



Industrial Pollution and Cancer: An Important Public Health Issue

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Received: 25/09/2026

| Accepted: 01/10/2026

Abstract:

Industrial pollution is a key contributor to environmental contamination. It causes environmental damage and imposes high costs on society, human health, and safety. Exposure to air pollution from industrial operations can increase cancer risk. The industries that produce the most pollution include textiles, cement, glass, plastic, sugar, tanning, and petroleum. Little or no attention is paid to this grave issue of industrial pollution. Many industries do not have a systematic approach for properly disposing of and draining their toxic effluent. The industrial sector, which accounts for approximately 50% of environmental contamination, should be held accountable for properly managing waste effluent. Pollution control has become a primary environmental concern today. This paper explores how environmental pollution affects the environment and society. People spend time in various microenvironments, such as homes, offices, public venues, other indoor spaces, transportation, and the outdoors. These locations contain substances that may act as carcinogens, and exposure levels influence their carcinogenic impact on the human body. A broad spectrum of observable effects provides strong evidence that the environment may play a crucial role in cancer. It is essential to raise public awareness of cancer etiology and the hazardous substances in the environment that may act as causative agents. We urgently need to understand how environmental factors and occupational pollutant exposure may contribute to cancer.

Keywords: Industrial pollution, cancer, cancer risks, industries, environment, carcinogen, public health.

Introduction:

Pollution, in its various forms, is the introduction of harmful contaminants into a biological ecosystem, negatively affecting the environment's ability to sustain itself, carry out normal regenerative processes, and support living organisms. Human activity is a major driver of increased pollution and ecosystem instability, resulting in fatalities from manufacturing operations, industrialization, and the ever-increasing demand to raise one's standard of living. According to a 2018 World Health Organization (WHO) report, air pollution from industrial activity (waste pollutants) causes one in every nine fatalities worldwide.

Environmental pollution has risen significantly in recent decades due to rapid economic expansion. It is mostly attributable to the manufacturing and industrial sectors, which form the basis of a country's economy. Industrial pollution negatively affects both biotic and abiotic environmental elements. It also jeopardizes people's safety, lives, and wealth while causing a slew of interconnected social issues. Industrial pollution alters numerous aspects of the environment, including energy patterns, radiation, and the environment's chemical and physical constituents. These factors affect water supply and influence agricultural output, directly or indirectly. Addressing industrial waste is vital to reduce or eliminate environmental pollution. Common industrial pollutants include chemical compounds, concrete, soil, ammonia gas, nitrogen compounds, gravel, scrap metal, oil, and heavy metals.

In 2015, cancer was the second leading cause of mortality, trailing only cardiovascular disease, accounting for nearly 8.7 million deaths worldwide. Cancer is the uncontrolled growth and proliferation of cells in the body, and it is one of the leading causes of death around the world. Approximately 100 forms of cancer are classified by the tissue or organ in the human body that is damaged. Cancer, a multifactorial disease, results from a variety of environmentally induced changes in the genome. Cancer is characterized by uncontrolled replication, failure to respond to growth-inhibiting signals, persistent angiogenesis, resistance to apoptosis, and the ability to invade other tissues.

Cancers can now be treated using both standard conventional procedures, including surgery, radiation therapy, and chemotherapy, and nonconventional or complementary therapeutic methods, such as hormone therapy, immunotherapy, nanotherapy, and so on. These well-established approaches target tumors and either impede or halt cell proliferation, but they cannot provide complete protection. Nonetheless, conventional cancer treatment procedures have negative side effects and mostly harm normal cells, tissues, and organs. The optimal cancer therapy approach depends on several factors, including the type and stage of malignancy, age, treatment frequency, medication dose, and patient health. Gene therapy, targeted silencing by siRNAs, the production of apoptosis-

inducing genes, and wild-type tumor suppressors are among the molecular-based techniques that have recently received increased attention.

Biological Aspects of Cancer :

Evidence accumulated to support the idea that cancer was the result of uncontrolled cell division, but the cause was unknown. Cancer development cannot be predicted because the complexity and proportion of carcinogenic factors, and how they interact with an individual, are poorly understood. The earliest clues came from epidemiologic observations. Carcinogenesis is a multiple-step process. One characteristic of chemical or physical carcinogenesis is the usually long period between contact with the carcinogen and the appearance of a tumor [7,9]. The latent periods of occupational cancers may extend from one to several years and in many cases to several decades. Initiation and promotion are two stages in tumor development. Initiation is caused by chemical, physical, or biological agents in the environment which irreversibly and heritably alter the cell genome [9,10].

Initiation: The first stage of carcinogenesis has been labeled initiation since 1947 [11]. Several experiments suggest that initiation involves irreversible genetic changes that predispose susceptible normal cells to malignant evolution and immortality [12,13]. Neoplasia initiation is essentially irreversible changes in appropriate target somatic cells. In the simplest terms, initiation involves one or more stable cellular changes arising spontaneously or induced by exposure to a carcinogen. This is the first step in carcinogenesis, in which the cellular genome undergoes mutations that create the potential for neoplastic development [14,15], predisposing the affected cell and its progeny to subsequent neoplastic transformation. The human DNA sequences responsible for transformation are called oncogenes. Although activating more than one oncogene appears necessary for neoplastic transformation, the data imply that initiation may occur with one-hit kinetics [16]. However, full expression of such neoplasia-initiating mutations invariably requires interaction with other later-arising gene mutations and/or changes to the cellular environment, but the initiating mutation creates the stable potential for pre-neoplastic cellular development in cells with proliferative capacity [17,18]. Each oncogene is closely associated with a normal DNA sequence present in the cellular genome, the proto-oncogene. At least five different mechanisms are considered for the conversion of proto-oncogenes to active oncogenes:

Overexpression of proto-oncogene following acquisition of a novel transcriptional promoter. The oncogene then acquires activity because its transcripts are produced at much higher levels than those of the related normal proto-oncogene. Over-expression due to amplification of the proto-oncogene or oncogene. Increased gene copies lead to corresponding increases in transcripts and gene product. This influences transcription levels and, in turn, the amount of gene product. Juxtaposition of the oncogene and immunoglobulin domains, following chromosomal translocations, that appears to result in deregulation of the gene. Alteration in the structure of the oncogene protein. This is the best-documented mechanism for oncogene proteins encoded by the ras genes [19].

