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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".

Human Health Risk Assessment of PFAS: A Critical Review

Per- and polyfluoroalkyl substances (PFAS), often called "forever chemicals," are synthetic compounds widely used in consumer and industrial products for their water- and oil-repellent properties. Their strong carbon-fluorine bonds make them highly resistant to degradation, leading to global environmental persistence. PFAS have been detected in soil, water, air, and biota across all continents, raising concerns about human exposure and health risks.

Dietary intake, particularly through fish and seafood, is considered the primary non-occupational exposure pathway, as these compounds bioaccumulate and biomagnify in aquatic food webs. Vulnerable populations, such as pregnant women, infants, and subsistence fishers, face heightened risks due to cumulative exposure.

PFAS enter the environment through industrial discharges, landfill leachates, wastewater effluents, and firefighting foams. They persist in soil and water for decades, with half-lives exceeding thousands of years in soil and tens of thousands in water. Long-range atmospheric transport further contributes to global contamination. Monitoring studies reveal PFAS in multiple matrices: dust > leachate > wastewater > groundwater > surface water > sediment > soil > fish > drinking water. Fish often exhibit higher PFAS concentrations than surrounding water due to bioaccumulation, making them a critical exposure source.

Fish consumption accounts for up to 99% of PFOS and 84% of PFOA intake in some populations. Studies across Europe, Asia, Africa, and North America confirm PFAS contamination in both freshwater and marine species, with levels varying by geography, species, and farming practices. Kinetic bioaccumulation models incorporating uptake, elimination, and metabolism rates have improved predictions of PFAS levels in fish. Monte Carlo simulations enhance exposure assessments by accounting for variability in consumption patterns and body weight.

PFAS exposure is linked to liver toxicity, immune suppression, developmental delays, and cancer. Rodent studies show hepatocellular hypertrophy, vacuolation, and necrosis at high doses, while gestational exposure correlates with neonatal mortality and impaired lung maturation. PFOS and PFOA are associated with reduced birth weight, delayed sexual maturation, and neurodevelopmental changes.

The No Observed Adverse Effect Level (NOAEL) for developmental toxicity can be as low as 0.003 mg/kg/day, and tolerable weekly intake values set by EFSA are 4.4 ng/kg/week for combined PFAS. Biomonitoring indicates serum PFAS levels often exceed 20 ng/mL in exposed populations, a threshold linked to increased health risks.

Human health risk assessment involves four steps: hazard identification, hazard characterization, exposure assessment, and risk characterization. Current models include physiologically based pharmacokinetic (PBPK) models, fish consumption models, and probabilistic approaches using Monte Carlo simulations. These models estimate cumulative exposure and identify high-risk groups. However, data gaps persist for emerging PFAS, mixture toxicity, and chronic low-dose effects.

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Pollution and Obesity: Unveiling the Hidden Link

Obesity is a global health crisis traditionally linked to poor diet and inactivity, but growing evidence shows environmental pollution plays a significant role. Industrialization and urbanization have introduced pollutants such as airborne particulates, noise, artificial light, persistent organic pollutants (POPs), and microplastics into daily life. These contaminants disrupt metabolism, hormonal balance, and inflammatory pathways, increasing obesity and chronic disease risk.

This review summarizes recent evidence on how pollutants influence weight gain and metabolic disorders, with particular concern for vulnerable groups like pregnant women, children, and the elderly.

Air pollution, comprising particulate matter, nitrogen dioxide, ozone, and polycyclic aromatic hydrocarbons, affects nearly everyone worldwide. Fine particles like $PM_{2.5}$ trigger oxidative stress and inflammation, disrupting lipid metabolism and promoting fat accumulation. These changes, along with elevated inflammatory markers (TNF-α and IL-6), impair energy regulation. Prenatal exposure is linked to higher BMI and waist-to-hip ratios in children, while long-term exposure in adults correlates with increased body weight and waist circumference, especially in women. Air pollution also reduces physical activity by impairing lung function and exercise capacity.

Noise pollution from traffic is a major health concern after air pollution. Chronic exposure activates stress pathways, raising cortisol and catecholamines that promote insulin resistance and fat storage. Every 10 dB increase in traffic noise can raise obesity risk by up to 20%, especially for abdominal fat. Nighttime noise disrupts sleep, further increasing metabolic stress and contributing to higher BMI in children and postpartum weight retention in women.

