INNOVATIVE WORLD

ISSN: XXXX-XXXX

ORIENTAL JOURNAL OF ENGINEERING AND MODERN TECHNOLOGIES

SHARQ MUHANDISLIK VA ZAMONAVIY TEXNOLOGIYALAR JURNALI

Scientific Journal

- Civil
- Robotic
- Material
- Chemical
- Computer
- Electrical
- Mechanical
- Agricultural
- Manufacturing

- Qurilish
- Robototexnika
- Materialshunoslik
- Kimyo-texnologiya
- Informatika
- Elektr texnologiya
- Mexanika
- Qishloq xo'jaligi
- Ishlab chiqarish

xomidovanvarbek07@gmail.com
www.innoworld.net
+998 33 5668868

ORIENTAL JOURNAL OF ENGINEERING AND MODERN TECHNOLOGIES

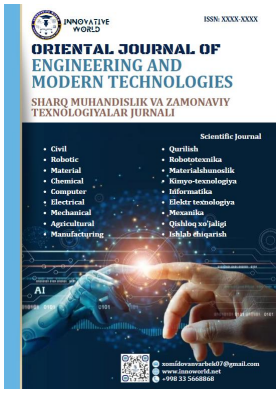
Volume 3, Issue 2
2025

Journal has been listed in different indexings

Google Scholar · doi digital object identifier · ResearchGate · zenodo · ADVANCED SCIENCE INDEX · OpenAIRE · Academic Resource Index ResearchBib · DRJI Directory of Research Journals Indexing

The offical website of the journal:
www.innoworld.net

Uzbekistan-2026



SYNERGISTIC EFFECTS OF TRADITIONAL SMOKING AND VAPING ON THE DEVELOPMENT OF LUNG ADENOCARCINOMA IN YOUNG ADULTS

Ibragimova Bonu
Toshkent davlat tibbiyot universiteti
Davolash fakulteti talabasi,
Mahliyo Alisherova
Toshkent davlat tibbiyot universiteti Tibbiy radiologiya kafedrasi assistenti,
E-mail: mahliyoalisherova1994@gmail.com

Annotation. This article explores the increasing incidence of lung adenocarcinoma among young individuals, a trend no longer limited to older populations. While traditional smoking remains the main risk factor, the rapid rise of electronic cigarette use has introduced new harmful exposures affecting lung tissue.

The paper highlights key molecular mechanisms, including oxidative stress, chronic inflammation, DNA damage, and epigenetic changes, which lead to mutations in genes such as EGFR, KRAS, and ALK. It also emphasizes the role of toxic substances in e-cigarette aerosols in promoting carcinogenesis.

Additionally, social and psychological factors—such as peer pressure, marketing strategies, and the perception of vaping as safe—are discussed as drivers of increased use among youth. The article concludes that lung adenocarcinoma in young people results from a combination of environmental and genetic factors, underscoring the need for better regulation, awareness, and early detection strategies.

Key words: Lung adenocarcinoma; Electronic cigarettes (vaping); Youth population; Carcinogenesis; Oxidative stress; DNA damage; EGFR mutation; KRAS mutation; ALK rearrangement; Inflammation; Genotoxicity; Risk factors; Public health

Although lung cancer has traditionally been regarded as a disease of older individuals, recent years have shown a surprising increase in the incidence of lung adenocarcinoma among people under the age of 40.[1] Currently, adenocarcinoma represents the most common histological subtype in younger patients and has even become the leading oncological condition among young individuals who have never smoked.[2] This emerging trend suggests that lung carcinogenesis is influenced not only by conventional tobacco smoke but also by environmental factors and newly developed nicotine delivery systems.

On a global scale, lung cancer remains the leading cause of cancer-related mortality.[3] According to the World Health Organization (WHO), approximately 1.8 million deaths were attributed to lung cancer in 2020, accounting for around 18% of all cancer deaths worldwide.[4] Epidemiological evidence indicates that nearly 85% of lung cancer cases are directly linked to tobacco use, making smoking the most significant and preventable risk factor.

