL and VLDL), and reduced high-density lipoprotein (HDL) cholesterol - are fundamental drivers of endothelial dysfunction, plaque formation, and subsequent cardiovascular complications [21][22].

A 2021 study by Majid et al. evaluated 525 participants aged 21–45 years without established CVD, demonstrated that both sole e-cigarette users and combustible cigarette users had significantly higher TG and lower HDL levels than never-users [21]. Additionally, "dual users" demonstrated an even more pronounced atherogenic lipid profile, with elevated TG and VLDL alongside reduced HDL [21]. Notably, the extent of these metabolic alterations appears to be strongly influenced by the type of device used. Users of earlier-generation e-cigarettes exhibited highly unfavorable lipid patterns, whereas individuals using certain pod-based devices occasionally had lipid and glucose profiles more comparable to those of never-users. This highlights that device characteristics, e-liquid formulations, nicotine delivery mechanisms, and exposure duration critically modulate the severity of metabolic outcomes [21].

Advanced lipidomic analyses have provided further mechanistic insight into how e-cigarettes disrupt lipid metabolism. A targeted study by Middlekauff et al. in 2020, which evaluated more than 1,000 lipid species, revealed that even when global, absolute lipid concentrations appear stable, profound compositional shifts occur at the molecular level [22]. Notably, these alterations exhibit significant sex-specific differences. Female e-cigarette users demonstrated increased levels of diacylglycerols (DAG) and triacylglycerols (TAG), both of which are strongly linked to metabolic dysfunction and elevated cardiovascular risk. Moreover, decreased levels of certain plasmalogens were detected in female users, indicating compromised antioxidant protection and heightened susceptibility to oxidative stress [22]. Although male e-cigarette users showed global lipid profiles largely comparable to non-smokers, alterations in individual lipid species were still observed. These findings emphasize that vaping induces specific, insidious metabolic changes that may significantly propel long-term cardiovascular disease development, highlighting the critical relevance of advanced lipidomic profiling in evaluating the true cardiovascular risk of e-cigarettes [22].

Glycemic Control and Diabetes Pathogenesis

Metabolic disturbances, particularly alterations in glucose homeostasis, are emerging as significant long-term consequences of electronic cigarette use. Although e-cigarettes have been widely perceived as lacking the severe metabolic detriments of combustible tobacco, recent comprehensive reviews and large-scale population studies demonstrate a clear association between vaping, poor glycemic control, and an elevated risk of developing diabetes [24].

The primary mechanism linking e-cigarette use to impaired glycemia is nicotine-induced metabolic dysregulation. Nicotine in e-liquids markedly reduces insulin sensitivity and activates AMP-activated protein kinase (AMPK) in adipose tissue. This activation increases lipolysis and the release of free fatty acids into the circulation, which drives systemic insulin resistance - the fundamental precursor to type 2 diabetes. Furthermore, emerging evidence from animal models indicates that inhaling nicotine-free e-cigarette aerosols can also induce hyperglycemia and contribute to insulin resistance. This suggests that other aerosolized constituents, such as propylene glycol, vegetable glycerin, and flavoring agents, may independently disrupt metabolic homeostasis and cellular glucose uptake. [23]

Large-scale population studies provide robust evidence linking e-cigarette use to an increased risk of impaired glucose homeostasis, prediabetes, and diabetes. Data from the U.S. Behavioral Risk Factor Surveillance System (BRFSS) offer detailed insights into these trends. An analysis of the 2016–2018 BRFSS dataset revealed that current e-cigarette users were 22% more likely to be diagnosed with prediabetes than those who had never used these products [24]. Most alarmingly, the study showed that "sole e-cigarette users" - those who had never smoked combustible tobacco - had a 54% higher risk of prediabetes than completely tobacco-naïve individuals. A more recent analysis of the 2020–2022 BRFSS data found that sole e-cigarette users maintained a statistically significant 7% higher probability of developing prediabetes [24].

A comprehensive analysis of the 2021-2022 Korean Community Health Survey demonstrated that exclusive e-cigarette users face a 15% increased risk of physician-diagnosed diabetes than non-smokers. For context, exclusive traditional smokers in the same cohort exhibited a 22% higher risk [25]. This indicates that while vaping may present slightly lower risks than combustible tobacco, it independently and significantly contributes to the pathogenesis of diabetes, largely driven by the metabolic effects of aerosolized components [25].

Crucially, the metabolic consequences are most severe among "dual users" - individuals who concurrently use both e-cigarettes and traditional combustible tobacco. Rather than serving as a transitional harm-reduction tool, the combined use of these products markedly increases the risk of metabolic dysfunction. U.S. population-level data indicate that dual users have substantially elevated odds of prediabetes, with a 28% increased risk compared to non-smokers - a rate significantly higher than that of exclusive users of either product. Furthermore, this group faces a 9% increased probability of receiving a definitive diabetes diagnosis. [24] Evidence from the Korean Community Health Survey explicitly identifies dual users as the highest-risk demographic among all studied groups, demonstrating a staggering 39% increased risk of developing diabetes. [25] This synergistic effect highlights that combining electronic and conventional nicotine delivery systems drastically worsens glycemic outcomes. Ultimately, these findings refute the narrative that e-cigarettes are a metabolically benign alternative, confirming that they exert a detrimental effect on glycemic control and, when combined with traditional smoking, lead to the worst possible health outcomes regarding diabetes.

