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Obesity and Environmental Pollutants: Emerging Crosstalk in Cancer Risk and Metabolic Dysregulation

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ABSTRACT

Obesity and environmental pollution are converging global health challenges that collectively drive the rising incidence of cancer and metabolic diseases. Adipose tissue, once regarded as a passive fat depot, is now recognized as a dynamic endocrine and immune organ, capable of storing lipophilic pollutants and modulating systemic homeostasis. Persistent organic pollutants, heavy metals, endocrine-disrupting chemicals, and particulate matter accumulate in adipose tissue where they exacerbate oxidative stress, chronic inflammation, and hormonal disruption. These processes intersect with obesity-associated insulin resistance, dysregulated adipokine secretion, and immune dysfunction, creating a permissive microenvironment for carcinogenesis. This review synthesizes evidence on the molecular, cellular, and systemic mechanisms by which obesity and pollutants interact to elevate cancer risk. Special emphasis is placed on oxidative stress and immune dysregulation as shared pathways, the modulating role of antioxidants and lifestyle factors, and the need for integrated public health approaches. Understanding this crosstalk is essential for designing preventive strategies in rapidly industrializing societies disproportionately affected by both obesity and environmental pollution.

Keywords: Obesity; Environmental pollutants; Cancer; Immunity; Oxidative stress

INTRODUCTION

The dual epidemics of obesity and cancer represent some of the most pressing challenges to global health. Obesity prevalence has tripled in the last four decades, with over 650 million adults classified as obese worldwide [1]. Parallel to this trend, cancer remains a leading cause of morbidity and mortality, with lifestyle and environmental exposures contributing substantially to its burden [2]. In parallel, industrialization and urbanization have increased human exposure to environmental pollutants, including persistent organic pollutants (POPs), heavy metals, and particulate matter [3]. These toxicants are increasingly recognized as carcinogens and endocrine disruptors. Importantly, obesity and pollutants do not act independently; rather, they interact at multiple biological levels. Obese adipose tissue can serve as a reservoir for pollutants, while pollutants themselves can promote obesity through "obesogenic" mechanisms [4]. This review highlights emerging evidence on how obesity and environmental pollutants converge to promote metabolic dysregulation and cancer risk, with attention to shared mechanistic pathways and preventive opportunities.

2. Obesity and Cancer: Pathophysiological Links

Obesity is a well-recognized risk factor for multiple malignancies, including breast, colorectal, endometrial, kidney, and pancreatic cancers [5]. The relationship is not merely associative but is increasingly explained through mechanistic studies that highlight endocrine, metabolic, and immunological disturbances. The obese state alters systemic homeostasis, leading to a biological environment that favors tumor initiation, progression, and recurrence [6].

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2.1 Adipose Tissue as an Endocrine Organ

Adipose tissue functions as a metabolically active organ rather than a passive fat store. It secretes numerous adipokines, cytokines, and growth factors that regulate energy metabolism, immunity, and cellular proliferation [7]. In obesity, secretion patterns shift unfavorably: leptin levels rise, promoting inflammation, angiogenesis, and tumor cell proliferation, while adiponectin levels decline, reducing its protective anti-inflammatory and anti-proliferative effects [8]. Elevated leptin also enhances aromatase activity, increasing estrogen production and thereby raising risk for hormone-dependent cancers such as breast and endometrial cancer [9]. Other adipokines like resistin and visfatin further contribute to insulin resistance and inflammatory signaling, compounding the pro-carcinogenic environment [10].

2.2 Chronic Inflammation and Insulin Resistance

A defining feature of obesity is chronic, low-grade inflammation originating within adipose tissue [11]. Hypertrophic adipocytes attract macrophages and T cells, which release tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP) [11]. These mediators induce DNA damage, promote angiogenesis, and support survival of malignant cells [11]. Simultaneously, obesity is strongly associated with insulin resistance. The resulting hyperinsulinemia activates insulin and insulin-like growth factor-1 (IGF-1) signaling cascades, stimulating cell proliferation and inhibiting apoptosis [13]. Together, inflammation and hyperinsulinemia create a permissive setting for neoplastic transformation and progression.