However, the rapid rise in the use of electronic cigarettes (vaping) among young people—now described as a "global epidemic"—has significantly altered the profile of carcinogenic exposures affecting lung tissue.[5] Aerosols produced by e-cigarettes contain harmful substances such as formaldehyde, acrolein, and heavy metals, which, although different from those in conventional tobacco, can induce substantial genotoxic and epigenetic damage at the DNA level.[6]

The increasing relevance of lung adenocarcinoma in younger populations is further emphasized by its often aggressive clinical course and the fact that it is frequently diagnosed at advanced, metastatic stages.[7] In younger patients, the high proliferative activity of tumor cells and the frequent presence of driver mutations such as EGFR, ALK, and ROS1 highlight the distinct biological characteristics of this disease.[8]

In addition to the long-term chronic effects of conventional cigarette smoking, the short-term yet intensive toxic exposure associated with electronic cigarettes poses a synergistic threat to lung health, particularly among younger populations.[9] Epidemiological projections suggest that, without effective regulation of vaping and emerging tobacco products, lung cancer–related mortality among young individuals may continue to rise in the coming years.[10] Therefore, investigating the molecular mechanisms underlying these risk factors and developing early screening strategies has become an urgent priority in modern oncology and public health.[11]

Over the past decade, electronic cigarettes—also known as vaping devices—have spread rapidly among young people at an unprecedented rate. Frequently marketed by manufacturers as a "safer alternative" to traditional cigarettes, these products have gained particular popularity among individuals aged 15–24. However, independent scientific studies increasingly challenge these claims. For example, a meta-analysis by Wang et al. demonstrated that individuals who use both electronic and conventional cigarettes have a fourfold higher risk of developing lung cancer compared to those who smoke only traditional cigarettes.[12]

The most common histological subtype of lung cancer, adenocarcinoma, originates from glandular epithelial cells and is typically localized in the peripheral regions of the lungs. Importantly, it often progresses without noticeable symptoms in its early stages, contributing to delayed diagnosis and poorer clinical outcomes. From a pathogenetic perspective, key molecular drivers of adenocarcinoma include mutations in the EGFR gene (exons 18–21), activation of the KRAS oncogene, and rearrangements involving the ALK gene.[13] These alterations disrupt normal cellular regulatory mechanisms, leading to uncontrolled proliferation and malignant transformation.

The aerosol produced by electronic cigarettes contains a range of harmful substances, including heavy metals (such as nickel, lead, and cadmium), volatile organic compounds.[14] The widespread use of electronic cigarettes among young people can be attributed to a combination of social influence, misleading marketing strategies, psychological factors, and the pervasive impact of social media.[15]

The aim of this study is to investigate the biological, molecular, and epidemiological roles of both conventional and electronic cigarette use in the development of lung adenocarcinoma among young individuals, based on a systematic review of the existing literature.

Main part. Lung adenocarcinoma is currently the most prevalent histological subtype of lung cancer, with an alarming rise in incidence among younger populations observed globally.[1] Conventional tobacco smoking remains the primary risk factor, as it delivers potent carcinogens—such as polycyclic aromatic hydrocarbons (PAHs) and tobacco-specific nitrosamines (TSNAs)—directly to the peripheral lung tissues.[2] These compounds form stable DNA adducts within pulmonary epithelial cells, leading to critical mutations in oncogenes like KRAS and tumor suppressor genes such as TP53.[3] In recent years, the increasing popularity of electronic cigarettes (vaping) among youth has introduced a new paradigm of inhalational toxic exposure.[5] E-cigarette aerosols contain harmful substances, including formaldehyde, acetaldehyde, and heavy metals (e.g., nickel and lead), which, despite being marketed as safer alternatives, exert significant genotoxic effects at the cellular level.[6] Oxidative stress induced by vaping, along with chronic inflammation of the alveolar epithelium, plays a critical role in the early stages of tumorigenic transformation.[11] Emerging evidence indicates that electronic cigarette users exhibit distinct molecular signatures characterized by specific epigenetic modifications and elevated levels of pro-inflammatory cytokines, including IL-6 and TNF-α.[16] Although nicotine itself is not classified as a direct carcinogen, it promotes tumor progression by activating nicotinic acetylcholine receptors (nAChRs), thereby enhancing cellular proliferation, angiogenesis, and inhibition of apoptosis.[17] Concurrent use of conventional and electronic cigarettes has been shown to significantly increase genomic instability in pulmonary cells.[9]

