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Research Article

Role of Environmental Toxicants and their exposure in the Etiology of Cancer

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Abstract
Cancer, a leading cause of morbidity and mortality worldwide, is a concerning disease with its multifaceted causality. Substantial contributors to the etiology of cancers are environmental factors. Environmental toxicants, including pesticides, heavy metals, air pollutants, persistent organic pollutants, and emerging contaminants such as microplastics, are ubiquitous in modern ecosystems and human environments. Exposure to these toxicants over a long period of time, even at minimal doses, has been strongly linked to carcinogenesis through diverse molecular and cellular mechanisms. This review summarizes current evidence on sources and routes of exposure to environmental toxicants and critically examines their role in cancer etiology. Key mechanisms suchas genotoxicity, oxidative stress, epigenetic dysregulation, endocrine disruption, and immune modulation are discussed. Epidemiological and experimental findings supporting the association between toxicant exposure and site-specific cancers are highlighted. Emerging concepts, including the exposome, cumulative risk assessment, and gene–environment interactions, are also addressed. Understanding the contribution of environmental toxicants to cancer development is essential for risk assessment, preventive strategies, and public health policy formulation.

Keywords: Environmental toxicants; carcinogenesis; cancer etiology; genotoxicity; exposome; oxidative stress.

INTRODUCTION

Cancer, a condition commonly prevailing throughout the world, is a major cause of mortality. The etiology of cancer is complex, with multiple factors involved ranging from genetic susceptibility to environmental factors (Vineis and Fecht, 2014). Evidence shows that genetic traits are responsible for less that around 30% cases, and environmental factors contribute majorly to 70-90% cases (Wu et al., 2016; Wu et al., 2018). These environmental factors include lifestyle and diet choices, air and water pollution, environmental toxins, radiation, occupational hazards, etc. Emerging evidence indicates that environmental pollution plays a significant rolein the global cancer burden, specifically in low and middle-income countries where regulatory controls may be limited (Landrigan et al., 2018). All the conditions to which a human is exposed throughout his lifetime are termed the exposome. The exposome concept has further expanded understanding by emphasizing the cumulative impact of lifetime environmental exposures on disease development, including cancer (Wild, 2005). Evaluating the contribution of environmental toxicants to cancer etiology is therefore critical for risk assessment and prevention strategies (Vineis& Wild, 2014).

Since carcinogenesis is a multistep process, multiple complementary exposures may act together to constitute the exposome for an individual. But it might be possible that these exposures do not predict carcinogenicity if they occur individually. Hence, it is important to note that synergistic effects of multiple disruptive exposures play a key role in the manifestation of different hallmarks of cancer (Goodson et al., 2015). Lack of established threshold exposure values for all of these environmental toxicants increases the risk of cancer development by compromising cellular homeostasis and impairing effective immune surveillance and deregulating inflammation (Humphrey et al., 2019; Loxham et al., 2015).

Environmental Toxicants and Route of Exposure

Environmental toxicants encompass a diverse group of chemical and physical agents that contaminate air, water, soil, and food. Many of these agents have been classified as

carcinogenic or potentially carcinogenic based on human and experimental evidence (IARC, 2019). Widespread exposure occurs through inhalation of polluted air, ingestion of contaminated food and water, and dermal contact with consumer products or occupational materials (WHO, 2013). Agriculture and industry setups are cancer-prone environments because of the repeated and prolonged exposure to toxicants (Alavanja et al., 2013).

The International Agency for Research on Cancer (IARC) has categorised environmental toxicants based on their reported carcinogenicity on humans. The airborne exposome includes regulated "criteria" pollutants like particulate matter (PM2.5 and PM10) and nitrogen dioxide, alongside thousands of hidden organic compounds like volatile organiccompounds (VOCs) and polycyclic aromatic hydrocarbons (PAHs). Humans are surrounded by these substances and are exposed to them primarily through inhalation, reasons being outdoor air pollution, second-hand smoke, radon, and industrial emissions. Increased incidents of lung cancer have been linked to PM 2.5, as research shows that exposure to PM 2.5 results in epigenetic alterations, disrupted gene expression in respiratory tract cells(Wang et al., 2025; Mukherjee et al., 2021). Industrial and traffic-dense environments release petroleum products, and exposure to benzene can happen in such conditions. Inhalation of benzene or solvents in industrial settings has been correlated to hematologic cancers (Mangiaterra et al., 2025; Aslam et al., 2022).

