CRI/ICEIT
NEWSLETTER

VOL. 36 NO. 3 – July 2026
ISSN 0858-2793
BANGKOK, THAILAND

Chulabhorn Research Institute

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".

PFAS and One Health Integration

Per- and polyfluoroalkyl substances (PFAS) are a large group of synthetic chemicals widely used in industrial applications and consumer products because of their exceptional chemical stability and resistance to degradation. These same properties, however, have led to their persistence, global distribution, and bioaccumulation in environmental compartments, wildlife, and human populations. PFAS contamination is now ubiquitous, including in remote regions, demonstrating their capacity for long-range transport and long-term environmental retention.

Exposure to PFAS occurs through interconnected pathways such as contaminated drinking water, food webs, and maternal transfer, resulting in parallel exposure scenarios for humans and animals. Toxicological and epidemiological studies consistently show similar adverse health outcomes across species, including immunotoxicity, reproductive and developmental effects, and carcinogenicity. These convergent effects reflect shared biological vulnerabilities and highlight the limitations of approaches that address human, animal, or environmental health in isolation.

The One Health framework, which recognizes the intrinsic interdependence of human, animal, and environmental health, provides a robust and integrative model for understanding and addressing PFAS contamination. Applying this perspective enables coordinated risk assessment, monitoring, and regulatory strategies that more effectively protect public health, wildlife, and ecosystems simultaneously.

The purpose of this review is to synthesize current knowledge on PFAS within an explicitly One Health context. It seeks to integrate evidence on the anthropogenic origins of PFAS, their environmental persistence, and global distribution with data on shared exposure pathways and health outcomes across humans and wildlife.

By focusing on convergent toxicological effects and common vulnerabilities, the review aims to demonstrate the value of cross-species data in risk assessment. In addition, it highlights populations and ecosystems that face disproportionate risks and critically examines the limitations of existing regulatory approaches, particularly in light of continued PFAS use and the emergence of replacement compounds. Ultimately, the review aims to support the development of integrated intervention strategies grounded in One Health principles.

This review is based on a comprehensive synthesis of the scientific literature addressing PFAS contamination, exposure, health effects, and regulatory responses. Emphasis was placed on studies that explicitly link environmental contamination with animal and human health outcomes, thereby reflecting the interconnected structure of the One Health triad.

Evidence from environmental monitoring, wildlife biomonitoring, toxicological experiments, and human epidemiological studies was evaluated collectively to identify shared exposure pathways, common mechanisms of toxicity, and inter-dependencies between health domains. The One Health framework served as the organizing principle for integrating findings and identifying areas where coordinated approaches are most urgently needed.

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PFAS and One Health Integration

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The reviewed evidence demonstrates that PFAS contamination arises predominantly from industrial activities, firefighting foams, and consumer products, resulting in widespread and long-lasting environmental presence. Their chemical stability prevents natural degradation, leading to persistent contamination of water bodies, soils, sediments, and food webs. As a result, exposure pathways converge across species, with drinking water, dietary intake, and maternal transfer representing dominant routes for both humans and wildlife. Long-chain PFAS tend to bioaccumulate in organisms, whereas short-chain compounds exhibit high mobility, further expanding exposure scenarios.

Across these shared exposure pathways, PFAS induce similar adverse health outcomes in multiple species. Immunotoxicity is among the most consistently observed effects and is characterized by suppressed immune responses, altered antibody production, and increased susceptibility to infections. Reproductive and developmental toxicity, including impaired fertility, altered growth, and adverse pregnancy outcomes, is evident in both human populations and wildlife. Certain PFAS have also been linked to carcinogenic effects, reinforcing concerns about long-term and cumulative risks. The consistency of these effects across taxa underscores the biological relevance of cross-species extrapolation and supports the integration of wildlife and experimental data into human health risk assessment.

Vulnerability to PFAS exposure is unevenly distributed. Pregnant women, infants, and communities dependent on contaminated water or traditional diets often face higher exposure and greater susceptibility. Wildlife species, particularly apex predators, accumulate high PFAS concentrations and experience adverse effects that can influence population dynamics. These species function as sentinels, providing early warnings of environmental contamination, but they can also serve as sources of human exposure through food chains. This bidirectional relationship further illustrates the inseparability of ecosystem health and public health.

