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REVIEW

The risk of lung cancer from vaping or e-cigarette usage: a systematic review

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Background: Active tobacco smoking remains the primary etiological factor for lung cancer, the leading cause of cancer-related mortality worldwide. The use of electronic cigarettes (ECs) has recently emerged as a potential public health concern due to its suspected association with respiratory and oncogenic outcomes. This study aimed to evaluate the association between EC use and the incidence of lung cancer through a systematic review of existing scientific literature.

Methods: A comprehensive literature search was conducted in PubMed, the Cochrane Library, Embase, ScienceDirect, Web of Science, Scopus, and Google Scholar up to June 2024. Original research articles of any study design that reported on the association between vaping or EC use and the risk of lung cancer were included. The quality of studies was assessed using the critical appraisal checklists for studies created by the Joanna Briggs Institute.

Findings: Of 2252 identified citations, 5 articles were selected for qualitative analysis. These were primarily non-randomised observational designs published between 2019 and 2024. Sample sizes ranged from 3162 to 4 329 288 participants of both genders. A potential association between EC use and the risk of developing lung cancer was observed. Individuals who concurrently use conventional cigarettes and ECs exhibit an increased likelihood of lung cancer incidence.

Conclusion: This systematic review suggests a potential association between EC use and an increased risk of lung cancer, particularly among dual users; however, causality cannot be established due to heterogeneity and limited longitudinal data.

Key words: vaping, electronic cigarettes, conventional cigarettes, lung cancer, lung function

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INTRODUCTION

Electronic cigarettes (ECs) are battery-operated devices that use lithium-ion batteries to produce aerosols that typically include 4-24 mg of nicotine in each puff, along with flavourings, glycerin, and propylene glycol.[1] ECs are heavily promoted as a more affordable, healthier, socially acceptable alternative to conventional cigarettes (CCs), and are often marketed as effective smoking cessation aids.[2-4] Vaping, or the use of ECs, has become increasingly prevalent worldwide. Despite limited empirical evidence, ECs are widely advertised as a harmless method of quitting smoking and are accessible to people of all ages. However, the impact of vaping on lung health remains uncertain, and further research is urgently needed to clarify any potential risks.[2,5,6]

While cigarette use has declined globally, ECs have gained popularity as both smoking cessation tools and recreational nicotine delivery systems.[7] A notable increase in EC use has been observed among teenagers and young adults, attributed to sensory appeal, affordability, and greater accessibility. These factors may contribute to heightened susceptibility to nicotine dependence.[8] The United States Preventive Services Task Force recommends using evidence-based cessation therapies rather than ECs, which are not approved by the United States Food and Drug Administration as smoking cessation aids. Respiratory symptoms are among the earliest health effects of vaping in children.[9,10] In older adults, reduced lung function has been associated with asthma and chronic bronchitis symptoms among younger EC users. In addition to nicotine's impact, the fine particulate matter and flavouring agents in EC aerosols may independently impair pulmonary function.[6]

Although the effects of ECs on inflammation and pulmonary function have been extensively studied, further research is necessary to reach definitive conclusions about their long-term impact on human health.[11,12] Governmental advisory groups have stated that EC use may pose a lower health risk than traditional combustible cigarette smoking, as ECs do not produce harmful combustion byproducts.[13] However, recent concerns about vaping-associated lung injury—known as e-cigarette or vaping-associated lung injury (EVALI)—have increased public awareness of its potential risks, including inflammation, pneumonia, and lung tissue damage.[11-15] Despite emerging evidence, major knowledge gaps persist regarding the long-term respiratory safety of vaping, particularly its association with lung cancer. Therefore, this systematic review was undertaken to synthesise and evaluate existing literature on the relationship between EC use and the incidence of lung cancer.