Artificial light at night disrupts circadian rhythms, affecting CLOCK gene and metabolic regulators (PPARα), which impairs lipid oxidation and promotes fat storage. High nighttime light exposure is linked to a 12–33% higher obesity risk, especially in older adults and men. Sleep disruption and melatonin suppression amplify these effects, making light pollution a significant obesogenic factor.

Persistent organic pollutants (POPs) like Polychlorinated biphenyls (PCBs), Polybrominated diphenyl ethers (PBDEs), Perfluorooctane sulfonate (PFOS), and Dichlorodiphenyldichloroethylene (DDE), accumulate in fat tissue for decades, disrupting endocrine signaling, induce mitochondrial dysfunction, and activate adipogenic pathways. Prenatal exposure raises BMI and adiposity in children, while adult exposure links to insulin resistance and dyslipidemia. POPs also alter gut microbiota composition, influencing energy harvest and metabolic homeostasis.

Microplastics, now ubiquitous in food and water, penetrate biological barriers and accumulate in organs. Preclinical studies show they promote adipocyte differentiation, systemic inflammation, and gut dysbiosis, hallmarks of obesity. Polystyrene particles upregulate pro-inflammatory markers and impair lipid metabolism, suggesting a synergistic effect with high-fat diets.

The link between pollution and obesity underscores an urgent need for integrated strategies. Policy measures targeting air quality, noise control, and light management, alongside public education on reducing plastic use, are critical. Vulnerable populations require tailored approaches, and future research should prioritize long-term studies exploring cumulative pollutant exposure, genetic susceptibility, and epigenetic modifications.

In conclusion, environmental pollutants act as hidden drivers of obesity through complex biological pathways involving oxidative stress, endocrine disruption, and microbiome alterations. Addressing this nexus demands a multidisciplinary approach, bridging nutrition science, toxicology, urban planning, and public health, to curb the dual burden of pollution and obesity.

Source: Current Obesity Reports, Vol. 14, Article 69, September 2025.

Human Health Risk Assessment of PFAS: A Critical Review

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Regulatory limits vary widely across regions. The U.S. EPA recently set maximum contaminant levels for PFOS and PFOA in drinking water at 4 ng/L, while the EU Drinking Water Directive allows up to 100 ng/L for the sum of 20 PFAS. For fish products, EFSA recommends limits as low as 0.63 μg/kg, though monitored levels often exceed these thresholds in contaminated areas. Harmonization of global standards remains a challenge.

Critical gaps include insufficient data on short-chain and emerging PFAS, lack of standardized sampling protocols, and limited understanding of cumulative exposure. Detoxification strategies for fish products are underexplored, with most research focusing on water treatment technologies such as activated carbon, advanced oxidation, and biodegradation. Climate change impacts on PFAS persistence and bioaccumulation also warrant investigation.

In conclusion, PFAS contamination in fish represents a significant public health concern due to their persistence, bioaccumulation, and toxicity. While regulatory measures and predictive models have advanced, inconsistencies in global standards and data gaps hinder effective risk management. Future research should prioritize long-term cohort studies, cumulative exposure models, and innovative remediation strategies to safeguard food safety and human health.

Source: Science of The Total Environment, Vol. 1000, Article 180428, October 2025.

Low-to-Moderate Arsenic Exposure and Cardiovascular Disease: A Global Perspective

Arsenic is a naturally occurring metalloid found in the earth's crust, present in both inorganic and organic forms. Human exposure primarily occurs through drinking water and diet, especially in regions where groundwater contamination is prevalent.

Globally, an estimated 200 million people are exposed to varying levels of inorganic arsenic, ranging from low concentrations below 10 μg/L to moderate levels between 10 and 100 μg/L and high levels above 100 μg/L. Countries such as Bangladesh, India, China, Vietnam, and Thailand are among the most affected, while millions of individuals in the United States also exceed the WHO and EPA guideline of 10 μg/L due to private wells.

Dietary sources, particularly rice and seafood, further contribute to arsenic intake. Historically, arsenic was used in pesticides, medicine, and even warfare, but its toxicological significance is now well recognized.

