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Molecular Epidemiology of Uranium Exposure: Omics Approaches in Cancer Research

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Abstract
Uranium poses significant occupational and environmental risks due to its dual chemical toxicity and radioactivity. A well-established correlation exists between uranium exposure, including radon progeny, and lung cancer in miners. This systematic review synthesizes published omics-based studies from 2000 to 2024, utilizing databases including PubMed, Scopus, ScienceDirect, and Web of Science, with keywords encompassing "uranium exposure," molecular epidemiology, omics, and cancer risk. Molecular epidemiology employing high-throughput omics technologies has substantially advanced our understanding of uranium-induced carcinogenic mechanisms, revealing high frequencies of somatic mutations in tumor suppressor genes such as TP53 and oncogenes including KRAS, dysfunctions in DNA repair pathways, and epigenetic alterations characterized by global DNA hypomethylation and gene-specific hypermethylation affecting RASSF1A and CDKN2A. MicroRNAs including miR-21, miR-34A, and miR-155 demonstrate significant dysregulation following uranium exposure. Metabolomics studies reveal altered lipid metabolism, disrupted amino acid turnover, and elevated oxidative stress markers. Integrated multi-omics analyses establish molecular linkages to apoptosis, mitochondrial dysfunction, and perturbations in energy metabolism pathways. Biomarkers such as micronuclei frequency and chromosomal aberrations demonstrate strong associations with cumulative exposure levels and cancer risk. Human epidemiological studies face methodological challenges including limited sample sizes and heterogeneous exposure assessment approaches, yet multi-omics molecular epidemiology offers substantial potential for improved risk assessment, early detection, and prevention strategies in exposed populations.

Keywords: uranium exposure; molecular epidemiology; omics analysis; genotoxicity; epigenetic alterations; cancer risk

Introduction

Exposure to uranium and its decay products remains a critical public health issue because of its dual chemical and radiological toxicities, as well as its potential contribution to carcinogenic risk in exposed populations (Faqir et al., 2025). Uranium and its decay progeny include radon gas (^{222}Rn) and subsequent alpha-emitting isotopes that are encountered not only in occupational settings such as mining, milling, and processing of uranium ores, but also in environmental or residential contexts through contaminated air, water, or soil (Zhang et al., 2022). The relevance of uranium exposure to cancer is underscored by consistent findings of increased lung cancer incidence and mortality among uranium miners, and by emerging concerns regarding low-dose chronic environmental exposures outside of mining (Tirmarche et al., 2004). Concurrently, high-throughput omics technologies—including genomics, transcriptomics, epigenomics, proteomics,

and metabolomics—offer transformative opportunities to characterize molecular perturbations induced by uranium or radon exposure, enabling comprehensive investigations into exposure-biomarker-disease relationships within a molecular epidemiology framework (Brugge & Buchner, 2019).

This review addresses a critical question with relevance for both the scientific community and populations exposed to uranium: How have omics-based techniques been applied to understanding the molecular effects of uranium exposure, and what are the implications of such findings for our understanding of cancer risk? The review is structured to systematically examine this question across multiple dimensions (Gueguen et al., 2017; Wang et al., 2024). The review opens by introducing the context of the relationship between uranium exposure and cancer risk. Next, we summarize the current state of omics-based studies focusing on the molecular effects of uranium exposure towards cancer. This serves as the transitional phase between our understanding garnered from the scientific community and the application of such understanding towards improving exposure assessment and risk prediction. In the final stage of our review, we outline concepts for future directions in the molecular epidemiology of uranium-induced cancer. As our review strives to bridge the clinical applicability of molecular findings with the experiences of affected populations, we ensure navigation of the scientific literature in a manner that maintains both relevance and academic integrity.

In pursuit of these goals, the review has been designed to encompass both comprehensiveness and accessibility. After introducing the background information that represents the foundation of our review and understanding the toxicological and epidemiological basis of health hazards due to uranium exposure, the review has been divided based on methodologies as well as targets. Such sections include mutational patterns, transcriptomic and epigenomic changes, metabolomic patterns, as well as the incorporation of multi-omics information. Special effort has been made to highlight exposure-biomarker-disease patterns derived from molecular changes, emphasizing how these molecular signatures translate to population-level health outcomes. The final section incorporates the findings from the referenced studies to highlight the advancements achieved in the field despite the remaining uncertainties. Finally, the review concludes by focusing on the latest insights representing the indispensable role of omics in the comprehension of the health hazards due to uranium exposure.