METHODS

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.[16]

Search strategy

A comprehensive literature search was conducted across five databases—PubMed, the Cochrane Library, Embase, Web of Science, and Google Scholar—from 2003 to 2024. The search terms used included combinations of the following keywords: "vaping", "e-cigarettes", "vapor cigarettes", "lung cancer", "lung carcinoma", and "pulmonary function". Only original research articles were retrieved. All relevant keyword permutations were employed.

A PICO (population, intervention, comparison, and outcome) framework guided the selection criteria:

- P: participants of either gender
- I: vaping or electronic cigarette use
- C: non-smokers or control group
- O: incidence of lung cancer

Study selection

After duplicate removal, titles and abstracts were screened based on pre-defined eligibility criteria. Full-text articles of all potentially relevant studies were independently reviewed.

Eligibility criteria

Studies were included if they met the following conditions:

- Design: randomised controlled trials, prospective or retrospective cohort studies, and cross-sectional studies
- Focus: reported on vaping or EC use and lung cancer

Exclusion criteria were as follows:

- Studies not related to the research question or lacking sufficient data
- Studies without results
- Non-English-language publications
- Case reports, commentaries, guidelines, editorials, book chapters, letters to the editor, narrative reviews, and meta-analyses

Reference lists of relevant systematic reviews and meta-analyses were manually screened to identify additional eligible studies. Both published and unpublished works, including grey literature, were included.

Study quality assessment

The Joanna Briggs Institute (JBI)'s critical appraisal checklists for studies were used to evaluate study quality.[17] Risk of bias was classified as high when the score of 'yes' responses was 49% or lower, moderate when between 50% and 69%, and low when 70% or higher. All included studies were categorised based on their level of risk: low risk, high risk, or with some concerns. Any disagreements between independent reviewers (SM, AG, AK, AT, and RW) were resolved through discussion and consensus.

Data analysis

Given the wide variation in study designs, populations, and measured outcomes, a meta-analysis was not feasible. Therefore, a qualitative synthesis approach was adopted, focusing on directionality and consistency of associations rather than pooled effect estimates. The heterogeneity was anticipated due to differences in population characteristics, smoking history, and definitions of EC use (Table 1). When available, adjusted odds ratios (ORs) or hazard ratios (HRs) controlling for confounders such as age, gender, and CC use were extracted and compared qualitatively.

RESULTS

Identification and description of studies

A total of 2252 citations were identified through database searching: 149 from PubMed, 388 from Embase, 34 from the Cochrane Library, 988 from Google Scholar, and 693 from Web of Science. After removing 365 duplicate records, 1887 articles remained for title and abstract screening. Of these, 1822 studies were excluded based on relevance. Sixty-five articles underwent full-text review, and following the application of inclusion and exclusion criteria, 60 were excluded. Five studies were included in the final qualitative synthesis. The study selection process is illustrated in the PRISMA flow diagram (Figure 1).

Five research articles were identified that highlighted different research related to the effects of vaping or EC use on the risk of lung cancer. The publishing years were 2019 through 2024. All five studies were non-randomised observational studies (cohort, case-control, or cross-sectional). Two studies each were conducted in the United States and Korea, and one in Russia. The total number of participants ranged from 3162 to 4 329 288, with both genders being included in the majority of the research. A wide range of age groups was included in the research assessed in this review, with the majority of the studies focusing on younger individuals.

The extracted data of the included studies presented in this review are summarised in Table 1. Substantial variability was observed across the included studies in terms of study design, geographical setting, population characteristics, exposure definitions, and analytic endpoints. The principal sources of heterogeneity are summarised in Table 1. The studies encompassed diverse methodological frameworks ranging from large population-based cohorts to cross-sectional surveys, and differed in how EC use was defined, reported, and measured. Outcome ascertainment and comparator groups also varied widely, precluding quantitative pooling of data.