Chronic exposure to high levels is linked to cardiovascular disease, cancers, and neurological disorders. However, uncertainty persists regarding the health effects of low-to-moderate exposure, which is often considered "safe" under current regulatory standards. Emerging evidence suggests that even these levels may elevate cardiovascular risk, raising critical questions for public health and policy.

The present systematic review addresses this gap by synthesizing evidence on low-to-moderate arsenic exposure and specific cardiovascular outcomes, including stroke, ischemic heart disease, acute myocardial infarction, and heart failure.

Nineteen observational studies were analyzed, spanning diverse regions such as the United States, Bangladesh, Taiwan, Chile, Italy, Denmark, China, and Spain. Exposure was assessed using water arsenic levels and urinary biomarkers.

The findings reveal important associations. Urinary arsenic was consistently linked to an increased risk of stroke incidence, while water arsenic showed a suggestive association with stroke but no clear link to stroke mortality. For ischemic heart disease, the evidence strongly supports a connection with water arsenic exposure, and mortality risk appeared higher when ecological studies were excluded. Acute myocardial infarction also showed an elevated mortality risk under similar conditions, although its incidence was less consistently associated.

Evidence for heart failure was limited, as only a few studies examined this outcome and found no significant effect. Analyses stratified by sex indicated that both men and women may be vulnerable, with some indication of greater risk for myocardial infarction mortality among men. Sensitivity analyses confirmed that differences in study design influenced the strength of associations, underscoring the need for standardized methodologies in future research.

Mechanistic insights show that arsenic contributes to cardiovascular pathology through several interconnected processes. A primary pathway is oxidative stress, where arsenic generates reactive oxygen species that damage endothelial cells and impair nitric oxide signaling, leading to vascular dysfunction and stiffness. It also promotes chronic inflammation and interferes with angiogenesis, accelerating atherosclerosis. Epigenetic changes such as DNA methylation and histone modifications alter gene expression, while disruptions in microRNA profiles affect myocardial contractility and vascular remodeling. These effects increase arterial stiffness and left ventricular workload, predisposing to hypertrophy and heart failure. Additionally, arsenic impairs calcium signaling and mitochondrial function, compromising cardiac energy metabolism. Together, these mechanisms heighten risks of hypertension, ischemic events, and cardiac dysfunction even at low-to-moderate exposure levels.

The review's strengths include its comprehensive scope, inclusion of urinary biomarkers, and rigorous sensitivity analyses. Limitations involve heterogeneity in exposure assessment, outcome definitions, and reliance on observational designs, which restrict causal inference. Future research should harmonize exposure metrics, incorporate alternative biomarkers such as toenails, and apply dose-response modeling to clarify risk thresholds.

From a public health perspective, cardiovascular disease accounts for one-third of deaths in the United States and remains the leading global cause of mortality. Even modest risk increases linked to arsenic exposure could have substantial population-level impacts. These findings underscore the urgency of revisiting regulatory standards and implementing mitigation strategies in arsenic-endemic regions.

In conclusion, low-to-moderate arsenic exposure is associated with increased risks of stroke, ischemic heart disease, and mortality from ischemic heart disease and acute myocardial infarction.

While uncertainties remain for heart failure and myocardial infarction incidence, the cumulative data call for stricter guidelines, improved monitoring, and targeted interventions to reduce arsenic-related cardiovascular burden worldwide.

Source: Environmental Health, Vol. 24, Article 29, May 2025.

Review of Evidence on Wi-Fi 2.4 GHz Radiation, Oxidative Stress, and Alzheimer's Disease

The potential health impact of electromagnetic radiation from wireless communication systems, particularly Wi-Fi operating at 2.4 GHz, has been a subject of scientific debate. While oxidative stress is widely recognized as a contributor to neurodegenerative diseases such as Alzheimer's disease (AD), the direct link between Wi-Fi exposure and AD remains inconclusive. This systematic review explores whether prolonged exposure to 2.4 GHz Wi-Fi radiation could influence oxidative stress and modulate genes associated with AD pathogenesis.

