Environmental Pollution exposure unlocks the pathogenesis of Lung Cancer: An Update

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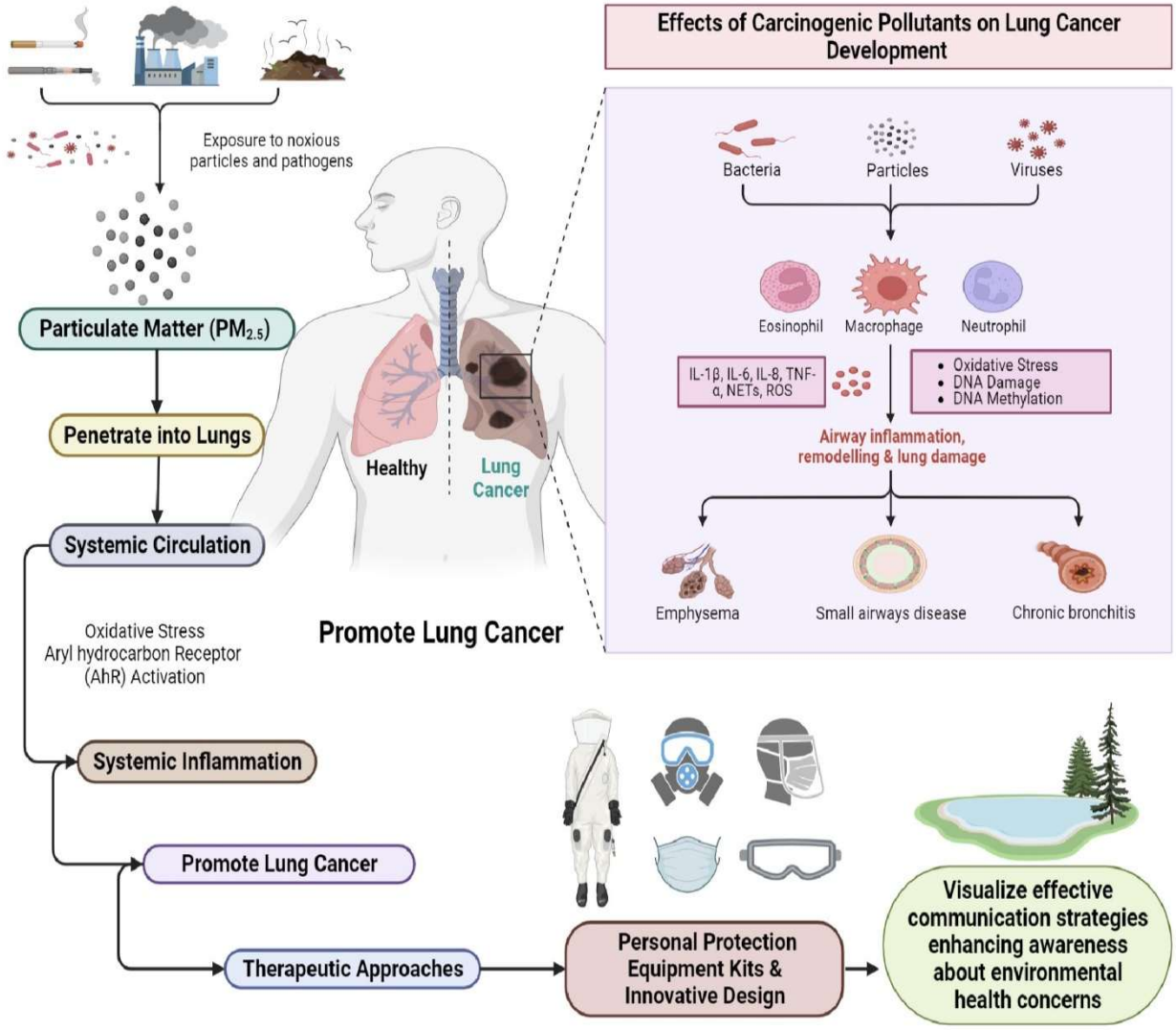
ABSTRACT

Air pollution and lung cancer are global issues. This study examines how $PM_{2.5}$ and PAHs cause and accelerate lung cancer. Due to environmental and occupational contaminants, lung cancer is common among nonsmokers. Global and national regulations are needed to govern air pollution emissions and design PPE to avoid lung injuries, considering physiological systems. The analysis highlights the need for effective preventative measures and laws to reduce the influence of air pollution on lung cancer rates. It provides extensive insights into the link between cancer-causing substances and lung cancer. The work discusses genetic predispositions, environmental exposures, and occupational risks that cause lung cancer, focusing on tobacco smoke and secondary exposure. Lung cancer risk is rising due to air pollution, prompting preventative and regulatory measures. Carcinogenic pollutants cause multiple cancers. Benzene, formaldehyde, arsenic, PVC, PAHs, nitrosamines, and other carcinogenic pollutants are discussed in this work. To reduce cancer risk, regulations, public health initiatives, and personal preventive measures should be implemented. They are controlling cancer-causing contaminants. Legislation, personal safety, public awareness, and education are included. Education, public-private partnerships, community participation, and media exposure are also encouraged—a significance of regular updates and monitoring in informing the public about environmental health advances. Individuals, communities, and governments are working to reduce cancer-causing toxins and improve the environment.

Keywords: $PM_{2.5}$ (Fine Particulate Matter), PAHs (Polycyclic Aromatic Hydrocarbons), PPE (Personal Protection Equipment), PVC (Polyvinyl Chloride), Nitrosamines, Public Awareness



Highlights

1. The research investigates the impact of $PM_{2.5}$ and PAHs from air pollution on the occurrence and progression of lung cancer, specifically in those who do not smoke.
2. Lung cancer is affected by genetic predispositions, mutations, environmental exposures, and occupational hazards, with smoking being a prominent risk factor. The presence of carcinogenic pollutants, particularly particulate matter originating from combustion, as well as polycyclic aromatic hydrocarbons (PAHs), is increasingly linked to the progression of lung cancer.
3. The presence of carcinogenic air pollutants, including PAHs, nitrosamines, and $PM_{2.5}$, has a substantial influence on the development and advancement of lung cancer. Among these pollutants, $PM_{2.5}$ is specifically recognized as the leading contributor to lung cancer cases among those who do not smoke.
4. Extended exposure to cancer-causing substances, namely air pollution, has been associated with the development of lung cancer and other respiratory diseases. This exposure may also lead to lung cancers, such as colorectal and prostate cancers.