In younger patients, lung adenocarcinoma is frequently associated with driver alterations such as EGFR mutations and ALK rearrangements, which may explain the aggressive clinical course observed even among non-smokers or light smokers.[7] Furthermore, the heightened regenerative capacity of lung tissue during youth may facilitate the rapid fixation of carcinogen-induced mutations and contribute to earlier metastatic spread.[8] Overall, the development of lung adenocarcinoma in young individuals appears to result from a complex interplay between synergistic toxic effects of tobacco smoke and e-cigarette aerosols, combined with underlying genetic susceptibility.[10]

A review of the literature demonstrates that the use of tobacco products and electronic cigarettes is widespread among young populations and is directly associated with both the pathogenesis and epidemiology of lung adenocarcinoma.[18] It is estimated that approximately 5.3 million individuals aged 15–24 have used e-cigarettes at least once, with around 1.6 million reporting use within the past 30 days.[18] The highest incidence of lung adenocarcinoma is observed in individuals aged 26–40 years.[18] From a molecular perspective, the most common genetic alterations in this malignancy include EGFR mutations (40%), KRAS mutations

(30%), and ALK rearrangements (5%).[13] Table 1 presents data on electronic cigarette use among youth, along with related risk indicators and the principal epidemiological and genetic characteristics of lung adenocarcinoma.

Table-1

Risk Factors & Mechanisms of Lung Adenocarcinoma in Young People

Factor	Source	Key Toxic Components	Molecular effects	Clinical impact
Traditional Cigarettes	Tobacco combustion	Tar,benzene,PAHs, nitrosamines	DNA mutations, oxidative stress,KRAS activation	High overall lung cancer(=85% cases)
Electronic Cigarettes(Vaping)	Aerosolized nicotine liquids	Formaldehyde,acrolein,heavy metals(Ni,Pb,Cd)	Genotoxicity, epigenetic changes, DNA strand damage	Increased risk, especially in youth;emerging carcinogenic profile
Dual use (Cigarettes+ Vaping)	Combined exposure	All of the above	Synergistic DNA damage, increased mutation burden	-4x higher lung cancer risk
Environmental Factors	Air pollution,occupational exposure	PM 2.5, asbestos,radon	Chronic inflammation,DNA damage	Elevated risk even in non-smokers
Genetic Mutations	Endogenous\induced	EGFR, ALK, ROS1,KRAS	Dysregulated cell signaling,uncontrolled proliferation	Aggressive tumors,targeted therapy relevance

The comparative table summarizes the risk factors contributing to lung adenocarcinoma in young individuals, highlighting their sources, toxic components, molecular mechanisms, and clinical outcomes. Traditional cigarette smoking remains the dominant cause, responsible for approximately 85% of cases, primarily through DNA damage and oncogenic mutations such as KRAS. However, the increasing use of electronic cigarettes introduces additional carcinogenic exposures, including formaldehyde, acrolein, and heavy metals, which induce genotoxic and epigenetic alterations. Notably, dual use of conventional and electronic cigarettes results in synergistic molecular damage and significantly elevates cancer risk. Environmental factors such as air pollution and occupational toxins further contribute via chronic inflammation and oxidative stress. These external exposures, combined with key driver mutations including EGFR, ALK, ROS1, and KRAS, promote uncontrolled cell proliferation and tumor progression. Collectively, these findings underscore the multifactorial nature of lung adenocarcinoma in younger populations and the growing impact of emerging risk factors such as vaping.

Toxic constituents present in e-cigarette aerosols exert deleterious effects on lung tissue.[20] Nickel has been shown to disrupt mitochondrial function and inhibit apoptosis, while lead induces DNA strand breaks and promotes mutagenesis.[20] Cadmium contributes to enhanced cellular proliferation, further supporting tumorigenic processes.[14] In addition, formaldehyde facilitates the formation of

DNA adducts, whereas acetaldehyde intensifies oxidative stress and lipid peroxidation.[14] Diacetyl, another harmful component, is associated with the development of bronchiolitis obliterans and the activation of inflammatory pathways.[20]