Aflatoxins are one of the most potent naturally occurring carcinogens. Exposure to these toxins can happen through ingestion via contaminated food, stored in humid conditions (Gemede et al., 2025; Jedua et al., 2026). Chronic aflatoxin exposure has been associated with increased risk of gallbladder, gastrointestinal, and liver cancers in some populations (Foerster et al., 2016).

Prolonged exposure to arsenic via contaminated groundwater is also linked to skin and bladder cancers (Shaji et al., 2021). Phthalates, which are widely used in consumer products, are reported to be endocrine-disrupting chemicals, and major routes of exposure are contaminated food and water, inhalation of indoor dust particles containing phthalate residues, and dermal absorption from cosmetics and personal care products(Ali et al., 2021; Pagoni et al., 2020). Epidemiological and pre-clinical studies have shown links between high levels of urinary phthalate metabolites and high risks of hormone-dependent cancers (Zheng et al., 2025).

Microplastics, emerging pollutants from plastic degradation, are increasingly implicated in gastrointestinal health risks (Bora et al., 2024). Primary exposure occurs through ingestion (via contaminated food and water), and inhalation of airborne microplastic particlespotentially contributes to colorectal carcinogenesis (Zuri et al., 2025. Major exposure routes include ingestion (via contaminated water and food), inhalation of industrial or occupational emissions, and, to a lesser extent, dermal contact through polluted soil or dust (Mileti et al., 2023). Pesticides promote carcinogenesis through endocrine disruption, genotoxicity, oxidative stress, and epigenetic alterations (Muñoz-Bautista et al., 2025). Exposure to pesticides primarily occurs through dermal absorption during handling and application, inhalation of aerosols or contaminated air, and ingestion of pesticide residues on crops and food products (Tudi et al., 2022; Kori et al., 2019, 2020).

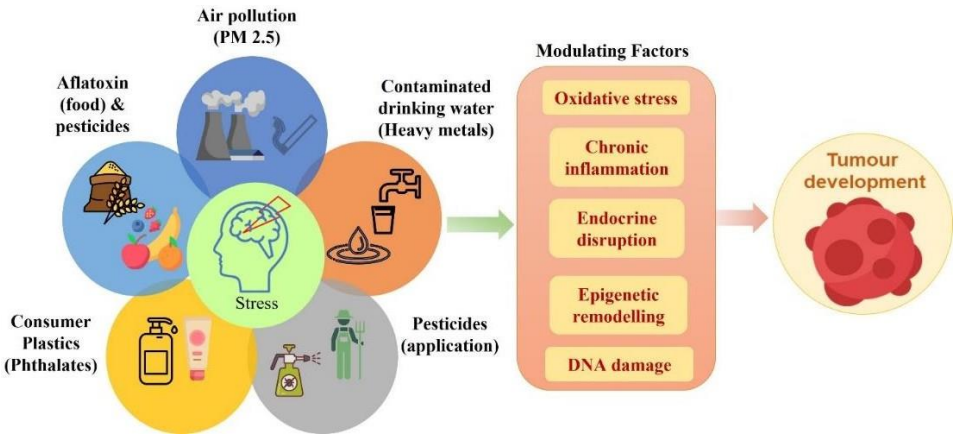

Figure: 1. Exposure to Environmental Toxicants and Modulating Factors Involved in the Prognosis of Tumour Generation

Toxicokinetic and Bioaccumulation

The carcinogenic effects of the environmental toxicants depend on their toxicokinetic properties (absorption efficiency, metabolic transformation, and elimination). Each toxicant has its own distinct rate of absorption, distribution, metabolism, and excretion. The primary homeostatic mechanism is xenobiotic metabolism, where Phase I oxidation takes place, mediated by cytochrome P450 enzymes. Phase II conjugation is supported by enzymes (e.g., glutathione S-transferases). While these processes generally detoxify chemicals for excretion, they can sometimes bioactivate pro-carcinogens into highly reactive intermediates, such as the metabolite benzo[a]pyrene-7,8-diol-9,10-epoxide produced from benzo(a)pyrene (Lang et al., 2025).