Alzheimer's disease is a multifactorial neurodegenerative disorder characterized by progressive memory loss, synaptic dysfunction, and neuronal death. Its development involves complex interactions among genetic, environmental, and lifestyle factors. Oxidative stress plays a central role in neuronal damage and accelerates processes such as β-amyloid accumu-lation and tau hyperphosphorylation. Genes like APOE, GSK3B, ABCA7, PICALM, CLU, TREM2, SORL1, and PRNP are strongly implicated in AD risk. Heat shock proteins (HSPs), including HSP27 and HSP70, act as molecular chaperones that protect against oxidative and proteotoxic stress, suggesting a potential neuroprotective role.

Wi-Fi technology operates within international safety limits for electro-magnetic field exposure, primarily based on thermal effects. However, non-thermal biological effects remain under investigation. Experimental studies have reported that 2.4 GHz radiation can induce oxidative stress, alter antioxidant enzyme activity, and modify gene expression in neuronal and other tissues. Animal models exposed to Wi-Fi signals showed increased malondialdehyde (MDA) levels, reduced glutathione (GSH), and impaired antioxidant enzyme activity, indicating oxidative imbalance. Histo-pathological changes in cardiovascular, renal, and reproductive systems have also been documented under prolonged exposure.

Evidence suggests that Wi-Fi radiation may influence genes involved in DNA repair, metabolism, and apoptosis, such as POLD4, FEN1, EXOG, and ITM2B. Altered expression of ITM2B, which regulates amyloid precursor protein (APP) processing, could favor β-amyloid accumulation, a hallmark of AD. Similarly, dysregulation of GSK3B may exacerbate tau phosphorylation and neuroinflammation. Although direct interactions between AD-related genes and those affected by Wi-Fi exposure have not been conclusively demonstrated, shared pathways involving oxidative stress and protein homeostasis indicate a plausible link.

Current evidence does not establish a definitive causal relationship between Wi-Fi exposure and Alzheimer's disease. However, findings highlight that chronic exposure to 2.4 GHz electromagnetic fields may impair antioxidant defenses and influence cellular processes relevant to neuro-degeneration. The interplay between environmental factors like electromagnetic radiation and genetic predisposition warrants further investigation. Under-standing these mechanisms is critical for assessing long-term health risks and developing preventive strategies.

Further research should employ longitudinal human studies and advanced molecular analyses to clarify whether Wi-Fi exposure contributes to AD onset or progression. Investigating gene-environment interactions, oxidative stress biomarkers, and protective roles of heat shock proteins could provide valuable insights. Additionally, exploring therapeutic interventions targeting oxidative stress pathways may help mitigate potential risks associated with electromagnetic radiation.

Source: Frontiers in Neurology, Vol. 16, Article 161643, October 2025.

Air Pollution and Bone Health: Susceptibility from Pregnancy to Childhood

Air pollution is a major global health concern, traditionally linked to respiratory and cardiovascular diseases, but emerging evidence suggests it may also affect skeletal development.

Osteoporosis, which impacts one in five people worldwide, originates partly from early-life factors. Bone accrual during childhood and peak bone mass in early adulthood are critical for lifelong bone health, making early exposures particularly important.

The present study, embedded in the Generation R cohort in the Netherlands, explored whether air pollution during pregnancy and early childhood influences bone mineral density (BMD) and area-adjusted bone mineral content (aBMC) at age six.

Researchers estimated daily concentrations of nitrogen dioxide (NO_2), particulate matter (PM_{10}, $PM_{2.5}$), and $PM_{2.5}$ absorbance at residential addresses from conception to 5.5 years using land-use regression models. Bone outcomes were assessed via DXA scans in nearly 6,000 children. Using distributed lag models, the study identified critical windows of susceptibility rather than relying on average exposure measures.

Findings revealed that exposure to $PM_{2.5}$ and $PM_{2.5}$ absorbance between approximately one and four years of age was consistently associated with lower BMD and aBMC. For example, a 5 μg/m³ increase in $PM_{2.5}$ corresponded to a 2.5-3% reduction in BMD at age six. Similar associations were observed for NO_2 and $PM_{2.5}$ with aBMC during the same period.