Promotion: Researchers introduced the concept of promotion after discovering chemical substances with low carcinogenic activity that could still induce cancer development under experimental conditions [20]. Promoter compounds do not interact directly with DNA and exert biological effects without metabolic activation [21,22]. These agents increase cell proliferation in susceptible tissues, contribute to fixing mutations, alter gene expression, and disrupt cellular growth control [Gomes,23]. The transformed [initiated] cell can remain harmless unless and until it is stimulated to proliferate further, upsetting cellular balance. The subsequent changes in an initiated cell that lead to neoplastic transformation may involve more than one step and require repeated, prolonged exposure to promoting stimuli [24]. The expression of the initial mutation depends not only on interactions with other oncogenic mutations but also on factors that may temporarily alter specific gene expression patterns, e.g., cytokines, lipid metabolites, and certain phorbol esters. This may enhance cellular growth potential and/or decouple intercellular communication processes that restrict cellular autonomy and thereby coordinate tissue maintenance and development [25].

Progression is the process by which successive changes in the neoplasm produce increasingly malignant subpopulations. Molecular mechanisms of tumor progression are not fully understood, but mutations and chromosomal aberrations are thought to play a role. Repeated exposure to carcinogenic stimuli or selection pressures favoring autonomous clonal derivatives may accelerate the process. Initiated cells proliferate, rapidly increasing tumor size. As the tumor grows, cells may undergo further mutations, increasing heterogeneity in the cell population. In the first phase of progression, sometimes called neoplastic conversion, pre-neoplastic cells transform into a state in which they are more committed to malignant development [26]. Dynamic cellular heterogeneity, a feature of malignant development, may often result from the early acquisition of gene-specific mutations that destabilize the genome. Examples are mutations of the p53 gene [27] or DNA mismatch repair genes [28]. Many tumor types develop transforming DNA sequences as they progress from normal to cancerous states. An elevated mutation rate established relatively early in tumor development may therefore generate variant cells at high frequency within a premalignant cell population. Such variant cells, with the capacity to evade constraints that restrict aberrant cell proliferation, tend to be selected during tumorigenesis [21]. The mechanism of promotion remains poorly understood. These changes trigger cell proliferation, an apparently necessary process in the “fixation” or expression of tumor initiation. Molecular genetic mechanisms are implicated in tumor progression, leading to chromosomal rearrangements or mutations that activate proto-oncogenes. Thus, it appears that, of the three stages of carcinogenesis initiation, promotion, and progression. Initiation most certainly, and progression most likely, involve molecular genetic changes and lead to particular cancer in a person [9,29]. The environment in which we live has three fundamental sets of components: physical (energy of one form or another), Chemical (matter, i.e., substances, whether natural or man-made), and Biological (living things). Humans can be affected by these hazards through different media, e.g., air, water, and soil. The influence they can exert on human

health is complex and may be modulated by our genetic makeup, psychological factors, and perceptions of the risks they present. Every human disease is linked to the environment in one way or the other. Diet and nutrition, infectious agents, toxic chemicals, physical factors, and physiological stress all play a role in the onset or progression of human diseases [3,10].

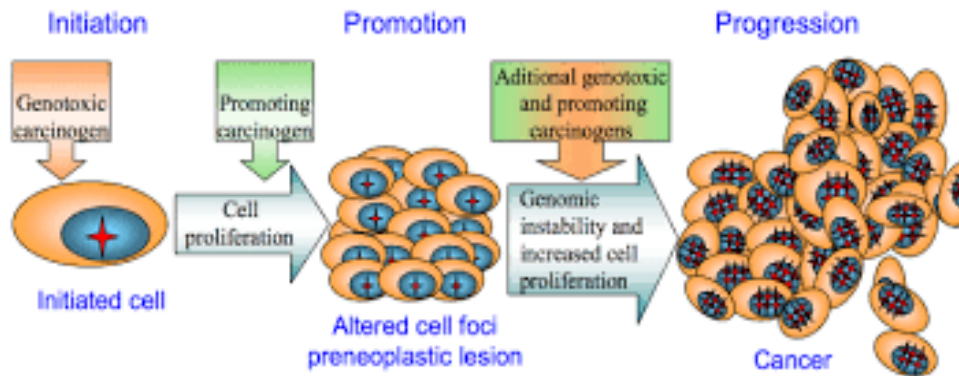


Figure 1: Overview of carcinogenesis

Involvement of Chemical factors of the environment in cancer:

Historically, the chemical carcinogen or group of carcinogens that has received the most attention has been tobacco smoke, and deservedly so. However, strong evidence also shows that industrial and manufacturing chemical exposures, agricultural pesticide exposures, indoor and outdoor air pollution, and chemical contamination of water can cause cancer in our living and working environment [3,81]. Carcinogenic exposures from consumer products, predominantly man-made, that people encounter involuntarily in daily life may come from paints in buildings, pesticides applied in gardens and playgrounds, chemicals used at home for cleaning or in schools, toys, etc. Tobacco is the most widely disseminated carcinogen in the world. Numerous epidemiological studies have shown the linkage between oral, lung, bladder, and breast cancer development and extensive use of tobacco, either in the form of smokeless tobacco or cigarette smoking [82,83,84].