Uranium is a naturally occurring metal and an environmental contaminant in air, water, and soil; thus, it poses threats to the environment and human health (Brugge & Buchner, 2019). The principal isotopes of uranium are ^{238}U (99.3%), ^{235}U (0.7%), and traceable ^{234}U (Zielinski et al., 2022). The main routes of human exposure occur through consumption of drinking water and food containing uranium, from which the metal can accumulate in body tissues. Of the heavy metals, uranium is distinctive due to its property of forming complex compounds (Bleise et al., 2003), which raises concern about its toxicity and its potential to induce cancer (Kreuzer et al.,

2018). This has prompted substantial scientific effort to define and understand its biological effects (Field & Steck, 2021).

Uranium exhibits dual toxicity: chemical and radiological. The chemical toxicity manifests primarily as nephrotoxic heavy metal behavior. Its effects concentrate in the renal system, impairing mitochondrial functions and disrupting redox balance, resulting in kidney cell damage and subsequent tubular necrosis with serious health implications for affected individuals (Vicente-Vicente et al., 2020). Conversely, the radiological aspect of toxicity stems from DNA damage produced by alpha particles emitted during uranium decay. This ionizing radiation component has profound health implications due to the resultant carcinogenic effects (Goodhead, 2018). For miners of uranium as well as other workers involved in the extraction of the substance, the picture can be even more complex. They are often at risk not only from uranium but from silica dust, diesel exhaust, as well as other radionuclides. This creates confounding factors when attempting to identify health risks specifically attributable to uranium exposure (Rage et al., 2022).

Pooled epidemiologic studies in 11 cohorts of uranium miners reveal a linear dose-response relationship in lung cancer risk, encompassing 7,754 deaths in 119,709 miners, and demonstrating an excess relative risk of 0.44 per 100 WLM radon exposure (Lubin et al., 1995). In a case-control analysis among Navajo miners, lung cancer risk was observed to be substantially elevated, with a relative risk of 28.6 (95% CI 13.2-61.7), compared to unexposed subjects despite the low prevalence of smoking (Roscoe et al., 1995). Systematic reviews have confirmed the pattern of excess cancer mortality in miners across Canada, France, the Czech Republic, and China, supporting the IARC classifications of uranium and radon progeny as Group 1 human carcinogens (International Agency for Research on Cancer, 2012; Tomášek et al., 2017). While the pattern has been suggestive for other cancer sites, including leukemia, renal cancer, and cancers of the upper aerodigestive tract, the confidence level is somewhat lower (Kreuzer et al., 2023).

Molecular epidemiology bridges exposure analysis, biomarker identification, and health outcome assessment through omics-based approaches to determine systems-level effects of uranium and radon exposure (Grison et al., 2022). Omics data can be integrated within the Adverse Outcome Pathway (AOP) framework to establish mechanistic links between molecular initiating events such as DNA damage and TP53 disruption, and their progression through cellular-level responses manifested during cancer development (Ankley et al., 2010). Uranium toxicity encompasses multiple mechanistic pathways including DNA damage, oxidative stress induction, epigenetic modifications, miRNA dysregulation, mitochondrial dysfunction, and metabolic perturbations (Ghosh et al., 2021; Guseva Canu et al., 2020). In summary, uranium exposure represents a complex chemical and radiological hazard that has been definitively confirmed as a lung cancer risk factor. Omics-based molecular analyses integrated within epidemiological frameworks possess the capacity to detect biomarkers indicative of dose-

response relationships and elucidate mechanisms underlying uranium-induced carcinogenesis (Salomaa et al., 2024).

