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INTERNATIONAL CENTRE FOR ENVIRONMENTAL AND INDUSTRIAL TOXICOLOGY (ICEIT)

CRI's ICEIT has been designated as a
"UNEP Centre of Excellence for Environmental and Industrial Toxicology".

AI in Environmental Research and Health: Current Applications and Future Prospects

Artificial intelligence (AI) has emerged as a transformative force in environmental research, addressing the limitations of traditional paradigms in tackling complex ecological challenges. With global climate change and environmental degradation accelerating, AI technologies, particularly machine learning (ML) and deep learning (DL), offer powerful tools for data processing, real-time monitoring, and predictive modeling. This commentary explores AI applications across five major domains: water pollution treatment, air pollution control, solid waste management, soil remediation, and environmental health. It highlights how AI-driven approaches enhance computational efficiency, reduce decision-making time by over 60%, and enable intelligent solutions for sustainability.

Applications and Key Findings: In water pollution treatment, AI models predict material performance, optimize wastewater treatment processes, and forecast pollutant distribution globally. For instance, ML algorithms such as CatBoost (Categorical Boosting model) and LSTM (Long Short-Term Memory model) have achieved high accuracy in predicting resin efficiency for PFAS removal and real-time effluent quality in wastewater plants. Similarly, Random Forest models have mapped global fluoride hazards using extensive datasets, aiding early risk assessment.

Air pollution control leverages AI for material screening and greenhouse gas mitigation. Hybrid approaches combining density functional theory (DFT) and ML enable accelerated identification of adsorbent materials and gas sensors for CO_2, CH_4, and N_2O, achieving near-perfect predictive performance. These innovations support the design of advanced environmental materials and global emission control strategies.

Solid waste management benefits from AI through systematic optimization and intelligent sorting. Hybrid neural networks integrate domain knowledge with ML to simulate policy interventions, improving recycling rates significantly. Sensor-based classification systems, employing spectroscopic and visual recognition technologies, achieve over 90% accuracy in waste identification. ML frameworks also automate composting workflows, predicting compost maturity and optimizing operational parameters.

In soil remediation, ML predicts pollutant immobilization efficiency and models global contamination trends. Random Forest algorithms forecast arsenic concentration changes in Chinese soils through 2040, while integrated models predict global soil inorganic carbon distribution, revealing vulnerabilities in acidification and climate change. These insights provide information for carbon cycle management and ecological protection strategies.

Environmental health applications include AI-driven toxicological modeling and risk prediction. Quantitative structure–activity relationship (QSAR) models combined with ML assess combined exposure risks of phthalates and organophosphates, while advanced algorithms identify potential obesogens and simulate pollutant migration patterns. DL models analyze spectral data for wastewater source tracing with near-perfect accuracy, and integrated spatio-temporal attention mechanisms predict pollutant dynamics in aquatic systems.

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Cardiotoxicity from Oral Cadmium Exposure: A Systematic Review

Cadmium (Cd) is a pervasive environmental contaminant and nonessential heavy metal that enters the human food chain primarily through soil contamination and subsequent accumulation in crops, animal tissues, and seafood. Its prolonged biological half-life and strong affinity for metallothionein proteins result in significant bioaccumulation, particularly in renal and skeletal tissues.

The Food and Drug Administration (FDA) developed an oral Cd toxicological reference value (TRV) range of 0.21–0.36 μg/kg bw/day, based on similar points of departure (POD) for the bone and kidney (urinary Cd 0.5 μg/g creatinine) and 50 μg/g in the kidney cortex.

However, cardiovascular disease (CVD) remains the leading cause of global mortality, and emerging evidence suggests that chronic low-level Cd exposure may contribute to CVD pathogenesis through mechanisms such as oxidative stress, endothelial dysfunction, and mitochondrial impairment.

This systematic review evaluates whether the current TRV provides adequate protection against cardiovascular outcomes and identifies the most sensitive endpoints associated with oral Cd exposure.