Despite increasing regulatory attention, PFAS remain a global concern due to their continued production, persistence in the environment, and the introduction of replacement compounds that may present similar or unknown hazards. Current regulatory frameworks often address PFAS on a compound-by-compound basis and lack coordination across sectors and regions. A One Health perspective reveals the limitations of fragmented approaches and emphasizes the need for harmonized, class-based regulation, integrated monitoring systems, and coordinated intervention strategies that simultaneously protect human health, wildlife, and ecosystems.

Future research must adopt transdisciplinary approaches that fully embrace One Health principles. Priority areas include improving the assessment of PFAS mixtures to better reflect real-world exposures and conducting long-term and multigenerational studies to capture chronic and developmental effects across species. Advances in integrated surveillance systems linking environmental measurements, wildlife biomonitoring, and human health data are essential for early detection and effective risk management. Additionally, thorough evaluation of replacement PFAS compounds is necessary to prevent unintended substitution with equally persistent and toxic alternatives. Strengthening international collaboration and regulatory harmonization will be critical for managing PFAS as a global challenge. Collectively, these efforts are essential for developing sustainable, equitable, and effective solutions to PFAS contamination.

Source: Archives of Toxicology, Vol. 100, Pages 1769–1787, May 2026.

Combined Effects of Benzo[a]pyrene and Environmental Pollutants

Benzo[a]pyrene (BaP) is a ubiquitous polycyclic aromatic hydrocarbon generated mainly through incomplete combustion processes, including vehicle emissions, industrial activities, cigarette smoke, and high-temperature cooking.

Although BaP is a well-established human carcinogen, real-world exposure rarely occurs in isolation. Humans are commonly co-exposed to BaP alongside other environmental contaminants such as heavy metals, particulate matter (PM), microplastics, organic pollutants, and physical factors including ultraviolet (UV) radiation. These co-exposure scenarios may produce synergistic, additive, or antagonistic toxic effects that differ substantially from those caused by BaP alone. Understanding these interactions is critical for accurate environmental health risk assessment and disease prevention.

This review systematically evaluates the toxicological interactions and combined effects of BaP with other environmental pollutants, including metals and metalloids, particulate matter, organic contaminants, electromagnetic radiation, and air pollution indicators. It summarizes current evidence on associated health outcomes, underlying molecular mechanisms, and knowledge gaps to improve understanding of the risks posed by combined environmental exposures.

The review conducted a retrospective literature search covering studies published from 1996 to April 2026 focusing on BaP co-exposure, combined exposure, and synergistic effects. Only *in vitro* and *in vivo* experimental studies were included, encompassing cellular models and animal studies such as rodents, zebrafish, and *Caenorhabditis elegans*.

The results demonstrate that BaP frequently interacts with co-existing environmental pollutants through multiple molecular pathways, producing diverse biological outcomes. Co-exposure with heavy metals such as arsenic, cadmium, lead, and aluminum altered BaP

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Heavy Metal Toxicity in Clinical and Environmental Health

Heavy metal are among the most important environmental pollutants affecting human health worldwide. Mercury (Hg), lead (Pb), cadmium (Cd), and arsenic (As) are naturally present in the Earth's crust, but industrialization, mining, fossil fuel combustion, agriculture, and improper waste disposal have dramatically increased their release into the environment. Because these metals are persistent and non-biodegradable, they accumulate in soil, water, air, plants, animals, and ultimately in human tissues.

Exposure can occur through contaminated food and drinking water, inhalation of polluted air, tobacco use, occupational activities, and household sources. Long-term exposure is associated with neurological, renal, cardiovascular, developmental, and carcinogenic effects, making heavy metal toxicity a major concern in environmental medicine and public health.

This article aimed to provide a comprehensive, evidence-based overview of the major toxic heavy metals, focusing on their environmental sources, human exposure pathways, mechanisms of toxicity, diagnostic approaches, clinical management, and prevention strategies. An additional goal was to integrate clinical toxicology with public health practice to support informed decision-making by healthcare professionals and policy-makers.

Scientific evidence published through 2025 was synthesized from epidemiological studies, clinical investigations, toxicological research, case reports, systematic reviews, and public health guidelines. Information was analyzed regarding environmental contamination, exposure assessment, toxicokinetics, disease manifestations, biomonitoring methods, chelation therapy, preventive measures, and emerging developments in precision toxicology.