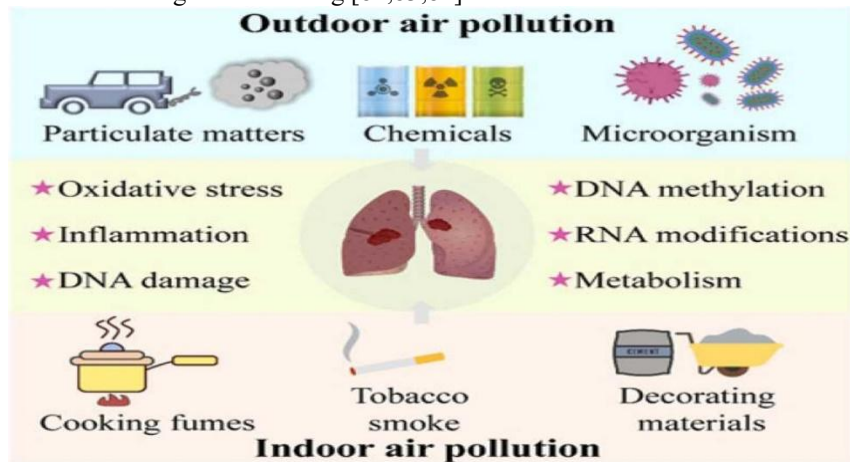


Figure 2: Consequences of air pollution (Yueguang Xue et al., 2022)

Response of cells to chemical injuries:

Getting cancer from a chemical depends on the following things:

[i] The kind of chemical you were exposed to; [ii] How much of the chemical you were in contact with; [iii] How long the contact lasted; [iv] How often you were exposed; [v] When you were exposed; [vi] How you were exposed and your general health. How does chemical exposure affect the body? The human body has defenses to guard against all sorts of harmful exposures, including those that may lead to cancer. When something enters the body, it often goes through a process that helps the body use it more easily or get rid of it. This process is called metabolism. Depending on how a chemical is processed, or metabolized, in our body, three types of carcinogens exist: Chemicals that can cause cancer (direct-acting carcinogens), Chemicals that do not cause cancer unless they are changed when they are metabolized (procarcinogens), and Chemicals that do not cause cancer by themselves but can act with another chemical to cause cancer (cocarcinogens). Some carcinogenic agents can directly or indirectly interfere with nucleic acids, mainly DNA, causing DNA alterations. These alterations may lead to cancer. However, cells can often repair DNA damage. If the damage is extreme, the cells may die. Unrepaired DNA damage can lead to gene mutations. These mutations can cause cancer, and if inherited, the progeny may be susceptible to cancer. Because cancer has a long latency period, determining which exposure, if any, may have led to a mutation is difficult. Therefore, we know very little about specific causes of cancers.

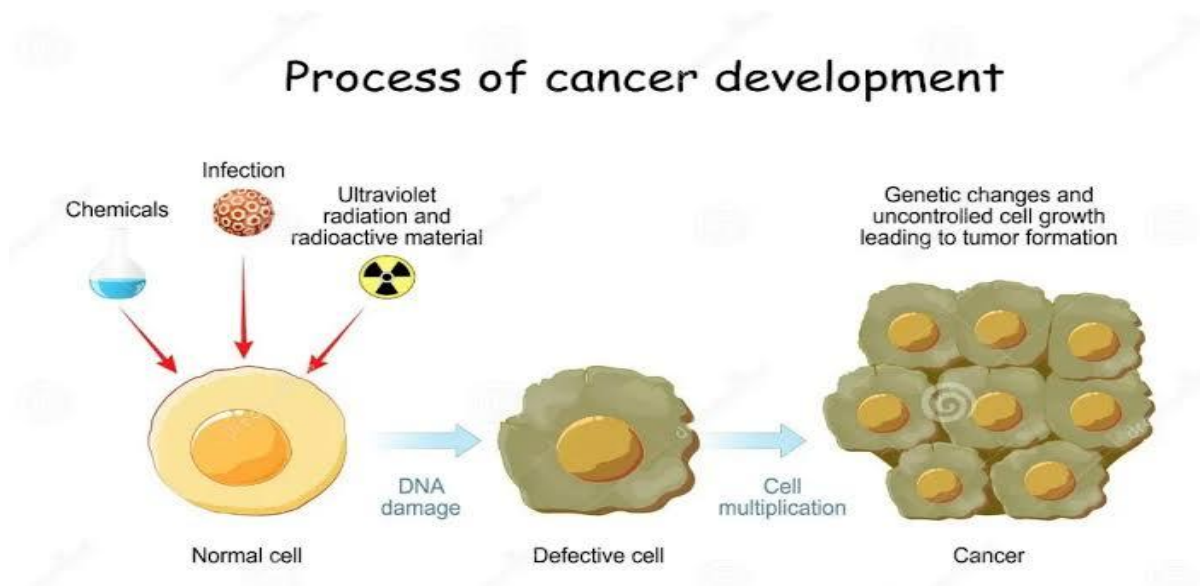


Figure 3. Schematic representation of cancer development and progression

Sources of Industrial Pollution and Carcinogenic Agents:

The International Agency for Research on Cancer (IARC) classifies these pollutants as known or probable human carcinogens.

