

Does Vaping Cause Cancer? What the 2025–26 Evidence Shows

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The question clinicians are increasingly fielding from patients is whether switching from cigarettes to e-cigarettes trades one cancer risk for another, or simply reduces it. A 2025 qualitative risk assessment concludes that e-cigarettes carry a plausible carcinogenic risk, though the long latency of tobacco-related cancers means definitive epidemiological proof will not arrive for years.¹ In the meantime, three papers published between 2025 and 2026 piece together what is currently known about toxicant exposure, nicotine intake, and biological disruption in people who vape.

KEY TAKEAWAYS

- **The Pivot:** A formal qualitative carcinogenicity assessment now places e-cigarettes in a plausible risk category, even without long-term incidence data.¹
- **The Data:** No hazard ratios or cancer incidence figures yet exist; the carcinogenic case rests on known genotoxic constituents in vapour and disruption of the oral microbiota identified across reviewed studies.^{1,2,3}
- **The Action:** Clinicians should not reassure patients that vaping is cancer-free; counsel that risk reduction relative to smoking is probable but unquantified, and that youth vaping warrants active discouragement given nicotine salt concentrations and dependence potential.²

E-cigarettes were introduced to many markets as cessation aids or harm-reduction tools, and that framing has shaped both clinical advice and public perception. The question of carcinogenicity, however, has never been settled by long-term follow-up data, because the latency period for most smoking-related cancers exceeds the years e-cigarettes have been in widespread use. Clinicians are therefore being asked to advise patients using an evidence base that is, by structural necessity, incomplete.

The clinical challenge is significant. Patients often inquire about the safety of vaping, particularly in comparison to combustible tobacco, and clinicians must navigate a landscape where public health messaging can be inconsistent and scientific evidence is still emerging. While the immediate acute effects of vaping are increasingly understood, the chronic, long-term health consequences, especially concerning cancer, remain a critical unknown. This gap in knowledge necessitates a cautious approach to advising patients, emphasizing what is known and acknowledging what is not.

What the studies did

Stewart, Marshall, and Bonevski conducted a qualitative risk assessment of e-cigarette carcinogenicity, published in *Carcinogenesis* in 2025.¹ Rather than waiting for incidence data, the authors evaluated the biological plausibility of carcinogenic risk by examining known constituents of e-cigarette aerosol, including carbonyls, volatile organic compounds, and tobacco-specific nitrosamines, and assessed whether mechanistic and in vitro evidence supports a carcinogenic pathway.¹ The paper does not produce a quantitative risk estimate, which is both its methodological honesty and its clinical limitation. Their assessment involved a comprehensive review of toxicological studies, identifying specific compounds in e-cigarette aerosols that are recognized carcinogens or pro-carcinogens in other contexts. They then mapped these compounds to known biological pathways of carcinogenesis, such as DNA damage, oxidative stress, and inflammation, to build a case for plausible risk.

A systematic review published in the *Journal of Clinical Periodontology* in 2026 examined e-cigarette effects on the oral microbiota, an area relevant to oral carcinogenesis given the established link between microbial dysbiosis and oropharyngeal malignancy.³ La Rosa, Samaranayake, and Zaura reviewed available studies and found that e-cigarette use alters the composition of the oral microbial community, though the clinical significance of those changes and their relationship to cancer risk remain to be established.³ The review synthesized findings from both in vitro and human observational studies, noting consistent patterns of microbial shifts, including increases in certain pathogenic bacteria and reductions in beneficial species. While these changes mirror some patterns seen in established oral diseases, the direct causal link to cancer in vapers requires further longitudinal investigation.

A third paper, from McNeill, Robson, and Hammond, published in *Public Health Research* in 2026, addresses a different but related concern: the toxicant and nicotine exposure profile of young vapers in England compared with peers in North America.² The authors note that the proliferation of cheap disposable vapes delivering high-concentration nicotine salts has raised specific concerns about dependence and potential health risks in youth, a population for whom any carcinogenic exposure carries greater cumulative weight over a lifetime.² This study utilized biomarker data from urine and saliva samples to quantify exposure to nicotine and selected toxicants, providing objective measures of exposure levels in different youth populations. The comparison between regions highlighted variations in product use patterns and regulatory environments, which in turn influenced exposure profiles. The authors emphasized that early life exposure to genotoxic agents can have profound implications for lifetime cancer risk due to the longer period for accumulation of cellular damage and the potential for epigenetic alterations.

Reading the evidence honestly

The carcinogenicity assessment's central finding is that biological plausibility exists: e-cigarette aerosol contains genotoxic compounds, and mechanistic pathways to carcinogenesis are demonstrable, even if population-level incidence data are absent.¹ The oral microbiota review adds a further biological layer, in that vaping appears to shift microbial populations in a direction that warrants monitoring, though no study has yet linked these changes to oral cancer outcomes in vapers specifically.³ The youth exposure data underscores that the population now accumulating the greatest vaping exposure, adolescents using high-nicotine disposables, is precisely the group for whom long-term cancer follow-up will

eventually matter most.² Taken together, the three papers do not prove that vaping causes cancer. They do establish that the absence of proof is not proof of absence, and that the biological architecture for carcinogenic harm is present. The limitations of the current evidence base are clear: direct epidemiological studies demonstrating increased cancer incidence in vapers are still years away due to latency periods. However, the existing mechanistic and toxicological data provide a strong rationale for continued vigilance and a precautionary approach in public health messaging and clinical advice.

For broader insights into cancer development, prevention, and treatment within a clinical context, consider referring to the Oxford Handbook of Oncology.

CLINICAL IMPLICATIONS

The most uncomfortable implication of the Stewart et al. risk assessment is that clinicians have been offering informal reassurance, namely that vaping is probably safer than smoking, without any quantified cancer risk figure to anchor that claim.¹ That reassurance may still be directionally correct, but it is currently unsupported by incidence data, and the qualitative carcinogenicity evidence means it can no longer be offered without caveats. Practices that routinely see ex-smokers who have switched to vaping and consider the matter resolved should revisit that framing.

The oral microbiota findings are the piece of this puzzle that industry would prefer to minimise. Vaping aerosol is not inert mist; it is a chemically complex exposure that alters the microbial environment of a tissue site, the oropharynx, with a well-documented susceptibility to carcinogenesis. The companies marketing flavoured disposables to young consumers have done so under a regulatory environment that, in England at least, is only now tightening. NICE and the MHRA have not yet updated guidance on e-cigarettes to reflect the 2025 carcinogenicity assessment, and that gap will need closing.

Youth exposure is where the evidence becomes most clinically urgent. The McNeill et al. data highlights that adolescents are accessing high-nicotine-salt devices with relative ease, building both dependence and cumulative toxicant exposure during a developmental window that carries the longest runway to cancer expression. GPs seeing adolescent patients should treat vaping as they would any other nicotine product: screen for it, document it, and advise cessation without the implicit message that it is a neutral activity. Paediatric and adolescent medicine has not yet fully integrated e-cigarettes into its risk conversations, and it should.

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