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Combined Effects of Metals and PFAS on Diabetes

Diabetes is a major global public health challenge, and increasing evidence suggests that environmental contaminants may contribute to its development alongside traditional risk factors such as diet, obesity, and physical inactivity.

Heavy metals and per- and polyfluoroalkyl substances (PFAS) have been implicated in metabolic dysfunction through mechanisms involving oxidative stress, inflammation, endocrine disruption, impaired glucose homeostasis, and pancreatic β-cell dysfunction. However, humans are exposed to these contaminants as complex mixtures rather than as individual chemicals.

Previous studies have largely evaluated single pollutants, limiting understanding of the combined effects of environmental exposures on diabetes risk.

The present study aimed to investigate the combined associations of serum PFAS compounds: perfluorooctanoic acid. (PFOA) and perfluorooctane sulfonate (PFOS), and blood metals (lead, cadmium, and mercury) with prevalent diabetes among U.S. adults.

Specific objectives included evaluating individual and joint exposure effects, identifying key contributors within the exposure mixture, assessing nonlinear exposure-response relationships, and comparing findings across multiple mixture-analysis approaches.

Data were obtained from 1,648 adults participating in the 2017-2018 National Health and Nutrition Examination Survey (NHANES) who had direct measurements of all five environmental biomarkers.

Diabetes status was based primarily on self-reported physician diagnosis. Survey-weighted logistic regression was used as the primary analysis while accounting for NHANES complex sampling design. Complementary mixture-analysis methods included Bayesian Kernel Machine Regression (BKMR), Weighted Quantile Sum (WQS) regression, and quantile g-computation.

The survey-weighted prevalence of diabetes was 11.4%. Logistic regression showed significant inverse associations between blood lead and cadmium with diabetes, while PFOS, PFOA, and mercury were not significantly associated.

BKMR identified lead and PFOS as the most influential contributors to the exposure-response surface. Exposure-response relationships for lead and cadmium were nonlinear and non-monotonic, indicating that conventional linear models may not adequately capture their effects. WQS regression and quantile g-computation consistently showed an overall inverse association between the environmental mixture and diabetes.

The findings highlight the importance of evaluating environmental chemicals as mixtures and demonstrate that mixture-based methods can reveal complex patterns that may be overlooked by traditional regression analyses. However, the cross-sectional design precludes causal inference, and observed inverse associations may reflect reverse causation, residual confounding, or survivor bias rather than protective effects.

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Prenatal PFAS Exposure and Offspring Health Outcomes

Per- and polyfluoroalkyl substances (PFAS) are a large group of persistent synthetic chemicals widely used in industrial processes and consumer products because of their water- and oil-repellent properties. Increasing evidence indicates that several PFAS compounds, particularly legacy compounds such as perfluorooctanoic acid (PFOA), perfluorooctane sulfonate (PFOS), perfluorohexane sulfonate (PFHxS), and perfluorononanoic acid (PFNA), can cross the placental barrier and expose the developing fetus during critical windows of development.

The Developmental Origins of Health and Disease (DOHaD) framework suggests that prenatal environmental exposures may alter developmental programming and influence disease susceptibility throughout life. This review evaluates current evidence linking prenatal PFAS exposure to offspring health outcomes and considers implications for developmental and intergenerational risk assessment.

The review aims to provide an evidence-based synthesis of the health effects of prenatal PFAS exposure on offspring. It seeks to evaluate epidemiological evidence across immune, metabolic, neurodevelopmental, endocrine, hepatic, renal, and cardiovascular domains; distinguish outcomes supported by strong versus limited evidence; summarize mechanistic pathways involved in developmental toxicity; assess challenges associated with emerging PFAS substitutes and mixture exposures; and propose a conceptual framework for developmental hazard assessment and future risk evaluation.

The strongest human evidence links prenatal PFAS exposure with impaired immune function and altered growth and metabolic outcomes. Consistent associations were observed between prenatal exposure and reduced vaccine antibody responses, increased susceptibility to infections, lower birth weight, accelerated childhood weight gain, and greater risk of obesity.