Animal studies consistently demonstrated that oral Cd exposure (1–5 ppm in drinking water) induced significant increases in systolic and diastolic blood pressure, with some effects observed at concentrations as low as 0.1 ppm over extended durations. Additional findings included cardiac hypertrophy, fibrosis, and oxidative stress.

Epidemiological evidence provided moderate confidence that Cd exposure is associated with atherosclerosis, myocardial infarction, cardiovascular mortality, and elevated blood pressure.

For instance, urinary Cd levels ≥0.5 μg/g creatinine were linked to increased risk of atherosclerosis and myocardial infarction, while blood Cd levels above 0.5 μg/L correlated with cardiovascular mortality. Associations with stroke and heart failure were inconsistent and classified as "not classifiable."

Mechanistic data support biological plausibility, implicating oxidative stress, endothelial dysfunction, and mitochondrial impairment as key pathways for Cd-induced cardiotoxicity. Importantly, the lowest observed adverse effect levels (LOAELs) for cardiovascular endpoints corresponded to the point of departure (POD) used for the current TRV, suggesting that the TRV remains protective.

Overall, the synthesis of animal and epidemiological evidence indicates moderate confidence that chronic oral Cd exposure contributes to increased blood pressure, atherosclerosis, myocardial infarction, and cardiovascular mortality, rendering these endpoints as presumed hazards to human health. The current TRV of 0.21–0.36 μg/kg bw/day appears protective for these cardiovascular effects.

Nevertheless, limitations such as variability in biomarker conversion factors, residual confounding in observational studies, and population-specific data underscore the need for further research. Future investigations should prioritize refining dose-response models, improving biomarker-based exposure assessment, and elucidating mechanistic pathways underlying Cd-induced cardiovascular toxicity. These efforts will enhance risk assessment frameworks and inform regulatory decisions aimed at safeguarding public health.

Source: Food and Chemical Toxicology, Vol. 207. Article 115778, January 2026.

AI in Environmental Research and Health: Current Applications and Future Prospects

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Despite remarkable progress, AI integration in environmental research faces challenges, including data scarcity, overfitting in small-sample models, and uneven geographical coverage of observational data. Proposed solutions include embedding domain knowledge into data generation, employing active learning frameworks, and leveraging data augmentation techniques such as variational autoencoders and generative adversarial networks.

Transfer learning and pre-training strategies enhance model generalization across regions, while global monitoring networks combining satellite and ground sensors can mitigate spatial and temporal biases.

In conclusion, AI is transforming environmental research through advanced data integration, predictive analytics, and real-time monitoring. Its application in areas such as water and air pollution control, solid waste management, soil remediation, and environmental health has significantly improved efficiency and accuracy. AI-driven methods accelerate material discovery, optimize treatment processes, and enable global pollutant mapping and health risk assessment.

However, challenges remain, including data scarcity, overfitting in small-sample models, and uneven geographical coverage. Solutions include embedding domain knowledge into models, using active learning and data augmentation, and applying transfer learning to improve generalization. Global monitoring networks combining satellite and ground sensors can further reduce spatial and temporal biases.

In the future, AI is poised to become central to sustainable environmental governance. Its ability to synthesize multi-source data and support evidence-based decisions will advance carbon neutrality and ecological restoration. Continued collaboration and innovation will ensure AI evolves into a transformative engine for environmental sustainability.

Source: Environment International, Vol. 203, Article 109788, September 2025.

PFOS Exposure and Chronic Kidney Disease: A Multimodal Investigation

Perfluorooctane sulfonate (PFOS), a synthetic perfluoroalkyl substance (PFAS), has been widely used since the mid-20th century in firefighting foams, textiles, and industrial applications for its water- and oil-repellent properties. Its exceptional chemical stability and environmental persistence, estimated half-life exceeding four decades, have led to global contamination. PFOS accumulates in soil, water, and living organisms, entering the human body through ingestion, inhalation, and dermal contact. Despite regulatory restrictions in Europe and North America, ongoing production in some regions continues to pose environmental and health risks. Epidemiological evidence increasingly implicates PFOS in systemic toxicity, including liver, immune, and developmental effects; however, its role in kidney disease remains poorly understood.

