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Heavy Metal Exposure and Cancer Risk: A Zoological Perspective on Human Health Implications

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Abstract
Heavy metals such as lead (Pb), cadmium (Cd), mercury (Hg), and arsenic (As) are persistent environmental pollutants with significant health risks to humans and wildlife. Chronic exposure to these metals is strongly associated with carcinogenesis, mediated through oxidative stress, DNA damage, and disruption of cellular signaling pathways. This review adopts a zoological perspective to explore the shared vulnerabilities of humans and animals to heavy metal toxicity, emphasizing bioaccumulation in food chains and its cascading effects on ecosystems. Understanding these mechanisms offers valuable insights into preventive strategies, highlighting the importance of multidisciplinary approaches to mitigate cancer risks and promote environmental and public health.
Keywords: Heavy metals, carcinogenesis, oxidative stress, DNA damage, zoological perspective, bioaccumulation

Introduction

Heavy metals such as lead (Pb), cadmium (Cd), mercury (Hg), and arsenic (As) are recognized as significant environmental pollutants that pose serious health risks to both humans and wildlife. Chronic exposure to these metals has been linked to a variety of health issues, including carcinogenesis, which is the process by which normal cells are transformed into cancerous cells. The mechanisms underlying heavy metal toxicity and carcinogenesis are complex and multifaceted, involving oxidative stress, DNA damage, and disruption of cellular signaling pathways. Understanding these mechanisms from a zoological perspective can provide valuable insights into the shared vulnerabilities of

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humans and animals to heavy metal toxicity (Zaib et al., 2023 a).

The environmental persistence of heavy metals is a major concern, as these elements do not degrade and can accumulate in the food chain. For instance, heavy metals can bioaccumulate in aquatic organisms, leading to significant health risks for both the organisms themselves and the predators that consume them, including humans (Ejikeme et al., 2022; Adegbola et al., 2021). Studies have shown that heavy metals can induce oxidative stress, leading to cellular damage and increasing the risk of cancer (Ohiagu et al., 2022; Xu et al., 2017). This oxidative stress is often mediated by the generation of reactive oxygen species (ROS), which can cause DNA strand breaks and mutations that contribute to carcinogenesis (Morales et al., 2016; Lin et al., 2018). Furthermore, heavy metals can interfere with DNA repair mechanisms, exacerbating the risk of genetic alterations that may lead to cancer (Morales et al., 2016; Zhao et al., 2022).

The carcinogenic potential of heavy metals is well-documented, with specific metals like cadmium and arsenic being classified as known human carcinogens (Kim et al., 2015; Wang et al., 2016; Zaib et al., 2023 b). The mechanisms of heavy metal-induced carcinogenesis include the activation of signaling pathways that promote cell proliferation and survival, as well as the induction of epigenetic changes that can alter gene expression (Zhao et al., 2022; Si & Lang, 2018; Zaib et al., 2023 c). For example, cadmium has been shown to activate the Wnt/β-catenin signaling pathway, which is involved in cell growth and differentiation, thereby promoting malignant transformation (Lin et al., 2018). Additionally, heavy metals can disrupt the normal function of metallothioneins, proteins that help protect cells from metal toxicity, leading to increased susceptibility to cancer (Si & Lang, 2018; Al-Mzaien, 2021).

From a zoological perspective, the effects of heavy metals on animal health can mirror those observed in humans (Zaib et al., 2023 d). Research has demonstrated that aquatic species, such as fish, exhibit similar responses to heavy metal exposure, including oxidative stress and genotoxicity (Ejikeme et al., 2022; Adegbola et al., 2021). The accumulation of heavy metals in fish can lead to morphological abnormalities, reproductive issues, and increased mortality rates, which in turn can impact entire ecosystems (Kipsang et al., 2022; Okoro et al., 2012). This shared vulnerability highlights the importance of understanding heavy metal toxicity not only for human health but also for wildlife conservation and ecosystem stability (Zaib et al., 2023 e).