2.3 Oxidative Stress as a Unifying Pathway

Another central link between obesity and cancer is oxidative stress. Excess nutrient intake increases mitochondrial activity and reactive oxygen species (ROS) production [14]. ROS damage DNA, proteins, and lipids, producing mutations that can initiate tumorigenesis [15]. Oxidative stress also enhances inflammatory signaling and genomic instability, further fueling the cycle of cancer development [16]. Thus, oxidative imbalance represents a unifying pathway that bridges obesity with cancer risk.

3. Environmental Pollutants and Carcinogenesis

Environmental pollutants represent another critical determinant of cancer risk, acting through mutagenic, epigenetic, and hormonal pathways [17]. Unlike lifestyle risks, pollutant exposure is often involuntary and long-term, making its health impact particularly concerning [18].

3.1 Categories of Pollutants

Persistent organic pollutants (POPs) such as polychlorinated biphenyls (PCBs), dioxins, and organochlorine pesticides are highly lipophilic and bioaccumulate in adipose tissue, disrupting endocrine and metabolic functions for decades [19]. Heavy metals including arsenic, cadmium, and lead act as genotoxic agents, inducing oxidative stress, altering DNA methylation, and directly damaging genetic material [20]. Endocrine-disrupting chemicals (EDCs) like bisphenol A and phthalates mimic or antagonize natural hormones, interfering with reproductive development and predisposing to hormone-sensitive cancers [21]. Airborne particulate matter (PM2.5) represents another major class, provoking systemic oxidative stress and inflammation and showing strong epidemiological associations with lung and bladder cancers [22].

3.2 Mechanistic Pathways

The mechanisms by which pollutants promote carcinogenesis are diverse yet interconnected. DNA damage occurs through direct covalent adduct formation or indirectly through pollutant-induced ROS [24]. Epigenetic alterations, including aberrant DNA methylation and histone modification, lead to dysregulated oncogene and tumor suppressor gene expression [25]. Endocrine disruption alters cellular growth and differentiation pathways, particularly in reproductive organs [26]. Finally, many pollutants impair mitochondrial function, reducing ATP generation and increasing ROS production, which together compromise cellular integrity and accelerate malignant transformation [27].

4. Adipose Tissue as a Reservoir of Pollutants

Adipose tissue plays a unique role in the interaction between obesity and environmental pollutants. Because many toxicants are lipophilic, fat depots act as long-term reservoirs, sequestering pollutants for years or even decades [28]. This property has dual consequences: it can reduce immediate systemic exposure but simultaneously prolong body burden and increase the risk of release during metabolic changes [28].

4.1 Bioaccumulation

Persistent organic pollutants (POPs) such as polychlorinated biphenyls, dioxins, and organochlorine pesticides accumulate in adipose tissue due to their hydrophobic nature [29]. In obese individuals, the larger fat mass increases

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the storage capacity, leading to higher pollutant load [30]. This accumulation may contribute to the observed association between obesity and elevated circulating levels of POPs, even when external exposure is modest.

4.2 Pollutant Release During Lipolysis

When fat stores are mobilized during weight loss, fasting, or illness, pollutants are released back into the bloodstream [31]. This transient surge in circulating toxicants can have systemic consequences, including oxidative stress, hepatic injury, and endocrine disruption. This phenomenon highlights a paradox: while weight loss improves metabolic health, it may temporarily increase exposure to stored pollutants, complicating long-term risk assessment.

5. Crosstalk Between Obesity and Pollutants in Cancer Risk

The interplay between obesity and pollutants amplifies carcinogenic risk through shared and synergistic biological pathways.

5.1 Oxidative Stress Synergy

Both obesity and pollutants are independent drivers of reactive oxygen species production. When combined, the oxidative burden intensifies, causing more extensive DNA strand breaks, base modifications, and lipid peroxidation [32]. These changes accelerate mutagenesis and genomic instability, central hallmarks of cancer.

5.2 Chronic Inflammation

Obese adipose tissue is a chronic source of inflammatory mediators, while pollutants such as particulate matter and heavy metals stimulate similar cytokine cascades [33]. Together, they enhance systemic inflammation, supporting angiogenesis, tumor cell proliferation, and immune evasion.

5.3 Immune Dysregulation

Pollutants suppress adaptive and innate immunity, weakening antigen presentation and natural killer (NK) cell function [34]. Obesity compounds this by impairing T-cell responsiveness and reducing anti-tumor surveillance [35]. The net result is a compromised immune system that allows malignant cells to thrive.

