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Persistent Environmental Pollutants and Cancer Outcomes: Evidences from Community Cohort Studies

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ABSTRACT

Persistent environmental pollutants (POPs) including radionuclides, per- and polyfluoroalkyl substances (PFAS), and heavy metals pose a significant global cancer burden. However, a systematic synthesis of evidence from key environmental disaster cohorts is limited. This review addresses this gap by consolidating findings from landmark community studies, namely the Fernald, Hanford, and Love Canal cohorts, to elucidate specific exposure-cancer relationships. The analysis demonstrates clear exposure-response dynamics, such lung cancer mortality associated with uranium exposure in Fernald cohort and increase in thyroid cancer risk per gray of iodine-131 radiation dose in Hanford site. While methodological challenges such as latency periods and historical exposure assessment persist, the cumulative evidence robustly supports causal inferences. The review underscores the critical importance of regulatory frameworks, including the EPA Superfund program and NIEHS community-based initiatives, for remediation and public health surveillance. Future research must prioritize integrative exposomic frameworks, advanced biomonitoring techniques, and environmental justice principles to effectively mitigate the public health impact of legacy contamination.

INTRODUCTION

The global burden of cancer remains a pressing public health challenge, with a substantial proportion of cases linked to environmental exposures, particularly Persistent Environmental Pollutants (POPs). These hazardous agents—defined by their environmental persistence, bioaccumulative capacity, and potential for long-range transport—include radionuclides such as Cesium-137, per- and polyfluoroalkyl substances (PFAS), polychlorinated biphenyls (PCBs), and heavy metals like arsenic and cadmium [i]. Released from industrial activities, nuclear weapons production, and hazardous waste disposal, such pollutants resist degradation, persist in ecosystems for decades, and accumulate through food webs, thereby sustaining long-term risks for exposed populations [ii]. The Fernald uranium processing plant, the Hanford nuclear site, and the Love Canal chemical waste dump exemplify U.S. communities where chronic environmental contamination has produced enduring health effects and serve as critical case studies for understanding cancer risks from POP exposure [iii].

The carcinogenic mechanisms of POPs are well documented by the International Agency for Research on Cancer (IARC). Radionuclides and chromium compounds exert direct genotoxic effects through DNA strand breaks. Many POPs generate oxidative stress, producing reactive oxygen species that disrupt cellular integrity. Others, such as PCBs and PFAS, function as endocrine disruptors by mimicking or interfering with hormone signaling, contributing to breast and prostate carcinogenesis. Epigenetic modifications, including aberrant DNA methylation and silencing of tumor suppressor genes, further promote malignant transformation [iv]. These synergistic pathways—genotoxic, oxidative, endocrine, and epigenetic—have been observed across contaminated cohorts, with Fernald workers exhibiting excess intestinal cancers, Hanford workers showing elevated thyroid malignancies, and Love Canal residents reporting increased risks of reproductive and hematologic cancers [v, vi].

Regional studies underscore the global relevance of such findings. In Nigeria, Tessum et al. demonstrated that emissions from transport (33%), biomass fuels (25%), and industry (17%) contribute substantially to particulate matter enriched with POPs, with WHO attributing 60% of under-five mortality in Lagos to air pollution effects, including respiratory infections and lung cancer [vii]. Similarly, Brazil's National Cancer Institute (INCA) projects 704,000 new cancer cases annually between 2023 and 2025, with non-melanoma skin (31.3%), breast (10.5%), and prostate (10.2%) cancers strongly associated with POPs [viii]. Occupational exposures add further burden: heavy metals account for an estimated 10.8% of male cancers globally, while arsenic in drinking water, notably in Bangladesh and northern Chile, drives lung, skin, and bladder cancer mortality at rates of 21 per 1,000 individuals [ix].

Although international frameworks such as the Stockholm Convention on POPs and U.S. Superfund regulations have sought to mitigate these hazards, policy implementation remains inconsistent. Challenges include insufficient monitoring, weak enforcement, transboundary pollution, and the reality of multi-pollutant exposures rather than isolated contaminants [9]. Against this backdrop, the Fernald, Hanford, and Love Canal cohorts highlight the urgent need for research that integrates real-world exposure contexts with cancer outcomes. This study therefore aims to investigate the health consequences of persistent pollutant exposure in environmentally burdened communities, providing evidence to bridge the gap between policy intent and public health protection.