Interestingly, prenatal exposure to NO_2 and $PM_{2.5}$ absorbance showed a weak positive association with bone outcomes, possibly reflecting model uncertainty or complex biological interactions. Sex-stratified analyses indicated stronger adverse effects in boys, suggesting potential biological

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Exposure to Diethyl Phthalates and Its Impact on Female Reproductive Health

Diethyl phthalate (DEP), a common plasticizer found in consumer products and cosmetics, is increasingly recognized as an environmental endocrine disruptor. Human exposure occurs through ingestion, inhalation, and skin contact, raising concerns about its long-term effects on reproductive health. While previous studies have linked DEP to male reproductive toxicity and metabolic disorders, its impact on female fertility has remained less understood.

The present study investigated how chronic DEP exposure affects ovarian reserve and oocyte maturation in female mice, offering insights into mechanisms that may also apply to humans.

Female fertility relies on a finite pool of primordial follicles formed during fetal development and gradually depleted throughout life. Their depletion can lead to premature ovarian insufficiency (POI), a condition associated with infertility and hormonal imbalance. The ovary's ability to produce hormones and mature eggs depends on mitochondrial integrity, which supports energy production and meiotic progression. Disruption of these processes compromises egg quality, fertilization success, and embryo development.

In this study, female C57BL/6J mice were exposed to DEP at 10 mg/kg and 100 mg/kg via oral gavage for eight weeks. Oocyte collection and hormonal assays were performed alongside histological and transcriptomic analyses to assess ovarian function.

Results showed a significant reduction in primordial follicles and ovulated oocytes, along with lower rates of first polar body extrusion, indicating impaired meiotic competence. High-dose DEP increased oocyte fragmentation, though estrogen and progesterone levels remained unchanged, suggesting toxicity occurs independently of systemic hormone imbalance.

Further analysis revealed mitochondrial dysfunction and oxidative stress in DEP-treated oocytes. Mitochondria were abnormally clustered, and reactive oxygen species (ROS) levels were elevated, contributing to spindle disorganization and chromosome misalignment. These structural abnormalities explain the reduced maturation competence and increased fragmentation rates.

Transcriptomic profiling identified hundreds of differentially expressed genes, with disruptions in PI3K-AKT, TGF-β, and Wnt signaling pathways, as well as genes linked to actin filament organization and cell cycle regulation. Several of these genes overlapped with those associated with ovarian function in POI patients, suggesting a mechanistic link between DEP exposure and follicle depletion.

In conclusion, DEP impairs ovarian reserve and oocyte quality through mitochondrial dysfunction and oxidative stress rather than hormonal disruption. These findings raise concerns about the reproductive risks posed by chronic exposure to phthalates, even at doses far exceeding typical environmental levels.

Future research should examine long-term reproductive outcomes, including pregnancy rates and offspring health, assess dose-response effects at environmentally realistic levels, and explore protective strategies like antioxidants to counteract DEP-induced damage.

By elucidating the cellular and molecular consequences of DEP exposure, this study contributes to a growing body of evidence that environmental chemicals can exert profound effects on reproductive health, warranting stricter regulation and public awareness.

Source: Ecotoxicology and Environmental Safety, Vol. 303, Article 118956, September 2025.

Air Pollution and Bone Health: Susceptibility from Pregnancy to Childhood

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differences in inflammatory response during early childhood.

Mechanistically, air pollution may impair bone health through systemic inflammation, oxidative stress, endocrine disruption, and vitamin D deficiency. These pathways could alter osteoblast and osteoclast activity, reducing bone accrual during critical growth phases. The observed associations are clinically relevant, even modest reductions in early bone mass can increase lifetime fracture risk, as a 10% increase in peak bone mass is estimated to delay osteoporosis onset by over a decade.

