CRI/ICEIT
NEWSLETTER

VOL. 34 NO. 3 – July 2024
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".

Environmental Factors Affecting Female Fertility

Female fertility is influenced by a complex interplay of biological and environmental factors, and recent research has highlighted the significant role of environmental exposures in reproductive health. This review synthesizes evidence on how environmental factors, including climate, temperature, radiation, air and water pollutants, nutrition, lifestyle habits, and endocrine-disrupting chemicals (EDCs), affect both assisted and non-assisted female fertility. EDCs such as heavy metals, plasticizers, parabens, pesticides, industrial chemicals, and their by-products can interfere with hormonal signaling, induce oxidative stress, and cause epigenetic changes, potentially impacting fertility across generations.

The objective of this review was to examine the direct and indirect effects of environmental factors and EDCs on female fertility outcomes, including oocyte maturation, ovulation, embryo transport, implantation, and pregnancy success.

Studies included *in vitro* and *in vivo* experiments on animals and humans, as well as epidemiological investigations.

The findings reveal that environmental factors exert both detrimental and protective effects on fertility. Animal studies show that exposure to bisphenol A (BPA) and its analogs disrupts folliculogenesis, impairs oocyte maturation, and alters steroidogenesis, while phthalates and pesticides induce oxidative stress, apoptosis, and follicular atresia. Heavy metals such as cadmium and lead impair ovarian function and implantation.

In humans, epidemiological data link air pollution, particularly $PM_{2.5}$, NO_2, and SO_2, to reduced conception rates and poorer outcomes in assisted reproduction. Proximity to high-voltage power lines increases unexplained infertility risk, and long working hours correlate with reduced fertility. Nutritional factors also play a critical role: diets high in trans fats, animal protein, and processed foods are associated with ovulatory dysfunction, whereas adherence to the Mediterranean diet improves conception rates and embryo quality. Obesity and excessive exercise negatively affect ovulation and endometrial receptivity, while moderate exercise and weight loss restore fertility potential.

In assisted reproduction, exposure to EDCs such as BPA and phthalates correlates with decreased oocyte yield, poorer embryo quality, and lower implantation and live birth rates. Industrial chemicals like PCBs and dioxins further compromise ovarian reserve and uterine receptivity. Emerging evidence suggests that EDCs can induce transgenerational epigenetic modifications, altering gene expression related to steroidogenesis and folliculogenesis, thereby affecting fertility in subsequent generations.

In conclusion, environmental factors, including chemical exposures, lifestyle habits, and dietary patterns, significantly influence female fertility through mechanisms involving hormonal disruption, oxidative stress, inflammation, and epigenetic changes. Recognizing these risks is essential for developing preventive strategies, improving reproductive outcomes, and safeguarding future generations. Further well-designed studies are needed to clarify dose-response relationships and guide regulatory policies aimed at reducing harmful exposures.

Source: Endocrine, Vol. 86, Pages 58-69, July 2025.

Exploring BPA Alternatives: Environmental Levels and Toxicity Review

Bisphenol A (BPA) is widely recognized as an endocrine-disrupting chemical with adverse effects on human and ecological health. Regulatory restrictions on BPA have led to the introduction of BPA alternatives, marketed as safer substitutes. However, growing evidence indicates that many alternatives share similar endocrine-disruptive and toxic properties, raising concerns about their environmental and health impacts. These compounds are increasingly detected in water, sediment, and soil, yet systematic data on their occurrence and biological effects remain limited.

This review aimed to consolidate existing data on environmental levels of BPA alternatives across Europe, summarize current knowledge on their physicochemical properties, bioactivity, and ecotoxicity, and identify research gaps to guide future monitoring and toxicity assessment, including new approach methodologies.

A systematic literature review was conducted to collect monitoring data on BPA and 25 alternatives in European water, sediment, and soil. Physico-chemical properties such as hydrophobicity and acidity were compiled from experimental and predictive models. *In silico* approaches, quantitative structure-activity relationship (QSAR) models, and predictive tools were evaluated for ecotoxicity predictions. Experimental data from *in vitro* bioassays and *in vivo* studies on microbial, invertebrate, and vertebrate models were synthesized to assess endocrine disruption, oxidative stress, and other endpoints.

