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Environmental Pollution and Cancer: Immunotoxic Mechanisms and Emerging Therapeutic Insights

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ABSTRACT

Environmental pollution remains a critical global health concern, exerting profound effects on human well-being through its role in cancer development and immune dysfunction. Exposure to air pollutants, heavy metals, pesticides, plasticizers, and other contaminants has been linked to the induction of oxidative stress, chronic inflammation, and genotoxicity, ultimately promoting carcinogenesis. Beyond mutagenic effects, pollutants disrupt immune homeostasis by impairing innate and adaptive immune responses, suppressing antitumor immunity, and enhancing susceptibility to tumor progression. Immunotoxic pathways involve dysregulation of cytokine networks, altered T-cell signaling, macrophage polarization, and impaired natural killer (NK) cell function. This review synthesizes current evidence on the mechanistic links between environmental pollution, immunotoxicity, and cancer, while also highlighting epidemiological data underscoring global disease burdens. Furthermore, we explore emerging therapeutic insights, including antioxidant interventions, immunomodulatory agents, nanoparticle-based detoxification systems, and policy-driven preventive measures aimed at mitigating pollutant exposure. By integrating mechanistic, clinical, and therapeutic perspectives, this article underscores the urgent need for multidisciplinary approaches to address pollution-driven immunotoxicity and cancer risk.

Keywords: Environmental pollution, Cancer, Immunotoxicity, Oxidative stress, Inflammation

INTRODUCTION

Environmental pollution has emerged as one of the most pervasive and pressing global health challenges of the 21st century [1]. The rapid pace of industrialization, urbanization, and agricultural intensification has significantly increased the release of pollutants into the environment [2]. According to the World Health Organization (WHO), more than 12 million deaths annually are linked to environmental risk factors, with a considerable proportion associated with cancers [3]. Unlike traditional risk factors such as genetics or lifestyle, environmental pollution poses a universal threat because of its ubiquitous presence in air, water, soil, and food chains [4]. Cancer, a leading cause of global morbidity and mortality, is strongly influenced by long-term environmental exposures. Pollutants act as initiators and promoters of carcinogenesis through multiple mechanisms, including direct DNA damage, epigenetic modifications, and immune system dysregulation. Importantly, cancer development is not solely dependent on mutagenic events but also on alterations in the tumor microenvironment [5]. Immunotoxicity-the impairment of immune competence by toxic substances-plays a central role in enabling transformed cells to evade immune surveillance, sustain proliferation, and establish malignancies [6]. Emerging evidence indicates that exposure to pollutants such as particulate matter, volatile organic compounds (VOCs), heavy metals, pesticides, and plasticizers leads to chronic oxidative stress and inflammation [7,8]. These conditions promote oncogenic mutations while simultaneously weakening host defense mechanisms. For instance, long-term air pollution exposure is now considered an independent risk factor for lung and colorectal cancers, even in nonsmokers. Agricultural workers chronically exposed to pesticides show higher incidences of lymphomas and leukemias. Industrial contamination of

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drinking water with arsenic and cadmium has been linked to increased cases of bladder, kidney, and skin cancers [9,10].

Given the scale of the problem, it is crucial to investigate how environmental pollutants contribute to cancer through immunotoxic mechanisms. By exploring the pathways that link pollution-induced immune alterations to tumor initiation and progression, researchers can better identify preventive and therapeutic interventions. This review aims to synthesize current understanding of these interconnections, highlight epidemiological evidence, and examine emerging strategies to mitigate the cancer burden associated with pollution-driven immunotoxicity.

2. Environmental Pollution and Cancer Development

Environmental pollutants encompass a broad range of chemical and physical agents, many of which have established carcinogenic potential [2]. Their effects vary depending on chemical composition, route of exposure, dose, and duration. Four key categories of pollutants are consistently implicated in cancer risk: air pollutants, heavy metals, pesticides, and endocrine-disrupting plasticizers.

2.1 Air Pollution

Air pollution, particularly fine particulate matter (PM2.5 and PM10), is one of the most studied environmental risk factors for cancer. Inhaled particles penetrate deep into the respiratory tract, where they generate reactive oxygen species (ROS), damage epithelial cells, and trigger chronic inflammatory responses [11]. Epidemiological studies link long-term exposure to high PM concentrations with lung cancer, and recent evidence suggests associations with breast, colorectal, and pancreatic cancers [12]. Mechanistically, pollutants activate oncogenic pathways such as NF-κB, p53, and MAPK while simultaneously impairing immune surveillance, enabling mutated cells to persist and proliferate [13].