Evidence for neurodevelopmental effects, including reduced cognitive performance and attention-deficit/hyperactivity disorder-related behaviors, was suggestive but less consistent. Similarly, endocrine effects involving thyroid hormone disruption and altered pubertal development were supported by moderate evidence.

Emerging findings also indicate potential impacts on liver function, blood pressure regulation, renal health, and cellular aging, although these outcomes require further confirmation. Mechanistically, PFAS may affect offspring health through placental transfer, receptor-mediated signaling, thyroid hormone transport interference, oxidative stress, inflammation, immune dysregulation, mitochondrial dysfunction, and epigenetic reprogramming. However, substantial uncertainties remain due to differences in exposure assessment, co-exposures, sex-specific susceptibility, compound-specific toxico-kinetics, and limited long-term follow-up.

Future research should prioritize repeated exposure measurements throughout pregnancy, long-term birth cohort follow-up, and improved characterization of emerging PFAS alternatives. Greater integration of human-relevant experimental systems, multi-omics technologies, mechanistic biomarkers, and physiologically based pharmacokinetic/toxicokinetic (PBPK/PBTK) modeling is needed to improve causal inference and estimate fetal internal doses. Research should also address real-world mixture exposures and identify developmental windows of heightened vulnerability. Regulatory frameworks should incorporate developmental and intergenerational considerations, particularly for pregnant women and infants.

In conclusion, prenatal PFAS exposure is a significant developmental health concern, with the strongest evidence linking exposure to impaired immune function and adverse growth-related outcomes. Although evidence for neurodevelopmental, endocrine, and other health effects is still emerging, biological mechanisms support potential long-term consequences. Future risk assessments should incorporate developmental susceptibility and lifelong health impacts to better protect vulnerable populations.

Source: Frontiers in Public Health, Vol. 14, Article 1892134, July 2026.

Combined Effects of Metals and PFAS on Diabetes

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Future research should employ prospective cohort studies with repeated exposure measurements to establish temporal relationships and causality. Larger studies are needed to better characterize nonlinear dose-response relationships and interactions among contaminants.

Further methodological development is also required to improve convergence and implementation of survey-weighted mixture models such as design-aware BKMR. Mechanistic studies should investigate biological pathways linking PFAS-metal mixtures with diabetes development.

This study demonstrates that associations between environmental contaminants and diabetes are complex, nonlinear, and best evaluated within a mixture framework. Lead and PFOS emerged as the most influential mixture components, while lead and cadmium showed significant associations in survey-weighted analyses. Consistent findings across BKMR, WQS regression, and quantile g-computation support the importance of mixture-based approaches for environmental health research. Although causal relationships cannot be established, the results underscore the need to consider concurrent exposures when evaluating environmental determinants of diabetes.

Source: Journal of Xenobiotics, Vol. 16, Issue 4, Article 128, July 2026.

From Animal Models to NAMs: A Shift in Developmental Immunotoxicity Testing

Developmental immunotoxicity (DIT) has emerged as a critical field in toxicology because the developing immune system is substantially more sensitive to chemical exposures than the mature immune system. Immune development begins during early embryogenesis and continues through fetal life, infancy, childhood, and adolescence, involving tightly regulated processes of hematopoiesis, immune organ formation, and immune cell maturation.

Disruptions during these sensitive developmental windows can result in long-term alterations in immune competence, increased susceptibility to infections, allergies, autoimmune diseases, and impaired responses to vaccination. Growing evidence from human epidemiological studies and experimental research has highlighted the need for improved approaches to identify chemicals capable of disrupting immune development.

Traditional animal-based testing methods provide valuable information but face ethical, logistical, and translational limitations, driving the transition toward human-relevant New Approach Methodologies (NAMs).

This narrative review provides a comprehensive overview of human immune system development, critical windows of susceptibility to developmental immunotoxicants, and current evidence linking environmental, pharmaceutical, and dietary exposures to immune disruption. It also evaluates advances in new approach methodologies (NAMs), including physiological mapping, multi-omics technologies, epidemiological research, and advanced human-relevant *in vitro* models, for improving developmental immunotoxicity (DIT) assessment and supporting regulatory decision-making.

