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The Impact of Long-Term Exposure to Air Pollution on Cancer Risk

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Abstract:

Air pollution is recognized as a critical global health challenge, contributing to approximately 7 million deaths annually and ranking among the most severe environmental crises confronting humanity. A substantial portion of the global population resides in regions where air pollution levels, driven by emissions from industrial activities, power generation, vehicular traffic, and residential combustion, significantly exceed the World Health Organization's recommended air quality guidelines. Ambient air pollution has emerged as a pressing global public health issue due to its widespread presence and severe adverse effects on human health, particularly its association with cancer.

Extensive evidence from epidemiological studies, experimental research in laboratory animals, and mechanistic investigations has established a strong link between air pollution particularly exposure to particulate matter (PM) and an increased risk of cancer incidence and mortality. Long-term exposure to PM and other air pollutants contributes to oxidative stress, inflammation, DNA damage, and epigenetic modifications, all of which are implicated in carcinogenesis.

In this study, we explore the intricate relationship between air pollution and the occurrence of specific cancers, including lung, bladder, and breast cancer, while also elucidating the underlying molecular mechanisms that drive pollution-induced carcinogenesis. By analyzing recent research findings and mechanistic data, we aim to enhance the understanding of how chronic exposure to air pollution contributes to cancer development. Addressing this issue requires global efforts to implement stringent air quality regulations, promote clean energy alternatives, and increase public awareness to mitigate the long-term health impacts of air pollution.

Keywords: Air pollution, Lung cancer, Particulate matter, Breast cancer

Introduction

Particulate matter (PM) is regarded as one of the most detrimental airborne pollutants, originating from both natural and human-caused sources or generated by atmospheric interactions (1). In the past few years, West Asia has seen desert dust storms, resulting in an increase in the frequency of dusty days and the daily average concentration of particulate matter with an aerodynamic diameter smaller than 10 μm (PM10) (2). Air pollution from the environment has been

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associated with a variety of health consequences, such as increased rates and death from heart disease, lung disease, and malignant illnesses (3). The respiratory and cardiovascular impacts of contaminants in air exposure have been thoroughly shown in both the industrial and general populations. Air pollution (AP) is a pervasive and intricate amalgamation of both solid and liquid particulates and gasses (4). PM and nitrogen oxides (NOx), particularly nitrogen dioxide (NO_2), are significant major constituents of pollution in the air. PM is often classified by size, differentiating particles with an aerodynamic diameter less than 10μm (PM10) from those with an aerodynamic diameter less than 2.5μm (PM2.5) (5). The former may be breathed, while the latter, classified within the PM10 fraction, can penetrate the lung alveoli and, in their tiniest form, enter the bloodstream. PM may originate from heating sources, vehicular traffic, industrial activities, and the agricultural sector, or it may be of natural origin (6).

AP is very detrimental, and even minimal concentrations pose a threat to human wellness. AP is currently regarded as a critical worldwide health concern and is accountable for an increasing array of health effects (7). Rapid development may result in increased exposure to harmful levels of environmental contaminants. Outdoors AP has long been a significant health issue, and the situation deteriorates daily (8). The primary contributor to AP in large urban areas is an inadequate transportation infrastructure. The level of AP in densely populated cities exceeds that of other regions, potentially causing significant adverse effects on human health by disrupting water and land environments (9). Tehran, Beijing, Sao Paulo, Shanghai, Cairo, Bangkok, Mexico City, and Jakarta are the most polluted regions globally. A significant sixteen percent of fatalities connected to illnesses that are not transmitted are attributable to AP (10). Ambient AP, recognized as a cancer-causing agent for humans, has been linked to the onset of several cancers. Numerous research examining industrial AP contact have shown a correlation between AP and an increased risk of lung cancer (11). The mechanisms behind this connection are thought to entail the onset and development of carcinogenesis via damage to the DNA, inflammatory processes, oxidative stress, and interruption of cell processes (12). Nonetheless, the correlation between air pollution and various cancer forms stays mostly ambiguous. Several observational investigations have attempted to investigate possible correlations between AP and cancers, including breast, bladder, and skin cancers; nevertheless, the results have been intricate and sometimes conflicting (13). The purpose of this study is to investigate the relationship between long-term exposure to AP and some cancers.

