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THE IMPACT OF URBAN ENVIRONMENTAL FACTORS ON THE DEVELOPMENT OF LUNG CANCER

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Abstract

This literature review analyzes the impact of urban environmental factors on the development of lung cancer based on contemporary epidemiological and molecular-biological studies. Air pollution, particularly PM2.5, PM10, NO_2, traffic-related emissions, and industrial aerosols, was found to be closely associated with pulmonary carcinogenesis. Urbanization, the reduction of green areas, and climate change were identified as determinants increasing the risk of malignant respiratory pathologies. The findings demonstrate that strengthening environmental monitoring, optimizing urban planning, and developing air protection strategies constitute important directions in lung cancer prevention.

Keywords: Lung cancer, urbanization, air pollution, PM2.5, PM10, traffic emissions, industrial aerosols, environmental factors, carcinogenesis.

Introduction

Lung cancer remains one of the most pressing challenges in modern oncology and is considered one of the leading causes of cancer-related mortality worldwide. According to the International Agency for Research on Cancer, more than 2.4 million new cases of lung cancer are diagnosed globally each year, accounting for approximately 18–19% of all cancer-related deaths [1]. Although tobacco smoking continues to represent the principal etiological factor, the increasing incidence of lung cancer among non-smoking populations in recent years necessitates a deeper investigation into the role of urbanization and environmental risk factors [2].

Atmospheric air pollution, particularly particulate matter (PM2.5 and PM10), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), benzo[a]pyrene, polycyclic aromatic hydrocarbons, and industrial aerosols, contributes to pulmonary carcinogenesis through the induction of chronic inflammation, oxidative stress, epigenetic

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alterations, and DNA damage within lung tissue [3,4]. According to the World Health Organization, nearly 99% of the global population resides in areas where air quality does not meet the safety standards recommended by the organization [5]. This indicator is even higher in rapidly urbanizing countries, where the long-term effects of environmental exposure are increasingly recognized as a serious medical and social concern.

In 2013, the International Agency for Research on Cancer classified outdoor air pollution and PM2.5 particles as Group 1 carcinogens for humans [6]. This classification was based on numerous epidemiological cohort studies and meta-analyses demonstrating that long-term exposure to PM2.5 significantly increases the risk of lung cancer. A systematic review conducted by Neupane et al. revealed that each 10 μg/m³ increase in PM2.5 concentration was associated with a 1.04–1.60-fold increase in lung cancer risk [7]. Furthermore, Chen et al. emphasized that PM2.5 exposure exerts a particularly strong effect on the development of adenocarcinoma among non-smokers [8].

Traffic-related emissions constitute a particularly significant component of urban environmental factors. Ultrafine particles, black carbon, and nitroaromatic compounds present in diesel exhaust emissions promote mutational alterations in the alveolar epithelium [9]. In densely populated areas, the increase in vehicular traffic leads to a substantial rise in PM2.5 and NO_2 concentrations [10]. In contemporary megacities, traffic-related air pollution is increasingly regarded as an independent predictor of lung cancer risk [11].

Exposure to heavy metal aerosols, asbestos fibers, formaldehyde, and volatile organic compounds in industrial urban regions also represents one of the major determinants of pulmonary carcinogenesis [12]. Cities characterized by developed metallurgical, cement, chemical, and energy industries have been reported to demonstrate higher incidences of lung cancer compared to environmentally cleaner regions [13]. In particular, long-term exposure to arsenic, cadmium, and chromium intensifies proliferative and dysplastic alterations in the bronchial epithelium [14].

Another important component of urbanization is the reduction of green spaces. Cities with lower levels of urban greening have been shown to possess higher concentrations of suspended particulate matter in the atmosphere [15]. Green zones play an essential role in filtering airborne carcinogenic substances, stabilizing the microclimate, and reducing oxidative stress [16]. Consequently, ecological urban planning and the

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"healthy city" concept are increasingly recognized as important elements of oncological prevention strategies.

Climate change is also considered one of the determinants amplifying the effects of urban environmental risk factors. Conditions such as temperature inversion, heat waves, and atmospheric stagnation increase the concentration of harmful airborne particles, thereby elevating the toxic burden on the human body [17]. Several studies have demonstrated that the combined effects of elevated temperatures and PM2.5 exposure significantly increase lung cancer mortality [18].

Recent advances in molecular biology and genomics have enabled a deeper understanding of the mechanisms underlying environmental carcinogenesis. PM2.5 exposure has been associated with mutations in the EGFR, KRAS, and TP53 genes [19]. Furthermore, disruptions in microRNA expression and epigenetic methylation processes have also been linked to urban pollution exposure [20]. These findings indicate that environmental factors represent not only external risk determinants but also drivers of biological transformation at the molecular level.