Association of vaping or e-cigarette use with risk of lung cancer

The objective of the included studies was to determine whether vaping or EC use is associated with lung cancer. Although definitive evidence remains limited, five studies reporting such an association were identified. A recent nationwide population-based study by Kim and colleagues[18] found that switching from CCs to ECs increased both the risk of lung cancer and lung cancer-specific mortality. In total, 4 329 288 individuals with a history of traditional smoking participated in the National Health Screening Program during 2012-2014 and again in 2018, with follow-up until December 2021. During this period, 6351 lung cancer-specific deaths and 53 354 lung cancer cases were recorded. Ex-smokers (cigarette smokers) who used ECs and were at least 5 years younger than non-users showed a higher risk of lung cancer-specific death [adjusted HR (aHR) 2.69, 95% confidence interval (CI) 1.12-6.46]. Compared with individuals with <5 years since quitting (YSQ) who did not use ECs, ex-smokers with EC use had higher risks of lung cancer (aHR 1.23, 95% CI 1.09-1.39) and lung cancer-specific death (aHR 1.71, 95% CI 1.10-2.66). Among individuals aged 50-80 years with at least 20 pack-years (PY) of smoking history, ex-smokers who used ECs had increased risks of lung cancer (aHR 1.65, 95% CI 1.05-2.58) and lung cancer-specific death (aHR 4.46, 95% CI 1.85-10.75). Similar associations were found in ex-smokers with <5 YSQ who used ECs (aHR 1.26, 95% CI 1.03-1.54).[18]

Herriges and colleagues[19] used nationally representative data from the United States to examine the prevalence of bladder and lung cancer among individuals with varied smoking histories. Compared with never smokers, both bladder and lung cancer were more prevalent in all categories with a history of smoking. At diagnosis, patients with a history of EC use were significantly younger [mean 56.87, standard deviation (SD) 9.86 versus mean 65.00, SD 12.60 years, $P = 0.001$]. Multivariable logistic regression showed increased odds of lung cancer associated with EC use (OR 1.614, $P = 0.007$) and cigarette smoking (OR 4.589, $P < 0.001$).[19] Paek and colleagues[14] examined smoking and EC use among Korean cancer survivors using data from the 2013-2018 National Health and Nutrition Survey. Propensity score matching produced a sample of 1260 cancer survivors and 5040 individuals in the matched non-cancer population. Former smoking was more prevalent among survivors (25.2% versus 19.9%), and current smoking was less common (6.7% versus 10.6%). No difference was found in lifetime EC use (2.4% versus 2.7%, $P = 0.529$). Successful quitting, defined as abstaining from both cigarettes and ECs, was significantly associated with cancer type, depression (OR 0.276, 95% CI 0.087-0.872), and problem drinking (OR 0.442, 95% CI 0.207-0.940).[14]

Gambaryan and colleagues[20] investigated the relationship between heated tobacco product and EC use and major respiratory conditions. Of the respondents, 14% had chronic bronchitis, 4.3% had chronic obstructive pulmonary disease, 3.1% had asthma, 1.5% had emphysema, 1.1% had lung cancer, and 1.3% had tuberculosis. Current EC use was significantly associated with lung cancer: OR 4.9 (95% CI 1.6-14.8) and OR 7.2 (95% CI 1.9-27.4).[20] Kizhakke Puliyakote and colleagues[13] used magnetic resonance imaging to measure ventilation−perfusion ($\dot{V}/\dot{Q}$) mismatch in asymptomatic EC users. Compared with controls, vapers showed a significantly greater mismatch at baseline [Log

Table 1. Basic characteristics and source of heterogeneity of the included studies