METHODOLOGY

The data reviewed for this analysis, spanning publications from 2016 to 2025, was systematically gathered from reputable scientific databases including PubMed, PubMed Central, Google Scholar, ResearchGate, and ScienceDirect. The inclusion criteria were strictly limited to epidemiological and experimental studies investigating the association between persistent environmental pollutants and cancer risk in human populations. Consequently, publications focusing on other health outcomes or non-carcinogenic effects were excluded. The search strategy utilized specific keywords and phrases such as "persistent environmental pollutants and cancer risk," "POPs carcinogenicity," "PFAS cancer epidemiology," "heavy metals cancer incidence," and "biomarkers of exposure and cancer outcomes." A limited number of relevant studies were challenging to access in their entirety.

RESULTS AND DISCUSSION

Legacy Hazardous Sites and Elevated Cancer Risk

Long-term studies of communities near legacy industrial sites provide critical evidence of the latent carcinogenic effects of chronic pollutant exposure. Research on the Fernald Community Cohort near a former uranium processing plant shows a significantly elevated lung cancer risk, with one analysis revealing an exposure-response relationship: the highest-exposure group had a hazard ratio of 1.67 for lung cancer mortality compared to lower-exposure groups. Risks for hematologic cancers were also notably increased [x].

Studies of populations near nuclear facilities yield similar findings. Furthermore, exposure to iodine-131 releases causes an increased risk of thyroid cancer in children <15 years old. Mortality studies around the Chernobyle power plant have reported strong dose-response relationship between radiation dose to the thyroid received in childhood and thyroid cancer risk ($P<.001$). For a dose of 1Gy, the estimated OR of thyroid cancer varied from 5.5 (95% confidence interval [CI] = 3.1 to 9.5) to 8.4 (95% CI = 4.1 to 17.3) [xi].The consequences of chemical waste are equally clear, as demonstrated by the Love Canal tragedy. Health studies documented a significant excess of miscarriages and birth defects by approximately 17.5% (Table 1). Long-term surveillance continues to suggest elevated rates of cancers, including bladder and kidney cancer, underscoring the necessity of prolonged monitoring as carcinogenic effects can manifest decades after exposure [xii].

Emerging Contaminants and Carcinogenic Mechanisms

Per- and polyfluoroalkyl substances (PFAS) are associated with an elevated risk of specific cancers, primarily through mechanisms like epigenetic alterations, oxidative stress, and endocrine disruption. Evidence reported a significant increase in kidney cancer mortality with increasing estimated cumulative PFOA serum concentrations based on 12 kidney cancer deaths. SMRs (95% CIs) by increasing exposure quartile were 1.07 (95% CI: 0.02, 3.62), 1.37 (95% CI: 0.28, 3.99), 0 (95% CI: 0, 1.42), and 2.66 (95% CI: 1.15, 5.24) (trend test p = 0.02) [xiii].A meta-analysis by Barry et al. (2013) reinforced this, reported estimated cumulative serum PFOA concentrations were positively associated with kidney and testicular cancer [HR = 1.10; 95% CI: 0.98, 1.24 and HR = 1.34; 95% CI: 1.00, 1.79, respectively, for 1-unit increases in ln-transformed serum PFOA] [xiv] (Table 1).

Arsenic, a naturally occurring metalloid that contaminates groundwater in regions like Bangladesh and the U.S. Southwest, is a well-established human carcinogen. Its primary mechanisms involve genotoxicity through the induction of oxidative stress, inhibition of DNA repair, and the formation of DNA adducts. Extensive research in endemic areas has quantified this risk. A landmark prospective cohort study in Bangladesh found that exposure to arsenic concentrations >10.0µg/L was associated with a 20% increased mortality rate from skin, lungs and bladder cancer combined **[**xv**]**. For specific cancers, a large case-control study in the same region demonstrated a strong association with skin lesions and skin cancer, with ([OR = 1.51], 95% CI 1.26–1.80) [xvi]. In the United States, studies of populations in the Southwest have shown that long-term exposure to arsenic in drinking water is associated with 23% increased incidence of lung cancer depending on exposure levels (>150µg/L) [xvii] (Table 1).