Environmental monitoring revealed that BPA remains dominant in environmental samples, but alternatives such as bisphenol S (BPS), bisphenol F (BPF), bisphenol AF (BPAF), and bisphenol A diglycidyl ether (BADGE) are increasingly detected.

In European freshwater, BPA reached several micrograms per liter, while BPS and BADGE were found at even higher levels in some cases.

Sediment samples contained BPA and BPAF at significant concentrations, and soil studies revealed BPA and BPF at detectable levels, with other alternatives rarely found.

These findings indicate widespread contamination and potential mixture exposures. Alternatives exhibit wide variability in hydrophobicity and acidity, influencing bioavailability and toxicity.

In vitro assays reveal estrogen receptor activation as a common mode of action for BPA and several alternatives, alongside androgen receptor antagonism and mitochondrial toxicity.

Oxidative stress emerges as a conserved pathway, with BPA and alternatives reducing antioxidant enzyme activity and inducing reactive oxygen species. Ecotoxicity studies demonstrate impacts across taxa, including microbial communities, invertebrates, and vertebrates such as zebrafish and amphibians, with early developmental stages being particularly sensitive.

BPA alternatives are ubiquitous in aquatic and terrestrial environments and exhibit endocrine-disruptive and oxidative stress-related effects similar to BPA. Despite regulatory efforts, monitoring remains inadequate, and toxicity data are fragmented, focusing on a few chemicals and endpoints.

Future research should expand environmental surveillance to include mixtures and realistic exposure scenarios, develop advanced methodologies integrating computational models and omics, and investigate overlooked endpoints such as immunotoxicity and microbiome disruption.

For well-studied alternatives like BPS, existing evidence already supports regulatory action to mitigate environmental and health risks.

Source: Environmental International, Vol. 189, Article 108728, July 2025.

Ambient Air Pollution and Urological Cancer Risk

Urological cancers, which include prostate, bladder, kidney, and testicular cancers, represent nearly thirteen percent of all cancers worldwide and impose a considerable public health burden. While environmental exposures such as cadmium and arsenic have long been recognized as risk factors, the role of ambient air pollution has remained uncertain.

The International Agency for Research on Cancer has classified particulate matter as a human carcinogen, raising concerns about its potential contribution to cancers beyond the respiratory system. Given the global prevalence of air pollution and its far-reaching health implications, understanding its association with urological cancers is essential for developing effective prevention strategies.

This study was designed to synthesize epidemiological evidence on whether exposure to major air pollutants contributes to the risk of urological cancers.

The primary objective of this research was to evaluate the association between major air pollutants, particularly fine particulate matter ($PM_{2.5}$, PM_{10}, nitrogen dioxide (NO_2), and other regulated pollutants, and the risk of individual and overall urological cancers.

To accomplish this objective, the researchers carried out a systematic review and meta-analysis that incorporated thirty-seven studies in the qualitative review and twenty-one in the quantitative synthesis.

These studies represented data from eighteen countries and utilized diverse methodological approaches, including cohort, case-control, and ecological designs. For the meta-analysis, a random-effects model with robust variance estimation was employed, focusing on standardized exposure increments of 5 µg/m³ for $PM_{2.5}$ and 10 µg/m³ for NO_2.

(Continued on page 4)

Cadmium Exposure and Osteoclastogenesis: Mechanistic Insights

Cadmium (Cd) is a highly toxic environmental pollutant associated with osteoporosis and skeletal fragility. Cd-induced bone loss was attributed to kidney dysfunction and altered calcium-phosphate metabolism. However, recent evidence suggests that Cd directly targets bone tissue, reducing bone formation and enhancing resorption.

Bone remodeling is a dynamic process involving a balance between osteoblasts (bone-forming cells) and osteoclasts (bone-resorbing cells).

Osteoclastogenesis refers to the differentiation of osteoclasts from monocyte/macrophage precursors under the influence of key signaling molecules such as RANKL (Receptor Activator of Nuclear Factor κB Ligand) and M-CSF (Macrophage Colony-Stimulating Factor). This process is tightly regulated by transcription factors, including NFATc1, which acts as a master regulator of osteoclast differentiation. Reactive oxygen species (ROS) also play a critical role in modulating osteoclast signaling pathways, particularly under stress conditions.