Reference	Year	Country	Study design	Sample size	Study population	Exposure/ intervention	Comparator	Outcomes	Heterogeneity source/ comment	Quality of studies
Kim et al.[18]	2024	Korea	Cohort	4 329 288	Ex-smokers ≥5 YSQ without EC use, ≥5 YSQ with EC use, <5 YSQ without EC use, <5 YSQ with EC use, and current smokers without and with EC use	EC	Smokers and non-smokers with and without EC use	During the follow-up, 53 354 individuals developed lung cancer, and 6351 LCSD events occurred. Switching to EC use after conventional smoking cessation was associated with a higher risk of lung cancer and related mortality	Comprehensive dataset; detailed adjustment; population exclusively East Asian	Fair
Herriges et al.[19]	2022	USA	Cross-sectional	85 187	Subjects from the National Health Interview Survey database between 2016 and 2018	EC	EC smokers and non-smokers	EC use was associated with increased ORs of 1.614 ($P = 0.007$) for lung cancer diagnosis	National survey; self-reported EC exposure; limited confounder control	Fair
Paek et al.[14]	2022	Korea	Case-control	32 242	1260 CS and 5040 non-cancer populations using the propensity score matching method	EC and CC	EC or CC	Regarding conventional smoking, the proportion of ex-smokers was higher (25.2% versus 19.9%) than current smokers (6.7% versus 10.6%) in the CS group than in the propensity-matched non-cancer population ($P < 0.001$)	Focused on survivors; high matching precision; limited cancer-specific endpoint	Good
Gambaryan et al.[20]	2020	Russia	Cross-sectional	11 625	6569 smokers, 2377 former smokers, and 2679 non-smokers	EC and HTP	EC and HTP	Current EC use was significantly associated with lung cancer: OR 4.9 (95% CI 1.6-14.8)	Population-level dataset; exposure self-reported; no pack-year data	Fair
Akinboro et al.[10]	2019	USA	Cross-sectional	3162	3162 with a lifetime history of a smoking-related cancer	EC	None	The prevalence of current EC use was 3.18% (95% CI 2.40% to 3.96%). The prevalence of EC use was highest among patients and survivors of lung cancer, 4.65% (95% CI 1.49% to 7.97%)	Cancer survivor cohort; EC use not stratified by duration or intensity	Fair

CC, conventional cigarettes; CI, confidence interval; CS, cancer survivors; EC, electronic cigarettes; HTP, heated tobacco products; LCSD, lung cancer-specific death; OR, odds ratio; YSQ, years since quitting.

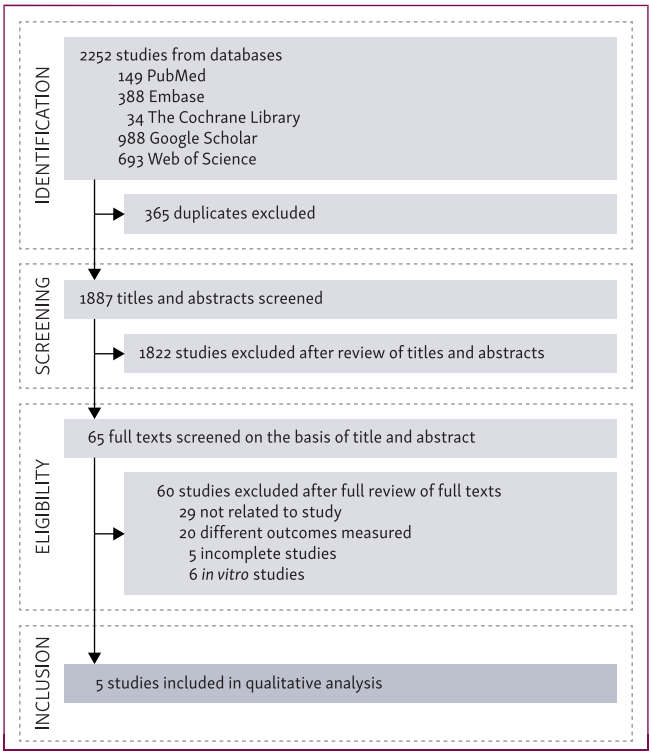

Figure 1. **Flow chart depicting the process of selecting or rejecting studies.**