5. The study explains how exposure to cancer-causing pollutants like $PM_{2.5}$ and PAHs may cause harm to DNA, resulting in mutations and the activation of cancer-causing genes, while also deactivating genes that limit tumor growth.
6. To avoid and regulate exposure to cancer-causing chemicals, it is crucial to implement regulatory measures, use personal protective equipment, raise public awareness, and provide education. To improve public awareness and education on environmental health literacy, it is recommended to use public-private partnerships, involve the community, and increase media exposure.

1. Introduction

In the era of globalization, cancer is the biggest problem that humanity is facing right now. Currently, among all types of carcinomas, lung cancer appears as the second most prevalent in the United States and is the primary cause of cancer-related death.[1] Cases of lung cancer are elevated substantially; studies revealed that in 2018, there were 2.09 million (11.6% of total cases) new cases worldwide, and a 1.76 million mortality rate (18.4% of total cancer deaths).[2] Lung cancer primarily arises from genetic predispositions, mutations, and environmental exposures, with smoking being the most significant risk factor, but the role of carcinogenic pollutants, particularly combustion-derived particulate matter (PM) and polycyclic aromatic hydrocarbons (PAHs), is gaining ground in the development of lung cancer.[3] Particles having a circumference that is less than 2.5 μm, referred to as fine particulate matter ($PM_{2.5}$), present a significant threat to human health. The main method of absorption for $PM_{2.5}$ is by inhaling, which enables the particles to enter the bloodstream through the lung alveoli. Oxidative stress and the presence of reactive oxygen or nitrogen species (ROS, RNS) in respiratory function contribute to the development or worsening of several illnesses by stimulating the synthesis of substances that promote inflammation in the lungs. $PM_{2.5}$ emitted into the atmosphere by automobiles and industrial facilities is well-documented to increase the likelihood of developing lung cancer and its associated mortality.[4] Studies have demonstrated the carcinogenic properties of PAHs, which are involved in DNA damage, mutations, and tumor initiation and promotion.[5] The potential involvement of the aryl hydrocarbon receptor (AhR) in regulating the metabolism and genotoxicity of PAHs suggests its essential role in lung cancer development associated with air pollution. Furthermore, it underscores the increasing prevalence of lung cancer among non-smokers, with air pollution identified as a major cause. The International Agency for Research on Cancer (IARC) classifies outdoor air pollution and $PM_{2.5}$ as carcinogenic to humans, contributing to a large number of lung cancer deaths globally. The review also emphasizes the significant impact of reducing air pollution, which is expected to result in a substantial reduction in lung cancer deaths.[6] Furthermore, the investigation considers the physiological mechanisms associated with lung damage triggered by air pollution, highlighting the need to implement both domestic and international standards that limit the release of air pollutants and ensure the accessibility of personal protective equipment (PPE) as preventive measures. The rising number of cases of lung cancer, particularly among those who do not smoke, may be attributed to occupational and environmental exposures. This focuses on the need to execute primary preventive strategies for addressing the widespread problem.[7] Overall, the review provides comprehensive insights into the association between carcinogenic pollutants, particularly PAHs, and the development of lung cancer, emphasizing the significant role of air pollution as a major cause of lung cancer mortality. It also underscores the crucial need for effective preventive measures and policies to mitigate the impact of air pollution on lung cancer incidence and prevalence.[5,7,8]

2. Causes of lung cancer

The primary factor influencing the development of lung cancer is cigarette smoking. Approximately 80-90% of cases of lung cancer are attributed to smoking since tobacco smoke contains several carcinogens.[9] Secondhand smoke is a significant factor in the development of lung cancer due to its inclusion of several hazardous chemicals that are also found in tobacco smoke.[9,10] Radon, also called an innate radioactivity gas found in dirt and stones, may lead to the development of lung cancer if breathed in, as well as occupational exposure to chemicals such as arsenic, diesel fumes, asbestos, and other related substances. One's genetic makeup and family history may also impact the probability of developing lung cancer. The primary cause of lung cancer is smoking however, it may also occur in those who have never smoked or have had little exposure to secondhand smoke.[9–11]

2.1. Exposure to carcinogenic pollutants

Exposure to carcinogenic pollutants, such as certain chemicals and radiation, can contribute to the development of cancer. Some of the common carcinogenic pollutants include:

- Aflatoxins
- Arsenic
- Asbestos
- Benzene
- Formaldehyde
- Radon
- Tobacco Smoke
- Polyvinyl chloride (PVC)
- Polycyclic Aromatic Hydrocarbons (PAHs)
- Nitrosamines

These chemicals are present in air, water, food, and several other household goods. As an example, common carcinogenic air pollutants in the United States include formaldehyde, benzene, acetaldehyde, and carbon tetrachloride.[12–14] To mitigate the risk of cancer, it is important to comprehend the mechanisms by which exposure to these contaminants heightens the risk and to implement preventive actions accordingly.[13,14]

3. Significance of carcinogenic substances to lung cancer progression

The presence of cancer-causing contaminants greatly influences the occurrence of lung cancer. Polluted air includes carcinogens such as PAHs and n-nitroso compounds, specifically nitrosamines, which are linked to the development of lung cancer.[13] PM2.5 is considered the primary factor responsible for lung cancer in those who have never smoked, highlighting the cancer-causing impact of air pollution.[12–14] Moreover, the presence of compounds such as arsenic, asbestos, beryllium, cadmium, coal and coke fumes, silica, and nickel, both in work environments and in the general surroundings, may significantly elevate the likelihood of getting lung cancer. To mitigate the likelihood of developing lung cancer, it is crucial to comprehend and restrict one's contact with these harmful substances.[8,12–14]