The review synthesizes evidence from published human and animal studies, *in vitro* and stem cell-based models, organoids, organ-on-chip platforms, zebrafish assays, and high-dimensional immune profiling technologies. Particular emphasis is placed on mechanistic insights and the development of Integrated Approaches to Testing and Assessment (IATA) based on NAMs to facilitate the replacement of animal testing.

The review demonstrated that immune development occurs through sequential stages involving the yolk sac, fetal liver, bone marrow, thymus, spleen, and lymph nodes, each representing potential windows of vulnerability.

Epidemiological and experimental evidence identified several developmental immunotoxicants, including PFAS, heavy metals, air pollutants, glucocorticoids, chemotherapeutics, bisphenol A, phthalates, and nutritional imbalances. These exposures were associated with impaired vaccine responses, altered lymphocyte development, dysregulated immune programming, and increased disease susceptibility later in life. Advances in single-cell transcriptomics have improved understanding of human immune ontogeny, while physiological maps such as the Human Immune System Development Map provide mechanistic frameworks for identifying key developmental events and adverse outcome pathways.

Emerging NAMs, including stem cell-derived immune models, zebrafish assays, organoids, and microphysiological systems, offer promising alternatives to animal testing by providing more human-relevant mechanistic data.

However, no single assay can adequately capture the complexity of immune development, emphasizing the need for integrated test batteries and complementary approaches.

Future progress requires comprehensive human reference datasets covering prenatal and postnatal immune development, expanded use of single-cell and multi-omics technologies, improved physiological maps, and validation of mechanistically relevant NAMs.

Standardization of protocols, establishment of performance criteria, development of internationally accepted test batteries, and stronger integration of epidemiological data with experimental models are essential.

Collaborative efforts among academia, regulatory agencies, and international organizations will be necessary to facilitate regulatory acceptance and implementation of NAM-based DIT assessment frameworks.

The field of developmental immunotoxicology is undergoing a paradigm shift from reliance on animal models toward human-relevant NAMs. Advances in immune system mapping, stem cell technologies, organoids, organ-on-chip systems, and systems immunology provide unprecedented opportunities to understand and predict the effects of chemicals on immune development.

Despite significant progress, important challenges remain, including limited human reference data, insufficient standardization, and the lack of validated test batteries. Addressing these gaps will enable the development of predictive, mechanistically informed, and ethically sustainable approaches for assessing developmental immunotoxicity and protecting human health, particularly during vulnerable stages of life.

Source: Archives of Toxicology, Vol. 100, Pages 3819-3840, September 2026.

Prenatal BTEX Exposure and the Risk of Preterm Birth

Preterm birth (PTB), defined as delivery before 37 completed weeks of gestation, remains a major contributor to neonatal morbidity and mortality worldwide. Environmental exposures during pregnancy have emerged as important determinants of adverse birth outcomes, with increasing attention directed toward air pollutants. Among these, benzene, toluene, ethylbenzene, and xylene (BTEX) are ubiquitous volatile organic compounds generated from traffic emissions, industrial activities, and fuel combustion. Despite growing concerns regarding their reproductive toxicity, epidemiological evidence linking prenatal BTEX exposure to preterm birth remains limited, particularly in large population-based settings.

The present study aimed to examine the extent to which prenatal exposure to ambient BTEX compounds contributes to variation in gestational duration and the risk of preterm birth. Furthermore, the investigation sought to characterize sensitive windows of exposure during pregnancy and to identify populations that may be disproportionately vulnerable to the adverse effects of BTEX. To address these objectives, a nationwide retrospective birth cohort study was conducted using data from 4,321,599 singleton live births enrolled in the National Free Preconception Health Examination Project between 2010 and 2015 across 337 Chinese cities.

The study demonstrated that higher prenatal exposure to BTEX was significantly associated with both reduced gestational duration and an increased risk of preterm birth. Among the individual compounds, toluene exhibited the strongest association with adverse birth outcomes. The findings further indicated that exposure during late pregnancy, particularly the third trimester, represented the most sensitive period for the occurrence of preterm birth.