Table 1: Sources of industrial pollution and carcinogenic agents

Industry Source	Key Pollutants	Carcinogenic Agents (IARC Classification)	Associated Cancers
Chemical Manufacturing	Benzene, formaldehyde, vinyl chloride, dioxins	Benzene (Group 1), Formaldehyde (Group 1), Vinyl chloride (Group 1)	Leukemia, nasopharyngeal cancer, liver angiosarcoma
Petroleum Refineries	Benzene, PAHs, toluene, xylene	Benzene (Group 1), PAHs (Group 1)	Lung, bladder, skin, and hematological cancers
Metal Processing and Smelting	Arsenic, cadmium, chromium (VI), nickel compounds	Arsenic (Group 1), Cadmium (Group 1), chromium (VI) (Group 1), Nickel (Group 1)	Lung, skin, prostate, bladder, nasal cavity cancers.
Textile and Dye Industries	Aromatic amines, azo dyes, formaldehyde	Benzidine (Group 1), Azo dyes (Some Group 2B/ 3), Formaldehyde (Group 1)	Bladder, nasal, and nasopharyngeal cancer
Agrochemical and Pesticide	Organochlorines, glyphosate, chlorpyrifos	Glyphosate (Group 2A), Organochlorines (Various IARC ratings)	Non-Hodgkin lymphoma, leukemia
Plastics and Rubber	Butadiene, styrene, acrylonitrile	Butadiene (Group 1), Acrylonitrile (Group 2A), Styrene (Group 2B)	Lung, leukemia, and possibly oesophageal cancers
Industry Source	Key Pollutants	Carcinogenic Agents (IARC Classification)	Associated Cancers
Pulp and Paper Industry	Dioxins (e.g., TCDD), chlorinated compounds, formaldehyde	Dioxins (TCDD - Group 1), Formaldehyde (Group 1)	Soft-tissue sarcoma, non-Hodgkin lymphoma, nasopharyngeal

Mechanisms of Carcinogenesis:

Industrial pollutants can induce cancer through various mechanisms, including:

DNA damage: Many contaminants can directly damage DNA, causing mutations that may cause or accelerate cancer development. DNA adducts (chemical alterations), strand breaks, and chromosomal abnormalities are just a few of the ways this damage can manifest [74]. Certain contaminants disrupt DNA repair processes, further increasing mutation risk.

Oxidative stress: Pollutants produce reactive oxygen species (ROS), such as hydrogen peroxide and superoxide radicals, that can damage proteins, lipids, and DNA. This oxidative damage can cause cellular dysfunction, inflammation, and mutations, all of which contribute to cancer development [75].

Inflammation: Pollution exposure can cause chronic inflammation, which can promote tumor growth. Growth factors and signaling molecules (cytokines) released by inflammatory cells can stimulate angiogenesis (the creation of new blood vessels that nourish tumors), metastasis (the spread of cancer cells), and cell proliferation [76].

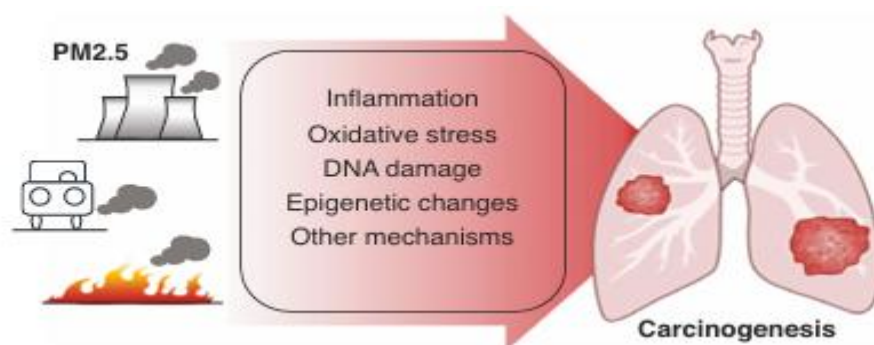
Epigenetic alterations: Pollutants can influence gene expression without changing the DNA sequence, leading to abnormal cellular function and cancer. These changes can include DNA methylation (the addition of methyl groups to DNA), histone modifications (changes to the proteins that wrap DNA), and microRNA expression variations [77]. Epigenetic alterations can influence the expression of genes involved in cell development, differentiation, and apoptosis (programmed cell death).

Endocrine disruption: Certain pollutants can alter the endocrine system, raising the risk of hormone-sensitive tumors [78]. These endocrine-disrupting chemicals (EDCs) can mimic or inhibit natural hormone functions, causing abnormal cell growth and development. Examples include industrial chemicals, insecticides, and heavy metals.

Receptor activation: Some pollutants can directly activate or inhibit biological receptors, such as growth factors and hormones. This abnormal receptor signaling can impair normal cellular processes and contribute to cancer formation [79].

Impaired cell signaling: Pollutants can disrupt cell signaling pathways that regulate cell growth, proliferation, and survival.

These



abnormalities can result in uncontrolled cell proliferation and the formation of cancer [80].

Figure 4: Mechanisms of PM 2.5-induced carcinogenesis (Meng Wang et al., 2025)

Divergence of Exposure:

Industrial pollution exposes people to hazardous substances through multiple channels, directly affecting their health. Factory-emitted airborne contaminants like benzene, formaldehyde, and dioxins have been associated with lung, nasopharyngeal, and haematologic cancers. Contaminated water is generated by the dumping of industrial waste into bodies of water, which exposes people to carcinogenic substances that increase their risk of bladder, liver, and gastrointestinal cancer. Soil pollution occurs when toxic industrial waste seeps into the ground, disrupting agriculture and bringing toxins into the food chain [60]. Occupational exposure poses a considerable risk to manufacturing workers, as inadequate protective measures can lead to direct, prolonged contact with carcinogens. Farmers have shown elevated risks of stomach cancer in the majority of worldwide studies [58].

Additionally, contaminants may accumulate in animal and plant tissues, contaminating food and exposing humans, particularly to bioaccumulative pollutants such as mercury and PCBs. Some industrial pollutants can contaminate consumer products such as plastics and textiles, causing low-level but prolonged exposure. Transplacental exposure allows certain pollutants to pass through the placenta, potentially exposing developing fetuses and raising the risk of childhood cancer. Finally, dermal exposure occurs when carcinogens, such as PAHs and some pesticides, penetrate the skin after contact with contaminated soil, water, or industrial waste.