3.1. Mechanisms of action of carcinogenic pollutants

The study specifically identifies two carcinogenic air pollutants: n-nitroso compounds such as nitrosamines, and PAHs.[8,13] $PM_{2.5}$, which contains substantial amounts of PAHs, is thought to enhance the invasion and spread of lung cancer cells by activating the AhR in human bronchial epithelial cells (BEAS-2B) and inducing oxidative stress. Significantly, women have a higher mortality rate than men for lung cancer caused by $PM_{2.5}$.[8,15,16]

Evidence demonstrates that $PM_{2.5}$ not only induces inflammation and oxidative stress at the local level but also triggers systemic inflammation by profoundly infiltrating the lungs and terminal Bronchioles.

PM_{10} has genotoxic and mutagenic properties, which elevate the susceptibility to lung cancer by causing inflammatory harm, generating reactive oxygen species, and inducing oxidative DNA damage. Long-term exposure to PM_{10} in the A549 human non-small cell lung cancer (NSCLC) has been shown in Fig.2, line has been linked to a reduction in the mitotic index and the proportion of cells in the G2/M stage. Significantly, prolonged exposure to air pollution might heighten the likelihood of developing lung cancer in those with a preexisting genetic predisposition.[15,16] It was discovered that increased exposure to $PM_{2.5}$ and a high polygenic risk score, which quantifies the genetic susceptibility to lung cancer, had a synergistic effect on the development of the illness.[16] Furthermore, those who do not smoke and those with a limited history of smoking are more prone to being linked with exposure to $PM_{2.5}$ and the development of lung cancer caused by EGFR mutations. A study including 33,000 patients from four different groups discovered a significant association between exposure to $PM_{2.5}$ and the development of EGFR mutant-driven lung cancer. Air pollution facilitates cancer development by infiltrating the lung epithelium with macrophages. These macrophages then produce interleukin-1, which triggers a state resembling progenitor cells in EGFR-mutant lung alveolar type II epithelial cells.[16,17] Significantly, the administration of anti-interleukin-1 medicine has the potential to diminish the intensity of this process.[16,17] The consequence of PM2.5 air pollution on the development of lung cancer. Patients with an inherited defect or other influencing characteristics that make them more susceptible to lung cancer are extremely vulnerable to the adverse effects of prolonged exposure to air pollution. The primary factors leading to this phenomenon include carcinogens, oxidative stress, stimulation of the AhR, genotoxic and mutagenic effects, and the elevated invasion and metastatic potential of lung cancer cells.[17] as shown in Figs 1&2.