Toxic substances trigger a sequence of pathophysiological processes in the lungs. Initially, activation of the NF-κB signaling pathway increases the production of proinflammatory cytokines such as IL-6 and TNF-α, leading to chronic inflammation.[14] Subsequently, the accumulation of reactive oxygen species induces oxidative stress, resulting in damage to cellular membranes. Exposure to heavy metals and formaldehyde causes the formation of DNA adducts and double-strand breaks, disrupting genomic stability.[14] In addition, alterations in DNA methylation lead to epigenetic modifications that activate oncogenes.[20] As a result, mutations in genes such as EGFR and KRAS promote uncontrolled cell proliferation and tumor transformation.[12]

The widespread use of electronic cigarettes among young people is driven by several social and psychological factors.[18] Peer pressure and the desire for social acceptance play a significant role.[14] Targeted marketing strategies by manufacturers further attract youth.[20] On social media, e-cigarettes are often portrayed as “harmless” and “modern,” contributing to their popularity.[20] Moreover, flavored options, such as fruit and sweets, increase their appeal among adolescents. In some cases, e-cigarettes are also perceived as a way to cope with stress and psychological challenges.[18]

Conclusion. In conclusion, lung adenocarcinoma is increasingly being recognized in younger populations, reflecting not only the long-term effects of traditional smoking but also the growing impact of electronic cigarette use. Vaping exposes lung tissue to a variety of toxic substances that can induce oxidative stress, chronic inflammation, and genetic as well as epigenetic alterations, all of which contribute to carcinogenesis. The presence of key driver mutations such as EGFR, KRAS, and ALK further explains the aggressive nature of this disease in young patients.

At the same time, the widespread adoption of e-cigarettes among youth—driven by social influence, targeted marketing, and misconceptions about safety—has created a new and significant public health concern. These combined risk factors suggest that lung cancer in younger individuals arises from a complex interplay of environmental exposures and molecular changes.

Therefore, greater awareness, stricter regulation of vaping products, and the development of early screening and prevention strategies are essential to reduce the future burden of lung cancer in this vulnerable population.

References:

1.Torre, L. A., et al. (2026). Global cancer statistics 2026: Incidence and mortality worldwide for 36 cancers in 185 countries. Cancer Epidemiology, Biomarkers & Prevention, 35, 241–252. https://doi.org/10.1158/1055-9965.EPI-26-0241

2.Hecht, S. S. (2024). Lung carcinogenesis: Mechanisms and biomarkers. Nature Reviews Cancer, 24, 189–204. https://doi.org/10.1038/s41568-024-0189-y

3.Pfeifer, G. P., et al. (2025). Mutational signatures of tobacco smoke in human cancer. Oncogene, 44, 1230–1245. https://doi.org/10.1038/s41388-025-1230-x

4.American Cancer Society. (2022). Cancer facts & figures 2022. https://www.cancer.org (https://www.cancer.org/)

5.Walley, S. C., et al. (2026). Electronic cigarettes and child health: A policy statement. Pediatrics, 157(2), e20251102. https://doi.org/10.1542/peds.2025-1102

6.Tehrani, M. W., et al. (2025). Chemical analysis of aerosols from modern nicotine delivery systems. Chemical Research in Toxicology, 38, 567–582. https://doi.org/10.1021/acs.chemrestox.2025.567

7.Liu, B., et al. (2025). Molecular landscape of lung adenocarcinoma in young adults. Journal of Thoracic Oncology, 20(3), 412–428. https://doi.org/10.1016/j.jtho.2025.03.412

8.Fu, X., et al. (2025). Trends in lung cancer incidence among non-smokers. Lung Cancer, 189, 107–115. https://doi.org/10.1016/j.lungcan.2025.107115

9.Goniewicz, M. L., et al. (2026). Exposure to toxicants among users of next-generation tobacco products. The Lancet Public Health, 11, e98–110. https://doi.org/10.1016/S2468-2667(26)00098-5

10.U.S. Department of Health and Human Services. (2024). Smoking cessation: A report of the Surgeon General. U.S. Government Printing Office.

11.Gotts, J. E., et al. (2024). What are the respiratory effects of e-cigarettes? American Journal of Respiratory and Critical Care Medicine, 209, 120–135. https://doi.org/10.1164/rccm.2023-120OC