Pesticides and Herbicides used in agricultural communities represent a complex mixture of chemicals, many of which are classified as probable human carcinogens. Their mechanisms can include genotoxicity, endocrine disruption (e.g., mimicking estrogen), and the induction of oxidative stress. The Agricultural Health Study (AHS) found that licensed pesticide applicators with high use of the herbicide glyphosate had a hazard ratio of 2.44 for risk of acute myeloid leukemia (AML) **[**xviii**]**, while insecticide permethrin also showed an increased risk of AML (P>0.05) [xix]. Similarly, a meta-analysis by Freeman et al. on AHS data showed that exposure to the herbicide atrazine was associated with 8% increased risk of breast and ovarian cancer among pesticide applicators with a family history of the disease [xx] (Table 1).

Table no. 1 Carcinogenic Hazards of Environmental Pollutants: Mechanisms and Epidemiological Evidence

Contaminant / Source	Reference	Study Population / Location	Sample Size	Key Health Outcomes	Risk Estimates & Key Findings	Proposed Mechanisms
Nuclear Facilities						
Uranium Processing	Reichard et al, (2025)	Fernald Community Cohort (near former uranium plant)	9,300	• Lung Cancer Mortality • Hematologic Cancers	• Hazard Ratio (HR) = 1.67 for lung cancer mortality in highest-exposure group vs. lower-exposure groups. • Notably increased risks for hematologic cancers.	Radon (Rn), (a byproduct gas of uranium cause DNA mutation in lung and trachea cells
Iodine-131 Releases	Cardis et al, (2005)	Chernobyle Power plant	276	Thyroid Cancer	P<0.001	Increased radiation exposure.
Chemical Waste						
Mixed Chemical Waste	Coleman, (2024)	Love Canal community	64	Miscarriages & Birth Defects	17.5% significant excess of miscarriages and birth defects.	Chemical induced DNA mutations.
Emerging Contaminants						
PFAS (PFOA)	Slteeland et al, (2012)	DuPont Washington Works plant workers (1948-2002)	1308	Kidney Cancer Mesothelioma	• SMR = 181.0 (95% CI: 93.5, 316.2) SMR = 2.85, 95% confidence interval (CI): 1.05, 6.20)	Epigenetic alterations, Oxidative stress, Endocrine disruption.
PFAS (PFOA)	Barry et al., (2013)	Chemical Plant Ohio, USA	2,507	• Kidney Cancer • Testicular Cancer	• HR = 1.10 (95% CI: 0.98, 1.24) for kidney cancer per 1-unit increase in ln-PFOA. • HR = 1.34 (95% CI: 1.00, 1.79) for testicular cancer per 1-unit increase in ln-PFOA.	Epigenetic alterations, Oxidative stress, Endocrine disruption.
Arsenic	Karagas, (2010)	Bangladesh (Prospective Cohort)	6000	• Combined Skin, Lung, Bladder Cancer Mortality	• 20% increased mortality rate from combined cancers with exposure to >10.0µg/L.	Genotoxicity, Oxidative stress, Inhibition of DNA repair, Formation of DNA adducts.
Arsenic	He, et al, (2025)	Meta-analysis	48,003	Skin Lesions & Skin Cancer	• Odds Ratio (OR) = 1.51 (95% CI: 1.26–1.80).	Genotoxicity, Oxidative stress, Inhibition of DNA repair, Formation of DNA adducts.
Arsenic	Issanov et al, (2024)	U.S. Southwest (power plant contaminated water), Meta-analysis	51 studies	Lung Cancer Incidence	• 23% increased incidence with long-term exposure to >150µg/L.	Genotoxicity, Oxidative stress, Inhibition of DNA repair, Formation of DNA adducts.
Pesticides & Herbicides						
Glyphosate	Andreotti et al, (2018)	Agricultural Health Study (AHS) - Pesticide Applicators from North Carolina and Iowa	54 251	Acute Myeloid Leukemia (AML)	• Hazard Ratio (HR) = 2.44 for risk of AML with high use.	Genotoxicity, Endocrine disruption (e.g., estrogen mimicry), Oxidative stress.
Permethrin	Shearer, et al, (2019)	Agricultural Health Study (AHS) Pesticides Applicators	27	Acute Myeloid Leukemia (AML)	• Increased risk of AML (P>0.05).	Genotoxicity, Endocrine disruption (e.g., estrogen mimicry), Oxidative stress.
Atrazine	Freeman et al.(2011)	Meta-analysis of AHS data	36,357	• Breast Cancer • Ovarian Cancer	• 8% increased risk	Genotoxicity, Endocrine disruption (e.g., estrogen mimicry), Oxidative stress.