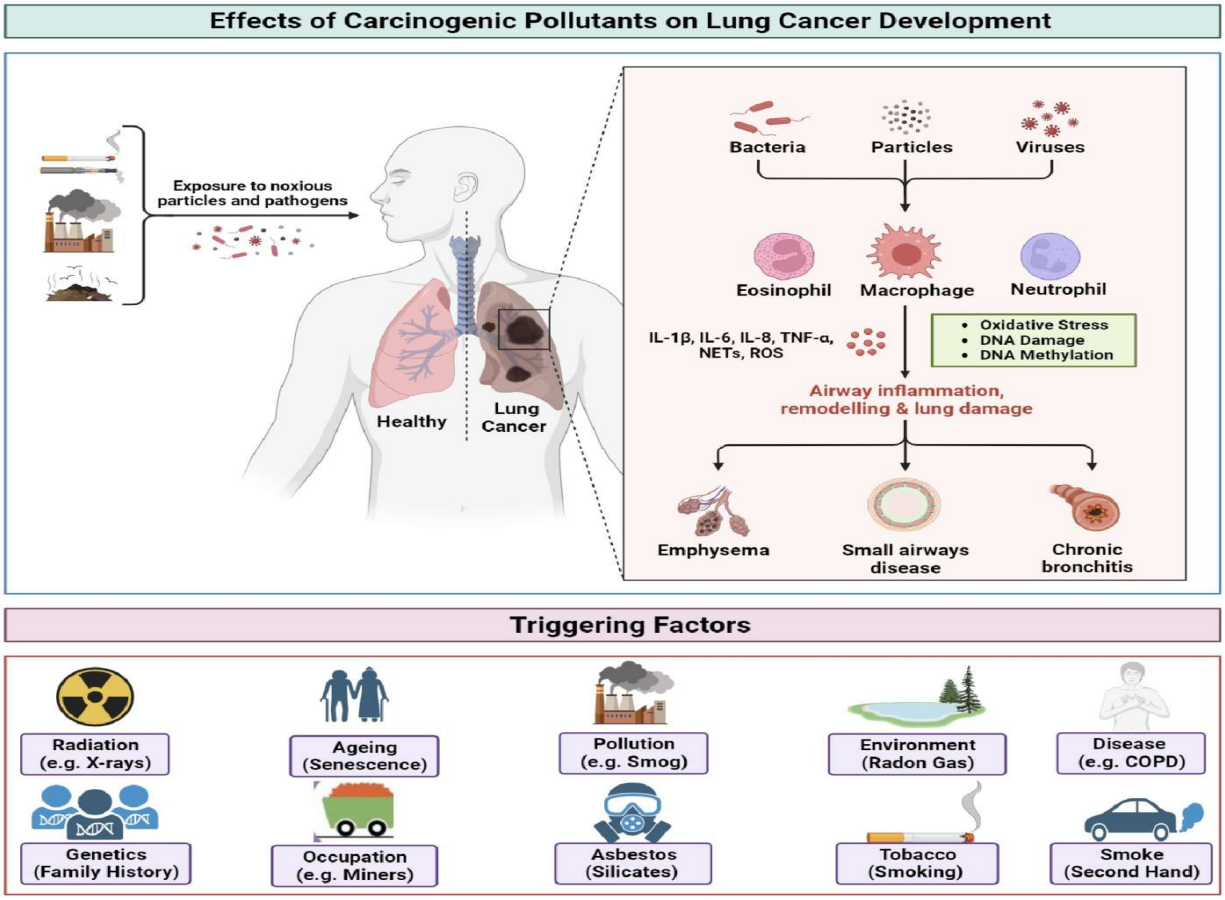


Figure 1: Effect of Carcinogenic Pollutants on Lung Cancer Development

3.2. Impact of long-term exposure to carcinogenic pollutants on lung health

Prolonged exposure to carcinogenic contaminants, such as air pollution, may profoundly affect lung health, possibly resulting in the onset of lung cancer. Outdoor air pollution, namely particle pollution, has been categorized as a class I human carcinogen and is linked to the onset of lung cancer and chronic obstructive pulmonary disease (COPD).[18,19] Exposure to some air pollutants, such as PM, ozone, nitrogen dioxide (NO_2), PAHs, and volatile organic compounds (VOCs), has been associated with acute worsening of respiratory symptoms and the occurrence of lung cancer.[18] Furthermore, there is a substantial correlation between the development of lung cancer and the presence of some harmful substances such as radon, asbestos, arsenic, and diesel fumes.[18–20] Moreover, research has shown that prolonged exposure to air pollution, including $PM_{2.5}$ and NO_2, might potentially heighten the likelihood of non-pulmonary malignancies, such as colon and prostate cancers, among elderly individuals.[21–23] It should be emphasized that even small amounts of air pollution exposure might increase individuals' vulnerability to acquiring various types of cancers.[22] Hence, it is important to minimize the risk of lung cancer and other respiratory ailments by avoiding exposure to these carcinogenic chemicals.[22,24,25]

3.3. Link between exposure to carcinogenic pollutants and lung cancer development

Exposure to carcinogenic contaminants, such as air pollution, may increase the likelihood of acquiring lung cancer. Outdoor air pollution, namely particle pollution, has been categorized as a class I human carcinogen and is linked to the onset of COPD.[21,26] Exposure to some air pollutants, including PM, ozone, NO_2, PAHs, and VOCs, has been associated with acute worsening respiratory symptoms and the occurrence of lung cancer.[26] Furthermore, the inhalation of some contaminants, such as radon, asbestos, arsenic, and diesel emissions, has been closely linked to the development of lung cancer.[12,14,26] Prolonged exposures to air pollution, namely PM2.5 and NO_2 might potentially elevate the likelihood of developing non-lung malignancies, such as colorectal and prostate cancers, in elderly individuals.[27] Even small amounts of air pollution exposure may render individuals vulnerable to the development of certain cancers.[28] Hence, it is important to decrease the level of contact with these cancer-causing chemicals to mitigate the likelihood of developing lung cancer and other respiratory ailments.[24,25,28,29]

4. Effects of carcinogenic pollutants on the body

Prolonged exposure to carcinogenic substances may lead to an increased susceptibility to cancer, among other detrimental health consequences. For instance, air pollution consists of carcinogenic substances that have been associated with several types of cancer, such as pancreatic, lung, bone, stomach, breast, and liver cancers.[30,31] Studies have shown a correlation between these contaminants and the development of COPD, particularly particle pollution. Interaction with certain air pollutants is associated with both lung cancer and acute respiratory complications, as demonstrated by data.[30] The pollutants included NO_2, PM, ozone, and VOCs. Furthermore, there exists significant evidence establishing a connection between lung cancer and exposure to certain pollutants, including radon, asbestos, arsenic, and diesel fumes.[32,33] In addition, elderly individuals may have an increased susceptibility to non-pulmonary malignancies such as colorectal and prostate cancers as a result of prolonged exposure to air pollution, including NO_2 and $PM_{2.5}$. Individuals may have an increased susceptibility to developing these malignancies even when exposed to low amounts of air pollution.[34] To minimize the likelihood of developing cancer and other respiratory diseases, it is necessary to prevent contact with these harmful substances that might cause cancer.[14,31–34]

4.1. Role of pollutants in DNA damage and mutation

Outdoor air pollutants have the potential to induce DNA damage and mutations, resulting in a range of health problems, including asthma, heart disease, and lung cancer.[35] The primary ways in which contaminants induce DNA damage include:

- **Particulate matter (PM)**: A significant component of ambient air pollution, it has the potential to induce oxidative DNA damage.[36] Small particles have a larger carcinogenic potential compared to large particles. Additionally, air with a greater concentration of small particles has a stronger mutagenic effect. PM can produce reactive oxygen species (ROS) on its surface, modify the functioning of mitochondria or NADPH-oxidase, and stimulate inflammatory cells that can generate ROS and reactive nitrogen species.[36]
- **Oxidative stress:** The damage to DNA caused by oxidative stress is believed to be a significant mechanism by which urban particulate air pollution exerts its effects. This damage may be associated with an increased risk of cancer. It may be used as an indicator of oxidative stress, which is significant for various health conditions induced by particulate air pollution.[36]
- **DNA methylation (DNAm):** Exposure to air pollution has been linked to alterations in DNAm, which might potentially contribute to the development of diseases. Nevertheless, the precise processes by which air pollution affects DNAm remain incompletely comprehended.[37] Research has shown that being exposed to airborne PM in the surrounding environment is linked to respiratory and cardiovascular illnesses as well as cancer. The oxidative stress caused by PM can occur through various mechanisms, including the direct production of ROS from the particle surface, the presence of soluble compounds like transition metals or organic compounds, the dysfunction of mitochondria or NADPH-oxidase, and the activation of inflammatory cells that can generate ROS and reactive nitrogen species. This oxidative stress can lead to DNA damage, which may increase the risk of cancer and serve as an indicator of oxidative stress related to other health issues caused by air pollution from particles.[36,37] Outdoor air pollution, namely particulate matter, may induce DNA damage and mutations via several pathways, including oxidative stress and alterations in DNAm. These impacts might potentially result in an increased susceptibility to cancer and other health complications. Additional investigation is required to get a deeper comprehension of the processes that underlie the impacts of air pollution on DNA, as well as to devise efficacious measures for reducing these hazards.[38,39]