Subgroup analyses suggested greater susceptibility among mothers with lower socioeconomic status, overweight or obesity, and among pregnancies involving male fetuses. These observations highlight potential interactions between environmental exposures and biological or social determinants of health. The consistency of the findings after adjustment for other common air pollutants supports the independent contribution of BTEX exposure to adverse pregnancy outcomes.

Several biological mechanisms may explain these associations. Experimental and epidemiological evidence suggests that BTEX compounds can induce oxidative stress, systemic inflammation, immune dysregulation, and placental dysfunction, all of which may contribute to premature initiation of labor. Collectively, the findings strengthen the growing body of evidence linking exposure to volatile organic compounds with impaired reproductive health.

Future research should focus on improving individual-level exposure assessment, identifying precise windows of vulnerability throughout gestation, and elucidating the biological mechanisms underlying BTEX-induced reproductive toxicity. Further studies are also needed to distinguish between different subtypes of preterm birth and to evaluate the effectiveness of public health interventions aimed at reducing maternal exposure to BTEX.

This large-scale cohort study provides robust evidence that prenatal exposure to BTEX is associated with shortened gestational duration and an elevated risk of preterm birth. The results identify late pregnancy as a particularly sensitive exposure window and highlight vulnerable populations that may require targeted protection. Reducing ambient BTEX pollution should therefore be considered an important strategy for improving maternal and infant health outcomes.

Source: Environmental Science & Technology, Vol. 60, Issue 31, Pages 21429-21439, August 2026.

Long-term Air Pollution Exposure and Hyperuricemia

Hyperuricemia, characterized by elevated serum uric acid concentrations, is an increasingly important public health concern because it contributes to the development of gout, chronic kidney disease, hypertension, cardiovascular disease, and metabolic disorders. Although genetic susceptibility, diet, alcohol consumption, and lifestyle factors are well-established determinants of serum uric acid levels, growing evidence suggests that environmental exposures may also influence disease risk.

Ambient air pollution has been linked to systemic inflammation, oxidative stress, and metabolic dysfunction, all of which may affect uric acid metabolism. However, evidence regarding the association between long-term air pollution exposure and hyperuricemia in the general population remains limited, particularly in areas with relatively low pollution levels.

This study aimed to investigate whether long-term exposure to major ambient air pollutants, including particulate matter ≤2.5 μm ($PM_{2.5}$), particulate matter ≤10 μm (PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and carbon monoxide (CO), is associated with elevated serum uric acid concentrations and an increased risk of hyperuricemia independent of renal function and other established risk factors.

A cross-sectional analysis was conducted using data from 20,117 adults who underwent routine health examinations between 2015 and 2019. Annual average concentrations of $PM_{2.5}$, PM_{10}, NO_2, SO_2, and CO were estimated using the Community Multiscale Air Quality modeling system and assigned according to participants' residential addresses. Hyperuricemia was defined as serum uric acid levels exceeding 7.0 mg/dL in men and 6.0 mg/dL in women.

Hyperuricemia was identified in 15.3% of participants. Long-term exposure to all five air pollutants was significantly associated with an increased risk of

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From Personal Monitoring to Annual Exposure: Estimating Outdoor Air Pollution Exposure

Long-term epidemiological studies of air pollution commonly rely on ambient monitoring data, whereas personal exposure studies often collect only short-term measurements. This creates a disconnect between estimates of individual exposure and assessments of chronic health effects. Furthermore, personal exposure includes contributions from both indoor and outdoor pollution sources, whereas most epidemiological evidence and air quality guidelines are based on outdoor air pollution.

This study aimed to develop and evaluate a framework for estimating annual personal exposure to outdoor-origin air pollutants using short-term personal monitoring data. The analysis was based on 12,563 person-days of exposure measurements collected from 337 participants across four London-based studies conducted between 2015 and 2019, encompassing diverse demographic and occupational groups, including older adults with chronic obstructive pulmonary disease, schoolchildren and teachers, professional drivers, and healthy adults.

Personal exposure measurements were first apportioned into outdoor- and indoor-generated components using established analytical approaches. Annual exposure estimates for $PM_{2.5}$, NO_2, O_3, and black carbon (BC) were subsequently derived by integrating hourly ambient pollutant concentrations, pollutant-specific infiltration efficiencies, microenvironmental adjustment factors, and individual time-activity patterns.