standard deviation of the V/Q distribution (LogSDQ̇) 0.61 (0.12) versus 0.43 (0.12), $P = 0.01$], which worsened after vaping [LogSDQ̇ 0.73 (0.16), $P = 0.03$]. Akinboro and colleagues[10] assessed EC use among survivors of smoking-related cancers in the United States. Current EC use was reported by 3.18% (95% CI 2.40% to 3.96%) of respondents. Current EC users were 83 times more likely to also be current cigarette smokers than never-EC users (relative risk ratio 82.89, 95% CI 16.54-415.37). Among individuals reporting only one smoking-related cancer diagnosis, EC use prevalence was highest in cervical (5.25%, 95% CI 3.19% to 7.31%), lung (4.65%, 95% CI 1.49% to 7.97%), pancreatic (4.12%, 95% CI 0.00% to 12.91%), and bladder cancers (3.77%, 95% CI 0.86% to 6.67%).[10]

Study quality assessment

Two reviewers independently evaluated the quality of each included study. Most studies in this analysis were judged to have low to moderate risk of bias, as indicated by a high proportion of 'yes' responses on the JBI appraisal tool. A third reviewer was consulted to resolve any disagreements. The distribution of quality assessment outcomes is presented in Figure 2.

Across the five studies, four reported positive associations between EC use and lung cancer risk, while one found no significant relationship after adjusting for age and smoking status. Despite varying methodologies, the direction of effect was consistently positive, particularly among dual users. Given the methodological differences, the extent and quality of statistical adjustment for potential confounders were also inconsistent (Table 2). Three studies applied multivariable adjustment for age, sex, and smoking history, whereas two provided only descriptive analyses without accounting for cumulative tobacco exposure or dual use. This variability underscores the heterogeneity of the available evidence and justifies the use of a qualitative synthesis rather than a meta-analysis in the present review.

DISCUSSION

CC smoking is well established as a major cause of lung cancer and other respiratory diseases.[7] However, as traditional tobacco use declines, the global use of ECs has risen sharply.[2,6,21] ECs are marketed as safer alternatives and effective smoking cessation aids, yet evidence to support these claims remains limited and inconsistent. Although they deliver fewer combustion-related toxicants, the long-term pulmonary risks of ECs, including their potential association with lung cancer, are still uncertain. Current epidemiological data are insufficient to establish causality, but accumulating mechanistic and clinical evidence suggest possible carcinogenic effects.[6]

This systematic review identified five studies examining the relationship between EC use and lung cancer. The direction of association was consistent across most reports, indicating a potential increase in lung cancer risk among EC users, particularly among dual users of ECs and CCs. Kim and colleagues[18] demonstrated that individuals who switched from CCs to ECs after cessation exhibited higher lung cancer incidence and mortality than non-EC users, even after multivariable adjustment. Herriges et al.[19] also reported an elevated risk among EC users compared with never smokers. Gambaryan and colleagues[20] found that EC

Reference	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	% of yes	Risk
Kim et al.	√	√	U	√	U	U	√	√	62.5%	Moderate
Herriges et al.	√	√	X	√	X	X	U	√	50.0%	Moderate
Paek et al.	√	√	√	√	X	X	√	√	75.0%	Low
Gambaryan et al.	√	√	X	√	X	X	U	√	50.0%	Moderate
Akinboro et al.	√	√	U	√	X	X	U	√	50.0%	Moderate

Q1 Were the criteria for inclusion in the sample clearly defined? Q2 Were the study subjects and settings described in detail? Q3 Was the exposure measured in a valid and reliable way? Q4 Were objective, standard criteria used for measurement of the condition? Q5 Were confounding factors identified? Q6 Were the outcomes measured in a valid and reliable way? Q8 Was appropriate statistical analysis used? √ = Yes. X = No. U = Unclear.