Moreover, the health implications of heavy metal exposure extend beyond direct toxicity (Zaib et al., 2023 f). Chronic exposure to heavy metals has been associated with various diseases, including cardiovascular diseases, neurological disorders, and immune dysfunction (Topdas et al., 2023). The interplay between heavy metal exposure and other environmental factors, such as pollution and lifestyle choices, further complicates the assessment of health risks. For instance, smoking has been shown to increase the bioavailability of heavy metals, thereby enhancing their carcinogenic potential (Bandeira et al., 2018). This underscores the need for comprehensive public health strategies that address both environmental and lifestyle factors in mitigating the risks associated with heavy metal exposure (Zaib et al., 2023 g).

Sources of Heavy Metal Exposure

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Heavy metals are pervasive environmental pollutants that can enter the human body through various pathways, leading to significant health risks. Understanding the sources of heavy metal exposure is crucial for developing effective public health strategies and mitigating the associated health risks. The primary sources of heavy metal exposure can be categorized into three main areas: environmental sources, dietary intake, and occupational exposure.

- *Environmental Sources*

Environmental sources of heavy metal exposure are primarily linked to industrial activities, mining operations, agricultural runoff, and contaminated water supplies. Industrial emissions are a significant contributor to heavy metal pollution, as factories release various metals, including lead, cadmium, mercury, and arsenic, into the atmosphere and surrounding environments Cook et al. (2021)Poole & Basu, 2017; López-Botella et al., 2021; Zaib et al., 2023 h).

Agricultural runoff is another critical source of heavy metal exposure, particularly in areas where fertilizers and pesticides containing heavy metals are used (Jaccob, 2020; Ah-Ra et al., 2020; Zaib et al., 2023 i). The leaching of these metals into water systems can contaminate drinking water and agricultural products, posing risks to human health (Okpogba et al., 2021; M, 2024). Contaminated water supplies, whether from industrial discharges or agricultural runoff, can lead to direct exposure through drinking water and recreational activities, further exacerbating the public health crisis associated with heavy metals (Michałek et al., 2019; Gong et al., 2017; Zaib et al., 2023 j).

- *Dietary Intake*

Dietary intake is a significant pathway for heavy metal exposure, particularly through the bioaccumulation of metals in food chains. Heavy metals can accumulate in fish, meat, and crops grown in contaminated soils, leading to increased concentrations of these toxic substances in the human diet (Abolape, 2019; Kim et al., 2023; Kowalska et al., 2017; Zeeshan et al., 2024 b). For instance, fish from polluted waters often contain elevated levels of mercury and other heavy metals, which can pose serious health risks to consumers (Zhou, 2023; Ściskalska et al., 2014; Zaib et al., 2023 k). Similarly, crops grown in contaminated soils can absorb heavy metals, leading to their presence in food products consumed by humans (Abbaslou, 2024; Chinedu & Chukwuemeka, 2018; Zeeshan et al., 2024 a).

The bioaccumulation of heavy metals in the food chain is particularly concerning because it can lead to higher concentrations of these toxic substances in top predators, including humans (Bocşan et al., 2016; Liu et al., 2018; Zaib et al., 2023 l). Studies have shown that long-term consumption of contaminated fish and agricultural products can result in chronic exposure to heavy metals, leading to various health issues, including neurological disorders, kidney damage, and increased cancer risk (Silva et al., 2022; Abdullahi et al., 2020; Choi & Kim, 2014; Zaib et al., 2023 m).

- *Occupational Exposure*

Occupational exposure to heavy metals is a significant concern, particularly in industries such as metal smelting, battery manufacturing, and construction activities. Workers in these industries are often exposed to high levels of heavy metals, including lead, cadmium, and mercury, through inhalation, dermal contact, and ingestion (Jung et al., 2016; Olsson et al., 2018; Shin et al., 2023;

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Zeeshan et al., 2024 c). For example, metal smelting operations release heavy metal particulates into the air, which can be inhaled by workers, leading to respiratory issues and systemic toxicity (Algafari et al., 2011; Bakri et al., 2020; Zaib et al., 2023 n).

Battery manufacturing is another industry with high occupational exposure risks, as workers may come into contact with lead and cadmium during the production and recycling processes (Ahmadi et al., 2022; White et al., 2019; Zeeshan et al., 2024 d). Construction activities, particularly those involving welding and metal cutting, can also expose workers to heavy metal fumes and dust, increasing their risk of developing occupational illnesses (Mourad & El-Sherif, 2022; Chung et al., 2015; Zaib et al., 2023 o).