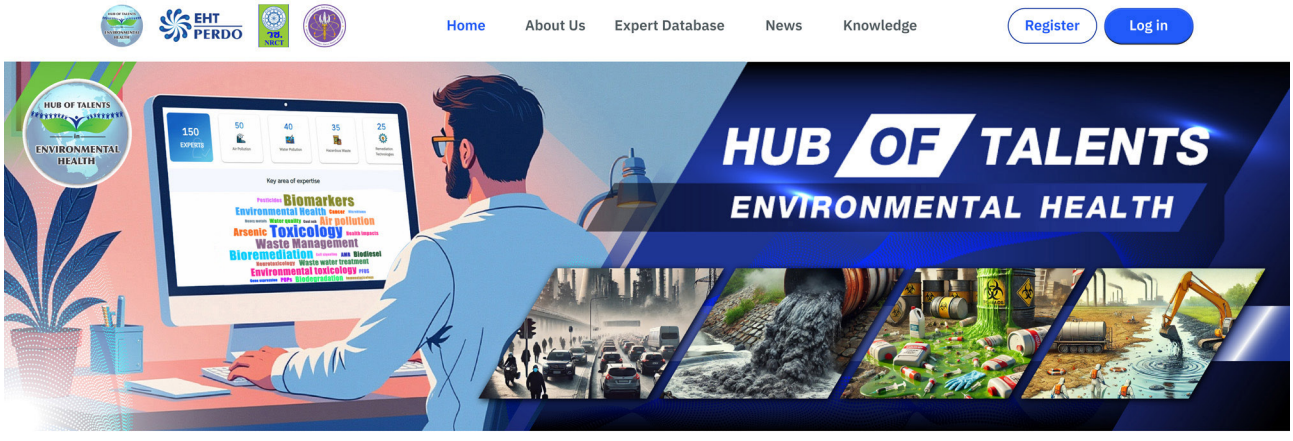
This study is the first to examine air pollution exposure from conception to early childhood with monthly resolution and bone health outcomes in a large cohort. Strengths include fine-scale exposure assessment, gold-standard dual-energy X-ray absorptiometry (DXA) measurements, and adjustment for socioeconomic and lifestyle factors. Limitations include reliance on residential exposure estimates and potential residual confounding. Nonetheless, the findings underscore the importance of early-life environmental exposures in shaping skeletal health and highlight air quality improvement as a potential strategy to reduce osteoporosis risk later in life.

In conclusion, childhood (particularly ages one to four) appears to be a critical window when air pollution adversely affects bone development. These results call for replication in other cohorts and further investigation into sex-specific effects and long-term consequences. Improving air quality may not only benefit respiratory and cardiovascular health but also contribute to stronger bones across the lifespan.

Future research should explore pollutant composition, cumulative exposure, and interventions to mitigate risks during vulnerable developmental stages. The potential for improved air quality to reduce osteoporosis prevalence adds weight to the argument for stronger environmental policies worldwide.

Source: Environment International, Vol. 203, Article 109739, September 2025.

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



A database of experts in various fields of environmental health in Thailand to support national and international networking.

https://www.envihealthhub.com

Science, research and innovations are moving at a fast pace. Coordination and collaboration allow for exchange of information and ideas across the globe. The collaboration of research scientists with similar interests allows for specialization, both at the level of the institution and the nation, which in turn fosters international collaborations whereby each country can share the expertise from their network of institutions, possibly becoming regional hubs of expertise and information.

Thailand is well-known on the international arena, with many geographical and logistical advantages to becoming a hub, especially for the Southeast Asian region. This has led to 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 government, public or private, that can be centers of knowledge and training, focusing in areas in which Thailand has the greatest expertise, such that Thailand can be a hub of expertise for the region.

In order to foster linkages, both at the local and national levels, as well as at the international levels, by creating these hubs, one of the first steps is **the development of a platform for collaboration in the form of a database of experts 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 experts 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.

This Center of Excellence has 5 members, including the Chulabhorn Research Institute (CRI), the Chulabhorn Graduate Institute (CGI), Mahidol University (MU), Burapha University (BUU), and Thammasat University (TU).

The areas of environmental health initially included are (a) the health impacts of air pollution, (b) the impacts of water pollution, (c) toxic chemicals and hazardous waste, as well as (d) remediation technologies for addressing environmental health issues.

Development of the database involves creation of a web-based front end, i.e., a website, that provides information in the form of news and articles on environmental health, as well as news and announcements on activities, e.g., training courses, meetings and conferences on related topics. Importantly, the website will provide a search function for registered users to identify experts in various fields of environmental health. The searchable information is linked from the database of experts, with information on areas of interest, research and expertise. There will also be links to other similar databases at the national, regional and global levels.