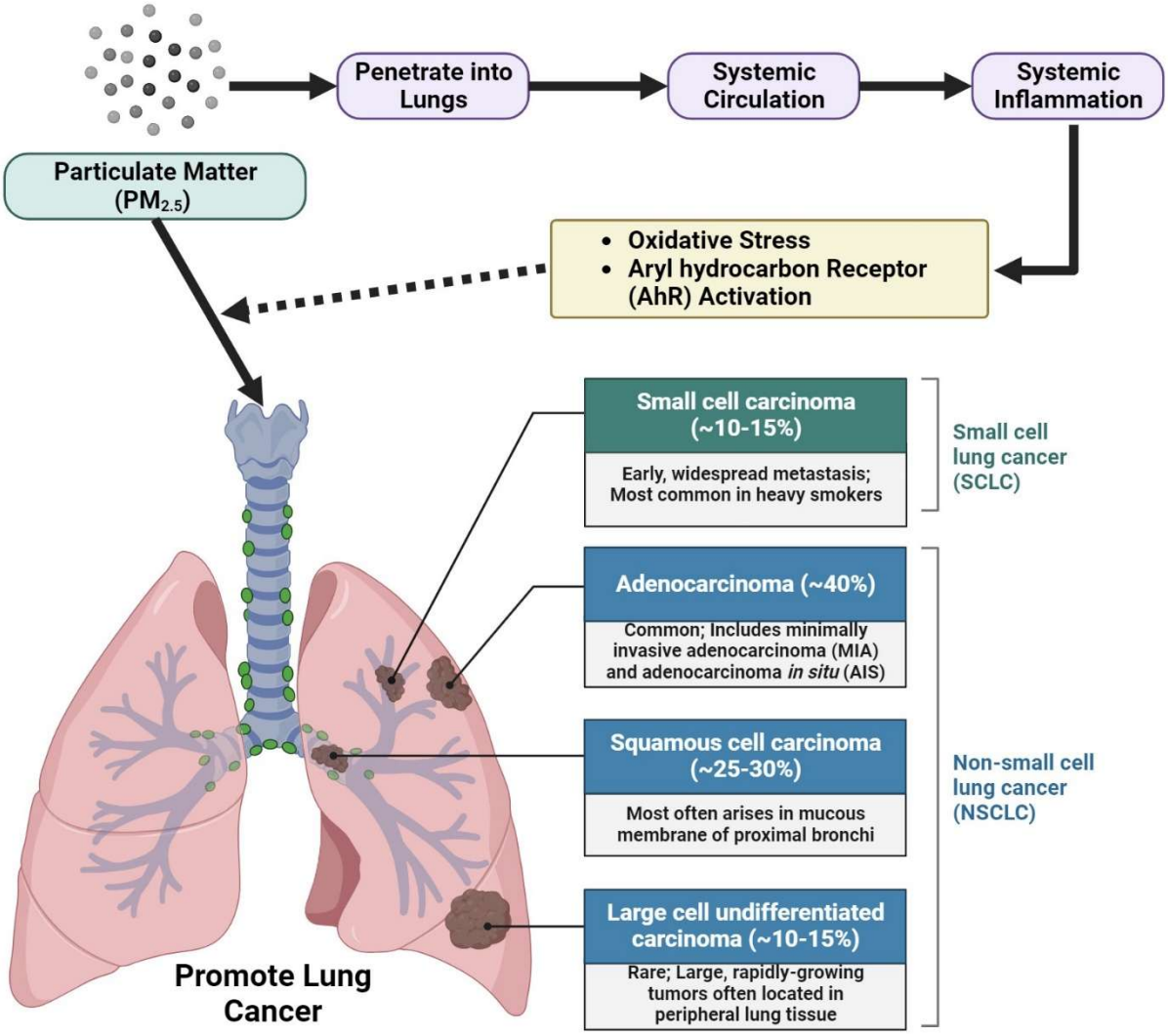


Figure 2: Activation Components of Lung Cancer Development

4.2. Activation of oncogenes and inactivation of tumor suppressor genes

Oncogenes are genetic sequences that, when altered or excessively activated, may stimulate the progression of cancer by inducing aberrant cellular proliferation and division. Activation may occur via many processes, including chromosomal translocation, point mutation, and gene amplification.[40] A proto-oncogene undergoes mutation or amplification, resulting in its transformation into an activated oncogene, which causes unrestricted cell proliferation.[41] Conversely, tumor suppressor genes often impede cell proliferation or facilitate programmed cell death. Disruption of these genes may result in unregulated cellular proliferation and contribute to the onset of cancer.[42] The activation of oncogenes and the silencing of tumor suppressor genes are crucial processes in the beginning and advancement of tumors.[40–42] Alterations in these genes may result in aberrant cell growth, suppression of programmed cell death, and the formation of new blood vessels, playing a role in the progression of cancer.[40] Comprehending the function of oncogenes and tumor suppressor genes is essential for the development of innovative anti-cancer strategies. Oncogenes promote aberrant cell growth, while tumor suppressor genes impede it. The initiation of oncogenes and the deactivation of tumor suppressor genes are pivotal

Occurrences in the progression of cancer and comprehending these mechanisms are crucial for formulating efficacious cancer therapies.[40–42]

5. Examples of carcinogenic pollutants

Certainly, here are some examples of carcinogenic pollutants:

5.1. Tobacco smoke

Tobacco smoking is a primary contributor to the development of lung cancer and is linked to several other forms of cancer, such as oral, throat, esophageal, pancreatic, bladder, and cervical cancer. Tobacco smoke is a well-recognized and primary factor in the development of lung cancer. The substance comprises more than 7,000 chemical compounds, with over 250 of them recognized as detrimental and at least 69 known to induce cancer via impairing DNA. PAHs, nitrosamines, and benzene are the main cancer-causing substances found in tobacco smoke. The presence of carcinogens in tobacco smoke causes harm to the DNA inside lung cells, resulting in genetic alterations that may lead to the unregulated proliferation of cells, a characteristic feature of cancer.[8,31]

5.2. Radon

Radon is a naturally occurring radioactive gas found in the Earth's soil. It is colorless, odorless, and tasteless. As it infiltrates homes and structures via the ground, it has the potential to reach hazardous levels. Radon, a radioactive gas, may seep from the ground and into buildings. Exposure to elevated levels of radon gas over a prolonged duration is recognized to be a causative factor for the development of lung cancer. While this gas occurs naturally, it becomes hazardous to health when indoor concentrations reach levels that may lead to the development of lung cancer if inhaled. Radon, as it deteriorates, emits radioactive particles that might potentially damage lung tissue if inhaled. Prolonged exposure to high levels of radon significantly raises the probability of acquiring lung cancer.[8,31]

5.3. Asbestos

Asbestos, a naturally occurring mineral group, has exceptional heat resistance and durability, making it a crucial ingredient in many construction and industrial products. Asbestos exposure may lead to the development of mesothelioma, asbestosis, and lung cancer. Asbestos comprises minuscule fibers that, upon inhalation, may get lodged in the lungs and lead to the development of cancer. Inhalation of asbestos fibers may result in their retention inside the lungs, leading to the development of fibrosis and chronic inflammation. Over time, this can worsen underlying respiratory problems, such as lung cancer.[8,30,31]

5.4. Benzene

Benzene, a white liquid, is an industrial waste derived from crude oil and is also found naturally in the oil. The chemical compound benzene is present in a diverse range of products, such as plastics, synthetic fibers, rubber, dyes, detergents, and resins. Prolonged exposure to elevated amounts of benzene is associated with the development of hematological malignancies and other blood-related disorders. Frequently used in the petrochemical, rubber, and oil refining sectors. Benzene is a well-established human carcinogen. Long-term exposure has been linked to the development of leukemia, namely acute myeloid leukemia (AML).[30,31,33]

5.5. Formaldehyde

Formaldehyde, a colorless gas with a strong and disagreeable odor, is used in the production of resin, disinfection, and preservation processes. It is present in tobacco smoke, building materials, and typical household products. Certain industries expose employees to hazardous compounds in the workplace, such as those involved in the production of plastics and textiles. The IARC has

designated it as a confirmed human carcinogen. Exposure to this human carcinogen increases the likelihood of developing nasopharyngeal carcinoma and leukemia.[8,30,31]

5.6. Arsenic

Arsenic, a naturally occurring element, is present in soil, water, and air. Environmental exposure is increased by the use of pesticides containing arsenic, industrial operations, and mining activities. When consumed as a result of contaminated food and beverages. Professional Extended duration of exposure is a potential hazard in the mining and smelting industries. Exposure to arsenic in water or other sources may lead to the development of several types of cancer, including skin, lung, bladder, and liver cancers.[43]

5.7. Polyvinyl chloride (PVC)

PVC, which is derived from the colorless gaseous compound vinyl chloride, is widely recognized as one of the most commonly used types of plastic. Individuals who deal with PVC or reside near vinyl chloride manufacturing facilities have a risk of exposure. Vinyl chloride, a component in PVC, is recognized as a human carcinogen. Prolonged inhalation exposure is associated with an increased susceptibility to liver cancer. Regarding humans, vinyl chloride is classified as a Group 1 carcinogen by the IARC. The current information indicates that those who are exposed to significant amounts of vinyl chloride in their workplace have a higher likelihood of developing liver cancer.[43–46]

5.8. Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs are generated as a result of the incomplete combustion of various organic substances such as coal, oil, gas, wood, and tobacco. They may be found in certain food sources or in air that has been contaminated. A ubiquitous byproduct of several industries, such as coal tar and aluminum production, as well as cigarette smoke and air pollution. Exposure to PAHs has been linked to the development of skin, lung, and bladder cancers. Furthermore, certain PAHs have been scientifically established or believed to be carcinogenic to people.[30,31]

5.9. Nitrosamines

Nitrosamines, a family of chemical compounds, may be formed by the combination of nitrogen-containing substances, nitrites, certain foods, and industrial processes. It is present in tobacco smoke, certain foods, and diverse production processes. Certain nitrosamines have been associated with increased susceptibility to gastric and esophageal cancer. The occurrence of human cancers in the digestive system and other organs is closely linked to certain nitrosamines, which are acknowledged as carcinogens.[30,31]

These examples emphasize the variety of cancer-causing contaminants and emphasize the need to understand their mechanisms of action to create efficient methods for prevention and reduction. These cancer-causing chemicals emphasize the many origins and possible health hazards linked to exposure. It is crucial to make efforts to reduce exposure to avoid health concerns, such as cancer, at both the individual and regulatory levels. To effectively combat the harmful effects of these pollutants, it is essential to implement regulatory measures, launch public health campaigns, and encourage the adoption of personal protective measures. This comprehensive approach will help minimize the occurrence of lung cancer and enhance respiratory well-being.