Chronic kidney disease (CKD) is a major global health challenge, characterized by progressive loss of renal function and associated with high morbidity and mortality. While diabetes and hypertension are well-known risk factors, emerging research highlights environmental nephrotoxins as significant contributors. Given the kidney's central role in blood filtration and xenobiotic clearance, it is highly vulnerable to persistent pollutants like PFOS. Understanding this link is critical for risk assessment and prevention.

To address this gap, a comprehensive approach was applied, integrating epidemiology, network toxicology, molecular docking, and animal experiments. Network toxicology combines bioinformatics, high-throughput data, and genomics to map interactions between chemicals, biological targets, and adverse outcomes, offering a systematic framework for toxicity prediction. Paired with multi-omics analysis, this method enabled the identification of key molecular targets and pathways involved in PFOS-induced renal injury.

Epidemiological analysis using data from the National Health and Nutrition Examination Survey (NHANES), involving 9,119 participants, revealed a significant association between serum PFOS levels and CKD prevalence. The relationship exhibited a U-shaped dose–response curve with an inflection point at 9.54 ng/mL. Higher PFOS exposure was linked to reduced estimated glomerular filtration rate (eGFR) and elevated renal biomarkers, indicating impaired kidney function. These findings suggest that both low and high PFOS exposure levels may influence CKD risk, with high doses exerting pronounced nephrotoxic effects.

Network toxicology identified 225 PFOS-related targets, 215 overlapping with CKD-associated genes. Enrichment analysis highlighted PI3K-AKT, MAPK, and Ras signaling pathways, implicating oxidative stress, inflammation, and lipid metabolism disruption. Protein–protein interaction analysis pinpointed four hub genes (ALB, PTGS2, AKT1, and IGF1) central to PFOS nephrotoxicity. A diagnostic model based on these genes achieved high predictive accuracy, underscoring their potential as biomarkers for early detection and risk stratification.

Molecular docking confirmed strong PFOS binding to these proteins, suggesting direct molecular interactions. *In vivo* experiments in mice demonstrated dose-dependent renal tubular damage, increased injury markers such as NGAL and KIM-1, and activation of the PI3K-AKT pathway. Histological analysis revealed tubular vacuolation and inflammatory infiltration, providing clear evidence of PFOS-induced renal pathology. These findings collectively establish a mechanistic link between PFOS exposure and CKD progression.

Beyond mechanistic insights, the study explored therapeutic possibilities. Screening via Coremine Medical identified *Astragalus membranaceus*, a traditional Chinese medicine, as a promising candidate. Docking studies showed its active compound, astragaloside IV, binds effectively to the four key proteins, suggesting potential renoprotective effects. This discovery opens avenues for natural product-based interventions in PFOS-related CKD, though clinical validation remains necessary.

The implications of these findings are significant. PFOS exposure represents an emerging environmental risk factor for CKD, with molecular evidence pointing to pathways that regulate inflammation, oxidative stress, and cell survival. The identification of key biomarkers offers opportunities for early diagnosis, while the therapeutic potential of *Astragalus membranaceus* provides a foundation for future intervention strategies.

Future research should focus on longitudinal cohort studies to confirm causality and assess temporal exposure patterns. Intermediate PFOS exposure levels need to be examined to validate the U-shaped dose–response trend observed in this study. Mechanistic investigations using *in vitro* and *in vivo* models are essential to clarify signaling cascades and gene–environment interactions. Additionally, studies should consider the combined effects of PFOS with other PFAS compounds to reflect real-world exposure scenarios. Addressing these areas will enhance understanding of PFOS nephrotoxicity and support the development of targeted strategies for prevention and treatment.

Source: Ecotoxicology and Environmental Safety, Vol. 302, Article 118770, September 2025.