Multiple exposure routes were identified for each metal. Mercury exposure occurs primarily through fish and seafood consumption, while lead exposure remains linked to contaminated paint, dust, water systems, batteries, and industrial emissions. Cadmium exposure commonly results from smoking, industrial activities, and contaminated food, whereas arsenic exposure is frequently associated with contaminated groundwater and rice-based products.

Despite differences in chemical behavior, these metals share several toxic mechanisms, including oxidative stress, mitochondrial dysfunction, enzyme inhibition, inflammation, and disruption of cellular signaling pathways. Mercury is particularly neurotoxic and readily crosses the blood-brain and placental barriers. Lead interferes with calcium signaling and heme synthesis, leading to neurological and hematological abnormalities. Cadmium accumulates in the kidneys and skeletal system, causing chronic kidney disease and bone damage. Arsenic disrupts cellular metabolism, impairs DNA repair mechanisms, and increases the risk of several cancers.

Health effects vary according to the type, dose, and duration of exposure. Common manifestations include cognitive impairment, developmental delays, peripheral neuropathy, anemia, hypertension, kidney dysfunction, osteoporosis, skin lesions, and cancer. Children, pregnant women, and workers in contaminated environments are particularly susceptible to adverse outcomes.

Accurate diagnosis relies on biomonitoring combined with detailed exposure history. Blood lead levels remain the standard marker of lead exposure. Mercury assessment requires species-specific interpretation using blood, urine, or hair samples. Urinary cadmium serves as an indicator of long-term body burden, while urinary arsenic speciation is necessary to distinguish toxic inorganic arsenic from less harmful organic forms. Unvalidated provoked urine testing was identified as a major source of diagnostic error and potential overtreatment.

The review emphasizes that treatment decisions should be guided by patient symptoms, exposure history, and clinical context rather than biomarker levels alone. Prevention and exposure control often provide greater benefits than pharmacological treatment. The authors also highlight the importance of health equity, environmental justice, evidence-based practice, and caution against misinformation and unnecessary detoxification therapies. Effective management requires cooperation among clinicians, public health agencies, environmental authorities, and communities.

Future efforts should focus on developing rapid and affordable speciation technologies, identifying clinically meaningful thresholds for low-level exposure, and designing safer chelators with greater selectivity for toxic metals. Advances in genomics, metabolomics, and microbiome research may contribute to precision toxicology approaches that enable individualized risk assessment and treatment. Additional implementation studies are needed to evaluate digital health tools, environmental remediation programs, and public health interventions aimed at reducing exposure and health disparities in high-risk populations.

In conclusion, mercury, lead, cadmium, and arsenic continue to pose substantial threats to human health, and effective management depends primarily on prevention, exposure reduction, accurate biomonitoring, and evidence-based clinical care, supported by future advances in diagnostics and precision toxicology.

Source: International Journal of Molecular Sciences, Vol. 27, Issue 8, Article 3513, April 2026.

Phthalate Exposure and Lipid Alterations in Taiwanese Children

Phthalates are widely used plasticizers and ubiquitous environmental contaminants that pose potential health risks, particularly in children, who are often exposed at higher levels and are more susceptible to environmental toxicants. Epidemiological studies have linked phthalate exposure to endocrine, reproductive, neurodevelopmental, cardiovascular, and metabolic disorders. However, the molecular mechanisms underlying these associations remain incompletely understood.

To address this knowledge gap, the present study investigated the relationship between phthalate exposure and serum lipidomic profiles in Taiwanese children using a targeted mass spectrometry-based lipidomics approach.

This cross-sectional study included 256 children aged 8-10 years from the Taiwan Birth Panel Study (TBPS) and the Taiwan Early-Life Cohort (TEC). Urinary concentrations of phthalate metabolites were determined as biomarkers of exposure, while serum phosphocholine-containing lipids, including phosphatidylcholines (PCs), lysophosphatidylcholines (lyso-PCs), ether-linked phosphatidylcholines, and sphingomyelins (SMs), were profiled using liquid chromatography coupled with tandem mass spectrometry. Statistical analyses, including partial least squares-discriminant analysis (PLS-DA) and multiple linear regression, were performed after adjustment for age, sex, body mass index, and household income.

Among all phthalate metabolites examined, mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP), a major oxidative metabolite of di(2-ethylhexyl) phthalate (DEHP), exhibited the strongest and most consistent associations with serum lipid alterations. Higher urinary MEHHP concentrations were associated with increased levels of several diacyl-PCs and lyso-PCs, whereas multiple ether-linked PCs and SMs were significantly decreased. These associations were particularly evident in the TBPS cohort, where 24 differential lipid species were identified.