2.2 Heavy Metals

Heavy metals such as arsenic, cadmium, chromium, and lead are persistent environmental contaminants with strong carcinogenic potential [3]. Chronic arsenic exposure through contaminated drinking water is a well-established cause of bladder, lung, and skin cancers. Cadmium exposure, common in industrial and agricultural areas, disrupts DNA repair mechanisms, induces oxidative stress, and alters immune signaling [14]. Chromium compounds, widely used in metallurgy and paints, cause genotoxicity and immunosuppression. Heavy metals are particularly concerning because they bioaccumulate in tissues and exert long-term effects, even after exposure ends [15].

2.3 Pesticides and Agricultural Chemicals

Pesticides remain indispensable in modern agriculture but pose serious health risks. Numerous studies link pesticide exposure to hematological malignancies such as non-Hodgkin's lymphoma, leukemia, and multiple myeloma [16]. Organophosphates, carbamates, and organochlorines interfere with immune cell signaling, impair T-cell proliferation, and alter cytokine profiles. In addition to direct DNA damage, pesticides act as immunotoxic agents that diminish tumor surveillance [17]. Occupational exposure among farmers, pesticide applicators, and rural residents highlights the pressing need for safer agricultural practices and stricter regulations.

2.4 Plasticizers and Endocrine Disruptors

Plasticizers such as bisphenol A (BPA) and phthalates are widely used in plastics, packaging, and personal care products [18]. These compounds mimic endogenous hormones, disrupt endocrine signaling, and promote tumorigenesis in hormone-sensitive tissues such as breast, prostate, and ovarian glands. Beyond their endocrine effects, plasticizers exert immunotoxic actions, including suppression of natural killer (NK) cell function and dysregulation of T-helper cell responses, thereby reducing antitumor immunity [19]. Chronic exposure through food packaging, medical devices, and household products makes these pollutants especially concerning for vulnerable populations, including children and pregnant women [20].

3. Immunotoxic Mechanisms Linking Pollution and Cancer

The immune system plays a dual role in cancer biology: it can protect against tumor development through immune surveillance, but it can also promote tumor progression when chronically dysregulated. Environmental pollutants often tip this balance by inducing immunotoxic effects that weaken antitumor responses and foster a pro-carcinogenic microenvironment.

3.1 Oxidative Stress and DNA Damage

Pollutants such as particulate matter, heavy metals, and pesticides elevate intracellular levels of reactive oxygen species (ROS) and reactive nitrogen species (RNS) [21]. These highly reactive molecules damage DNA, proteins, and lipids, leading to mutations and genomic instability. At the same time, oxidative stress activates transcription factors such as NF-κB and AP-1, which drive chronic inflammation, a recognized enabler of tumorigenesis [22]. Persistent oxidative damage can overwhelm DNA repair systems, allowing mutated cells to survive and proliferate [23].

3.2 Cytokine Dysregulation

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Environmental contaminants alter cytokine signaling pathways that regulate immune responses. For example, pesticide exposure has been shown to increase pro-inflammatory cytokines such as TNF-α and IL-6, while suppressing anti-inflammatory cytokines like IL-10 [24] This imbalance promotes chronic low-grade inflammation, which not only damages tissues but also creates a tumor-promoting environment. In addition, dysregulated cytokine networks recruit myeloid-derived suppressor cells (MDSCs) that inhibit effective T-cell responses against tumors [25].

3.3 T-Cell Dysfunction

T-lymphocytes are central to adaptive immunity, yet pollutants often impair their function. Heavy metals and endocrine disruptors can inhibit T-cell proliferation, skew T-helper (Th) cell differentiation, and reduce cytotoxic T-cell activity. Such impairments weaken immune surveillance, allowing malignant cells to escape detection [26]. Endocrine disruptors like BPA, for instance, shift the Th1/Th2 balance toward a Th2-dominant profile, which is less effective in antitumor defense [27].