Air Pollution and Breast Cancer Risks: Sensitive Periods

Air pollution is a widespread environmental exposure containing known mammary carcinogens and endocrine-disrupting compounds, which may influence breast cancer risk. Breast cancer remains the most common invasive cancer among women worldwide, and growing evidence suggests that pollutants such as nitrogen dioxide (NO_2) and fine particulate matter ($PM_{2.5}$) may contribute to this risk. Despite these findings, the role of exposure timing, particularly during biologically sensitive periods such as pregnancy and menopause, has not been fully understood. This study was designed to address this gap by examining both specific windows of susceptibility and long-term exposure patterns.

The primary objective was to determine whether exposure to NO_2 and $PM_{2.5}$ during key life stages, including the first birth, most recent birth, and the menopause transition, is associated with breast cancer incidence. Additionally, the study aimed to assess whether long-term exposure to these pollutants influences breast cancer risk overall and by estrogen receptor (ER) status and tumor invasiveness, as well as to identify potential sensitive periods using distributed lag non-linear models (DLNMs).

To achieve these aims, researchers conducted a prospective cohort analysis using data from the Sister Study, which enrolled 50,884 women across the United States between 2003 and 2009. Annual concentrations of NO_2 and $PM_{2.5}$ from 1990 to 2017 were estimated at participants' residential addresses using validated spatiotemporal models. Incident breast cancer cases, including invasive tumors and ductal carcinoma in situ (DCIS), were confirmed through medical records.

The findings revealed limited evidence that exposure during the first birth, most recent birth, or menopause transition significantly increased breast cancer risk. However, when long-term exposure was examined, a clear pattern emerged. A 10-ppb increase in NO_2 across lag years 1 to 11 was associated with a higher incidence of ER-positive breast cancer and DCIS. In contrast, $PM_{2.5}$ exposure during lag years 11 to 13 was linked to ER-negative breast cancer, with a hazard ratio of approximately 1.36 per 10-µg/m³ increase.

These results suggest hormonally mediated mechanisms for NO_2 and possible initiation-related pathways for $PM_{2.5}$. The DLNMs highlighted unique lag periods of susceptibility, underscoring the importance of considering exposure timing in breast cancer research.

In conclusion, this study provides compelling evidence that long-term exposure to NO_2 and $PM_{2.5}$ influences breast cancer risk differently by ER status. NO_2 appears to be associated with ER-positive tumors, while $PM_{2.5}$ may play a role in ER-negative tumors during specific lag periods. These findings emphasize the need for further research into the biological mechanisms underlying these associations and support the development of preventive strategies aimed at reducing air pollution exposure.

Source: Environment International, Vol. 206, Article 109966, December 2025.

Prenatal Glyphosate Exposure and Multigenerational Health Effects in Mice

Glyphosate is one of the most widely used herbicides globally, with applications exceeding 160 million kilograms annually in North America alone. Its extensive use in agriculture, particularly as a pre-harvest desiccant and in glyphosate-resistant crops, has led to its pervasive presence in the food supply. Residues are commonly detected in staple foods such as wheat, corn, soy, and oats, which form the foundation of the Western diet.

While glyphosate was initially considered safe for humans due to the absence of the Shikimate pathway in mammals, emerging evidence suggests indirect effects mediated through gut microbiota disruption, immune modulation, and endocrine interference. These concerns are particularly critical during prenatal development, a sensitive window when maternal exposures can shape offspring health trajectories through microbiome transfer, hormonal signaling, and immune programming. Previous studies have largely focused on high-dose exposures, limiting their relevance to real-world dietary intake.

This study addresses these gaps by investigating whether prenatal exposure to glyphosate at dietary-relevant levels and at the U.S. EPA's acceptable daily intake threshold can disrupt metabolic, immune, and neurobehavioral outcomes across generations in mice, including those genetically predisposed to colitis.

The study aimed to determine whether prenatal glyphosate exposure at human-relevant doses disrupts gut integrity, metabolic regulation, immune function, and neurobehavioral outcomes across generations, and to explore microbiome-mediated mechanisms underlying these effects.

F0 mice were exposed to glyphosate in drinking water during mating and gestation at two doses: 0.01 mg/kg/day (Average American Diet) and 1.75 mg/kg/day (EPA limit). Offspring (F1 and F2) were assessed for intestinal pathology, metabolic function, behavior, and gut microbiome composition using histology, cytokine profiling, hormone assays, metabolomics, and 16S rRNA sequencing. Correlational analyses explored microbe-metabolite interactions.