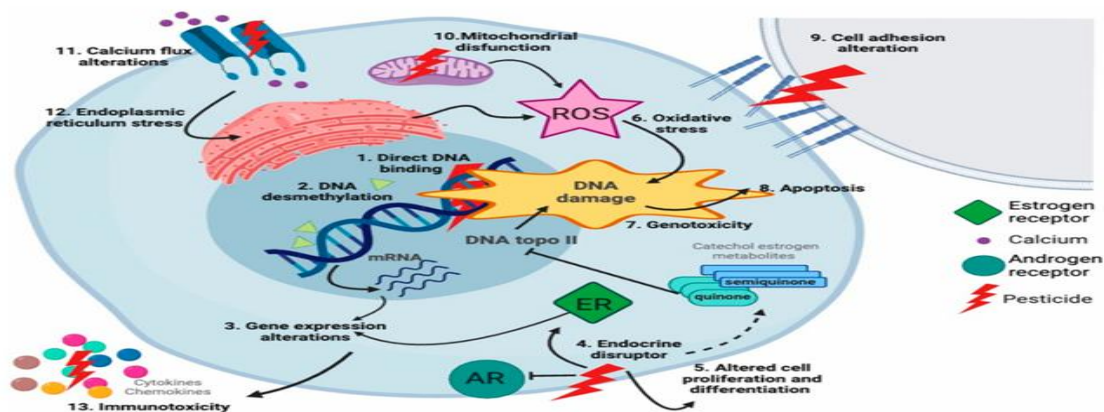


Figure 5: Mechanisms of pesticide-induced cancer (María del Pilar Navarrete-Meneses et al., 2024)

Exposure remains widespread for many key occupational carcinogens, such as asbestos, coal tar, arsenic, and silica, particularly in developing countries (Table 2).

Table 2: Cancers Associated with Various Occupations or Occupational Exposure

Sl.no	Cancer	Substances or processes
1.	Lung	Arsenic, asbestos, cadmium, coke oven fumes, chromium compounds, coal gasification, nickel refining, foundry substances, radon, soot, tars, oils, silica
2.	Bladder	Aluminum production, rubber industry, leather industry, 4-aminobiphenyl, benzidine
3.	Nasal cavity and sinuses	Formaldehyde, isopropyl alcohol manufacture, mustard gas, nickel refining, leather dust, wood dust
4.	Larynx	Asbestos, isopropyl alcohol, mustard gas
5.	Pharynx	Formaldehyde, mustard gas
6.	Mesothelioma	Asbestos
7.	Lymphatic and hematopoietic	Benzene, ethylene oxide, herbicides, X-radiation system
8.	Skin	Arsenic, coal tars, mineral oils, sunlight
9.	Soft-tissue sarcoma	Chlorophenols, chlorophenoxy herbicides
10.	Liver	Arsenic, vinyl chloride

Epidemiological Evidence:

Epidemiological research substantially supports the association between industrial pollution and many malignancies. Specific studies have linked leukemia and lymphoma to benzene exposure among petrochemical workers. Populations exposed to air pollution from major industries frequently have higher rates of lung cancer [70]. Smoking-adjusted relative risks for lung cancer in urban areas reported increased risks. The findings pertain mainly to smokers. For nonsmokers, case counts were generally too small to meaningfully interpret urban-to-rural differences [71]. Furthermore, communities exposed to arsenic-contaminated water have a higher incidence of bladder and colorectal cancer. There is additional evidence that there is a higher prevalence of breast cancer in locations with high industrial emissions, including endocrine-disrupting chemicals. In 2012, breast cancer was the leading tumor, in terms of new cases and deaths, in women worldwide, whereas prostate cancer ranked second in incidence and fifth as a cause of cancer death among men worldwide [73]. Asbestos exposure in occupational settings, such as construction and shipbuilding, is significantly linked to mesothelioma and lung cancer [72]. Pesticide exposure among agricultural and industrial workers has been linked to non-Hodgkin lymphoma and prostate cancer. Proximity to hazardous waste dumps, especially in areas without environmental laws, has been related to an increased risk of liver and pancreatic cancer. Overall, longitudinal cohort studies support this association, showing that long-term inhabitants of polluted industrial centers had higher overall cancer mortality rates than those living in less industrialized locations.

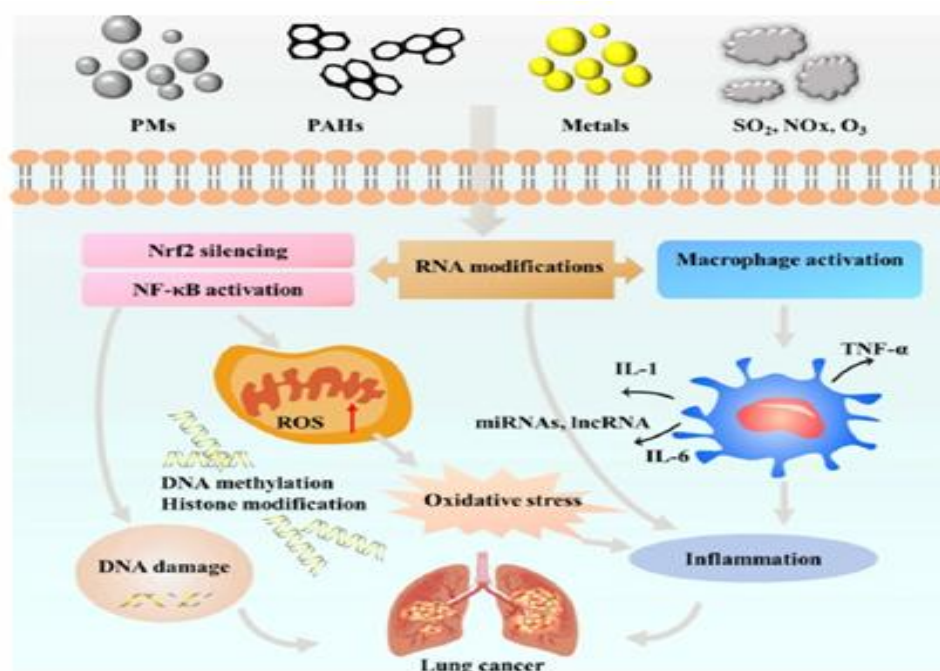


Figure 6: Mechanism of air pollution as a cause of lung cancer (Ida Ayu Jasminarti Dwi Kusumawardani et al., 2023)

Types of Studies Investigating Cancers:

Most studies used a geographical ecological design based on standardized mortality or morbidity ratios to identify potential over-incidence of death or morbidity. Poisson regression and similar statistical techniques were used to analyze the link between health indicators and exposure, accounting for confounding factors (mainly socio-economic). (Table 3).