Carcinogenic mechanism of air pollution

Inflammatory substances associated with AP, including tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and interleukin-1β (IL-1β), foster pro-inflammatory conditions that facilitate tumorigenesis (14). Inflammatory signaling networks, including nuclear factor-kappa B (NF-κB) and signal transducer and activator of transcription 3 (STAT3), are often active in cancer (15). These processes modulate the activity of genes associated with cell survival, growth, blood vessel development, and dissemination, hence creating a conducive environment for tumor formation. Inflammation fosters a tumor-promoting milieu by affecting several elements of the tumor's microenvironment, such as immune system cells, fibroblasts, endothelial cells, and the extracellular matrix (16). Additionally, pro-inflammatory and stromal cells have a role in the modification of the extracellular matrix, promoting tumor expansion and dissemination. Inflammation has a complex involvement in the emergence of cancer, affecting several facets of tumor origin, growth, and development (17). Long-term inflammation thus produces damage to DNA, establishes a tumor-promoting milieu, hinders immune surveillance, and enhances angiogenesis. A troubling element of air pollution is its capacity to produce mutagenesis, resulting in genetic modifications in living creatures (18). Laboratory rats situated close to contaminated manufacturing areas (1 km downwind from two integrated steel mills) exhibited a greater transmissible mutation rate at tandem-repeat DNA loci than those at a reference site 30 km distant, with this impairment mainly attributed to a rise in mutations inherited via the paternal germline (19). Mutations in essential regulatory genes may impair cellular processes, resulting in unregulated cell proliferation and the onset of cancer. The findings indicate that mitigating air pollution is essential to diminish its mutagenesis capacity on cells (20).

Breast cancer and air pollution

Present medical information indicates that breast cancer (BC) is the most often identified cancer among women internationally, resulting in significant morbidity and mortality, and is the main cause of cancer-related deaths in females globally (21). Based on the Global cancer prevalence statistics, the worldwide incidence of cancer is steadily growing, especially in developing nations, with an estimated 1.7 million new cancer cases and 521,900 deaths recorded globally in 2012. Globocan (2020) estimates 2261419 new BC cases (both sexes, all ages) in 2020, representing 11.7% of all new cases of cancer (22). BC killed an estimated 684996 people, accounting for 6.9% of all cancer fatalities. BC has a global prevalence rate of 47.8 and a death rate of

13.6 per one million individuals (23). Scientists at the National Institutes of Health discovered that residing in regions with elevated particle AP correlates with a higher prevalence of BC. The research, released in the Journal of the National Cancer Institute, is one of the biggest investigations currently underway examining the correlation between outside AP, particularly fine particulate matter, and the prevalence of BC (24). The study was conducted by researchers from the National Institute of Environmental Health Sciences (NIEHS) and the National Cancer Institute (NCI), both affiliated with NIH. Research in the Journal of the National Cancer Institute revealed that residing in regions with elevated particle AP correlates with an 8% rise in the prevalence of estrogen receptor-positive BC (25). Inconsistencies persist in the research about the correlation between AP and BC incidence. Crouse et al. identified a positive correlation between the amount of NO2 and BC risk with 95% confidence; however, other investigations reported positive relationships with limited, small, or null statistical significance (26). Hystad et al. identified a substantial correlation between levels of NO2 and premenopausal BC, whereas a marginal connection was seen between NO2 and postmenopausal BC in women (27). Datzman et al. identified substantial positive correlations between NO2, PM10, and BC occurrence via medical information from a local insurance provider in Germany, which encompasses about one million females and 9,577 cases (28). AP can raise the possibility of BC by engaging with estrogen receptors, inducing inflammatory and oxidative stress, and disturbing hormonal equilibrium. Certain research indicates that the impact of AP on BC risk is more pronounced in younger women (29). Garcia et al. examined the association between elevated BC possibility and various air pollutants, such as acrylamide, carbon tetrachloride, chloroprene, 4,4'-methylene bis (2-chloroaniline), propylene oxide, and vinyl chloride; however, the risk percentages were not significantly different (30). Furthermore, a review analysis synthesized the findings from 8 case-control investigations and 9 cohort investigations, indicating little evidence to substantiate a correlation between PM and BC incidence (25). Goldberg et al. indicated that exposure to atmospheric NO_2 and UFPs may elevate the likelihood of developing postmenopausal BC, particularly in women with positive estrogen and progesterone receptor expression (31). The varied results and inferences may be attributed to climate, cultural disparities, or population density; nevertheless, more investigations and analyses are required to establish a definitive association between AP and BC risk.