Certain toxicants exhibit bioaccumulation, where their total body burden increases with age, as seen with organochlorine pesticides (lipophilic compounds)(Jaishankar et al., 2014). Others target specific organs for deposition; for example, the prostate is a target organ for cadmium deposits. Some chemicals react with DNA to form pathogenic adducts that can persist in tissues for decades, serving as long-term biomarkers of exposure. Such persistence complicates exposure assessment and increases the likelihood of chronic cellular damage, thereby elevating cancer risk (Goodman et al., 2015).

Table 1. Exposure to Environmental Toxicants and Their Mechanism of Toxicity

Environmental Toxin	Sources / Settings of Exposure	Mechanism of Action / Carcinogenesis	References
Particulate Matter (PM2.5, PM10)	Inhalation via outdoor air pollution, industrial emissions, and second-hand smoke.	Causes epigenetic alterations and disrupts gene expression in respiratory tract cells. Induces oxidative stress leading to DNA base modifications, and activates innate immune responses that sustain chronic inflammation.	Wang et al., 2025; Mukherjee et al., 2021; Valavanidis et al., 2013; Gangwar et al., 2020
Nitrogen Dioxide, VOCs, & PAHs (e.g., Benzo(a)pyrene)	Inhalation of polluted air and industrial emissions.	Undergoes xenobiotic metabolism (Phase I oxidation and Phase II conjugation), which can bioactivate pro-carcinogens into highly reactive intermediates (e.g., benzo[a]pyrene-7,8-diol-9,10-epoxide).	Lang et al., 2025
Benzene and Solvents	Inhalation in traffic-dense environments and industrial settings.	Strongly correlated with the development of hematologic cancers and nervous system cancers.	Mangiaterra et al., 2025; Aslam et al., 2022
Aflatoxins (e.g., Aflatoxin B1)	Ingestion of contaminated food stored under warm and humid conditions.	Leaves a distinct mutational "fingerprint" by inducing a unique G to T transversion in the p53 gene, acting as a hallmark driver of liver, gallbladder, and gastrointestinal cancers.	Gemede et al., 2025; Jedua et al., 2026; Foerster et al., 2016; Mafe&Büsselberg, 2025
Heavy Metals (Arsenic, Cadmium, Nickel)	Ingestion of contaminated groundwater (Arsenic), inhalation of emissions, and dermal contact with polluted soil.	Triggers excessive reactive oxygen species (ROS) causing DNA mutations. Induces inheritable epigenetic alterations. Cadmium specifically acts as a "metalloestrogen," activating estrogenic pathways to promote uncontrolled cellular proliferation.	Shaji et al., 2021; Mandal et al., 2022; Salmerón-Bárcenas et al., 2025; Mafe&Büsselberg, 2025
Phthalates and BPA (Endocrine-	Ingestion (food/water), dermal absorption (cosmetics/personal	Acts as an endocrine disruptor that mimics or blocks natural hormones, interfering with hormonal signaling networks to	Ali et al., 2021; Pagoni et al., 2020/2022; Zheng et al.,