Cd exposure induces oxidative stress and mitochondrial dysfunction, which amplify osteoclastogenic signaling. Two transcription factors (NRF2 and NRF1) are central to this response. NRF2 generally protects against oxidative stress by activating antioxidant genes, while the long isoform of NRF1 (L-NRF1) promotes osteoclastogenesis through NFATc1 activation. Understanding this interplay is crucial for developing strategies to mitigate Cd-induced bone toxicity.

The study aimed to determine the role of NRF2 and L-NRF1 in Cd-induced bone loss, explore the molecular mechanisms underlying osteoclastogenesis under oxidative stress, and assess whether antioxidant interventions can mitigate Cd-related bone toxicity.

In vivo experiments involved male wild-type (WT), global *Nrf2* knockout ($Nrf2^{-/-}$), and myeloid-specific *Nrf2* knockout (*Nrf2*(M)-KO) mice exposed to Cd (50 or 100 ppm) in drinking water for 8 or 16 weeks. Bone architecture was analyzed using micro-computed tomography (micro-CT), histology (TRAP staining), and biomechanical testing. Plasma markers of bone turnover (TRAP5b, CTX-I, OCN, P1NP, RANKL, OPG) were measured by ELISA.

In vitro experiments used bone marrow-derived osteoclast progenitor cells (BM-OPCs) and RAW 264.7 macrophage cells cultured with Cd (10-20 nM) under osteoclastogenic conditions.

Genetic manipulations included *Nrf2* knockdown (KD), *Nrf2* overexpression (OE), and L-*Nrf1* silencing or overexpression via lentiviral transduction.

Reactive oxygen species (ROS) were quantified using DCFH-DA and MitoSOX assays. Antioxidant treatments (N-acetyl-L-cysteine, mitoquinone mesylate) were tested for their effects on osteoclast differentiation.

Bone loss and osteoclast activation: Cd exposure significantly reduced trabecular bone volume (BV/TV) and increased osteoclast number and surface in WT mice, with more severe effects in *Nrf2*-deficient mice. $Nrf2^{-/-}$ and *Nrf2*(M)-KO mice exhibited greater bone fragility, lower maximum load, and higher plasma TRAP5b and CTX-I levels compared to controls.

Cellular mechanisms: In vitro, *Nrf2*-deficient BM-OPCs and RAW cells showed exaggerated osteoclast differentiation under Cd exposure, marked by increased TRAP-positive multinucleated cells, NFATc1 protein, and osteoclastogenic genes.

Conversely, *Nrf2* overexpression suppressed these changes. Mechanistic studies revealed that Cd-induced ROS accumulation upregulated L-NRF1, which in turn activated NFATc1, amplifying osteoclastogenesis. Silencing L-*Nrf1* reversed this phenotype, while its overexpression worsened it.

Antioxidant intervention: Treatment with NAC or MitoQ reduced ROS levels, downregulated L-NRF1 and NFATc1, and mitigated osteoclast differentiation in Cd-exposed *Nrf2*-deficient cells. These findings confirm that ROS-mediated L-NRF1 activation is a key driver of Cd-induced osteoclastogenesis.

Implications: NRF2 and L-NRF1 act in a contradictory yet coordinated manner: NRF2 protects against oxidative stress, while L-NRF1 accelerates osteoclast differentiation under Cd exposure. This interplay highlights a novel redox-regulatory axis in bone metabolism. Targeting NRF2 activation or L-NRF1 inhibition may offer therapeutic strategies for Cd-induced osteoporosis.

Prolonged Cd exposure leads to bone loss through enhanced osteoclastogenesis mediated by ROS-dependent L-NRF1 activation and NFATc1 induction. NRF2 deficiency exacerbates this process, underscoring its protective role in bone homeostasis. Antioxidant treatments effectively attenuate these effects, suggesting potential interventions for environmental Cd toxicity.

Future research should focus on human-relevant exposure models, sex-specific responses, and combined analysis of osteoclast and osteoblast dynamics to fully elucidate Cd's impact on skeletal health.