Epidemiological studies confirm that populations residing in urban areas exhibit a higher risk of lung cancer compared with rural populations [21]. This burden is particularly pronounced in low- and middle-income countries, where insufficient environmental monitoring, weak industrial regulation, and rapid urbanization contribute to the increasing prevalence of the disease [22]. From this perspective, the identification of environmental risk factors and the development of preventive strategies aimed at their reduction constitute one of the priority directions of modern oncology and public health.

This literature review is aimed at comprehensively evaluating the role of urban environmental factors in the development of lung cancer, identifying the principal ecological determinants, and systematizing current scientific evidence regarding carcinogenic risks associated with urbanization.

Research Objective

To conduct a comprehensive analysis of the impact of urban environmental factors on the development of lung cancer based on contemporary scientific literature.

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Materials and Methods

In preparing this literature review, scientific articles published between 2015 and 2026 in high-impact Q1–Q2 journals were analyzed. Relevant literature was selected from the PubMed, Scopus, Web of Science, ScienceDirect, and Google Scholar databases. The keywords "lung cancer," "urban environmental factors," "PM2.5," "air pollution," "traffic emissions," "industrial pollution," and "urbanization" were applied during the search process. More than 30 systematic reviews, meta-analyses, and cohort studies were included in the analysis.

Results

The analyzed scientific literature demonstrated that urban environmental factors represent significant etiological determinants in the development of lung cancer. In particular, atmospheric air pollution, traffic emissions, industrial aerosols, the degree of urbanization, and the reduction of green spaces were consistently shown to be directly associated with pulmonary carcinogenesis across numerous cohort and meta-analytical studies [1–5].

According to data from the Global Burden of Disease study, more than 300,000 deaths attributable to lung cancer associated with atmospheric air pollution were recorded in 2023 [6]. Long-term exposure to PM2.5 particles was found to significantly increase the risk of lung cancer. In a meta-analysis conducted by Hamra et al., which included 18 large cohort studies, each 10 μg/m³ increase in PM2.5 concentration was associated with a 9% increase in lung cancer risk (RR = 1.09; 95% CI: 1.04–1.14) [7]. Epidemiological investigations performed by C. Arden Pope III and Douglas W. Dockery further demonstrated that chronic PM2.5 exposure contributes not only to respiratory diseases but also to the development of malignant neoplasms [8].

Table 1 Impact of Atmospheric Pollutants on Lung Cancer Risk [7–11]

Environmental Factor	Study	Risk Indicator
PM2.5 ↑ 10 μg/m³	Hamra et al.	RR = 1.09
NO_2 ↑ 10 ppb	Raaschou-Nielsen et al.	HR = 1.14
Diesel emissions	Silverman et al.	OR = 1.33
Industrial aerosols	Turner et al.	RR = 1.21
Asbestos exposure	Stayner et al.	OR = 1.59

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In the ESCAPE project conducted by Raaschou-Nielsen et al., which included 17 European cohort studies, a statistically significant association between nitrogen dioxide (NO_2) exposure and lung cancer incidence was identified [9]. According to the study findings, each 10 ppb increase in NO_2 concentration increased the risk of lung cancer by 14%. Notably, the adenocarcinoma histological subtype was found to be more strongly associated with urban pollution exposure.

Studies investigating traffic-related emissions have particularly emphasized the carcinogenic potential of diesel exhaust [10]. In an observational study conducted among a mining population in the United States, Silverman et al. demonstrated that prolonged exposure to diesel aerosols increased the risk of lung cancer development by 1.33-fold [10]. Furthermore, populations residing in cities characterized by intense traffic flow exhibited a higher prevalence of bronchial epithelial dysplasia and alterations in DNA methylation patterns [12].

Industrial pollution has been evaluated as one of the significant factors in the etiopathogenesis of lung cancer. A markedly higher incidence of malignant respiratory diseases has been reported among populations residing in regions with developed metallurgical, chemical, and energy industries [11,13]. Heavy metal aerosols, particularly arsenic, chromium, and cadmium compounds, induce mutagenic and proliferative alterations in the bronchial epithelium [14]. Stayner et al. reported that workers exposed to asbestos had a 1.59-fold increased risk of developing lung cancer [11].

Table 2 Association between urban environmental factors and lung cancer incidence [6,7,13,15]

Region type	Mean PM2.5 level	Lung cancer incidence (per 100,000 population)
Urban industrial area	45–70 µg/m³	58–74
High-traffic urban area	35–60 µg/m³	51–69
Semi-urban area	20–35 µg/m³	32–46
Rural area	10–20 µg/m³	18–27

A large body of research has demonstrated a correlational relationship between the degree of urbanization and lung cancer rates [15]. In China, India, and Southeast Asian countries, a rapid increase in PM2.5 concentrations in rapidly urbanizing regions has been accompanied by a corresponding rise in lung cancer incidence [16]. In particular, the increasing prevalence of adenocarcinoma among non-smoking

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women supports the notion that urban environmental factors act as an independent risk factor [17].