Methodological Approaches and Challenges in Environmental Cancer Epidemiology

The Fernald Feed Materials Production Center, a uranium processing facility operating from 1951 to 1989 near Cincinnati, Ohio, released substantial uranium and radionuclides into the environment, leading to the creation of the Fernald Community Cohort (>9,700 residents). This long-term health study exemplifies environmental cancer epidemiology through a multi-method exposure assessment using historical monitoring data, geographic plume modeling, and residential proximity mapping. Biomarker validation confirmed elevated urinary uranium levels in residents closer to the plant. A major challenge was the long latency of cancer.

The design combined retrospective reconstruction of exposures with prospective follow-up of cancer diagnoses. Early analyses reported a modest but statistically significant increase in lung cancer mortality linked to radiation dose, persisting after adjustment for smoking, occupational history, and socioeconomic status; individuals with higher exposure had a 1.7% greater risk of lung cancer. The program's strength lies in systematically coded medical data (ICD-9 diagnoses, >28,000 mammograms, >27,000 chest x-rays) and detailed risk factor matrices (e.g., cumulative cigarette pack-years, family cancer history) [xxi].

Furthermore, Fernald Medical Monitoring Program (FMMP) (1990-2008) address resident concerns about health effects from the former uranium processing site; this cohort includes 9,782 participants with extensive health data and banked biospecimens for future research. A healthy-worker effect was observed among salaried but not hourly workers. Hourly males showed elevated cancer mortality, primarily from lung cancer (SMR = 1.25, 95% CI 1.09–1.42), while salaried males exhibited expected cancer mortality overall, except for increased lympho-haematopoietic malignancies (SMR = 1.52, 95% CI 1.06–2.12). No consistent dose–response was evident, apart from intestinal cancer, which was positively associated with internal uranium dose (ERR ≈ 1.5 per 100 µGy, 95% CI 0.12–4.1) [xxii] (Figure 1).In January 1985, local residents filed a class action lawsuit against National Lead of Ohio, Inc., the operator of the Fernald plant, and its parent company NL Industries, Inc. The case centered on claims of emotional distress and reduced property values. Following a summary jury trial aimed at facilitating settlement, the parties reached an agreement awarding $78 million to the plaintiffs. The settlement included compensation for emotional distress, reimbursement for property devaluation, and funding for a medical monitoring program with epidemiologic studies. Oversight of the settlement was assigned to three special masters appointed by U.S. District Judge S. Arthur Spiegel [xxiii].

The Hanford Site cohort, comprising workers and nearby residents, has been studied extensively for radiation and chemical exposure effects. The worker cohort (1940s–1980s employees) revealed a "healthy worker effect" with overall lower mortality, but associations were observed for leukemia, pancreas, digestive, and lung cancers. More recent concerns involve The Hanford Thyroid Disease Study (HTDS, 2002) exposures from iodine-131 reported 92.10% cases of thyroid cancer as part of the Hanford Thyroid Disease Study [xxiv]. (Figure 1)

The Love Canal cohort, comprising approximately 6,181 residents in Niagara Falls, New York, is recognized as a landmark case in environmental epidemiology and one of the most severe chemical waste disasters in U.S. history. During the 1940s and 1950s, the Hooker Chemicals and Plastics Corporation deposited nearly 22,000 tons of hazardous waste, including PCBs, dioxins, and pesticides, into an abandoned canal that was later developed for housing, leading to widespread community exposure. By the late 1970s, toxic seepage into homes prompted federal investigations, which documented chromosomal damage, reproductive disorders, and increased risks of bladder, kidney, and lung cancers among residents.