Prenatal glyphosate exposure caused significant multigenerational effects, including gut barrier dysfunction, immune activation, metabolic disruption, and behavioral impairments. Healthy offspring showed goblet cell loss, crypt hyperplasia, reduced mucin-2, and disorganized tight junctions, alongside macrophage infiltration and a pro-

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Pharmacokinetics and Pharmacodynamics of PFOS and Its Role in Cancer Development

Environmental pollution driven by industrialization, urbanization, and agricultural practices has led to widespread ecological degradation. Among the most persistent pollutants is Perfluorooctane Sulfonate (PFOS), a synthetic compound widely used in industrial and consumer products such as firefighting foams and stain-resistant fabrics.

PFOS is classified as a persistent organic pollutant (POP) due to its chemical stability, resistance to degradation, and bioaccumulative properties. Its presence in ecosystems and food chains poses significant risks to human health, including endocrine disruption, oxidative stress, mitochondrial dysfunction, and immunotoxicity.

Emerging evidence links PFOS exposure to the development and progression of hormone-sensitive cancers (prostate, breast, and ovarian cancers) through mechanisms such as hormonal interference, chronic inflammation, and epigenetic modifications.

This review aims to summarize the pharmacokinetics and pharmacodynamics of PFOS, explore mechanistic pathways through which PFOS contributes to carcinogenesis, highlight epidemiological evidence linking PFOS exposure to cancer risk, and discuss regulatory measures and future research directions to mitigate PFOS-related health hazards.

PFOS exhibits unique pharmacokinetic behavior, binding strongly to serum proteins and accumulating in protein-rich organs such as the liver and kidney. It can cross the placenta, raising concerns about prenatal exposure.

Mechanistically, PFOS disrupts lipid metabolism via PPARα activation, induces oxidative stress through ROS generation, and interferes with mitochondrial function. It also binds non-covalently to DNA, causing structural distortions and impairing repair processes, while epigenetic alterations such as global DNA hypomethylation and histone modification activate oncogenes and silence tumor suppressor genes.

In cancer development, PFOS disrupts androgen receptor signaling in prostate cancer, promotes stem/progenitor cell self-renewal, and activates oncogenic pathways including PI3K/Akt and NF-κB.

In breast cancer, PFOS enhances estrogen receptor signaling, increases cyclin D1, and reduces adhesion molecules, facilitating metastasis.

In ovarian cancer, PFOS impairs steroidogenesis, alters histone acetylation, and promotes granulosa cell tumor growth.

Epidemiological studies associate elevated PFOS serum levels with increased cancer risk, particularly in occupationally exposed populations. However, findings vary by age, hormonal status, and geographic region, underscoring the need for longitudinal studies.

In conclusion, PFOS is a persistent pollutant with complex toxicological effects, including endocrine disruption, oxidative DNA damage, and epigenetic alterations. These mechanisms contribute to its potential role in carcinogenesis, especially in hormone-sensitive cancers.

While regulatory actions have reduced PFOS use in developed countries, global exposure remains a concern. Urgent measures are needed to strengthen monitoring, enforce regulations, and develop safer alternatives. Future research should focus on elucidating PFOS's molecular pathways, assessing cumulative exposure, and exploring targeted interventions to mitigate its health impact.

Source: Cancers, Vol. 17, Article 3507, October 2025.

Prenatal Glyphosate Exposure and Multigenerational Health Effects in Mice

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inflammatory cytokine profile (TNF-α, IL-1β, IFN-γ, IL-17), which shifted toward Th17 dominance in F2 mice.

Metabolic changes included impaired glucose tolerance, reduced insulin sensitivity, and lower glucagon-like peptide-1 (GLP-1), with altered leptin and ghrelin suggesting appetite dysregulation. Elevated serum lipopolysaccharide-induced chemokine and disrupted tight junctions point to gut barrier failure as a driver of these effects.