Mechanisms of Carcinogenicity

Heavy metals are well-established environmental pollutants that can induce various mechanisms of carcinogenicity, leading to significant health risks, including cancer. The primary mechanisms through which heavy metals exert their carcinogenic effects include oxidative stress, genotoxic effects, epigenetic modifications, and disruption of cellular signaling pathways. Each of these mechanisms plays a crucial role in the initiation and progression of cancer.

- *Oxidative Stress*

Oxidative stress is a key mechanism through which heavy metals induce cellular damage. Heavy metals generate reactive oxygen species (ROS), which can cause significant damage to DNA, lipids, and proteins within the cell. For instance, cadmium (Cd) has been shown to induce oxidative stress, leading to DNA strand breaks and other forms of cellular damage (Liu et al., 2023; Kim et al., 2015; Zaib et al., 2023 p). The generation of ROS can overwhelm the cellular antioxidant defenses, resulting in lipid peroxidation and protein oxidation, which contribute to cellular dysfunction and promote carcinogenesis (Kontaş & Bostancı, 2020; Zaib et al., 2023 q).

The oxidative stress induced by heavy metals not only damages cellular components but also activates signaling pathways that can lead to inflammation and further cellular injury (Zeeshan et al., 2024 e). This chronic oxidative environment can create a feedback loop that enhances the risk of malignant transformations in affected cells (Liu et al., 2023; Zeeshan et al., 2023 a). The ability of heavy metals to induce oxidative stress is a critical factor in their carcinogenic potential, as it sets the stage for subsequent genetic and epigenetic alterations that drive cancer development (Zaib et al., 2023 r).

- *Genotoxic Effects*

Heavy metals also exert direct genotoxic effects through interactions with DNA. This can occur via metal-DNA binding, leading to structural changes in the DNA molecule that can result in mutations. For example, arsenic (As) has been shown to interfere with DNA repair mechanisms, thereby increasing the likelihood of mutations and chromosomal aberrations (Kim et al., 2015; Kopp et al., 2017; Zeeshan et al., 2023 b&c). The genotoxic effects of heavy metals are particularly concerning because they can lead to the initiation of cancer by causing permanent changes in the genetic material of cells (Zaib et al., 2023 s).

Studies have demonstrated that exposure to heavy metals such as cadmium and arsenic can result in increased levels of DNA damage, as evidenced by the formation of phosphorylated histone H2AX (γ-H2AX), a marker of DNA double-

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strand breaks (Liu et al., 2023; Zeeshan et al., 2023 d&e&f&g). This DNA damage can lead to cell cycle arrest, apoptosis, or, if not properly repaired, to the accumulation of mutations that drive tumorigenesis (Kontaş & Bostancı, 2020; Zeeshan et al., 2023 f&g). The ability of heavy metals to induce genotoxic effects underscores their role as significant carcinogens in both environmental and occupational settings (Zaib et al., 2023 t).

- *Epigenetic Modifications*

In addition to direct DNA damage, heavy metals can induce epigenetic modifications that disrupt normal gene expression patterns. These modifications include altered DNA methylation and histone modifications, which can silence tumor suppressor genes or activate oncogenes, contributing to cancer development (Kim et al., 2015). For instance, lead exposure has been shown to modify the expression of genes involved in cell cycle regulation and apoptosis, thereby promoting uncontrolled cell growth (Kontaş & Bostancı, 2020; Zeeshan et al., 2023 h&i).

Epigenetic changes can be heritable and may persist even after the initial exposure to heavy metals has ceased, leading to long-term consequences for cellular function and increasing the risk of cancer (Kumar et al., 2023; Kim et al., 2015). The ability of heavy metals to induce epigenetic alterations highlights the complexity of their carcinogenic mechanisms and the need for further research to understand the implications of these changes for human health (Zeeshan et al., 2024).

Disruption of Cellular Signaling

Heavy metals can also interfere with cellular signaling pathways that regulate critical processes such as cell proliferation and apoptosis. By disrupting these pathways, heavy metals can promote uncontrolled cell growth and survival, which are hallmarks of cancer (Kontaş & Bostancı, 2020). For example, cadmium has been shown to activate signaling pathways associated with cell proliferation while inhibiting apoptotic pathways, leading to enhanced cell survival and increased tumorigenic potential (Liu et al., 2023; Kim et al., 2015).