Annual exposure estimates derived from the extrapolation framework were generally consistent with measurements obtained during monitoring campaigns, with stronger agreement observed when longer monitoring periods were available.

Comparisons across exposure metrics showed that conventionally assigned ambient concentrations, particularly those based on nearest-monitor approaches, tended to exceed both matched-scaled ambient estimates and personal exposure attributable to outdoor sources. This pattern was most pronounced for gaseous pollutants, including NO_2 and O_3

In contrast, infiltrated ambient pollution more closely reflected personal exposure, especially among individuals who spent the majority of their time indoors. These findings underscore the potential for exposure misclassification when ambient monitoring data are used as direct surrogates for personal exposure and highlight the importance of accounting for microenvironmental and behavioral factors in exposure assessment.

Further research is needed to evaluate the robustness and generalizability of the extrapolation framework using long-term datasets that include concurrent ambient, indoor, outdoor, and personal monitoring measurements. The integration of mobility and behavioral information, such as GPS tracking, activity diaries, and smartphone-based data collection, may improve the characterization of individual exposure patterns.

Additional studies should also investigate seasonal variation in pollutant infiltration, assess alternative monitoring strategies using repeated short-term campaigns across multiple seasons, and better characterize indoor sources contributing to overall exposure.

In conclusion, this study introduces a novel approach for estimating long-term personal exposure to outdoor-generated air pollutants from short-term monitoring data. By incorporating temporal, spatial, behavioral, and microenvironmental determinants, the framework advances current exposure assessment methods and strengthens the linkage between personal exposure science and environmental epidemiology. Although additional validation is warranted, the proposed methodology offers a promising tool for improving the accuracy of exposure estimation and enhancing future assessments of air pollution-related health risks.

Source: Environment International, Vol. 214, Article 110414, August 2026.

Long-term Air Pollution Exposure and Hyperuricemia

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hyperuricemia, with the strongest associations observed for PM_{10} and SO_2. Higher exposures to $PM_{2.5}$, PM_{10}, NO_2, and SO_2 were also associated with elevated serum uric acid concentrations. The associations were particularly pronounced among smokers, older adults, and individuals with dyslipidemia or central obesity.

These findings support the hypothesis that air pollution contributes to hyperuricemia through oxidative stress, inflammation, and metabolic disturbances that enhance uric acid production. Because the associations remained significant after adjustment for renal function, the observed effects appear to extend beyond impaired uric acid excretion. The results further suggest that even relatively low levels of air pollution may adversely affect metabolic health.

Future studies should employ longitudinal designs and more refined exposure assessment methods that incorporate personal activity patterns, indoor environments, and other influential factors. Research in diverse populations and settings is also needed to strengthen the evidence and improve generalizability.

This study expands current understanding of the health impacts of air pollution by highlighting its potential role in hyperuricemia, a metabolic condition that has received relatively limited attention in environmental health research. These findings support the inclusion of metabolic outcomes in air pollution risk assessments and highlight the importance of policies that reduce population exposure and the burden of chronic diseases.

Source: Clinical Rheumatology, Vol. 45, Pages 6927-6936, September 2026.

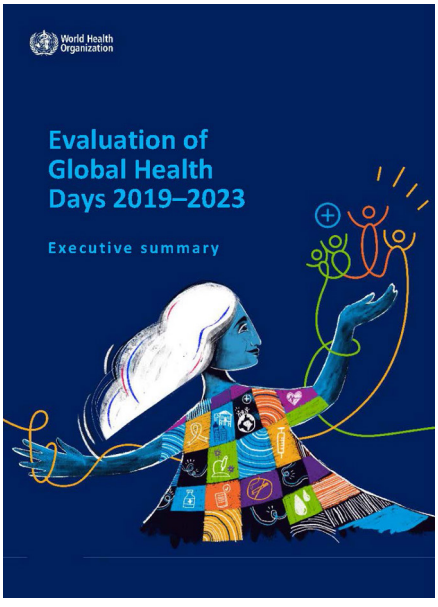
WHO Evaluation of Global Health Days 2019–2023: Executive Summary

The World Health Organization (WHO) conducts Global Health Days and Weeks as a core component of its mandate to raise awareness of priority health issues and mobilize action at global, regional, and national levels. These campaigns, some mandated by the World Health Assembly or United Nations bodies and others initiated by non-state actors, aim to promote advocacy, stakeholder engagement, and collective action on public health priorities.