Source: Environmental Health Perspectives, Vol. 132, No. 6, Article 067009, June 2024.

Heavy Metal Exposure and Its Impact on Brain and Gut Microbiota

Heavy metals such as lead (Pb), cadmium (Cd), mercury (Hg), and manganese (Mn) are persistent environmental contaminants that pose significant risks to human health. These metals can cross the blood-brain barrier (BBB), accumulate in neural tissue, and disrupt central nervous system (CNS) function. Infants and children are particularly vulnerable due to an underdeveloped BBB and prolonged brain development, increasing susceptibility to cognitive deficits and neurodevelopmental disorders.

Recent research highlights the gut-brain axis as a critical mediator of these effects, suggesting that heavy metal-induced gut microbiota dysbiosis may exacerbate neurotoxicity. This systematic review synthesizes evidence from animal studies to explore whether changes in gut microbiota contribute to brain dysfunction following heavy metal exposure.

The review aimed to evaluate the relationship between heavy metal exposure, gut microbiota alterations, and neuropsychological or molecular outcomes in animal models. Specifically, it sought to determine whether gut microbiota acts as a mediator in cognitive and behavioral impairments and to identify potential mechanisms underlying these interactions.

Sixteen studies met inclusion criteria, covering Pb (n=10), Cd (n=1), Hg (n=3), Mn (n=1), and combined Pb+Mn exposure (n=1). Animal models included rats, mice, zebrafish, carp, and fruit flies. Most exposures were oral, mimicking dietary ingestion. Gut microbiota was analyzed primarily through 16S rRNA sequencing, with some studies employing shotgun metagenomics.

The findings consistently indicate that heavy metal exposure alters gut microbiota diversity and composition, particularly affecting the phyla Firmicutes (gram-positive bacteria) and Proteobacteria (gram-negative bacteria), though directionality varied.

Pb exposure often reduced *Lactobacillus* abundance, correlating with depression-like behaviors and impaired memory in rats and fruit flies. Conversely, increases in Proteobacteria were linked to heightened anxiety and locomotor activity.

Hg exposure decreased brain-derived neurotrophic factor (BDNF) levels and induced neuroinflammation, while Mn exposure caused hippocampal degeneration and reduced microbial richness.

Behavioral outcomes included deficits in spatial memory, learning, and social interaction, alongside molecular changes such as reduced serotonin (5-HT) expression, increased oxidative stress markers, and activation of apoptotic pathways.

Mechanistically, gut microbiota may influence brain function through short-chain fatty acid (SCFA) production, modulation of neurotransmitter precursors (e.g., tryptophan for serotonin), and immune signaling. Dysbiosis can impair these pathways, amplifying neurotoxic effects.

Probiotic interventions demonstrated promising results: supplementation with *Lactobacillus* and *Bifidobacterium* restored microbial balance, increased SCFA levels, and mitigated behavioral and neurochemical impairments. For example, probiotics reversed Pb-induced depression-like behaviors and dendritic spine loss, while reducing pro-inflammatory cytokines and oxidative damage. These findings underscore the gut microbiota's potential role as both a mediator and therapeutic target in heavy metal neurotoxicity.

This review reveals a complex interplay between heavy metals, gut microbiota, and the CNS. Heavy metal exposure induces both direct neurotoxic effects and indirect consequences via gut dysbiosis, contributing to cognitive and behavioral impairments. Probiotic interventions show potential in mitigating these effects, suggesting novel strategies for prevention and therapy.

Future research should employ standardized methodologies, integrate multi-omics approaches, and explore microbiota-based interventions in human populations. Understanding this triad (heavy metals, gut microbiota, and brain) could inform public health policies and precision medicine approaches aimed at reducing neurodevelopmental risks associated with environmental contaminants.

Source: Environmental Pollution, Vol. 348, Article 123732, May 2024.

Ambient Air Pollution and Urological Cancer Risk

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The findings revealed consistent evidence linking air pollution to an increased risk of urological cancers. A 5 µg/m³ increase in $PM_{2.5}$ was associated with a six percent higher risk of overall urological cancer, a seven percent increase for bladder cancer, and a nine percent increase for kidney cancer, while prostate cancer showed a non-significant five percent increase.