Despite substantial progress in understanding uranium toxicity through single-omic methodologies, comprehensive integrative analyses synthesizing multiple omics layers remain limited. This review uniquely synthesizes multi-omics data linking uranium exposure to systemic carcinogenic pathways within a molecular epidemiology framework, providing a holistic perspective on molecular mechanisms and biomarker identification. The public health implications are particularly significant for uranium mining communities and populations exposed to uranium-contaminated water sources, where molecular biomarkers could enable early detection and targeted prevention strategies. By integrating genomic, epigenomic, transcriptomic, and metabolomic evidence, this review advances the field toward comprehensive risk assessment models capable of addressing both occupational and environmental exposure scenarios.

Method

This review paper adopts an integrative and narrative methodology to comprehensively synthesize and evaluate the current scientific evidence on the molecular epidemiology of uranium exposure, focusing particularly on the application of omics-based technologies in elucidating the pathways leading to carcinogenesis. The review systematically searched peer-reviewed publications across major databases, including PubMed, Scopus, ScienceDirect, and Web of Science, using a combination of keywords such as “uranium exposure,” “molecular epidemiology,” “omics,” “genomics,” “epigenetics,” “proteomics,” “metabolomics,” and “cancer risk.” The search encompassed literature published between 2000 and 2024, ensuring the inclusion of both foundational and recent studies relevant to uranium’s chemical and radiological toxicities. Studies were selected based on their direct investigation of molecular, cellular, or genomic alterations resulting from uranium or radon exposure, encompassing both human cohort analyses—such as those involving uranium miners and environmentally exposed populations—and experimental models that employed omics or biomarker-based methods.

Exclusion criteria removed studies lacking biological or molecular data, non-English publications without translation, and purely ecological assessments without health-related endpoints. From the selected studies, key information was extracted regarding study design, exposure quantification, omics platforms utilized, biological pathways affected, and health outcomes observed. The synthesis process categorized findings according to major omics domains—genomics, transcriptomics, epigenomics, proteomics, and metabolomics—allowing cross-comparison to identify converging evidence of uranium-induced molecular perturbations. This included alterations in DNA repair mechanisms, mutations in tumor-suppressor genes such as TP53 and KRAS, dysregulation of microRNAs, global DNA hypomethylation, and metabolic disturbances linked to oxidative stress and mitochondrial dysfunction.

A qualitative analytical framework was applied to interpret consistencies and divergences among findings while evaluating the biological plausibility and epidemiological relevance of each study. Recognizing methodological challenges such as limited sample sizes, heterogeneous exposure metrics, and confounding co-exposures (e.g., radon, silica, diesel exhaust), the review emphasizes the need for integrated multi-omics studies and longitudinal cohort investigations to better establish causality. Overall, this methodological approach enables a multidimensional understanding of uranium's toxicogenomic profile, providing a foundation for identifying biomarkers of exposure, susceptibility, and early disease, and advancing the development of improved cancer risk assessment and preventive strategies within the field of molecular epidemiology.

Results and Discussion

From the reviewed literatures, the major impact of uranium exposure on individuals as well as the community as a whole has become apparent, specifically within communities where individuals are directly involved in uranium mining operations (Guéguen et al., 2017). For those directly working in mines and their families, the risk of lung cancer transcends mere statistics—it represents a tangible health burden. In fact, individuals with greater long-term exposure to uranium and radon have been shown in genomic studies to harbor elevated frequencies of mutations in genes vital for protecting against cancer, like TP53 and KRAS, and exhibit unique mutational patterns such as the C>T transition at CpG dinucleotides, a signature associated with oxidative DNA damage (ICGC/TCGA Pan-Cancer Analysis of Whole Genomes Consortium, 2020; Kim et al., 2021). Another critical area of investigation encompasses transcriptomics and epigenomics of individuals exposed to uranium. Studies have demonstrated that uranium exposure alters cellular DNA repair mechanisms and modulates pathways involved in stress-induced apoptosis. Moreover, the epigenome has been found to exhibit characteristic alterations including widespread DNA hypomethylation and tumor suppressor gene promoter hypermethylation (Li et al., 2022; Schlomm et al., 2020).