6. Prevention and control of exposure to carcinogenic pollutants

Exposure to carcinogenic pollutants has the potential to induce cancer, however, there are preventive and control methods that may be implemented. Authorities can establish rules and laws aimed at safeguarding individuals from hazardous chemicals and overseeing adherence to these restrictions. Manufacturers and industries can customize their procedures to adhere to rules, while employers may implement safety measures to safeguard their employees. Individuals may further

Promote a more sustainable environment by adhering to health and safety protocols in their workplaces, as well as reducing air pollution via less reliance on automobiles.[47] The primary objective of the US Environmental Protection Agency (EPA) is to safeguard public health and mitigate the adverse effects of poisons and harmful substances on communities around the nation. The EPA's endeavors to evaluate and mitigate cancer risks encompass a broad spectrum of activities. These include addressing the impact of pesticides and toxic chemicals, minimizing air pollution, implementing measures to protect water resources, managing land and waste disposal, and responding to emergencies. Additionally, the EPA focuses on addressing the risks associated with per- and poly-fluoroalkyl substances (PFAS), conducting research on cancer and children's health, enforcing compliance with environmental regulations, promoting environmental justice, conducting environmental impact assessments, studying environmental economics, undertaking regional projects and geographic initiatives, and collaborating on international and tribal initiatives.[47,48] The National Institute for Occupational Safety and Health (NIOSH) issued a policy on chemical carcinogens, acknowledging that any degree of exposure to a carcinogen is unsafe. The major method to avoid occupational cancer is to minimize worker exposure to chemical carcinogens by the removal or replacement of these substances, as well as the implementation of engineering controls. The occurrence of cancer caused by exposure to environmental and occupational carcinogens may be avoided or minimized by reducing or eliminating such exposures.[47,48] Primary prevention has a crucial role in reducing the occurrence of lung cancer. Recognizing the well-accepted connection between tobacco smoking and lung cancer, the focus is on modifying behavior as a primary approach to decreasing smoking rates. Crucially, the conversation goes beyond tobacco to include the increasing worry about lung cancer in those who do not smoke, which is caused by environmental and occupational hazards. The rise in the number of young individuals diagnosed with lung cancer, especially those who do not smoke and have certain genetic abnormalities, highlights the need for effective strategies to prevent the disease from occurring in the first place. The recommended method comprises identifying and treating both tobacco and non-tobacco variables that contribute to the risk of lung cancer. This approach is cost-effective and can help reduce the burden of lung cancer on people and healthcare systems. This underscores the need for continuous research, public health campaigns, and education to enhance primary preventive methods and improve results in the presence of this substantial public health issue.[8,47,48]

6.1. Regulatory measures

Regulatory measures are essential for managing and limiting the level of exposure to cancer-causing contaminants. Governmental regulatory methods include the formulation of rules and laws aimed at managing and limiting the exposure to substances that might cause cancer. An example of this is the US-EPA, which formulates rules under the Clean Air Act and other legislations to restrict the presence of carcinogenic pollutants.[49] The NIOSH has issued a policy regarding chemical carcinogens, which highlights the importance of minimizing worker contact with these substances by either eliminating or replacing them and implementing engineering controls as the main strategies for preventing occupational cancer.[48,49] Employers have a legal obligation to evaluate and control the potential harm caused by cancer-causing substances in the workplace, to prevent workers from being exposed, and to implement protective measures for their employees. Engaging in certain behaviors, such as adhering to health and safety protocols at work and actively working to decrease air pollution, may also play a role in preventing exposure to cancer-causing contaminants.[8,48,49]

6.2. Personal protective measures

Personal protection equipment (PPE) is crucial for reducing the risk of coming into contact with dangers in different environments. PPE encompasses a range of products, including gloves, safety glasses, shoes, earplugs, hard hats, respirators, coveralls, vests, and full-body suits.[8,10] To guarantee the appropriate use of PPE, it must be designed, produced, and maintained in a safe, clean, and reliable manner. For optimal worker compliance, the PPE should be designed to provide a comfortable fit. Employers have the responsibility to provide suitable and sufficient PPE to their workers, impart training on its use and maintenance, and ensure timely replacement and upkeep as needed.[4,8,30,31] Within healthcare environments, PPE such as gowns, gloves, masks, and goggles serve as a tangible barrier to shield against contact with contagious agents. Employers are advised to uphold documented PPE policies and protocols, conduct yearly reviews, and make necessary updates as required. Proper instruction on the utilization of PPE and consistent upkeep are essential for guaranteeing its efficacy in reducing the risk of exposure to dangers.[4,8,30,31] is shown in Fig.3.