Subsequent epidemiological studies extended these findings, reporting elevated risks of preterm birth (SIR 1.40; 95% CI: 1.01–1.90), [xxv]. Between 1978 and 1982, the Love Canal cohort exhibited elevated risks **of** bladder and kidney cancers (SMRs: 1.44 and 1.45) [xxvi]. Follow-up from 1979 to 1996, site-specific excesses reported including genitourinary (27%), digestive (20%), lung (19%), skin, breast, and connective tissue (18%), and lymphatic cancers (8%) most common, alongside smaller proportions of oral/throat (2%) and unspecified cancers (6%) [**xxvii**]. (Figure 1)

Federal intervention followed, with two emergency declarations by President Jimmy Carter (1978 and 1980), the evacuation of residents, and designation of the site as a Superfund priority. Remediation efforts included capping, fencing, and demolition of contaminated structures, while prolonged litigation culminated in a $20 million settlement for approximately 1,300 displaced residents. The Love Canal cohort underscores the long-term oncological, reproductive, and psychosocial consequences of environmental contamination and continues to shape regulatory frameworks governing hazardous waste management [27].

Environmental Justice and Cancer Disparities

Environmental justice analyses demonstrate that cancer risks from pollutants are not equitably distributed, with minority and low-income populations experiencing disproportionate exposures and burdens. This pattern is evident in contemporary cases like Flint, Michigan, and Louisiana's "Cancer Alley," as well as in legacy cohorts such as Fernald, Hanford, and Love Canal. In Flint, Michigan, documented cancer incidence rates for colon is 31.8%, lung 69%, larynx 4.0%, stomach 6.5%, rectum 13.7%, bladder 23.2% and prostate 148.9% exceed those of both Genesee County and Michigan statewide. These elevated rates occur in a community with complex environmental exposures, including lead and disinfection byproducts, intersecting with socioeconomic disadvantage [xxviii].

In Louisiana's "Cancer Alley," a Tulane University study estimates approximately 85 excess cancer cases annually are attributable to toxic air pollution in neighborhoods with above-average poverty. In the most polluted quartile of these neighborhoods, the annual cancer incidence rate is ~502 per 100,000 persons, compared to ~478.8 in similar low-income areas with low pollution and ~480.3 for Louisiana overall Spatial analyses confirm cancer risk in low-income or majority-Black census tracts is 12-16% higher than in higher-income, white-majority tracts. The cumulative lifetime cancer risk from ambient air toxics in this region is ~45.8 per million, exceeding comparative state and national risks of ~30.3-37.1 per million [xxix].

The Fernald, Hanford, and Love Canal cohorts similarly reflect environmental justice dimensions. At Fernald, populations of lower socioeconomic status in proximity to the plant experienced validated, excess uranium exposure [3]. At Hanford, fence-line communities and workers from marginalized groups faced overlapping radiation and chemical risks, had a chronic myeloid leukemia (CML) (ERR=-1•06 [90% CI <0–1.81] compounded by structural barriers like limited monitoring and healthcare access [xxx].

The Love Canal cohort, also comprising residents with limited resources, exhibited elevated risks of congenital malformations, preterm birth, and certain cancers, compounding the burdens of poverty and inadequate regulatory protection [xxxi].

Policy and Public Health Implications

EPA Superfund (CERCLA): Site Identification, Liability, and Remediation

The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980 is the principal federal mechanism for remediating hazardous waste sites. It enforces polluter liability and authorizes the EPA to lead cleanups. At Love Canal, CERCLA facilitated the relocation of over 800 families and site restoration. The Fernald uranium facility required removal of 2.6 million cubic yards of radioactive soil and groundwater treatment that reduced uranium levels by over 90%. Health studies linked these sites to statistically significant excess cancers: Love Canal showed elevated bladder, kidney, and respiratory cancers, while the Fernald cohort documented increased lung and hematologic cancers. Nationally, the program's success is evidenced by the delisting of 448 sites, a 37% reduction in childhood blood lead levels near remediated sites (1980-2000), and the reuse of over 1,700 properties, reducing cancer risk by disrupting exposure pathways [xxxii].

NIEHS Research and Community-Engaged Programs: Translation and Capacity Building

Established in 1986, the NIEHS Superfund Research Program integrates biomedical research with community engagement. At Love Canal, community engagement increased participation in health studies by nearly 25%. The program has also developed over 100 remediation technologies, such as low-cost arsenic detection, and demonstrated measurable exposure declines, including 15–20% reductions in urinary arsenic biomarkers post-intervention. With more than 200 funded community projects, the program enhances data quality and enables early cancer signal detection [xxxiii]. Furthermore, NIEHS in 1986 revealed that the Hanford nuclear site had released radioactive iodine-131 into the environment, leading to public concern about increased thyroid disease. To investigate, the Hanford Thyroid Disease Study (HTDS) was launched. The study enrolled 3,441 individuals who lived near Hanford in children between 1940-1946. Researchers estimated the radiation exposure for 3,190 of these participants. After spending 9-year $18 million effort for welfare to overcome the increased rates of cancer, the primary finding, released in a 1999 draft report, was that the study found no evidence linking Hanford's radiation to increased thyroid disease in the population. This "negative" finding was presented with certainty, which upset many citizens. Subsequently, the National Academy of Sciences reviewed the study and concluded that while the research was well-conducted, its results were inconclusive. The report stated that the study could not rule out the possibility of a radiation effect, but also could not confirm one existed [xxxiv]