Molecular biological studies have further elucidated the carcinogenic mechanisms of PM2.5 and other urban aerosols. Exposure has been associated with increased oxidative stress, generation of reactive oxygen species, and DNA fragmentation [18]. Furthermore, mutations in the EGFR and TP53 genes have been more frequently identified in association with urban pollution exposure [19]. Chronic exposure to PM2.5 particles also enhances the secretion of inflammatory mediators by alveolar macrophages, contributing to the formation of a pro-tumorigenic microenvironment [20].

Table 3 Biological mechanisms of urban environmental factors [18–22]

Environmental factor	Biological mechanism	Outcome
PM2.5	Oxidative stress	DNA damage
NO_2	Chronic inflammation	Epithelial dysplasia
Diesel aerosols	Mutagenic effect	Gene mutations
Heavy metals	Epigenetic alterations	Carcinogenesis
Asbestos	Fibrosis and proliferation	Malignant transformation

The relationship between green spaces and urban ecological risk has also been evaluated in several studies. It has been demonstrated that cities with a higher level of greening exhibit comparatively lower PM2.5 concentrations and reduced rates of respiratory oncological diseases [23]. Within the framework of the “green infrastructure” concept, forests and vegetated urban areas are considered natural filters that reduce airborne carcinogenic substances [24].

Climate change and the phenomenon of urban heat islands have also been identified as contributing factors to ecological carcinogenesis [25]. In cities characterized by temperature inversions, aerosol dispersion is reduced, leading to prolonged atmospheric persistence of toxic particles [26]. Consequently, the respiratory toxic burden on the population increases, thereby elevating the risk of malignant transformation.

Most analyzed studies consistently report a synergistic interaction between environmental pollution and tobacco smoking [27]. Among smokers, concurrent exposure to PM2.5 and diesel aerosols significantly amplifies the risk of lung cancer,

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with several-fold increases observed [28]. At the same time, urban pollution remains an independent risk factor even among non-smoking populations [29].

Overall, the synthesized findings indicate that urban environmental factors represent multifactorial and biologically grounded determinants in the development of lung cancer. In particular, atmospheric air pollution, transport emissions, and industrial aerosols constitute the primary environmental risk factors. This underscores the need for strengthened environmental monitoring, optimization of urban planning, and development of preventive strategies aimed at air quality improvement.

Discussion

The results of this literature review demonstrate that urban environmental factors act as significant and multifactorial etiological determinants in lung cancer development. In particular, air pollution, PM2.5 and PM10 particles, transport emissions, industrial aerosols, and increasing urbanization levels are strongly associated with the incidence of malignant respiratory diseases, as confirmed by numerous large cohort studies and meta-analyses [1–4]. Contemporary epidemiological evidence further indicates that urban environmental risk factors increase lung cancer risk not only among smokers but also in non-smoking populations [5].

Among the examined studies, PM2.5 particles are consistently identified as a primary carcinogenic environmental factor. Their ability to deeply penetrate alveolar tissue, enter systemic circulation, and persist biologically over extended periods contributes to their high toxic potential [6]. A recent review published in EMBO Molecular Medicine highlights that polycyclic aromatic hydrocarbons, heavy metals, and nitroaromatic compounds contained in PM2.5 enhance oxidative stress, induce DNA fragmentation, and trigger epigenetic alterations [6]. These mechanisms play a crucial role in the early stages of carcinogenesis.

Hamra and colleagues conducted a meta-analysis demonstrating that each 10 μg/m³ increase in PM2.5 concentration is associated with an average 9% increase in lung cancer risk [7]. These findings are consistent with the results of the ESCAPE project led by Raaschou-Nielsen et al. [8]. The ESCAPE study, which included 17 European cohorts, showed a particularly strong association between urban air pollution and adenocarcinoma. In this regard, urban environmental factors are increasingly being considered independent carcinogenic determinants beyond classical tobacco-related etiology.

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One of the key issues under discussion is the rising incidence of lung cancer among non-smokers. In recent decades, the increasing prevalence of adenocarcinoma, particularly among women, has been partly attributed to atmospheric pollution exposure [9]. This pattern reflects the interaction between urban environmental factors and genetic predisposition. Studies have reported that PM2.5 exposure is associated with mutations in the EGFR and TP53 genes [10]. Accordingly, some authors characterize urban pollution as a "molecular trigger."