The observed lipidomic changes suggest that DEHP exposure may disrupt key pathways involved in lipid homeostasis. Increased diacyl-PCs may reflect altered lipoprotein metabolism, while elevated lyso-PCs have been linked to inflammation and oxidative modification of low-density lipoproteins, processes associated with cardiovascular disease. Reduced ether-linked PCs may indicate impaired peroxisomal lipid metabolism and dysregulation of peroxisome proliferator-activated receptor (PPAR) signaling, a central regulator of lipid metabolism. Furthermore, decreased SM levels suggest disturbances in sphingolipid metabolism and possible enhancement of ceramide formation, pathways implicated in neurotoxicity, nephrotoxicity, autophagy, and carcinogenesis.

Although the study was limited by its cross-sectional design, restricted lipid coverage, and potential residual confounding, it provides important molecular evidence linking phthalate exposure to lipid metabolic disruption in children.

Overall, the findings demonstrate that DEHP exposure is associated with significant perturbations of the pediatric serum lipidome and suggest mechanistic pathways through which phthalates may contribute to cardiovascular, metabolic, neurological, and renal health risks. Future longitudinal and mechanistic studies are needed to establish causality and evaluate the long-term health consequences of early-life exposure.

Source: Journal of Food and Drug Analysis, Vol. 34, Pages 78-87, June 2026.

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absorption, metabolism, and detoxification processes. These interactions promoted neurotoxicity, reproductive toxicity, and carcinogenesis through oxidative stress, DNA damage, apoptosis, epigenetic alterations, and disruption of cellular signaling pathways. Particularly strong evidence exists for synergistic carcinogenic effects between BaP and arsenic, which substantially increase lung cancer risk.

Particulate matter acts as an important carrier of BaP and can intensify its toxicity. Carbon-based particles, silica nanoparticles, asbestos, and metal nanoparticles were shown to enhance oxidative stress, inflammation, apoptosis, and DNA damage. Co-exposure frequently resulted in respiratory, cardiovascular, and developmental toxicity.

Emerging pollutants such as microplastics and nanoplastics further increased BaP bioavailability and promoted intestinal, renal, and aging-related damage through mitochondrial dysfunction, ferroptosis, and disruption of gut-organ communication pathways.

BaP also exhibited significant interactions with organic pollutants, including endocrine-disrupting chemicals, phthalates, PFASs, tobacco-related compounds, and food-processing contaminants. These mixtures caused liver injury, renal fibrosis, splenic inflammation, neurodevelopmental toxicity, endocrine disruption, and enhanced carcinogenicity. Key mechanisms involved oxidative stress, inflammatory activation, mitochondrial dysfunction, altered CYP1A1 enzyme activity, changes in macrophage polarization, and dysregulation of signaling pathways such as NF-κB, STAT3, Notch, and PI3K/Akt.

Physical environmental factors also modified BaP toxicity. Combined exposure to UV radiation and BaP enhanced skin carcinogenesis by increasing reactive oxygen species generation, DNA damage, mitochondrial dysfunction, and mutations in cancer-related genes. Evidence also suggested synergistic carcinogenic interactions between BaP and X-rays, radioactive particles, microwave radiation, and sulfur dioxide, although these areas remain less extensively studied.

Overall, the review highlights that oxidative stress, inflammatory responses, mitochondrial impairment, DNA damage,

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A Modernized Human Physiologically Based Toxicokinetic Model of Cadmium

Cadmium (Cd) is a toxic environmental heavy metal that poses a significant public health concern due to its persistence, bioaccumulation, and long biological half-life. Human exposure occurs primarily through contaminated food, ambient air, and cigarette smoking. Chronic Cd exposure has been associated with renal dysfunction, liver injury, osteoporosis, cardiovascular disease, and cancer.

Physiologically Based Toxicokinetic (PBTK) models are widely used to describe the absorption, distribution, metabolism, and excretion of chemicals in the body and support risk assessment. Most existing human Cd PBTK models are derived from the classic Kjellström and Nordberg (K&N) model developed in 1978.

The study aimed to modernize the long-standing K&N-based human Cd PBTK framework by incorporating physiologically realistic processes and improving its predictive accuracy.