Behavioral deficits (reduced locomotion and working memory) were most pronounced in F2 EPA-exposed mice, coinciding with decreased kynurenine and serotonin, indicating gut-brain axis disruption. Colitis-susceptible mice lacked overt behavioral changes but showed enteric neuroinflammation markers.

Microbiome shifts included depletion of *Akkermansia muciniphila* and enrichment of *Parabacteroides spp.,* with strong correlations to GLP-1 and kynurenine. Cyanobacterial blooms in susceptible mice raise concerns about neurotoxic metabolites. These effects occurred at doses far below regulatory limits, persisted into F2, and suggest transgenerational transmission, challenging current safety assumptions.

Prenatal glyphosate exposure at dietary-relevant levels disrupts gut barrier integrity, metabolic regulation, and neurobehavioral function across generations, likely through microbiome-mediated mechanisms.

These findings challenge current safety thresholds and underscore the need for mechanistic studies and regulatory reevaluation, particularly regarding developmental windows of susceptibility and transgenerational effects.

Source: Science of the Total Environment, Vol. 1002, Article 180437, November 2025.

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IARC Monographs Volume 135:
Perfluorooctanoic Acid (PFOA) and Perfluorooctanesulfonic Acid (PFOS)

PFOA is carcinogenic to humans (Group 1)

PFOS is possibly carcinogenic to humans (Group 2B)

This volume of the IARC Monographs provides evaluations of the carcinogenicity of two agents, **perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS), and their corresponding isomers and salts.**

These substances belong to the class of per- and polyfluoroalkyl substances (PFAS), which are characterized by extreme resistance to degradation due to the strength of the carbon–fluorine bond. First synthesized in the 1940s, PFOA and PFOS have been widely used in industrial and consumer applications.

PFOA has been integral to fluoropolymer manufacture and is found in surface coatings that impart stain, oil, and water resistance to household products, textiles, carpets, leather goods, and food packaging.

PFOS shares similar uses but is also employed in aqueous film-forming foams for firefighting, semiconductor fabrication, photolithography, electroplating, and various specialty applications such as insulation, dyes, and inks.

Both PFOA and PFOS are ubiquitous in the environment, with elevated concentrations near industrial sites, firefighter training areas, and waste disposal facilities. They contaminate food chains, particularly fish, seafood, and eggs, and persist in drinking water supplies.

Occupational exposure can result in serum concentrations exceeding 100,000 ng/mL, while levels in the general population typically range from 4 to 12 ng/mL. Environmental contamination of drinking water near polluted sites has been reported at concentrations reaching hundreds of micrograms per liter, underscoring the magnitude of exposure risk.

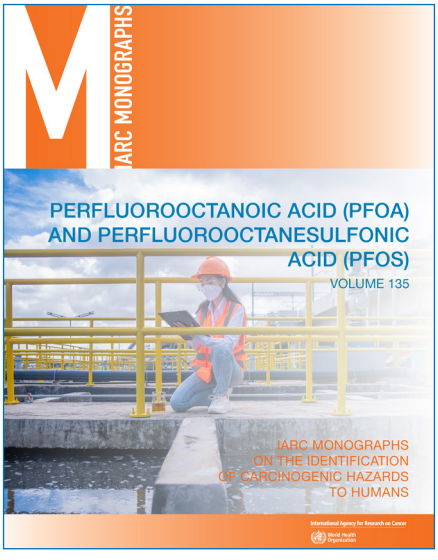
Epidemiological evidence demonstrates a consistent association between PFOA exposure and kidney cancer, supported by meta-analyses showing increased risk proportional to serum levels. Testicular cancer risk is also elevated in highly exposed cohorts, while evidence for other cancers, including prostate, breast, thyroid, and liver, remains limited or inconsistent.

For PFOS, human data are sparse; however, mechanistic studies and emerging epidemiological findings suggest carcinogenic potential, particularly in hormone-sensitive cancers such as breast and ovarian cancer. Recent research highlights PFOS's role in endocrine disruption, epigenetic modifications, and activation of oncogenic signaling pathways, including PI3K/Akt and WNT/β-catenin, which are implicated in tumor progression.