Figure 2. **Risk of bias assessed by using JBI critical appraisal checklist for the included studies.**

Table 2. Adjustment for key confounders in included studies

Reference	Adjusted for age	Adjusted for sex	Adjusted for smoking history/pack-years	Adjusted for other confounders	Adjustment quality
Kim et al.[18]	✔	✔	✔ (pack-years)	Comorbidities, socioeconomic status	High
Herriges et al.[19]	✔	✔	✔ (smoking status)	Race, education	Moderate
Paek et al.[14]	✔	✔	✔ (smoking category: current/ex/never)	Alcohol use, depression	High
Gambaryan et al.[20]	✔	✔	✖ (self-reported only)	None reported	Low
Akinboro et al.[10]	✔	✔	✖ (lifetime history only)	Cancer type	Moderate

use, even in the absence of cigarette smoking, was associated with serious respiratory outcomes, and dual users exhibited the greatest morbidity. Similarly, Chaiton and colleagues[7] reported increased respiratory symptoms among young never smokers who vaped. Although heterogeneity in study design, population, and endpoints precluded quantitative pooling, the consistency in direction of effect supports a potential causal link.

The findings align with emerging evidence from epidemiological and preclinical studies. While ECs are often promoted for harm reduction, prior systematic reviews have focused on short-term respiratory and cardiovascular outcomes rather than oncological endpoints.[2-6] The present synthesis extends that evidence, showing that EC use may not be harmless, particularly among dual users. In the Korean cohort, the increased risk persisted despite adjustment for age, sex, and smoking intensity, highlighting potential residual or synergistic carcinogenic effects.[18]

EC aerosols contain aldehydes, volatile organic compounds, and heavy metals at concentrations sufficient to induce oxidative stress and DNA damage.[6] Experimental research supports this biological plausibility. Canistro and colleagues[22] demonstrated in rats that EC exposure significantly increases the activity of cytochrome P450 (CYP) isoforms—particularly CYP1A1/2, CYP2B1/2, CYP2C11, and CYP3Aenzymes—that metabolize and activate many carcinogens. Such induction may raise susceptibility to CYP-mediated carcinogenesis. Excessive production of reactive oxygen species due to CYP activation can further promote DNA damage and malignant transformation.[22]

Emerging mechanistic studies have shown that EC aerosols can alter gene expression and proteomic signatures in ways similar to tobacco smoke. Tsai and colleagues[23] found that EC vapour induces chromosomal aberrations, DNA hypomethylation, and dysregulation of enhancer RNAs involved in tumour suppression. These molecular alterations were associated with enhanced oncogenic signalling and poorer immune regulation in lung squamous-cell carcinoma. Similarly, Park et al.[24] demonstrated that exposure of human bronchial epithelial cells carrying KRAS activation and TP53 silencing to EC aerosols promoted accelerated colony formation and altered expression of 263 genes, suggesting malignant transformation potential. *In vivo* data also support these findings—mice exposed to EC aerosols for 54 weeks developed lung adenocarcinomas.[25]

The rapid rise in EC use, particularly among adolescents and young adults, has created new challenges for tobacco control.[2,6] Although ECs are sometimes viewed as nicotine replacement tools, current data do not confirm their efficacy for sustained cessation.[6,21] Many users become dual users rather than exclusive EC users, perpetuating nicotine dependence and potentially compounding carcinogenic exposure.[26] Moreover, the growing perception of ECs as a 'healthier' option mirrors historical misrepresentations of filtered cigarettes, which were once marketed as safer alternatives despite similar health risks. Public health campaigns should therefore avoid equating 'reduced harm' with 'no harm'.

Several constituents of EC liquids, including propylene glycol, benzoic acid, and diethyl carbonate, have demonstrated cytotoxic and pro-inflammatory properties.[27-29] The outbreak of EVALI linked to tetrahydrocannabinol oils adulterated with vitamin E acetate further underscores the vulnerability of EC users to poorly regulated formulations.[11-15] Although EVALI is distinct from carcinogenesis, its occurrence illustrates the unpredictable toxicity of EC aerosols and the need for stricter quality control.