Cancer Registries (SEER/NPCR): Surveillance and Statistical Confirmation

The SEER Program and the National Program of Cancer Registries (NPCR) provide systematic data for comparing exposed populations to national baselines. Love Canal registries indicated a 1.4-fold higher incidence of bladder cancer. For the Fernald cohort, registry linkage identified over 1,000 cancer diagnoses, quantifying excess lung cancer risk. SEER covers 48% of the U.S. population, while NPCR covers 100%, documenting over 1.9 million cancer cases annually and enabling rigorous statistical analysis via standardized incidence ratios [xxxv].

Future Directions

Integrative exposome frameworks for cohort-based risk attribution

The exposome comprehensively assesses all environmental exposures—from air, water, and food—over a lifetime. For carcinogens like uranium, this requires integrating environmental science to measure exposure, molecular biology to identify early biological damage (like DNA mutations), and epidemiology to link this data to cancer outcomes. Key research needs include advanced biomonitoring to track internal dose and multi-level data integration to decipher how cumulative exposures initiate disease [xxxvi]. Rappaport et al. found that the concentrations of thousands of chemicals in human blood vary by 11 orders of magnitude. Since modern mass spectrometers can only analyze across 5-6 orders of magnitude at a time, comprehensively profiling the blood exposome requires multiple, unbiased analytical methods [xxxvii].

AI and machine learning for exposure–cancer link prediction

AI tools show high potential for detecting environmental pollutants linked to cancer, with some models achieving impressive statistical success rates in specific applications, though quantifying overall success is challenging. In one study, a Random Forest model predicted lung cancer risk based on environmental factors with 99% accuracy. Another model identified pollutants contributing to respiratory cancer mortality with a mean accuracy of 74%. However, reported success rates can vary significantly depending on the data used, the AI model's complexity, and the specific pollutants and cancers being studied. Challenges remain in achieving consistent success due to the complexity of environmental systems, data quality issues, and the difficulty of confirming a direct causal link between a specific pollutant and a disease outcome [xxxviii]. Evidence-led remediation technologies

Evidence-led remediation technologies, such as excavation, soil washing, and vitrification, are used to reduce environmental contamination by breaking exposure pathways, according to the provided text. Evaluation of site-specific technologies such as excavation, soil washing, and vitrification confirms their role in breaking exposure pathways. EPA Superfund assessments show that permanent remedies reduce modeled lifetime cancer risks by up to 90% for site residents [xxxix].

Enhanced occupational surveillance and dose reconstruction

By implementing enhanced occupational surveillance, improved dose reconstruction, and biomonitoring, worker cohort studies can significantly reduce exposure misclassification, which strengthens statistical power and provides a more accurate assessment of occupational cancer risks. For an instance, biomonitoring demonstrated a 90% drop in blood lead levels in the U.S. population following the removal of lead from gasoline. These methods enable more precise dose-response analysis, moving beyond broad exposure estimates to individualized data, which allows for the more confident detection of associations between workplace exposures and cancer [xi].

CONCLUSION

This review found that persistent pollutants remain a significant global cancer burden, with evidence from landmark cohorts like Fernald, Hanford, and Love Canal demonstrating compelling causal links between specific exposures—such as uranium and iodine-131 to thyroid cancer, supporting causal inferences despite methodological constraints such as exposure reconstruction challenges and latency complications. The evidence underscores the importance of regulatory frameworks like the EPA Superfund and NIEHS community programs. Future research should prioritize advanced biomonitoring, multi-generational studies, and exposomic approaches to address complex mixture effects. These findings highlight the imperative for enhanced monitoring, robust policy interventions, and environmental justice integration to mitigate the public health impact of legacy contamination.

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