The Chem HelpDesk

"Strengthening capabilities for sound chemicals management"

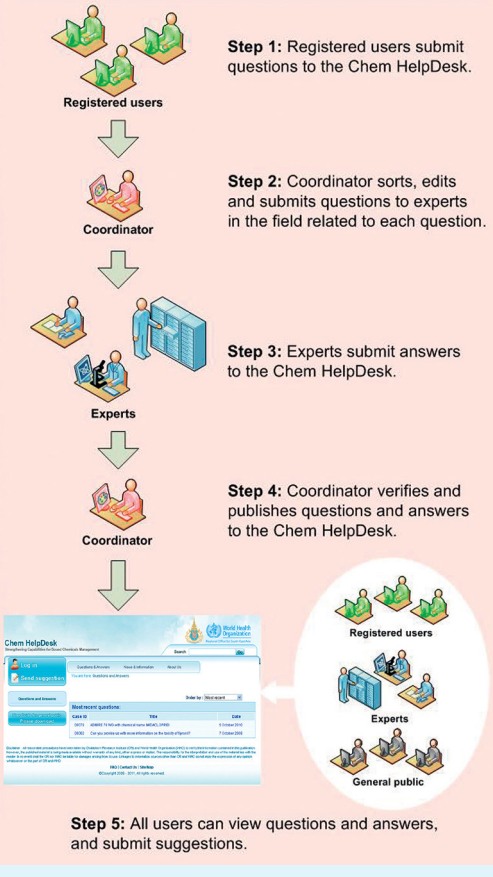
The Regional HelpDesk for Chemical Safety, or **Chem HelpDesk** was established as a joint-initiative between WHO SEARO and CRI, through the WHO Collaborating Center for Capacity Building and Research in Environmental Health Science and Toxicology. The aims of the Chem HelpDesk are to address the issue 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

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



WHO: Global Status Report on Neurology

Neurological conditions are the leading cause of ill health and disability, affecting over 1 in 3 people worldwide. **The Global status report on neurology** presents the first comprehensive global assessment of the public health response to neurological disorders under the Intersectoral global action plan on epilepsy and other neurological disorders 2022–31 (IGAP).

Drawing on data from 102 WHO Member States representing 71% of the world population, the report sets 2022 baseline values for the 10 global targets of IGAP.

It captures the global status across governance and advocacy, financing, service delivery and workforce, access to medicines and technologies, brain health promotion and disease prevention, and research and information systems.

The analysis reveals critical gaps and implementation barriers, underscoring the urgent need for bold and coordinated action to achieve IGAP targets by 2031. To accelerate progress, the report offers inclusive, evidence-based and actionable recommendations for policymakers, IGAP partners and the global neurology community, ensuring that people with neurological conditions and their families are at the heart of the response.

Source: WHO Publications. Global Status Report on Neurology, October 2025.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute, Scheduled for December 2025

	Training Course	Date	Duration	Closing Date
1	Environmental Health Risk Assessment and Management of Toxic Chemicals: Part I - Fundamentals of Risk Asesssment	December 8-12, 2025	5 work days	October 30, 2025
2	Environmental Health Risk Assessment and Management of Toxic Chemicals: Part II - Specific Issues in Risk Asesssment	December 15-17, 2025	3 work days	

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

Course Description:

Environmental Health Risk Assessment and Management of Toxic Chemicals

The fundamentals course is an integration of science and policy, and covers the principals of human health and ecological risk assessment; the risk assessment paradigm; the risk assessment and management processes, which start from identification of the hazard, assessment methods, the inherent uncertainties in each step, and 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. The course also teaches the application of risk assessment methods to various problems and describes the policy context in which decisions to manage environmental health risks are made. The applications of environmental impact assessment procedures for identifying and assessing risk are also 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.

The specific issues course will cover problem formulation, the mode of action and human relevance framework, benchmark dose modeling, an introduction to food safety, food chemicals data needs and risk assessment approaches, risk assessment of essential nutrients and the risk-benefit analysis, mode of action and human relevance, chemical-specific adjustment factors and an introduction to physiologically based pharmacokinetic modeling.

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 chemistry, biological sciences 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.

Fellowships:

A limited number of fellowships are available that will cover 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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