5.4 Metabolic Dysfunction and Hormonal Disruption

Pollutants such as endocrine-disrupting chemicals mimic estrogens or androgens, while obesity enhances endogenous hormone production, particularly estrogens from adipose aromatase activity [36]. Together, they amplify risk for hormone-dependent cancers. Moreover, pollutant-induced insulin resistance intensifies obesity-driven hyperinsulinemia, fueling IGF-1 signaling and tumor growth [37].

6. Antioxidants, Lifestyle, and Therapeutic Implications

Given the convergence of obesity and pollutant exposure on oxidative stress and inflammation, antioxidant strategies hold promise as mitigating interventions. Both dietary and endogenous antioxidants can help neutralize reactive oxygen species (ROS), reduce DNA damage, and restore redox homeostasis [38].

6.1 Dietary Antioxidants

Natural compounds such as vitamins C and E, carotenoids, polyphenols, and flavonoids have demonstrated protective effects against oxidative stress and pollutant-induced toxicity [39]. For instance, resveratrol and curcumin modulate nuclear factor erythroid 2–related factor 2 (Nrf2) pathways, enhancing cellular antioxidant defenses [40]. Diets rich in fruits, vegetables, and whole grains not only provide antioxidants but also improve metabolic health, thereby reducing the combined carcinogenic risk of obesity and pollutant exposure [41].

6.2 Pharmacological Approaches

Pharmacological antioxidants and agents targeting oxidative stress, such as N-acetylcysteine and mitochondrial-targeted compounds, are being investigated for their potential to reduce pollutant-induced carcinogenesis [42]. In obesity-associated cancers, drugs that improve insulin sensitivity, such as metformin, may indirectly attenuate oxidative stress and inflammatory signaling [43].

6.3 Lifestyle Modifications

Lifestyle interventions including weight reduction, regular physical activity, and avoidance of high-fat processed diets can alleviate obesity-related metabolic dysfunction. Exercise itself exerts antioxidant effects by upregulating endogenous defense systems and improving mitochondrial function [44]. Importantly, gradual weight loss strategies are recommended to minimize abrupt pollutant release from adipose stores.

7. Public Health Implications and Future Perspectives

The intersection of obesity and environmental pollutants in cancer risk underscores the need for integrated public health action.

7.1 Policy and Regulation

Strengthening regulations to limit human exposure to pollutants through restrictions on industrial emissions, pesticide use, and plastic additives remains fundamental. Public health initiatives must also emphasize obesity prevention through healthy food policies, physical activity promotion, and equitable access to nutritious diets.

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7.2 Risk Assessment in Vulnerable Populations

Certain groups, such as individuals in rapidly industrializing regions, low-income communities, and workers in high-exposure occupations, face disproportionately high risks [45]. These populations often also bear a higher burden of obesity, creating a compounded vulnerability. Targeted surveillance and intervention programs are urgently needed.

7.3 Research Directions

Future studies should explore the interactive mechanisms of pollutants and obesity at molecular and epidemiological levels, particularly focusing on long-term cohort studies. Advances in exposomics and systems biology could provide a more holistic understanding of combined exposures. Furthermore, research into safe weight loss strategies that minimize pollutant re-exposure is essential.

7.4 Health Education and Community Engagement

Raising awareness about the dual role of pollutants and obesity in cancer can empower individuals to adopt protective behaviors, such as dietary improvements and reduced use of plastics and pesticides. Community-based education and participatory approaches will be critical to ensure sustainable change.

CONCLUSION

Obesity and environmental pollutants represent converging epidemics that interact to amplify cancer risk and metabolic dysfunction. Adipose tissue serves both as a storage site and an active mediator of pollutant toxicity, linking obesity with pollutant-induced inflammation, oxidative stress, and immune suppression. These overlapping pathways underscore the inadequacy of addressing obesity and pollution in isolation. Instead, integrated strategies combining lifestyle interventions, antioxidant support, clinical surveillance, and robust environmental policies are essential for reducing long-term cancer risk in vulnerable populations. Understanding and intervening in this crosstalk is critical for global cancer prevention in the 21st century.

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