3.4 Macrophage Polarization

Macrophages exhibit remarkable plasticity, switching between pro-inflammatory M1 and tumor-promoting M2 phenotypes. Exposure to pollutants often drives polarization toward the M2 phenotype [28]. These M2-like macrophages secrete growth factors, promote angiogenesis, and suppress cytotoxic immune responses, thereby facilitating tumor progression. For example, diesel exhaust particles and arsenic have been reported to enhance M2 polarization in experimental studies [29].

3.5 Natural Killer (NK) Cell Suppression

NK cells represent the first line of defense against malignant transformation by directly killing tumor cells without prior sensitization [30]. However, several pollutants impair NK cell cytotoxicity by reducing perforin and granzyme expression. Decreased NK cell activity has been observed in populations chronically exposed to industrial chemicals, which correlates with elevated cancer risk [31,32].

4. Epidemiological Evidence

Epidemiological studies provide compelling support for the mechanistic links between environmental pollution, immunotoxicity, and cancer [33]. In urban centers with high air pollution levels, lung cancer incidence is significantly elevated even among non-smokers, highlighting the carcinogenic potential of airborne particles [34]. Cohort studies in Asia and Europe have demonstrated a dose-response relationship between particulate matter exposure and cancer mortality. Similarly, exposure to traffic-related pollutants is associated with increased breast cancer risk in women [35].

Occupational exposure studies further strengthen these associations. Agricultural workers exposed to pesticides show higher prevalence of non-Hodgkin's lymphoma, leukemia, and multiple myeloma compared to the general population [36]. Industrial workers chronically exposed to cadmium or arsenic face elevated risks of bladder, kidney, and skin cancers. In many of these cohorts, immune dysfunction markers-including altered cytokine levels, impaired NK cell function, and reduced T-cell activity-parallel cancer incidence, underscoring the role of immunotoxicity [37]

Importantly, epidemiological evidence also highlights health disparities. Communities located near industrial zones, waste incinerators, and contaminated water sources often face disproportionately high cancer burdens [38]. This underscores the need for environmental justice interventions to reduce exposure in vulnerable populations.

5. Emerging Therapeutic and Preventive Strategies

Given the intertwined roles of pollution, immunotoxicity, and cancer, research is increasingly directed toward strategies that restore immune competence and limit pollutant effects.

5.1 Antioxidant-Based Interventions

Antioxidants such as vitamins C and E, flavonoids, and polyphenols can mitigate ROS-induced oxidative stress [39]. Experimental studies show that diets rich in antioxidants reduce pollutant-induced DNA damage and inflammation. Pharmacological agents like N-acetylcysteine are also being investigated for their ability to replenish intracellular glutathione and counteract pollutant-driven oxidative stress [40].

5.2 Immunomodulatory Approaches

Cancer immunotherapies, including immune checkpoint inhibitors and adoptive T-cell transfer, offer promising strategies to reverse pollutant-induced immune suppression [41]. While primarily developed for established cancers, these therapies may eventually be adapted for populations with high environmental exposure risks. Cytokine-based therapies that restore Th1 responses or enhance NK cell activity are under preclinical exploration [42].

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5.3 Nanoparticle-Based Detoxification

Recent advances in nanomedicine suggest that engineered nanoparticles can adsorb or neutralize pollutants before they exert harmful effects [43]. Additionally, nanoparticle carriers can deliver antioxidants or immunomodulators directly to affected tissues, increasing therapeutic precision while minimizing systemic toxicity.

5.4 Policy and Public Health Interventions

Prevention remains the most effective strategy. Strengthening environmental regulations, promoting clean energy transitions, reducing pesticide dependency, and enforcing safe waste disposal practices are critical public health measures [44]. Community-level interventions, including clean water programs, urban air quality monitoring, and health education campaigns, are equally vital [45]. Such policies not only reduce cancer risk but also help restore immune resilience in exposed populations [46].

CONCLUSION

Environmental pollution represents a dual threat to health by directly inducing DNA damage and indirectly fostering carcinogenesis through immunotoxicity. The interplay between oxidative stress, chronic inflammation, and immune dysregulation provides a mechanistic basis for understanding how pollutants contribute to cancer. Advances in immunology, toxicology, and therapeutic innovation offer promising avenues for mitigating these effects. A multidisciplinary approach integrating biomedical research, clinical intervention, and environmental policy will be essential to reduce the global burden of pollution-driven cancers.

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