The **Evaluation of Global Health Days 2019-2023** was undertaken to support both accountability and organizational learning by assessing WHO's processes for planning and managing these campaigns, evaluating their contribution to observable changes between 2019 and 2024, and identifying opportunities to strengthen coordination, measurement, and learning.

The evaluation found that Global Health Days remained highly relevant and well aligned with WHO strategic priorities, the Sustainable Development Goal 3, and the Triple Billion targets. Campaigns effectively increased awareness, fostered partnerships, stimulated stakeholder engagement, and supported policy advocacy across multiple health areas. However, their overall effectiveness was limited by broad objectives, insufficiently defined target audiences, uneven coordination, and resource constraints.

Monitoring systems predominantly measured outputs such as media reach and engagement rather than changes in behavior, policy, or health outcomes, limiting evidence of long-term impact. The evaluation recommends a more strategic, results-oriented approach, including stronger campaign prioritization, improved monitoring and evaluation frameworks, enhanced partnerships, and greater investment in country-level implementation capacity to improve sustainability and impact.



Source: WHO Publications. Evaluation of Global Health Days 2019-2023: Executive Summary, August 2026.

Multi-Scenario Assessment of PAH Exposure and Cancer Risks in China

Polycyclic aromatic hydrocarbons (PAHs) are ubiquitous environmental contaminants generated primarily from incomplete combustion processes, including industrial activities, vehicle emissions, fossil fuel combustion, biomass burning, and petroleum-related operations. Once released, PAHs can accumulate in multiple environmental media such as air, water, soil, dust, and food, creating numerous pathways for human exposure. Because many PAHs possess carcinogenic, mutagenic, and toxic properties, they are recognized as priority pollutants by environmental regulatory agencies.

Although numerous studies have investigated PAH contamination in specific media, most have focused on single exposure routes, which may not accurately reflect real-world exposure conditions.

This study addressed this gap by developing an integrated multi-media and multi-pathway framework to evaluate human exposure and carcinogenic risks associated with PAHs across industrial, urban, and rural environments in China. The research also sought to evaluate uncertainty and variability in risk estimates through probabilistic modeling.

A systematic review of literature published between 2010 and 2024 identified 138 eligible studies reporting PAH concentrations in soil, water, air, dust, and food across China.

Exposure assessment included dietary ingestion, inhalation, soil and dust ingestion, and dermal contact with soil, dust, and water.

Average Daily Doses (ADDs) were calculated separately for adults and children using exposure parameters specific to the Chinese population.

Carcinogenic risks were estimated using benzo[a]pyrene equivalent concentrations (BaPeq) and Incremental Lifetime Cancer Risk (ILCR).

PAH concentrations varied substantially among environmental media and scenarios. Industrial areas exhibited the highest concentrations in air and water, while urban areas showed elevated levels in soil and dust. Rural areas generally had lower contamination levels.

Across all scenarios and age groups, dietary intake was the dominant exposure pathway, contributing 67% to 97% of total PAH exposure. Children consistently experienced higher exposure levels than adults because of lower body weight and greater relative contact with contaminated media.

Carcinogenic risk followed the order industrial > urban > rural, with children exhibiting higher risks than adults. Total ILCR values exceeded the commonly accepted threshold of 10^{-4} in industrial adults, industrial children, and urban children, indicating potential health concerns. These findings emphasize the

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The Chem HelpDesk

"Strengthening capabilities for sound chemicals management"