Metabolomics analyses further reinforce these molecular findings, demonstrating perturbations in lipid metabolism, amino acid metabolic pathways, as well as elevated markers of oxidative stress in both experimental animal models and human cohorts (Anwar et al., 2021; Barhoumi et al., 2020). By integrating information obtained through multiple omics disciplines, researchers have successfully identified convergent biological pathways implicated in cancer initiation and progression following uranium exposure (Wang et al., 2023).

Molecular epidemiology studies provide valuable tools and biomarkers for risk assessment and early detection. Cytogenetic biomarkers such as elevated frequencies of micronuclei and chromosomal aberrations have been shown to correlate with cumulative exposure to uranium and subsequent cancer risk (Anwar et al., 2021; Guéguen et al., 2021; Vineis et al., 2020). Biomarkers such as these serve as molecular sentinel indicators for early diagnosis and

individualized risk assessment. However, large-scale prospective human studies remain necessary to definitively link molecular indices to long-term health consequences. In this context, biobanks play instrumental roles, including the German Uranium Miners' Biobank (GUMB), as well as Canadian uranium worker biobanks, which provide essential biospecimens and exposure data for molecular epidemiological investigations (Canadian Nuclear Safety Commission, 2023; Gomolka et al., 2022).

Despite the progress made, important knowledge gaps remain. Human studies have been limited by constraints including small sample sizes, inconsistent exposure assessment methodologies, and confounding factors such as concurrent exposure to radon, silica, and tobacco smoke (Grison et al., 2022). Current omics studies predominantly represent preclinical investigations or cross-sectional analyses, which preclude determination of temporal sequences from initial exposure through disease onset (Anson-Cartwright et al., 2021). Comprehensive multi-omics analyses integrating genomic, epigenomic, transcriptomic, and metabolomic data from human cohorts remain scarce. Mechanistic pathways mediating uranium-induced carcinogenesis continue to require further elucidation (Salomaa et al., 2024).

To advance the field, several research priorities should be emphasized. First, longitudinal cohort studies incorporating precise uranium exposure measurements are essential. Such studies should integrate comprehensive molecular profiling encompassing genome, epigenome, transcriptome, and metabolome analyses, alongside evaluation of susceptibility and effect biomarkers. Second, multi-omics integration offers opportunities to develop predictive biomarker panels that can be embedded in molecular risk models, enabling individualized exposure-risk stratification among uranium workers and environmentally exposed populations. Third, identifying molecular markers capable of predicting cancer risk at early stages would represent a paradigm shift in cancer detection and prevention strategies.

Future directions should prioritize artificial intelligence-based integration of multi-omics datasets to identify complex molecular signatures and interaction networks. Personalized exposure biomarkers derived from individual molecular profiles could enable precision medicine approaches in occupational health surveillance. Furthermore, omics-guided environmental health policies could incorporate molecular biomarker monitoring into regulatory frameworks for uranium exposure limits and worker protection standards. The development of pathway-based adverse outcome models linking molecular initiating events to adverse health outcomes would strengthen mechanistic understanding and improve quantitative risk assessment methodologies.

Visual integration of these findings reveals a clear pathway from uranium exposure through DNA damage and oxidative stress, progressing to mutations in critical genes such as TP53 and KRAS, epigenetic dysregulation, and ultimately to cancer development. This mechanistic sequence, supported by converging evidence across multiple omics platforms, underscores the

value of integrated molecular epidemiology in understanding and preventing uranium-induced carcinogenesis.

Conclusion

Uranium exposure poses significant health risks, especially for populations near uranium mines and processing sites, due to its combined chemical toxicity and radioactivity that induce DNA damage and increase lung cancer risk. Advances in omics technologies have deepened understanding of uranium's impact on genomic stability, gene expression, epigenetic regulation, and metabolism, with molecular epidemiology studies demonstrating strong links between uranium exposure and genetic damage leading to cancer. However, prospective human studies that integrate comprehensive omics data with precise exposure assessments remain limited. Future research should prioritize multi-omics longitudinal studies combining detailed exposure monitoring and biological markers to enhance risk assessment and public health strategies. Incorporating artificial intelligence and systems biology into data integration could drive development of personalized exposure biomarkers and precision prevention, ultimately enabling translation of molecular insights into practical risk models and evidence-based policies to protect occupational and environmental health from uranium-induced carcinogenesis.

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