The study retained the basic eight-compartment structure of previous models but introduced several important modifications. Physiological blood flows were incorporated, and major organs such as the liver, kidney, and rest of the body were divided into blood and tissue sub-compartments. A filtrate compartment was added to the kidney to better represent glomerular filtration, tubular reabsorption, and urinary excretion. The model also included reversible binding of Cd with plasma proteins and metallothionein, improving the representation of Cd transport and storage.

Model calibration and validation were performed using U.S. population data, including body weight growth curves, dietary Cd intake estimates, smoking-related Cd exposure, blood and urinary Cd concentrations, and autopsy measurements of liver and kidney Cd concentrations. Sensitivity analyses were conducted to identify influential model parameters.

The updated model successfully reproduced observed Cd concentrations in blood, urine, liver, and kidney across age, sex, and smoking categories. Predictions generally fell within the accepted two-fold range of measured values and outperformed several previously published Cd PBTK models. The model accurately represented the proportions of Cd in red blood cells, plasma protein-bound fractions, and free plasma Cd without relying on empirical correction factors used in earlier models.

Simulations showed that Cd accumulates progressively with age, with the greatest body burden stored in the rest of the body, followed by the kidney and liver. Smoking substantially increased lifetime Cd accumulation, resulting in body burdens up to three- to four-fold higher than those of non-smokers. Estimated biological half-lives were approximately 15-16 years for the whole body, 24-35 years for the kidney, 7-10 years for the liver, and 7-8 years for blood during chronic exposure. These values are consistent with published toxicological evidence.

Sensitivity analysis identified gastrointestinal absorption and tissue exchange processes as key determinants of Cd burden. The findings emphasize the importance of iron status, particularly in women, because Cd absorption is influenced by intestinal iron transport mechanisms. The inclusion of physiological blood flow and portal liver entry significantly improved predictions for blood, liver, and kidney Cd levels, addressing major shortcomings of previous models.

There is a need to develop population-based PBTK models that incorporate interindividual variability and uncertainty. Such developments would improve identification of vulnerable subpopulations and strengthen the use of PBTK modeling as an advanced tool for Cd risk assessment, regulatory decision-making, and precision public health applications.

In conclusion, this study presents a major modernization of the traditional human Cd PBTK model by incorporating physiologically realistic blood flow, tissue-specific Cd transport, portal liver entry, and renal filtration mechanisms. The resulting model demonstrates superior predictive performance and provides a robust platform for future population modeling, exposure assessment, and health risk evaluation of cadmium exposure.

Source: Environment International, Vol. 209, Article 110150, March 2026.

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apoptosis, and altered xenobiotic metabolism represent central mechanisms underlying the majority of BaP co-exposure toxicities. The magnitude and direction of these effects depend on pollutant type, concentration, exposure duration, exposure sequence, and biological context.

Current evidence is largely derived from laboratory-based *in vitro* and animal studies, and therefore may not fully reflect real-world human exposure. Future research should prioritize chronic low-dose exposure models that better simulate environmental conditions and evaluate long-term outcomes, developmental toxicity, reproductive effects, and intergenerational health impacts.

Greater attention should also be paid to sex-specific differences and vulnerable populations. Mechanistic investigations should incorporate integrated multi-omics approaches, including transcriptomics, proteomics, metabolomics, and epigenomics, together with computational toxicology and systems biology methods.

A more comprehensive understanding of epigenetic and molecular regulatory networks is essential for identifying biomarkers of early exposure and developing accurate risk assessment strategies. Ultimately, improved knowledge of BaP co-exposure toxicity will support more effective environmental management, public health protection, and regulatory decision-making.

Source: iScience, Vol. 29, Article 116642, July 2026.

The Center of Excellence on Environmental Health and Toxicology (EHT) develops A Hub of Talents Database of Professionals in Environmental Health

Scientific collaboration is important for finding solutions to environmental health issues. Various countries and institutions can share expertise from their network of experts and professionals, possibly becoming regional hubs of expertise and information. Thailand has a national policy to develop a Hub of Talents and Hub of Knowledge. **The Hub of Talents** refers to research and academic institutions that can be centers of expertise and know-how, and that are internationally accepted. They should also have the infrastructure to support high-level research, whether by local researchers or in collaboration with foreign experts. **The Hub of Knowledge** refers to research and academic institutions, whether they be governmental, public or private, that can be centers of knowledge and training, focusing in areas in which Thailand has the greatest expertise.