Animal studies provide strong evidence of carcinogenicity. PFOA consistently induces liver tumors, including hepatocellular adenomas and carcinomas, as well as pancreatic acinar cell and Leydig cell tumors in rodents. PFOS, though less studied, has shown clear associations with liver tumors in experimental models. These findings are reinforced by mechanistic data showing that both compounds activate nuclear receptors such as PPARα, CAR, and PXR, disrupt lipid metabolism, and trigger oxidative stress and chronic inflammation.

Mechanistic evidence shows that PFOA and PFOS cause epigenetic changes, impair DNA repair, and generate reactive oxygen species, leading to oxidative DNA damage. They also suppress immune function, enabling tumor progression. PFOS further interacts with sex hormone receptors, amplifies proliferative signaling, and is linked to mitochondrial dysfunction, thyroid disruption, and reduced immune response.

Based on the integration of human, animal, and mechanistic evidence, the IARC Working Group classified **PFOA as carcinogenic to humans (Group 1) and PFOS as possibly carcinogenic to humans (Group 2B).** These classifications reflect the weight of evidence from multiple streams, including strong mechanistic data and positive findings in experimental animals.



Regulatory agencies have responded with stringent measures: the U.S. Environmental Protection Agency set drinking water limits at 0.004 ng/L for PFOA and 0.02 ng/L for PFOS, while the European Food Safety Authority established a tolerable weekly intake of 4.4 ng/kg body weight for combined PFAS. Despite these actions, global exposure persists due to environmental persistence and historical use, emphasizing the urgent need for continued monitoring, stricter regulations, and research into safer alternatives.

In conclusion, the evidence reviewed in this volume underscores the carcinogenic potential of PFOA and PFOS through multiple biological pathways, including genotoxicity-independent mechanisms such as epigenetic reprogramming, receptor-mediated signaling, and immune modulation. These findings highlight the importance of comprehensive risk assessment and proactive regulatory interventions to mitigate the long-term health impacts of PFAS exposure worldwide.

Source: IARC Monographs on the Identification of Carcinogenic Hazards to Humans Volume 135: Perfluorooctanoic Acid (PFOA) and Perfluorooctanesulfonic Acid (PFOS), February 2025.

Consultative Meeting on
"Regional Collaborations on Environmental Health to Address Common Issues of Public Health Concern"
December 7-8, 2025
at Chulabhorn Research Institute Convention Center, Bangkok, Thailand

Organized by Center of Excellence on Environmental Health and Toxicology (CoE EHT)
(Program: Hub of Talents in Environmental Health with Support of the National Research Council of Thailand)

This meeting was organized by the Center of Excellence on Environmental Health and Toxicology (CoE EHT), of which the Chulabhorn Research Institute (CRI, Bangkok, Thailand) is a member institution, through the Hub of Talents in Environmental Health Project under the National Research Council of Thailand (NRCT).

Co-chairs for the meeting were Professor *Khunying* Mathuros Ruchirawat (CRI) and Professor William Au (University of Texas Galveston Branch, USA). Attendees included invited international experts from Aarhus University, the Global Alliance for Health and Pollution (GAHP), the Health and Environmental Sciences Institute (HESI), University of Aberdeen, Utrecht University, and the World Health Organization (WHO); invited delegates from China, Indonesia, Malaysia, Nepal and Vietnam; and international participants from Bhutan, Ghana, India, Indonesia, Iran, Malaysia, the Philippines, Zambia and Thailand, with a total of 35 attendees from 17 countries.

As science, research and innovations are moving at a fast pace with limited technological and personnel resources, it is necessary to develop multi-country collaborations to capture opportunities and address environmental health problems effectively. A first meeting with this objective was held at CRI in June 2024, with the title of "International Conference on Environmental Pollutants and Toxicants Affecting Health".

The current meeting aimed to accelerate the progress in developing multi-country collaborations. The agenda for the meeting was drafted to stimulate

discussion and interactions among participants and to channel efforts in delivering actionable guidelines and tasks. Presentations by invited experts and existing collaborators provided leads for identification of new opportunities for collaborations.