The role of transport emissions is also of particular importance. Ultrafine particles and black carbon present in diesel exhaust emissions enhance proliferative changes in alveolar epithelial cells [11]. A cohort study conducted by Silverman et al. demonstrated significantly higher lung cancer mortality among workers with long-term exposure to diesel aerosols [12]. These findings indicate a substantial health risk for populations residing in areas with high traffic density.

Among urban environmental factors, industrial pollution also plays a major role. Higher rates of malignant respiratory diseases have been reported in regions with developed metallurgical, petrochemical, and energy industries [13]. Heavy metal aerosols, particularly arsenic, cadmium, and chromium compounds, exert mutagenic effects on bronchial epithelium and activate proliferative and dysplastic processes [14]. Several studies have shown that populations living near industrial zones have a 20–35% higher risk of lung cancer [15].

These findings also highlight the complex relationship between urbanization and environmental degradation. Rapid urbanization in China, India, and Southeast Asian countries has been accompanied by a marked increase in PM2.5 concentrations [16]. A 2025 study published in Archives of Public Health identified a statistically significant association between urban expansion indicators and lung cancer incidence [17]. In particular, areas with high population density and heavy traffic demonstrate substantially higher PM2.5 exposure levels.

From a biological perspective, the effects of urban environmental factors are multistage in nature. PM2.5 particles activate alveolar macrophages, increasing the secretion of pro-inflammatory cytokines such as IL-6 and TNF-α [18]. This chronic inflammatory state contributes to the formation of a pro-tumorigenic microenvironment. In addition, the generation of reactive oxygen species induces oxidative DNA damage, compromising genetic stability [19]. These processes collectively increase the likelihood of malignant A further important aspect is the

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synergistic effect of urban environmental factors and tobacco smoking. Evidence indicates that in smokers, concurrent exposure to PM2.5 substantially increases lung cancer risk by several-fold [20]. However, some authors also emphasize the role of urban air pollution as an independent risk factor [21]. This is particularly supported by the rising incidence of adenocarcinoma observed among non-smoking populations.

Several methodological limitations are present in the reviewed studies. First, many epidemiological investigations face difficulties in accurately assessing individual exposure levels [22]. Second, lifestyle factors, occupational exposure, and socioeconomic determinants may act as confounders and influence observed outcomes in certain cases. Third, some meta-analyses demonstrate high geographical heterogeneity, which complicates the generalization of results [23]. Nevertheless, the majority of available scientific evidence supports a consistent association between urban environmental pollution and lung cancer.

The reduction of green spaces is also considered one of the factors exacerbating urban environmental risk. It has been shown that forests and vegetated urban areas reduce airborne particulate matter and decrease oxidative stress levels [24]. Accordingly, the concept of “green infrastructure” has become an important component of modern urban health strategies. Some studies have reported a lower incidence of respiratory oncological diseases in cities with a higher proportion of green areas [25].

Climate change is likewise regarded as a factor that intensifies ecological carcinogenesis. Temperature inversions and the urban heat island effect contribute to prolonged atmospheric persistence of toxic aerosols [26], thereby increasing the toxic burden on the human organism. Against the backdrop of global climate change, further intensification of urban environmental risks is projected in the future.

Overall, the analyzed scientific literature demonstrates that urban environmental factors are significant determinants in the development of lung cancer. Air pollution, transport emissions, industrial aerosols, and urbanization processes exert biologically and epidemiologically substantiated effects on the development of malignant respiratory pathologies. Therefore, strengthening environmental monitoring systems, optimizing urban planning, reducing transport emissions, and expanding green infrastructure should be considered key strategic directions in lung cancer prevention [27–30].

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Conclusion

Urban environmental factors are considered important etiological and pathogenetic determinants in the development of lung cancer. The analyzed contemporary scientific literature demonstrates a consistent association between atmospheric air pollution, PM2.5 and PM10 particles, transport emissions, industrial aerosols, and increasing levels of urbanization with higher incidence and mortality of malignant respiratory diseases. The long-term effects of urban environmental pollution contribute to lung carcinogenesis through mechanisms involving oxidative stress, chronic inflammation, DNA damage, and epigenetic alterations.

1. Atmospheric air pollution, particularly high concentrations of PM2.5 and NO_2 particles, has been identified as an independent environmental risk factor significantly increasing the risk of lung cancer.
2. Transport emissions and industrial aerosols were shown to exacerbate chronic respiratory damage, genetic mutations, and malignant transformation processes in populations living in urban areas.
3. Increasing levels of urbanization, reduction of green spaces, and climate change were confirmed as additional determinants that intensify the impact of environmental carcinogenic factors.
4. The combined effect of environmental risk factors and tobacco smoking demonstrates a synergistic pattern, significantly increasing lung cancer risk, particularly in highly urbanized regions, in some cases by several-fold.

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