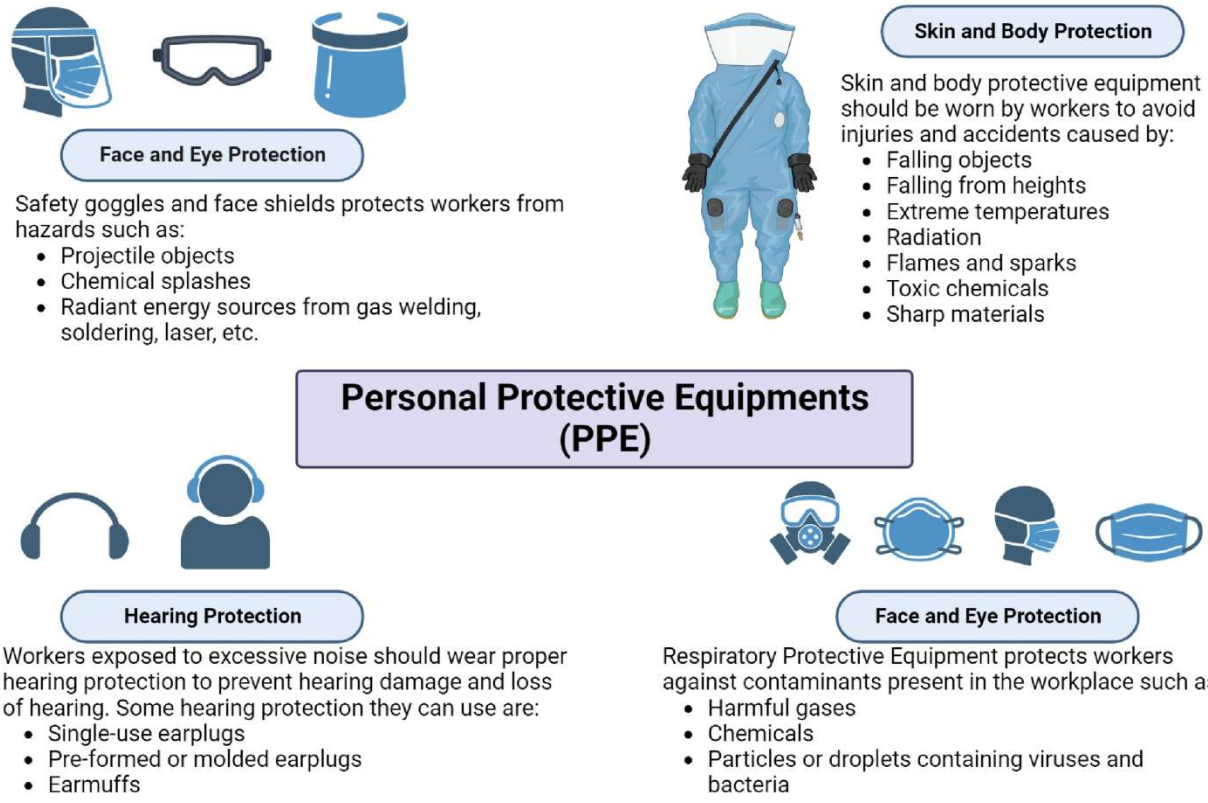


Figure 3: Various Personal Protective Equipment

6.3. Public awareness and education

Public awareness and instruction are vital in the prevention and management of exposure to cancer-causing contaminants. Increasing public knowledge of radon, for instance, is crucial in decreasing the incidence of lung cancer fatalities associated with radon exposure.[50] Efficient dissemination of information and educational initiatives may enhance individuals' comprehension of the hazards linked to carcinogens and empower them to adopt preventive measures for their safety and the safety of others.[49,50] Several difficulties and possibilities exist for enhancing environmental health literacy. These include:

- **Insufficient and delayed provision of precise information:** Current national protocols for environmental risk communication often rely on risk and crisis communication strategies, which

may be inadequate in effectively addressing the issues of air pollution and associated health risks.[51]

- **Skepticism towards scientific findings:** The prevailing political climate has resulted in a lack of faith in scientific research, which might impede efforts to safeguard public health from harmful substances.[49]
- **Targeting the appropriate demographics:** It is crucial to reach the specific groups of people who should be informed about environmental contaminants, since a significant number of individuals are still uninformed about the dangers linked with them.[49]

To enhance public awareness and education, the following steps may be implemented:

- **Educational programs:** Creating and executing educational campaigns that specifically target the dangers of carcinogenic pollutants and provide guidance on minimizing exposure may effectively increase public knowledge and encourage behavioral modifications.[50]
- **Engaging in public-private partnerships:** By collaborating with commercial organizations, non-profit groups, and government agencies, the message may be amplified, and a broader audience can be reached.[50,51]
- **Community engagement:** By actively involving local communities in workshops, seminars, and other activities, knowledge may be effectively shared, and a feeling of responsibility for environmental well-being can be nurtured.[50,51]
- **Enhanced media exposure:** The amplification of media coverage on matters about environmental health may effectively heighten public consciousness and motivate individuals to actively safeguard themselves and their surroundings.[50,51]
- **Consistent updates and surveillance:** Consistently informing the public about recent research discoveries and regulatory advancements can sustain interest and ensure the public remains well-informed about the most recent progress in environmental health.[50,51]

By enhancing public awareness and education, individuals, communities, and governments can collaborate to diminish exposure to cancer-causing pollutants and enhance overall environmental health.[49–51]

7. Discussion and Conclusion

Examining the link between $PM_{2.5}$ and PAHs emissions from specific industries and the occurrence of lung cancer in non-smokers. Examine the effects of different PPE on reducing exposure to $PM_{2.5}$ and PAHs, and explore how innovative PPE designs could potentially prevent lung cancer. Examine the gender-specific disparities in mortality rates, as well as the environmental and genetic factors that elevate the susceptibility to lung cancer triggered by exposure to $PM_{2.5}$ and PAHs. When evaluating the enduring effects of PM2.5 and PAHs exposure on human health, it is important to take into account other types of cancer, such as colon and prostate cancers, that are not related to the lungs. Explore the mechanism by which carcinogenic substances such as benzene and formaldehyde increase the likelihood of developing lung cancer by harming DNA and inducing mutations. Additionally, examine specific therapeutic approaches that might mitigate this risk. Analyze the impact of existing legislation on the prevalence of lung cancer resulting from air pollution, as well as the advantages and disadvantages of implementing these regulations worldwide. Analyze activities focused on raising awareness and educating people about environmental health literacy, and evaluate their effectiveness in decreasing exposure to cancer-causing chemicals. Specifically, analyze how successfully these campaigns target and engage certain demographic groups. Conduct a study on the efficacy of community engagement initiatives and private-public partnerships in educating the public about the dangers of carcinogenic pollutants. Evaluate the outcomes in terms of improvements in behavior and reduced exposure.

Investigate the influence of media exposure and strategies for communication on increasing awareness about environmental health concerns and the threats presented by carcinogenic pollutants.

8. Abbreviation and Description

Abbreviation	Description
PM	Particulate Matter
PM2.5	Fine Particulate Matter
PAHs	Polycyclic Aromatic Hydrocarbons
ROS	Reactive Oxygen Species
AhR	Aryl Hydrocarbon Receptor
COPD	Lung Cancer and Chronic Obstructive Pulmonary Disease
PPE	Personal Protection Equipment
PVC	Polyvinyl Chloride
NO2	Nitrogen Dioxide
NIOSH	National Institute for Occupational Safety and Health
DNAm	DNA methylation
VOCs	Volatile Organic Compounds
IARC	International Agency for Research on Cancer
EPA	Environmental Protection Agency

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