Disrupting Chemicals)	care), and inhalation of indoor dust.	drive hormone-dependent malignancies.	2025; Kabir et al., 2015
Microplastics	Ingestion (via food and water) and inhalation of airborne plastic particles.	Easily infiltrates biological systems, contributing to the total carcinogen burden and driving gastrointestinal and colorectal carcinogenesis.	Bora et al., 2024; Zuri et al., 2025/2023; Wang et al., 2021
Pesticides	Dermal absorption during handling/application, inhalation of aerosols, and ingestion of crop residues.	Promotes cancer via a combination of endocrine disruption, genotoxicity, oxidative stress, and epigenetic alterations. Lipophilic organochlorines bioaccumulate, increasing the body burden with age.	Muñoz-Bautista et al., 2025; Tudi et al., 2022; Jaishankar et al., 2014; Alavanja et al., 2013; Kori et al., 2019
Tobacco Smoke	Inhalation by both active and passive smokers.	Introduces over 60 known carcinogenic compounds into the body, inducing oxidative stress and DNA mutations.	Huang et al., 2017
Asbestos and Silica	Inhalation from environmental and occupational sources.	Activates innate immune responses to sustain low-grade, persistent inflammation. Releases pro-inflammatory cytokines (IL-6, TNF-α, IL-1β) that create a survival milieu for transformed cells and drive immune exhaustion.	Gangwar et al., 2020; Florescu et al., 2023

Mechanisms of Toxicant-Induced Carcinogenesis

Environmental toxicants exert carcinogenic effects by interfering with several biological mechanisms. The interconnected network of these mechanisms gets impaired due to the signalling cascade. Oxidative stress, DNA damage, epigenetic changes, endocrine disruption, chronic inflammation, disruption of calcium homeostasis and mitochondrial function are the effects of these toxicants. Heavy metals, industrial emissions, cigarette smoke, PM 2.5 induce oxidative stress resulting in increased reactive oxygen species production. Excessive reactive oxygen species produce base modifications in the DNA which can lead to mutations in tumor suppressor genes (Mandal et al., 2022).

Genotoxic environmental agents directly damage DNA and disturb the repair pathways and the accumulation of mutations, driving tumor initiation (Smith et al., 2016). Chronic reactive oxygen species in elevated levels not only damage DNA but also disrupt cellular signalling pathways, and favours genomic instability. Such conditions if sustained promote a pro-inflammatory microenvironment and support tumor progression (Klaunig et al. 2011; Valavanidis et al., 2013). Exposure to environmental toxicants can also result in epigenetic reprogramming through alterations in DNA methylation patterns, histone modifications. Changes in expression of non-coding RNA can disturb gene homeostasis, causing abnormal activation and silencing of pathways involved in tumor initiation and progression (Herceg&Vaissière, 2011). Heavy metal toxicants such as arsenic, nickel, and cadmium can alter gene expression and these changes could be inheritable (Salmerón-Bárcenas et al., 2025).

Endocrine-disrupting chemicals further complicate the toxic insult by interfering with hormonal signalling networks that are involved in the development of hormone-dependent malignancies. Endocrine-disrupting chemicals like phthalates and BPA mimic or block natural hormones (Kabir et al., 2015). Some toxic agents interfere with calcium signalling and damage mitochondria, resulting in metabolic modulation that supports the survival of cancer cells. Cadmium acts as a metalloestrogen which binds to estrogenic receptors to activate estrogenic signaling pathways in breast and uterine cells, thereby promoting uncontrolled proliferation (Mafe and Büsselberg, 2025).

Chronic inflammation represents another key route of toxin-driven carcinogenesis. Substances such as asbestos, silica, and air pollutants activate innate immune responses that sustain low-grade, persistent inflammation (Gangwar et al., 2020). Pro-inflammatory cytokines like IL-6, TNF-α, and IL-1β create a milieu that promotes proliferation,

angiogenesis, and survival of transformed cells (Florescu et al., 2023). Persistent cytokine signaling and oxidative stress not only promote DNA damage but also drive immune exhaustion, enabling malignant cells to evade cytotoxic T-cell recognition (Liu et al., 2022).

Epidemiological and Experimental Evidence

Over the years, epidemiological research has demonstrated correlations between exposure to environmental toxins and increased cases of cancer(Boffetta, 2006). Chronic exposure to air pollution has been linked to mortality due to lung cancer, and pesticide exposure has been associated with hematological and prostate malignancies (Turner et al., 2017; Alavanja et al., 2013).Pre-clinical studies provide supporting evidence for the fact that chronic toxicant exposure can induce malignant transformation and tumor formation (Hernández et al., 2013). These studies help in understanding the dose–response relationships and identification of molecular targets in carcinogenesis (Goodman et al., 2015).