From a clinical perspective, the findings emphasize that physicians should assess EC use in routine evaluations, particularly in patients with respiratory symptoms or high-risk profiles.[18] The incorporation of EC history into lung cancer screening programmes may be prudent, given the evidence of additive risk among former smokers who transition to ECs. The Korean cohort's finding of elevated lung cancer mortality among EC users who had quit conventional smoking underscores this point.[18]

Substantial heterogeneity was observed among the studies included in this review. Differences in design (cohort, cross-sectional, and case-control), population characteristics, definitions of EC exposure, and outcome assessment limited direct comparison and precluded formal meta-analysis. Some investigations relied on self-reported use of EC, while others used registry or database verification. Follow-up durations were generally short, and adjustments for confounding variables such as age, sex, PY, comorbidities, and socioeconomic factors varied considerably, affecting comparability and the strength of inference.

Despite these limitations, the qualitative synthesis consistently indicated a positive association between EC use and lung cancer risk. Most multivariable analyses that adjusted for key confounders reported higher ORs or HRs for EC users compared with non-users, particularly among dual users. Although causality cannot yet be established, these convergent trends highlight the need for more robust, longitudinal research with standardized definitions and comprehensive confounder control.

Mechanistic evidence complements these epidemiological findings. EC-induced carcinogenesis appears to involve

both shared and distinct molecular pathways compared with conventional smoking. While traditional cigarette smoke primarily activates intrinsic apoptotic cascades, EC vapour triggers FAS-ligand-dependent extrinsic apoptosis, with both mechanisms converging on common oncogenic targets.[26] The observed up-regulation of pro-inflammatory cytokines such as interleukin-6 and interleukin-8 supports a model in which chronic airway inflammation and oxidative stress act as initiating events in tumorigenesis.[14] Together, the molecular, epidemiological, and toxicologic data lend coherence to the observed association between EC use and lung cancer.

This review has inherent limitations reflecting those of the included studies. The small number of eligible reports and their observational design restrict causal inference. Short follow-up periods limit the assessment of the latency, which is a critical factor in cancer development, and self-reported exposure introduces recall bias.[19] Residual confounding from prior smoking, environmental exposures, or genetic susceptibility cannot be excluded.[14,18]

Future research should therefore emphasize large-scale, prospective cohort studies that clearly distinguish exclusive EC users from dual and former smokers, incorporate biochemical verification of nicotine exposure, and apply uniform outcome definitions. Integration of molecular biomarkers such as DNA adducts, oxidative stress indicators, and epigenetic modifications will help elucidate causal mechanisms. Establishing registries to track EC exposure and cancer incidence could further strengthen surveillance and data quality. Randomised controlled trials comparing EC-assisted cessation with approved pharmacological therapies are also warranted to evaluate efficacy and safety. Once a sufficient body of homogeneous data emerges, meta-analyses employing random-effects modelling will be critical to quantify risk precisely and confirm or refute the observed associations.

There is an urgent need to raise public awareness about the long-term risks of vaping, especially in relation to lung cancer. The portrayal of ECs as a 'healthier' alternative—with taglines such as 'safer than smoking'—mirrors the historical misrepresentation of filter-tipped cigarettes. In the absence of data from randomised controlled trials, such claims remain unsubstantiated and potentially misleading. Policymakers and public health professionals must address the misinformation surrounding EC safety. To clarify the causal relationship between EC use and lung cancer, future longitudinal studies with greater statistical power are required.

CONCLUSION

This systematic review indicates a possible association between EC use and increased lung cancer risk, particularly among dual users. The consistency across heterogeneous studies, combined with mechanistic plausibility, supports the need for heightened clinical vigilance and stronger regulatory oversight. However, evidence remains preliminary, and causality cannot yet be confirmed. Until long-term data become available, ECs should not be promoted as risk-free alternatives to CCs. Policymakers and clinicians must interpret current findings with caution and prioritise education, regulation, and research aimed at minimising harm and clarifying the true oncogenic potential of ECs. While current evidence indicates a possible link between EC use and lung cancer, the findings should be interpreted cautiously due to limited study numbers and potential confounding. Larger, long-term studies are required to confirm this association. The observed associations highlight potential oncogenic risks but fall short of proving a causal relationship.

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DISCLOSURE

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