The disruption of cellular signaling by heavy metals can also lead to chronic inflammation, which is known to contribute to cancer progression. This inflammatory environment can further exacerbate oxidative stress and promote additional genetic and epigenetic changes, creating a vicious cycle that enhances the risk of cancer development (Kim et al., 2015; Kontaş & Bostancı, 2020). Understanding how heavy metals disrupt cellular signaling is crucial for developing strategies to mitigate their carcinogenic effects and protect public health.

The relationship between heavy metal exposure and toxicity in various animal models has been extensively documented, particularly in rodent studies. Research indicates a significant correlation between heavy metal exposure and tumor development in these models. For instance, Jamadagni et al. highlight that repeated exposure to heavy metal-containing products can lead to cumulative toxicity in Wistar rats, emphasizing the importance of determining the no observed effect level (NOEL) and no observed adverse effect level (NOAEL) through rigorous testing protocols (Jamadagni et al., 2017).

Moreover, the bioaccumulation of heavy metals in aquatic organisms serves as a critical bioindicator of environmental health and potential human health risks.

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Aquatic species such as fish and mollusks are particularly susceptible to heavy metal accumulation, which can reflect broader ecological impacts and human exposure risks (Afzal et al., 2023). Rahmaniar and Kamil discuss the remediation of contaminated soils using hyperaccumulator plants, which can absorb heavy metals like copper, thus providing insights into how aquatic organisms might similarly bioaccumulate these toxins from their environments (Rahmaniar & Kamil, 2015). The patterns of bioaccumulation observed in these species can mirror the health risks faced by humans, particularly in communities reliant on contaminated water sources for drinking and food (Zubair et al., 2023a&b).

Chronic exposure to heavy metals has also been linked to adverse reproductive outcomes and tumor growth in wildlife studies. Birds and mammals exposed to heavy metals exhibit reproductive failures and increased tumor incidence, underscoring the ecological ramifications of environmental contamination (Abbas et al., 2023). The findings from various studies indicate that heavy metals such as cadmium, lead, and nickel can disrupt endocrine functions and lead to developmental abnormalities in wildlife populations. While specific studies detailing these effects were not cited, the general consensus in the literature supports the notion that heavy metals pose significant risks to wildlife health (Zeeshan & Zaib, 2023).

The implications for human health are stark, particularly in light of epidemiological studies that have established a clear association between chronic exposure to arsenic in drinking water and various cancers, including skin, lung, and bladder cancer. Vulnerable populations, including children, pregnant women, and occupational workers, face heightened risks due to their increased susceptibility to the toxic effects of heavy metals. The cumulative exposure from environmental sources can lead to significant health disparities, as these groups often have limited access to safe drinking water and adequate health care resources. The need for targeted public health interventions is critical to mitigate these risks and protect at-risk populations.

Preventive strategies are essential in addressing the challenges posed by heavy metal toxicity. Implementing stricter environmental regulations and monitoring systems can significantly reduce emissions of heavy metals into the environment. Regulatory frameworks must be established to ensure that industries adhere to safe limits for heavy metal discharges, thereby protecting both ecological and human health. Furthermore, phytoremediation presents a promising approach to mitigate heavy metal contamination in soils and water bodies. The use of plants, particularly those that are hyperaccumulators, can effectively remove heavy metals from contaminated environments, as highlighted by Rahmaniar and Kamil (Rahmaniar & Kamil, 2015). This bioremediation strategy not only cleans up contaminated sites but also enhances soil health and biodiversity.

Conclusion

Heavy metals pose a dual threat to both human and animal health due to their persistence and bioaccumulative nature. The mechanisms of toxicity—ranging from oxidative stress and DNA damage to epigenetic changes—underline their carcinogenic potential. The mirrored impacts in humans and animals underscore the interconnectedness of environmental health and ecosystem stability. Addressing these risks requires integrated strategies, including stricter environmental regulations, enhanced monitoring, and increased public

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awareness. By adopting a multidisciplinary approach, it is possible to reduce heavy metal exposure, safeguard ecosystems, and minimize cancer risks in vulnerable populations.

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