Lung cancer and air pollution

Lung cancer (LC) is the most prevalent malignancy and the main reason of death due to cancer globally (32). Tobacco use is the biggest recognized environmental factor, with 81.7% of LC occurrences attributed to it (33). Moreover, multiple investigations have shown that contact with outside pollutants, such as PM, NOx, ozone, and sulfur dioxide, could have an association with LC. LC is the predominant reason for worldwide cancer mortality and incidence, with around 2 million diagnoses and 1.8 million fatalities (34). Lung cancers are the second most prevalent diagnosis of cancer in both men and women, behind prostate and BC, respectively. Contaminated air comprises a minimum of two categories of carcinogens: polycyclic aromatic hydrocarbons (PAHs) and n-nitroso substances, including nitrosamines (35). Studies indicate that PM2.5 could encourage tumorigenesis in pulmonary tissues. PM2.5 exhibits elevated PAH concentrations, capable of inducing oxidative stress and activating aryl hydrocarbon receptors in human bronchial epithelial cells (BEAS-2B) (36). Stimulation of the aryl hydrocarbon receptor facilitates the spread and invasion of LC cells. Research revealed that death from LC attributable to PM2.5 is significantly greater in women than in males (37). Besides inducing local inflammation and oxidative stress, PM2.5 infiltrates the lungs and terminal bronchioles, subsequently entering the bloodstream and eliciting widespread inflammation. A recent study indicates that prolonged contact with PM10 in a human NSCLC cell line resulted in a reduction of the mitotic rate and the proportion of cells in the G2/M phase relative to a control group (38). Long-term contact to fine particulate AP may induce genotoxicity and mutagenicity. It can raise the likelihood of LC by proinflammatory destruction, the generation of reactive oxygen species, and DNA oxidation (39). Consequently, particularly in those with a significant genetic predisposition, extended exposure to AP may elevate the occurrence of lung tumors. Huang et al. established a synergistic impact of elevated genetic vulnerability (assessed using a polygenic risk rating derived from genome-wide correlation research) and increased PM2.5 exposure on lung cancer frequency (40). Outside air pollution has been associated with many epigenetic alterations, such as adjustments to post-translational histone modifications, 5-hydroxymethylation, and particularly DNA methylation (DNAm) (41). DNAm is a biochemical alteration that happens in cytosines, especially within the CpG context, and influences the expression of genes along with multiple other roles (42). As noted with TP53, hypermethylation facilitates gene silence, while DNA hypomethylation leads to chromosomal instability and the stimulation of retrotransposon sequences and repetitive motifs, including LINE-1 and Alu. DNA hypomethylation

additionally impacts essential chromosomal areas, including the subtelomeric and pericentromeric areas (43). Access to atmospheric air pollution, both short-term and long-term, correlates with aberrant DNA methylation. Additional research indicates that epithelial cells from humans exposed to PM2.5 have increased susceptibility to hypomethylation and transcriptional stimulation of multiple genes and microRNAs (miRNAs), hence altering cancer-related signaling networks (44). PM2.5 may generate alterations in long non-coding RNAs (lncRNA) like loc146880 through reactive oxygen species (ROS), facilitating autophagy and cancer in lungs (45).