Epidemiological studies, such as the multicentric ESCAPE project, have established significant links between outdoor air pollution and lung cancer, even at low levels of exposure. In Ontario, Canada, UV radiation, radon, and PM2.5 were identified as the top three contributors to the environmental cancer burden. Tobacco smoking remains the most impactful preventable exposure, containing over 60 compounds that increase cancer risk in both active and passive smokers (Huang et al., 2017).Experimental evidence provides causal insights through mutational signatures, molecular "fingerprints" left by specific toxins. For example, aflatoxin B1 induces a unique G to T transversion in the p53 gene, a hallmark of liver cancer. Similarly, arsenic exposure through groundwater is dose-related to increased incidences of skin, bladder, and lung cancers in regions like Bangladesh and Taiwan (Mafe and Büsselberg, 2025).

Organ-Specific Cancer Risk

Environmental carcinogens exert their toxic effects on each organ differently depending upon exposure route, metabolic activation, and tissue susceptibility. Air pollution mainly targets the respiratory system, and heavy metals and endocrine-disrupting chemicals are implicated in liver, kidney, breast, and reproductive cancers (Baan et al., 2008; Kabir et al., 2015). Lung Cancer is highly linked to tobacco, radon, air pollution, asbestos, nickel, chromium, and diesel exhaust. Genitourinary Cancers are associated with tobacco, aromatic amines, arsenic, obesity, hypertension, trichloroethylene, and certain pesticides. Hematologic Malignancies have been correlated with exposure to benzene. Contaminated food and water are linked to liver (aflatoxin), stomach (arsenic), and colorectal cancers (PM2.5, microplastics).

Emerging Perspectives and Public Health Implications

Emerging research underscores the concept of exposome, since an individual is rarely exposed to a single toxicant at one time (Sexton & Linder, 2011). In addition to existing contaminants, new ones, such as microplastics, have been added to the burden of carcinogens due to their capacity to infiltrate the biological systems (Wang et al., 2021). To effectively prevent cancer, it is necessary to have integrated environmental policies, improved exposure monitoring, and public health interventions aimed at reducing toxicant exposure (Landrigan et al., 2018).

The exposomic perspective recognises that the risk of cancer is formed by the combined exposures from conception to death. An emerging and interdisciplinary discipline integrating molecular epidemiology, basic research, and clinical medicine is environmental oncology. Multi-omics, predictive modelling, and advanced testing are transforming this field.

Multi-omics combines genomics, epigenetics, transcriptomics, and metabolomics to identify early indicators of response and exposure biomarkers. Predictive Modelling uses artificial intelligence to simulate multi-toxin interactions and predict cancer risks. Advanced testing utilizes 3D organoids and CRISPR screens to study human-specific responses to carcinogens (Gao, 2025).

Marginalised communities who work in extreme conditions face increased exposure to industrial effluents and hazardous waste. Recognising these environmental disparities is a critical requirement for modern public health policy. This should be taken into consideration while designing risk assessment models.

Prevention from the exposure of environmental toxins can be maximised through macro-level changes, such as sustainable urban infrastructure and the enforcement of comprehensive regulatory standards to limit the environmental presence of potent carcinogens.

CONCLUSIONS

The swing from a healthy cell to a malignant cell usually results from multiple factors over time, rather than a single event. It builds up throughout a lifetime of exposure to toxic agents. The environmental pollutants work together to interfere with our genetic and hormonal systems, leading to the development of cancer. There is a need to look beyond individual risks and address the environmental injustices that prevail worldwide by improving public health policies. By making the most use of newer technologies such as multi-omics and AI-driven predictive models, signs of abnormalities in the human body can be detected much earlier.To effectively reduce the global burden of cancer, there is a need to implement stricter regulations and create urban spaces that put people's health ahead of industrial convenience.

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Informed consent

There are no human participants in this article, and informed consent is not required.

Ethical Statement

This article does not contain any studies with human or animal participants.

Conflict of interest statement

Authors don't have any conflicts of interest.

Data availability statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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