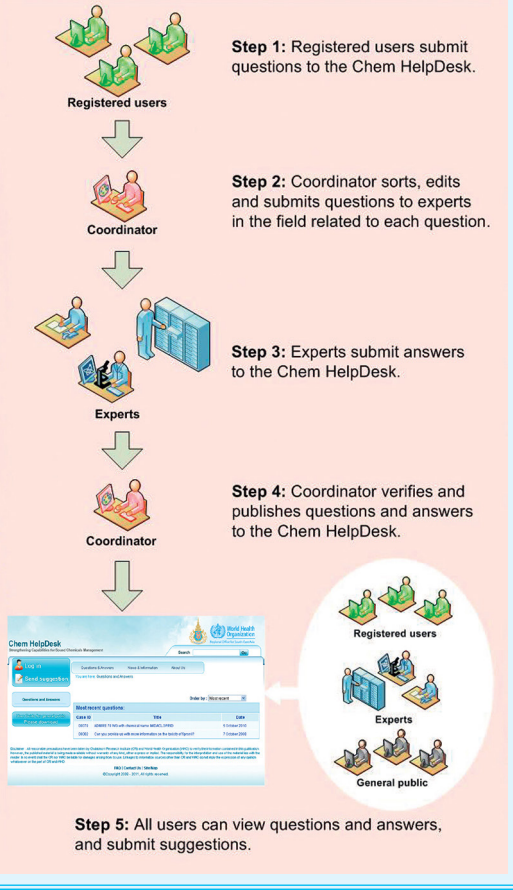
The Regional HelpDesk for Chemical Safety, or **Chem HelpDesk** was established as a joint-initiative between WHO SEARO and CRI, through the WHO Collaborating Center for Capacity Building and Research in Environmental Health Science and Toxicology. The aims of the Chem HelpDesk are to address the issue of the widening gap in the fields of chemical safety and chemicals management between developed and developing countries, and to empower countries in the South-East Asia Region to manage the import, manufacture and processing, storage, distribution, transport, use, recycling and disposal of chemicals, in ways that minimize significant adverse impacts on health and the environment.

The Chem HelpDesk is not-for-profit, and through its website provides cost-free answers to questions submitted by registered users. These answers are provided by experts in the respective fields, who supply technical and scientific advice as part of a Community of Practice (CoP). It is the aim of the Chem HelpDesk to benefit users and to help countries in areas of most need to protect human health and the environment through the safe use and management of chemicals.

In addition to the **"Questions & Answers"** service for registered users, the website also provides information on the safe use of chemicals, as well as a comprehensive list of links to other important websites related to chemicals management in the region. General users have access to the database of questions and answers, as well as all other information on the website.

For more information, please visit: http://www.chemhelpdesk.org
or e-mail: coordinator@chemhelpdesk.org

The functional workflow of the Chem HelpDesk is divided into 5 steps:



Multi-Scenario Assessment of PAH Exposure and Cancer Risks in China

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importance of considering both total exposure and toxic potency when evaluating health risks associated with PAHs.

Food ingestion was the largest contributor to carcinogenic risk, accounting for approximately 56% to 84% of total ILCR across scenarios. However, dermal exposure, particularly from dust, contributed disproportionately to carcinogenic risk despite its relatively smaller contribution to total PAH exposure. Sensitivity analysis showed that variability in BaPeq concentrations within environmental media was the primary source of uncertainty in risk estimates.

Future studies should incorporate more representative scenario-specific food contamination data, include Chinese-language literature to reduce selection bias, improve temporal resolution to capture changes in emissions and regulations, and develop more standardized monitoring datasets across environmental media. Enhanced characterization of dietary PAH exposure and children's dust-related exposure pathways is particularly important for improving risk assessments.

This study demonstrated that dietary intake is the predominant pathway of PAH exposure and carcinogenic risk across industrial, urban, and rural environments in China. Children consistently experience greater exposure and higher cancer risks than adults, while industrial settings present the greatest overall health concern.

The findings also reveal that dust-related dermal exposure can substantially influence carcinogenic risk, particularly among children. By integrating multiple environmental media, exposure pathways, and population groups, the proposed framework provides a robust approach for environmental health risk assessment and supports the development of targeted strategies to reduce human exposure to PAHs and other environmental contaminants.

Source: Environmental Monitoring and Assessment, Vol. 198, Article 902, August 2026.