Research Limitations

While the current body of literature provides compelling evidence linking electronic cigarette use to various cardiovascular and metabolic risk factors, several significant methodological limitations in the existing research must be acknowledged. Addressing these constraints is essential for accurately interpreting current data and for designing future studies.

A primary limitation of many large-scale epidemiological analyses - such as those using the Behavioral Risk Factor Surveillance System (BRFSS) and the Korean Community Health Survey (KCHS) - is their cross-sectional nature. Because these studies measure exposure and outcomes at the same time, they cannot establish temporal sequences or definitive causal relationships between e-cigarette use and the onset of metabolic or cardiovascular diseases. Consequently, the possibility of reverse causality - where individuals with existing health concerns switch to e-cigarettes as a perceived harm-reduction strategy - cannot be entirely ruled out.

Furthermore, many population-based studies rely heavily on self-reported data for both smoking status and medical diagnoses (e.g., prediabetes or diabetes). This methodology is highly susceptible to recall bias and misclassification. Additionally, in surveys using face-to-face interviews, respondents may underreport their vaping habits or concurrent combustible tobacco use due to social desirability bias, potentially skewing the true magnitude of the associated risks.

The e-cigarette market's extreme diversity poses a formidable challenge for researchers. Devices vary widely in power output and heating mechanisms, from early-generation "cig-a-likes" to advanced modifiable systems and modern pod-based devices. Moreover, e-liquids contain vastly different nicotine concentrations (including potent nicotine salts), varying ratios of propylene glycol to vegetable glycerin, and thousands of different flavoring agents. This extreme heterogeneity makes it exceedingly difficult to standardize exposure protocols across experimental studies or to extrapolate findings to all categories of e-cigarette users.

Disentangling the independent cardiovascular and metabolic effects of e-cigarettes is further complicated by the high prevalence of "dual users" and the fact that a vast majority of vapers are former combustible cigarette smokers. Although statistical adjustments (such as propensity score matching) are used, residual confounding from past tobacco exposure or concurrent intermittent smoking remains a significant limitation in isolating the precise pathological contribution of e-cigarette aerosols. Furthermore, many epidemiological datasets lack detailed information on the frequency, duration, and intensity of vaping sessions, limiting the ability to establish clear dose-response relationships.

Finally, much of the clinical and experimental data focuses exclusively on acute, short-term physiological changes, such as immediate spikes in heart rate, temporary endothelial dysfunction, or acute oxidative stress. Because widespread e-cigarette use is a relatively recent phenomenon, robust, long-term prospective cohort studies tracking the development of hard clinical endpoints - such as the actual incidence of myocardial infarction, stroke, or long-term diabetes complications - are still maturing.

Conclusions

This comprehensive review of the current literature provides compelling evidence that electronic cigarette (e-cigarette) use is a significant and independent cardiovascular and metabolic risk factor. Although these devices are often marketed as a safer alternative to traditional combustible tobacco products, findings from recent meta-analyses and large-scale population studies unequivocally demonstrate that they are not benign.

Key Findings:

Metabolic Dysregulation: Exposure to e-cigarette aerosols, particularly those containing nicotine, leads to significant disruptions in glucose and lipid homeostasis. Vaping is associated with an increased risk of prediabetes and type 2 diabetes, driven by nicotine-induced insulin resistance and unfavorable alterations in the lipid profile changes, including elevated LDL and triglycerides alongside reduced HDL cholesterol.

Cardiovascular Strain: E-cigarette use induces acute hemodynamic responses, marked by rapid increases in blood pressure and heart rate. This mechanism, driven by catecholamine release and sympathetic nervous system stimulation, increases myocardial oxygen demand and may accelerate the progression of atherosclerosis and chronic hypertension.

The Critical Danger of Dual Use: The most alarming evidence concerns "dual users" - individuals who combine e-cigarettes with traditional tobacco. This demographic faces the highest risk of diabetes, hypertension, and severe dyslipidemia, fundamentally refuting the narrative that e-cigarettes are a safe and effective harm-reduction tool for smokers who do not completely quit combustible tobacco.

Public Health and Youth: Given the rapid surge in vaping among adolescents, there is a substantial risk of a new wave of cardiometabolic diseases emerging in younger populations. Early exposure to nicotine and other toxic aerosol constituents can lead to permanent alterations in autonomic and vascular regulation.

Directions for Future Research:

Long-term prospective cohort studies are urgently needed to assess hard clinical endpoints, such as the incidence of myocardial infarction and stroke among exclusive e-cigarette users. Furthermore, future research should focus on distinguishing the metabolic and vascular effects of various device generations, power settings, and specific flavoring agents.

Final Takeaway:

Cardiovascular prevention strategies must strongly emphasize the complete cessation of all forms of nicotine delivery. Education for patients and healthcare professionals must be grounded in evidence showing that e-cigarettes, by inducing oxidative stress, systemic inflammation, and endothelial dysfunction, pose a real and documented threat to long-term cardiovascular health.

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