To foster linkages and create these hubs, one of the first steps is **the development of a platform for collaboration in the form of a database of professionals and institutions.** The main objective for this database is to allow the search for research scientists and institutions with the expertise and related facilities in various aspects that can be identified and contacted, whether it be to provide expert information to governmental, private, or other agencies that need it; to join in and/or support collaborative research; or to join in other research and academic activities that would be of use to network members, e.g., act as scientific and academic advisors on student academic committees.

This **database of professionals in environmental health** was developed by the Center of Excellence on Environmental Health and Toxicology, through the Hub of Talents in Environmental Health project of the National Research Council of Thailand. The Center of Excellence has 5 members, including the Chulabhorn Research Institute (CRI), the Chulabhorn Graduate Institute (CGI), Mahidol University (MU), Thammasat University (TU) and Burapha University (BUU).

Development of the database involved creation of a web-based front end that provides information in the form of news and articles on environmental health, news and announcements on activities, and a search function for registered users to identify professionals in various fields of environmental health. The searchable information is linked from the database of professionals, with information on areas of interest, research and expertise.

The database currently contains information on 109 professionals: health impacts of air pollution (22), impacts of water pollution (33), toxic chemicals and hazardous waste (35), and remediation technologies for addressing environmental health issues (19). Registration of professionals with the database is on-going. The top areas of interest covered by the expertise are currently Air Pollution, Environmental Toxicology, Wastewater Treatment, E-waste, Biomarkers of Effect, Water Quality and Environmental Science, though this is dynamic and changing as additional experts are registered with the database. In addition, the website also provides information through articles from the registered experts, which currently includes articles on the health impacts of air pollutants found in incense sticks, the benefits and potential impacts of mining rare earth elements, bioaccumulation of cadmium in the human body, the current situation of PFOS/PFOA contamination in the environment, the pressing issue of particulate matter in Thailand, and cadmium contamination in Thai durian.

FAO/WHO: Evaluation of Certain Contaminants in Food

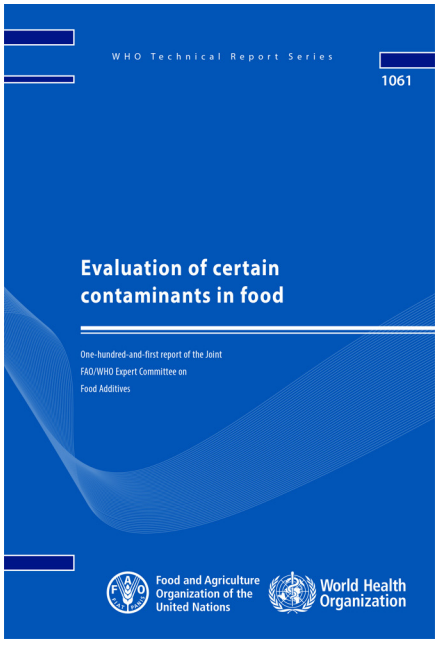
The one-hundred-and-first meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA) was held in Geneva from 15 to 21 October 2025 to evaluate the safety of selected food contaminants, with a specific focus on inorganic and organic arsenic species.

The report "**Evaluation of Certain Contaminants in Food**" presents a comprehensive re-evaluation of toxicological and exposure data, building on earlier assessments conducted at previous JECFA meetings. It incorporates newly available evidence on biochemical and toxicological mechanisms, human epidemiological studies, analytical methods, and patterns of occurrence and contamination in food.

JECFA applied a systematic risk assessment framework that includes hazard identification, dose–response assessment, exposure assessment, and risk characterization. This approach enables the Committee to determine the potential health effects of arsenic, understand how toxicity varies with dose, and estimate dietary exposure across populations. Particular attention was given to chronic dietary intake, which represents the main source of risk, especially in populations with high consumption of contaminated food or water.

The evaluation highlights that inorganic arsenic is of greatest concern due to its established carcinogenicity and association with multiple adverse health outcomes, including skin, lung, and bladder cancers, as well as cardiovascular and developmental effects. Organic arsenic species are generally considered less toxic, but their contribution to overall exposure remains relevant. The Committee reviewed factors influencing arsenic levels in food, including environmental contamination, agricultural practices, feed-to-food transfer, and the effects of processing and preparation.