Table 3: Studies Investigating Cancers

Reference	Country	Industrial background	Health outcome	Epidemiological design	Exposure assessment
Zambon et al., 2007 [35]	Italy	Industrial waste incinerators, Municipal solid waste incinerators, medical waste incinerators, thermal power plants, oil refinery industrial plants for the production of primary aluminum	Visceral and extra-visceral sarcoma	Case-control (72 cases and 405 controls)	Dispersion modeling (Industrial Source Complex Model in long-term mode, version 3 (ISCLT3))
Simonsen et al., 2010 [37]	United States	Petrochemical industries	Lung cancer (registry)	Case-control (455 cases and 437 controls)	Distance (0.5 miles, 1 mile, and 2 miles)
Yu et al., 2006 [36]	Taiwan	Oil refinery	Leukaemia	Case-control (171 cases and 410 controls)	Distance, based on previous studies (3 km radius from the geographic centroid of any of the four petrochemical complexes)

Table 3: Continued.....

Reference	Country	Industrial background	Health outcome	Epidemiological design	Exposure assessment
Edwards et al., 2006 [38]	United Kingdom	Iron and steel, chemical, and heavy engineering industries	Lung cancer (registry)	Case-control study (204 cases and 339 controls)	Distance, guided by a validation study using data from historical records
Petrauskaite et al., 2002 [39]	Lithuania	Production of mineral fertilizers, aluminum fluoride, and sulfuric acid	Lung cancer (mortality)	Case-control study (410 cases, 410 controls)	Distance, based on measurements of sulfuric acid and the prevailing wind (6 km)
Lopez-Cima et al., 2011 [40]	Spain	23 industrial installations reporting to the EPER	Lung cancer	Case-control study (626 cases, 626 controls)	Distance

Pascal et al., 2011 [34]	France	Oil refining, oil storage, petrochemical and organic chemical activities, chlorine chemistry, steel and metal working, chemical plants, waste incineration plant, port	All cancers, lung cancer, bladder cancer, breast cancer, multiple myeloma, malignant non-Hodgkin's lymphoma, and acute leukemia (hospitalizations)	Standardized incidence ratio	Coupling of a dispersion model (ADMS4), a meteorological model and kriging to assess the SO ₂ levels
Viel et al., 2011 [41]	France	13 municipal solid waste incinerators	Non-Hodgkin's lymphomas (registry)	Standardized incidence ratio	Dispersion model (Atmospheric Dispersion Model System version 3—ADMS 3) for each category of pollutants (dioxins, metals, and dusts)

Table 3: Continued.....

Reference	Country	Industrial background	Health outcome	Epidemiological design	Exposure assessment
Axelsson et al., 2010 [42]	Sweden	Industrial complex including a large cracker producing ethylene and propene	Leukemia, lymphoma, cancers of the lung, liver, and central nervous system, all cancers taken together (registry)	Standardized incidence ratio	Models (unspecified) of ethylene levels
Eitan et al., 2010 [43]	Israel	Petroleum refineries, oil-fired power plant, and several large petrochemical, chemical, and agrochemical industries	Lung cancer, bladder cancer, and non-Hodgkin's lymphoma	Standardized incidence ratio	Spatial interpolation of SO ₂ and PM ₁₀ routine monitoring data
Monge-Corella et al., 2008 [44]	Spain	18 EPER-registered paper, pulp, and board industries	Lung cancer (mortality)	Standardized incidence ratio	Distance (≤ 5 km from a paper, pulp, and board industry, ≤ 5 km from any other industrial installation, towns having no EPER-registered industry within 5 km of their municipal centroid (reference level))

Table 3: Continued.....

Reference	Country	Industrial background	Health outcome	Epidemiological design	Exposure assessment
García-Pérez et al., 2010 [45]	Spain	118 integrated pollution prevention and control (IPPC) category 2 metal production and processing installations which report their emissions to the EPER	Leukemia (mortality)	Standardized mortality ratio	See Monge-Corella
García-Pérez et al., 2009 [46]	Spain	57 combustion installations which report their emissions to the EPER	Lung, larynx, and bladder cancer (mortality)	Standardized mortality ratio	See Monge-Corella
García-Pérez et al., 2010 [47]	Spain	118 integrated pollution prevention and control (IPPC) category 2 metal production and processing installations that reported their releases to air and water in 2001	Tumors of the digestive system (mortality)	Standardized mortality ratio	See Monge-Corella
Ramis et al., 2009 [48]	Spain	452 industries reporting releases to air to the EPER, grouped by industrial sector	Non-Hodgkin's lymphomas (mortality)	Standardized mortality ratio	See Monge-Corella

Table 3: Continued.....

Reference	Country	Industrial background	Health outcome	Epidemiological design	Exposure assessment
Cambra et al., 2011 [49]	Spain	284 industries declaring emissions of pollutants to the EPER	Lung cancer (mortality), hematological tumors (mortality)	Standardized mortality ratio	Distance (<2 km, >2 km)
Yang et al., 2000 [50]	Taiwan	Kaohsiung oil refinery	Lung cancer (mortality)	Standardized mortality ratio	Distance
Tsai et al., 2009 [51]	Taiwan	Petrochemical industries	Bladder cancer (mortality)	Standardized mortality ratio	In each district, the number of employees of the industries divided by the population, in three classes

Lin et al., 2017 [85]	Taiwan / regional analyses	Petrochemical industrial complexes (PICs)	Lung cancer mortality / burden estimates	Ecological and registry-based analyses	Residential proximity / modeled exposure to PICs.
Barjoan et al., 2020 [86]	France (Nice area)	Domestic waste incineration plant vicinity	Cancer incidence (multiple sites)	Retrospective incidence analysis (2010–2014)	Local registry linkage; spatial analysis around incinerator.
Domingo et al., 2020 (review) [87]	Global (review)	Petrochemical industries / industrial emissions	Various cancers (leukaemia, bladder, lung, etc.)	Narrative review / synthesis	Summarizes studies using proximity, registry data, and pollutant measures.

