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Exploring cadmium-induced alterations in the expression profile of microRNAs



(a) General diagram of the miRNA biogenesis pathway. (b) The major routes of cadmium exposure and its toxic impacts. AGO, Argonaute; Cd, cadmium; Exp5, Exportin 5; miRNA, microRNA; Ran/GTP, Ras-related Nuclear protein/Guanosine Triphosphate; RISC, RNA-induced silencing complex; TRBP, TAR RNA-binding protein. Credit: Mostafa Rezaei-Tavirani, Nasrin Amiri-Dashatan, Mehdi Koushki, Masoumeh Farahani

Cadmium (Cd), a toxic heavy metal, has been identified as a significant environmental pollutant due to its widespread industrial use and persistence in the environment. Chronic exposure to Cd poses a considerable threat to human health, as it accumulates in various tissues over time, leading to numerous diseases.



Recent research, <u>published</u> in the journal *Gene Expression*, has highlighted the role of microRNAs (miRNAs) in the molecular mechanisms of Cd toxicity. miRNAs are small non-coding RNAs that regulate <u>gene expression</u> post-transcriptionally, influencing a wide range of biological processes. The review aims to summarize the current understanding of Cd-induced miRNA dysregulation and its implications for disease progression.

Heavy metals are classified into essential and nonessential categories. Essential metals, such as manganese, play crucial roles in biological processes, whereas nonessential metals like Cd, lead (Pb), and arsenic (As) are toxic even at low concentrations. These metals can enter the human body through inhalation, ingestion, or skin contact, and they tend to bioaccumulate in tissues such as bones, liver, and kidneys.

Cd, in particular, has a long biological half-life and is known to cause <u>oxidative stress</u>, DNA damage, and interference with cellular metabolism, contributing to various diseases including cancer, cardiovascular diseases, and kidney damage.

Cadmium exposure affects miRNA expression through several mechanisms. Cd can induce oxidative stress, leading to the generation of reactive oxygen species (ROS), which in turn can alter the expression of miRNAs. Additionally, Cd can interact with DNA and proteins, causing epigenetic changes that influence miRNA expression. These dysregulated miRNAs can disrupt normal cellular functions by targeting specific mRNAs for degradation or translational repression, thereby affecting critical biological pathways.

Numerous studies have documented the effects of Cd on miRNA expression in various cell types and tissues. For instance, exposure to Cd has been shown to upregulate miR-221, which is associated with immune system alterations and increased risk of cancer. Similarly, miR-363-3p



expression is increased in Cd-exposed renal cells, leading to changes in cell proliferation and apoptosis. These findings suggest that specific miRNAs could serve as biomarkers for Cd exposure and its related health effects.

Human studies have corroborated the findings from in vitro and animal models, demonstrating that Cd exposure leads to significant changes in miRNA profiles. For example, workers exposed to Cd in occupational settings exhibit altered levels of miRNAs such as miR-122-5p and miR-326-3p, which are linked to early detection of Cd toxicity. These miRNAs are involved in critical pathways that regulate cellular stress responses, inflammation, and apoptosis, highlighting their potential as biomarkers for monitoring Cd exposure and associated health risks.

The dysregulation of miRNAs plays a pivotal role in the pathogenesis of diseases induced by Cd exposure. Understanding the specific miRNAs affected by Cd and their target pathways provides valuable insights into the molecular mechanisms of Cd toxicity.

Although significant progress has been made, further research is needed to establish a consistent profile of miRNA expression in response to Cd exposure and to develop effective strategies for preventing and mitigating the adverse health effects of this toxic metal. Future studies should focus on large-scale analyses of miRNA changes in diverse populations to enhance our understanding of Cd-related diseases and improve public health interventions.

More information: Mehdi Koushki et al, Cadmium-induced Alterations in the Expression Profile of MicroRNAs: A Comprehensive Review, *Gene Expression* (2024). DOI: 10.14218/GE.2023.00126



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