JECFA updated exposure estimates, established health-based guidance values, and recommended improved monitoring, analytical methods, and preventive measures to reduce contamination, highlighting the need for ongoing evaluation and coordinated risk management.



Source: WHO Technical Report Series, No. 1061, Evaluation of Certain Contaminants in Food, May 2026.

FAO/WHO: Pesticide Residues in Food (Report 2025)

Pesticide Residues in Food: Report 2025 presents the findings of the Joint FAO/WHO Meeting on Pesticide Residues (JMPR), an internationally recognized expert body that provides independent scientific advice to the Food and Agriculture Organization of the United Nations (FAO), the World Health Organization (WHO), the Codex Alimentarius Commission, and national regulatory authorities. The report serves as a comprehensive reference for the evaluation of pesticide residues in food and their potential impacts on human health, supporting the development and revision of Codex Maximum Residue Limits (MRLs) and promoting harmonized international food safety standards.

The report reviews 38 pesticides, including newly assessed compounds and substances undergoing periodic re-evaluation, with comprehensive assessments covering toxicology, metabolism, residue behaviour, analytical methods, and dietary exposure.

Based on these evaluations, the JMPR recommends Acceptable Daily Intakes (ADIs), Acute Reference Doses (ARfDs), Maximum Residue Limits (MRLs), Supervised Trials Median Residue (STMR) values, and Highest Residue (HR) estimates for plant and animal commodities.

The report also addresses advances in pesticide risk assessment, including updated dietary exposure methods, New Approach Methodologies (NAMs), revised residue definitions, storage stability, and transparency in dietary burden calculations.

Chronic and acute dietary exposure assessments were conducted using internationally recognized models, while supporting annexes provide detailed intake estimates, livestock dietary burden calculations, and technical information for Codex and national regulatory authorities.

Source: WHO Publication, Pesticide Residues in Food: Report 2025, May 2026.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute
Scheduled for December 2026

	Training Course	Date	Closing Date
1	Environmental Health Risk Assessment and Management of Toxic Chemicals	November 30 - December 8	October 15
2	Beyond Fundamentals: Chemical Risk Assessment in Practice – Applications through Real World Case Studies	December 9-11	October 15

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

Course Description:

Course 1 The "Environmental Health Risk Assessment and Management of Toxic Chemicals" course is an integration of science and policy, and covers the principals of human health and environmental risk assessment; the risk assessment paradigm; the risk assessment and management processes, which start from problem formulation, hazard, identification and characterization, exposure assessment, and risk characterization; the inherent uncertainties in each step; the mode of action and human relevance framework; chemical-specific adjustment factors; risk assessment of complex mixtures and food chemicals and essential nutrients; the relationship between risk assessment and risk management; as well as the need for open, transparent and participatory acceptance procedures and credible communication methods. Emphasis will be placed on potential adverse health effects of human exposure to environmental hazards, although the principles of ecological risk assessment will also be covered. Importantly, the course teaches the practical application of risk assessment methods to various problems using case studies relevant to problems faced in developing countries.

Course 2 The "Beyond Fundamentals: Chemical Risk Assessment in Practice" course is designed to expand on core concepts and frameworks used to evaluate chemical hazards and exposures, characterize risk, and interpret assessment results for decision-making. The course will emphasize practical, real-world case examples and hands-on application of commonly used risk assessment tools and approaches and will consist of small-group breakout case study sessions, where participants will explore problem formulation, exposure assessment, toxicity assessment, and risk characterization, highlighting similarities and key differences between human health and ecological approaches. Case studies will cover both human health and ecological applications and will include a range of chemistries, exposure scenarios, toxicological endpoints, and regulatory contexts with global and regional applications.

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

Requirements:

Applicants must fulfill the following requirements:

- Approximately two (2) years' work experience related to the use of basic knowledge in biological or biomedical sciences, chemistry, or medicine.
- Hold a bachelor's degree 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.

Application: Interested persons may apply for

- **Course 1 only**
- **Course 2 only**, those who apply for Course 2 only should have the necessary background in fundamentals of risk assessment or should have completed Course 1 in previous years.
- **Course 1&2**, special consideration will be given to participants who plan to attend both training courses.

Fellowships:

A limited number of fellowships are available that will cover course fees, round-trip airfare, accommodation allowance, daily stipend, training materials, and health insurance.

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Calendar

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

The 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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