Table 3: Continued.....

Reference	Country	Industrial background	Health outcome	Epidemiological design	Exposure assessment
Tait et al., 2020 (systematic review) [88]	Global (review)	Waste incineration emissions	Cancer mortality associations (various sites)	Systematic review	Highlights heterogeneity and some distance-related associations.
Jephcote et al., 2020 (meta-analysis) [89]	Global (review)	Petrochemical industry exposures (fenceline communities)	Hematological malignancies (leukemia) incidence	Systematic review & meta-analysis	Estimates attributable cases in 'Cancer Alley' from included studies.
Baek et al., 2022 (meta-analysis) [90]	Global (meta-analysis)	Waste incinerator emissions (MSWIs)	Multiple cancers (pooled) — no consistent overall increased risk except for some sub-types	Meta-analysis of case-control & cohort studies	Pooled heterogeneous exposure metrics (distance, registry, modeling).
Choi et al., 2022 [91]	Korea	Three industrial waste incinerators (IWIs) near a small town	Cancer incidence (various types)	Retrospective cohort (1999–2017; registry linkage)	Residential history; regional cancer registry comparison.

Williams et al., 2020 [92]	United States (refinery areas)	Oil refineries/petroleum industry emissions	Increased incidence for some cancer types (localized disease noted)	Registry-based analyses/case–control components	Residential proximity (miles) to refineries.
Chen et al., 2016 [93]	China (petrochemical site study)	Petrochemical complex emissions	Estimated Excess Lifetime Cancer Risk (ELCR) from VOCs	Exposure assessment/risk estimation	Air monitoring + ELCR modeling; identifies benzene and 1,3-butadiene as major contributors.

Table 3: Continued.....

Reference	Country	Industrial background	Health outcome	Epidemiological design	Exposure assessment
Reeve et al., 2013 (spatial analysis) [94]	Australia / international methods	Large industrial zones / Kiryat Haim industrial zone example	Cancer incidence rates near industrial zones	Spatial ecological analysis	Double kernel density and zonal estimates to assess proximity effects.
Negri et al., 2020 (review) [95]	Italy / Europe (review)	Waste incinerators	Health impacts including cancer incidence	Systematic review of cohort & case–control evidence	Notes methodological limitations and mixed findings.
Boonhat et al., 2023 (meta-analysis) [96]	Global (meta-analysis)	Residential exposure to petrochemical industrial complexes	Pancreatic cancer (pooled increased risk RR≈1.3)	Systematic review & meta-analysis	Exposure defined mainly by residential proximity to petrochemical complexes.
Domingo 2025 (preprint / updated review) [97]	Global (preprint 2025)	MSWIs and industrial incinerators	Cancer incidence & mortality (synthesis up to 2024/2025)	Preprint narrative/systematic synthesis	Highlights heterogeneity and calls for updated harmonized exposure assessments.
Bottini et al., 2025 (meta-analysis) [98]	Global (2025 meta-analysis)	Residential exposure to MSWIs	Respiratory and cancer hospitalizations/ mortality (mixed results)	Meta-analysis of cohort and case–control studies up to 2024	Finds small increased risks for selected respiratory outcomes; heterogeneity persists.

Prevention and Control Strategies:

Addressing industrial pollution and its link to cancer requires a comprehensive, multi-faceted approach. Governments must enact and strictly enforce stringent rules on industrial emissions and waste disposal, including limits, permit requirements, and penalties for noncompliance, and revise regulations regularly to reflect new scientific understanding. Governments should incentivize industries to adopt cleaner technologies and methods, such as switching to renewable energy, using fewer harmful chemicals, and establishing closed-loop systems, possibly through tax incentives or subsidies [52,53]. To reduce contaminants in emissions, facilities must implement and maintain effective pollution-control methods such as filters, scrubbers, and wastewater treatment systems.

Zoning regulations, buffer zones around industrial operations, promoting personal protective equipment in work environments, and public health education are all essential exposure-reduction tactics. Education and public-awareness campaigns are essential to help communities protect themselves and demand change. Additionally, research and monitoring—with independent agencies making data accessible to the public—are crucial for tracking pollution levels in soil, water, and air and for identifying new carcinogens. International collaboration is needed to share best practices and develop treaties, since pollution transcends national boundaries [54]. Stricter rules, engineering controls, and routine health monitoring for employees exposed to carcinogens are all necessary to improve workplace safety. Promoting practices like organic farming and sustainable agriculture also helps reduce pollution from fertilizers and pesticides.

Lastly, urban planning and transportation measures, such as investing in public transportation, cycling infrastructure, and electric automobiles, can greatly reduce air pollution and its overall impact on the environment.

Populations at risk:

Several populations are particularly vulnerable to industrial contamination. These include residents living near industrial zones, who face risks because of their proximity to contamination sources and the resulting environmental degradation [65]. Industrial workers face a direct threat since they are routinely exposed to high levels of carcinogens, often without proper protection. Children and the elderly are especially vulnerable to pollution. For childhood cancer, genotoxicity and carcinogenicity studies are necessary, along with evaluations of other potential biological effects, including epigenetic modifications [62]. Furthermore, marginalized communities, particularly those in low- and middle-income nations, experience structural disadvantages, including a lack of access to required healthcare and legal protection. Workers in the informal sector, such as rubbish pickers and small-scale miners, often work in unregulated settings without safety inspections, which significantly increases their exposure to dangerous substances. People with pre-existing health disorders are especially vulnerable, as pollution may aggravate their diseases and increase cancer risk owing to weakened immune or respiratory systems [66]. Indigenous peoples living near industrial developments frequently endure disproportionate exposure as a result of encroaching on customary territories and a lack of political participation. The Global Burden of Disease study estimated that in 2019, 50.6% of cancer deaths in men and 36.3% in women were attributable to behavioral, environmental, occupational, or metabolic risk factors [61]. Finally, rural people living near extractive industries, including mining or oil drilling, often face long-term contamination of natural resources due to limited access to healthcare and environmental monitoring.