Bladder cancer and air pollution

In 2018, bladder cancer ranked as the 10th most prevalent cancer globally. The rates of incidence were elevated in males, for whom it ranked as the sixth most prevalent malignancy (46). The greatest rates of incidence were seen in the advanced nations. The majority of bladder cancers are classified as urothelial carcinoma (UC), with the rest including squamous cell carcinoma, sarcoma, lymphoma, and adenocarcinoma (47). Around 75 percent of these cases are non-muscle-invasive bladder cancer (NMIBC). Non-muscle invasive bladder cancer (NMIBC) has a significant frequency owing to its sluggish natural history and elevated relapse rate (48). Environmental hazards and comprehensive exposome (the whole of exposure from both inside and outside elements) have a substantial role in the progression of BC. Consequently, comprehending these risk variables is essential for protection (49). Continuous exposure to AP, especially PM2.5, is linked to a heightened chance of bladder cancer. A comprehensive review and meta-analyses indicated that a 5 µg/m3 rise in PM2.5 exposure correlates with a 6% greater likelihood of bladder cancer. The research identified indicative evidence of a correlation between prolonged contact with PM2.5 and the prevalence of bladder cancer (8). Separate research indicated that the correlation between zinc and PM2.5 underscores the significance of pollution from factories. A comprehensive review and meta-analysis revealed that a 10 µg/m3 rise in NO2 correlates with a 4% elevated risk of bladder cancer (50). Nevertheless, several investigations have not shown a correlation between NO2 and bladder cancer. The existing information about the correlation between the prevalence and morbidity of bladder and kidney cancer and AP contact primarily originates from industrial settings including contact with gasoline vapors, chlorinated solvents, asbestos, pesticides, and polycyclic aromatic hydrocarbons (PAHs) (51). A study and meta-analysis revealed an elevated incidence of urinary bladder cancer among motor vehicle drivers who were professionally subjected to significant levels of traffic-related AP (52). Nonetheless, although the level of airborne contaminants in the general public is significantly lower than that experienced by drivers and workers in factories, it is plausible to infer that long-term contact in the general population may be linked to an elevated likelihood of bladder cancers (53). Multiple pathways could account for the association between AP contact and bladder cancer. Current research on animals indicates that contact with PM2.5 may disrupt the angiotensin/bradykinin pathway, leading to premature kidney injury, oxidative stress, and/or inflammation, ultimately resulting in cancer (54). These biological results indicate that prolonged contact to air pollution may cause abnormalities in the urinary tract, potentially resulting in urological malignancies (54).

Discussion and conclusion

The urban air comprises a complicated amalgamation of oxidizing gases and particulates of varying sizes and compositions. Contact with AP elements may harm the biological molecules of people and animals, potentially leading to illnesses like malignancy (55). The International Agency for Research on Cancer has classed contact with entire diesel engine exhaust as probably dangerous to people, according to little human data and significant evidence from animal studies (56). Several epidemiological research has demonstrated correlations between fine particulate matter in AP and deaths from LC. Emission origins can involve natural phenomena like wildfires, volcanic eruptions, and dust storms (57). Anthropogenic pollutants include emissions from combustion methods utilized in heating, energy generation, industrial activities, and vehicular traffic. The concentration of contaminants in urban air around us is influenced by both local emissions and extended transportation as well as meteorological factors (58). Irrespective of the extent of air pollution contact, it is standard procedure to classify its components as gases and particles. AP poses a considerable and extensive risk to public health, with 99% of the global population inhaling polluted air, as reported by the World Health Organization (WHO) (59). The hazards linked to AP parallel those induced by cigarette smoking. Contact with air pollution may result in cancer, stroke, respiratory disorders, heart problems, and other health complications. Air pollution jeopardizes advancements in alleviating the global cancer burden by exacerbating the annual increase in avoidable cancer cases (60). Mitigating air pollution directly decreases the risk of lung cancer; however, the measures implemented—such as expanding green spaces, utilizing cleaner energy sources, and promoting active transportation (walking and

cycling)—also yield numerous health advantages, including improved dietary habits, enhanced physical activity, and reduced prevalence of other non-communicable diseases and co-morbidities, thereby diminishing the probability of developing additional cancers (61). AP may adversely affect the quality of life for those with cancer by intensifying respiratory symptoms, elevating tiredness, diminishing physical activity, and aggravating treatment side effects. It may also hinder cancer treatments by diminishing the efficacy of chemotherapeutic agents, exacerbating surgical difficulties, and possibly interfering with targeted medicines and immunotherapies (62). AP become a substantial factor in its occurrence. This analysis examines the correlation between air pollution—particularly particulate matter (PM2.5), industrial pollutants such as vinyl chloride and benzene—and the heightened risk of cancer (63). Air pollution may mechanistically induce cell damage by oxidative stress, inflammation, and genetic alterations, hence leading to cancer formation. Epidemiological data from cohort and regional studies indicates a favorable association between prolonged contact with air pollution and higher rates and death of cancer (64). Moreover, air pollution has been demonstrated to deteriorate survival results in people with cancer, especially those detected during the initial stages (64). The analysis underscores the requirement for more stringent air quality laws and pertinent studies on the fundamental mechanisms affected by air pollution. Mitigating contact with air pollution is essential for decreasing the chance of cancer and enhancing overall health results.

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Ethics approval and consent to participate

Not applicable

Conflict of Interest

The author declared no conflict of interest.

Consent for publication

Not Applicable

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