The meeting opened with agreement on a shared vision: a commitment to multi-country, synergistic collaboration to address environmental health challenges. Participants defined specific aims, including identifying strategic areas for joint projects; generating evidence on toxicity mechanisms and genomic signatures; characterizing environmental and human exposure through monitoring and biomonitoring; applying *in vivo*, *in vitro*, and novel methods; developing evidence-based and personalized health risk assessments; promoting integrated solutions; and establishing a framework for data and knowledge exchange.

A Database of Experts was discussed as a key collaboration tool, with proposed refinements including renaming it the Database of Professionals, ensuring data privacy, linking with international networks (GAHP, HESI, WHO), and designating country representatives as national contact points.

An electronic registration and update system was endorsed. Subsequent deliberations identified priority environmental health issues, culminating in a focused list of pollutants: particulate matter/air pollution, highly hazardous pesticides, heavy metals and metalloids, e-waste, PFAS and emerging persistent organic pollutants, and microplastics.

Collaborative topics were developed for $PM_{2.5}$ exposure, pesticides, and arsenic, addressing vulnerable populations, exposure pathways, health outcomes, monitoring, mitigation, and policy relevance. Participants emphasized the need for coordinated regional collaboration, capacity building, and shared methodologies. CRI was identified as a central partner, with specific interest in arsenic contamination and river-related exposures, and e-waste impacts. The meeting concluded with strong consensus and plans for follow-up collaborations.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute
Scheduled for February 2026

	Training Course	Date	Duration	Closing Date
1	Principles of Toxicology, Toxicity Testing and Safety Evaluation	February 16-20, 2026	5 work days	January 16, 2026

Course Coordinator: *Khunying* Mathuros Ruchirawat, Ph.D.

Course Description:

The Chulabhorn Research Institute (CRI) is aware of the importance of providing a training program to assist developing countries with human resource development in the fields of environmental health and toxicology, including the principles of toxicity testing. In February 2026, CRI is organizing a training course on "Principles of Toxicology, Toxicity Testing and Safety Evaluation" for participants from developing countries, primarily in the Asia/Pacific region.

This course presents the fundamental and basic concepts of toxicology, including mechanisms involved in chemical actions from the entrance of chemicals into the body until excretion (ADME); toxicokinetics; activation and detoxification mechanisms; dose-response relationships; types of harmful effects; biological and chemical factors that influence toxicity; and the principles for testing of the effects of various types of toxicants on human health, including novel testing methodology.

Participants who complete the courses will receive a Certificate of Completion for their professional portfolio.

Requirement:

Applicants must fulfill the following requirements:

- Approximately two (2) years' work experience related to the use of basic knowledge in chemistry, biological or biomedical sciences or medicine.
- Hold a bachelor's degree in biological sciences, chemistry, pharmacy, or medicine from a university/technical college.
- Demonstrate proficiency in English (speaking, reading and writing).
- Be in good health, both physically and mentally, and have a health certificate provided by an authorized physician. This form is also attached together with the Application Form. Pregnancy is regarded as a disqualifying condition for participation in the course.

Fellowships:

A limited number of fellowships are available that will cover course fee, round-trip airfare, accommodation (on site) and meals, training materials, and health insurance.

Submission:

Interested persons are required to submit your application through your respective organization, which will then forward the applications to the Royal Thai Embassy in your country.

Contact: Chulabhorn Research Institute (CRI)
54 Kamphaeng Phet 6 Rd.,
Lak Si, Bangkok 10210, Thailand
Tel: +66 2 553 8535
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Calendar

More information and application:
Please visit - https://www.cri.or.th/academic-activities-en/activity-calendar/

The CRI/ICEIT NEWSLETTER is published quarterly by the International Centre for Environmental and Industrial Toxicology of the Chulabhorn Research Institute. It is intended to be a source of information to create awareness of the problems caused by chemicals. However, the contents and views expressed in this newsletter do not necessarily represent the policies of ICEIT.

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