Environmental Fate and Toxicity of Polycyclic Aromatic Hydrocarbons (PAHs)

Polycyclic aromatic hydrocarbons (PAHs) are a broad class of organic contaminants primarily generated through the incomplete combustion of carbonaceous materials and petroleum-related processes. Their hydrophobicity, lipophilicity, and chemical stability contribute to extensive distribution across atmospheric, aquatic, and terrestrial environments, resulting in long-term persistence and accumulation in environmental compartments and biological systems. Numerous PAHs are recognized for their carcinogenic, mutagenic, and teratogenic properties, leading to their designation as priority pollutants by international regulatory agencies.

The environmental and health impacts of PAHs are determined not only by their concentrations but also by their molecular characteristics, which govern environmental transport, transformation, bioavailability, and toxicological activity.

This review provides an integrated analysis of the links between PAH formation, environmental fate, toxicological mechanisms, and human health implications, thereby advancing understanding of the processes underlying their environmental and public health significance.

This review examined the relationships among PAH molecular structure, physicochemical properties, environmental behavior, toxicological pathways, and associated health risks. A critical synthesis of evidence from environmental chemistry, combustion science, toxicology, exposure assessment, and risk assessment was conducted.

Findings from laboratory studies, environmental monitoring, mechanistic investigations, and epidemiological research were integrated to evaluate PAH sources, formation mechanisms, environmental fate, transformation processes, exposure pathways, and adverse health outcomes within a unified conceptual framework.

The review demonstrated that environmental behavior of PAHs is largely determined by molecular structure, particularly molecular weight and ring number. Low-molecular-weight PAHs are generally more volatile and mobile, facilitating atmospheric transport, whereas high-molecular-weight PAHs exhibit greater persistence, stronger adsorption to particulate matter, and higher toxic potential. Major environmental sources include transportation emissions, industrial processes, biomass burning, residential heating, and fossil fuel combustion, with the hydrogen-abstraction-acetylene-addition (HACA) mechanism recognized as a key pathway for PAH formation.

Following release, PAHs are transported through the atmosphere and redistributed to terrestrial and aquatic environments via deposition processes. Their strong affinity for organic matter and black carbon promotes accumulation in soils and sediments, where environmental persistence is often prolonged.

Although photochemical oxidation, microbial biodegradation, and other transformation processes can reduce PAH concentrations, their effectiveness is frequently limited by sequestration within environmental matrices. In addition, atmospheric reactions can generate oxygenated and nitrated PAH derivatives that may exhibit enhanced toxicity.

The toxic effects of PAHs are primarily associated with metabolic activation by cytochrome P450 enzymes, producing reactive metabolites that induce oxidative stress, DNA damage, and mutagenesis. These molecular mechanisms contribute to a range of adverse health outcomes, including cancer, cardiovascular disease, respiratory dysfunction, reproductive toxicity, neurodevelopmental disorders, and immunological impairment.

Human exposure occurs predominantly through inhalation of contaminated air, dietary intake, and dermal contact. Current risk assessment approaches may underestimate overall health risks because they focus on a limited number of priority PAHs while often excluding alkylated, oxygenated, and nitrated derivatives with significant toxicological relevance.

Future research should prioritize a more comprehensive evaluation of PAH mixtures, emerging PAH derivatives, and the mechanisms governing bioavailability under environmentally relevant conditions. Greater attention should also be directed toward non-cancer health outcomes, cumulative exposure assessment, and the influence of environmental change on PAH transport and transformation. The incorporation of advanced biomonitoring techniques, mixture-based toxicological approaches, and broader chemical coverage in regulatory frameworks would improve the accuracy of health risk assessments.

Overall, the evidence synthesized in this review confirms that PAHs remain important environmental contaminants because of their widespread occurrence, persistence, and capacity to induce diverse adverse health effects. Their environmental fate and toxicological potential are intrinsically linked to molecular structure and environmental interactions, underscoring the need for interdisciplinary research and more inclusive risk assessment strategies. A deeper understanding of these relationships will support the development of more effective regulatory policies and management practices aimed at reducing environmental contamination and protecting public health.

Source: Molecules, Vol. 31, Issue 13, Article 2211, June 2026.

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