The International Agency for Research on Cancer of the World Health Organization evaluates potential cancer risks to humans from a range of chemical exposures [55]. Half of the 60 chemicals and chemical mixtures the agency has evaluated as having sufficient evidence of carcinogenicity in humans represent occupational exposures, which tend to be concentrated among small groups of people chronically exposed at high levels. These include workplace exposures such as the rubber industry or coke production, as well as exposure to specific aromatic amines, petrochemicals, and metals. Excess risks were also observed in the meat, rubber, and leather footwear industries. For some occupations, such as cooks, restaurant personnel, and meat industry workers, diet may play a strong role [56,57]. How much cancer can be attributed to occupational exposure has been a controversial issue, but a few percent seems a reasonable estimate.

Technological Innovations in Pollution Control:

One of the main causes of environmental deterioration and health problems associated with cancer is industrial pollution. As the field advances, developers and policymakers must ensure equal access to technologies that improve population health [59]. Technological innovation is essential to reducing, and in some cases eliminating, cancer-causing pollutants from industrial processes. These developments aim to stop pollutant creation at the source and treat pollutants after they are created.

a. **Cleaner Production Techniques:** The use of integrated solutions to improve the efficiency and reduce environmental impact of industrial processes is known as "cleaner production." The goal is to reduce waste generation and avoid using hazardous substances [63,64].

i) **Process Optimization:** Manufacturing processes are being redesigned by industries to consume less energy and emit fewer emissions. For instance, optimizing chemical reactions can prevent the production of hazardous byproducts such as formaldehyde or benzene.

ii) **Material Substitution:** Replacing hazardous raw materials with safer ones. For instance, replacing organic solvents with water-based solvents in paint and textile manufacturing.

iii) **Energy Efficiency:** In order to reduce the dependency on fossil fuels and related pollutants, cleaner production frequently uses energy-saving technology like waste heat recovery systems and renewable energy sources (like solar-powered operations) [68].

iv) **Benefits:** By addressing pollution at its source, cutting operational expenses, and lowering long-term liabilities associated with harm to the environment and human health, this strategy is proactive.

- b. Industrial Waste Treatment: Industrial emissions and effluents contain a variety of carcinogens. Advanced waste treatment technology aims to eliminate or neutralize these compounds before they enter soil, water, or air.
- i) Chemical Treatment: Complex organic carcinogens like dioxins or polycyclic aromatic hydrocarbons (PAHs) can be broken down into less dangerous compounds by processes like oxidation (such as ozonation and chlorination).
- ii) Bioremediation: The degradation or detoxification of contaminants using microorganisms (such as fungi and bacteria). For example, some bacteria can degrade hazardous solvents such as trichloroethylene (TCE).
- iii) Advanced Filtration: Heavy metals and fine particulate matter are being removed from wastewater using technologies like membrane bioreactors, activated carbon filtration, and nanomaterials.
- iv) Incineration and Thermal Treatment: Hazardous organic compounds that would normally cause cancer can be safely destroyed by high-temperature incinerators, especially when equipped with sophisticated flue gas treatment systems [67].
- c. Real-Time Emission Monitoring: Real-time industrial emissions monitoring has grown more accurate, responsive, and available with the development of digital technologies. This significantly improves the ability to prevent harmful exposures.
- i) IoT (Internet of Things) Sensors: To continually assess the amounts of pollutants like volatile organic compounds (VOCs), particulate matter (PM_{2.5}/PM₁₀), sulfur dioxide, and nitrogen oxides, smart sensors are installed at emission locations (such as industrial smokestacks and wastewater outlets) [69].
- ii) AI-Powered Analytics: Real-time data analysis from these sensors, anomaly detection, emission spike prediction, and even automated remedial measures inside industrial systems are all possible with AI systems.
- iii) Remote Monitoring Platforms: Companies and regulatory agencies can monitor compliance, send out alerts, and act quickly in the event of harmful spills thanks to the transmission of data from emission sources to centralized dashboards.
- iv) Public Transparency: Additionally, some systems provide data to community apps or public platforms, increasing transparency and empowering local people to recognize and address environmental risks.

DISCUSSION:

A substantial body of research supports the relationship between the environment and human health. This supports increased primary prevention efforts to eliminate or reduce physical, chemical, and biological exposures to known carcinogens, as well as environmental interventions, including in the workplace, to reduce cancer incidence and the disease's clinical, economic, and social burdens. Epidemiological evidence in humans is sufficient to define many major cancer etiologies with strong, credible evidence. Many of these risks are avoidable, making it possible to lower cancer incidence. The study's findings show that industrial hazardous effluents harm the environment and socio-environmental conditions while also posing risks to human health and safety.

CONCLUSION:

Industrial pollution negatively affects environmental biotic and abiotic components. Dangerous chemicals and pollutants contaminate all natural resources, including air, water, and soil. When pollutants and toxic chemicals contact humans, they cause a range of health problems, including respiratory, neurological, and digestive illnesses. Pollution awareness needs to be raised urgently. Industrial pollution is a modifiable environmental risk factor for cancer, necessitating extensive public health interventions. Coordinated efforts among governments, researchers, and communities are essential to reduce exposure and protect vulnerable groups. Policymakers and environmental protection organizations must address this critical issue and protect the country from the harmful effects of pollution.

Conflicts of Interest:

The authors declare no conflict of interest.

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