For NO_2, a 10 µg/m³ rise was correlated with a three percent higher overall risk and a four percent increase for bladder and prostate cancer. $PM_{2.5}$ was primarily associated with prostate cancer, whereas other pollutants such as NO_x, ozone, sulfur dioxide, and black carbon demonstrated limited or inconsistent associations.

Subgroup analyses indicated stronger associations in cohort studies and among males, and sensitivity analyses confirmed the robustness of these results. Importantly, population attributable fraction estimates suggested that reducing $PM_{2.5}$ by 5 µg/m³ could prevent approximately six percent of urological cancer cases globally, with the greatest benefit in high-pollution regions such as China, India, and Egypt.

The biological plausibility of these associations is supported by several mechanisms. Fine particulate matter penetrates deep into the lungs and enters systemic circulation, delivering carcinogenic components such as heavy metals and polycyclic aromatic hydrocarbons to multiple organs.

(Continued on page 5)

Assessing Pesticides in the Atmosphere: Global Pollution, Health Risks, and Regulatory Insights

Pesticides have become indispensable in modern agriculture, with global usage reaching approximately 2.7 million tonnes in 2022. While their residues in soil, water, and food have been extensively studied, the presence of pesticides in the atmosphere remains a relatively neglected area of research.

Airborne pesticides originate from spray drift during application, volatilization from plants and soil, and long-range atmospheric transport. These pollutants can irritate the respiratory system, impair immunity, and increase the risk of chronic diseases, including cancer and neurodegenerative disorders.

Despite the existence of regulatory frameworks in regions such as the European Union, the United States, China, and Brazil, specific air quality standards for pesticides are rare, leaving a significant gap in global monitoring and control.

The study aimed to quantify atmospheric pesticide pollution across thirty-eight countries using a scoring approach, assess theoretical health risks for different population groups, evaluate regulatory performance through pesticide application intensity and existing standards, and propose strategies for global monitoring and harmonization.

Data were compiled from sixty peer-reviewed publications and official monitoring programs covering the period from 2010 to 2023. Pollution scores were calculated using log-transformed concentrations of detected pesticides across multiple sampling sites, with adjustments for mixtures to ensure robustness. Health risk assessments employed Target Hazard Quotient and Hazard Index for non-carcinogenic effects and Total Carcinogenic Risk for cancer risk, considering infants, children, and adults. Regulatory analysis included pesticide application intensity derived from FAO data and a review of air quality standards.

The results revealed striking regional disparities. Countries in Asia and Oceania generally exhibited higher atmospheric pesticide scores than those in the Americas, while Europe showed wide variability. Moldova, Armenia, and France ranked among the highest, whereas New Zealand, Slovenia, and Italy had the lowest scores.

China, with a score of 0.15, reflected extensive monitoring coverage and significant pesticide use, while the United States and Brazil recorded relatively low scores despite their large-scale pesticide consumption.

Health risk assessments indicated that non-carcinogenic risks were negligible, as all hazard indices were well below one. However, carcinogenic risk exceeded the acceptable threshold in China and Croatia, primarily due to alpha-hexachlorocyclohexane (α-HCH), a persistent compound classified as a probable human carcinogen.

Epidemiological evidence further linked pesticide exposure to thyroid and breast cancers, leukemia, Parkinson's disease, dementia, and endocrine disorders.

Regulatory analysis showed that pesticide application intensity varied widely, with Malta, the Netherlands, and Italy reporting the highest values. Surprisingly, high application intensity did not consistently correspond to high atmospheric pollution scores, suggesting that factors such as pesticide volatility and long-range transport play significant roles. Few countries have established air quality standards for pesticides; the United States remains an exception, offering screening levels and risk assessment tools.

Monitoring gaps were evident, as sampling sites were unevenly distributed, particularly in Nordic countries and large nations like the United States, where state-level scores varied dramatically.

In summary, this global assessment underscores the urgent need for comprehensive airborne pesticide monitoring networks, harmonized air quality standards, and coordinated international regulatory efforts.

Although current exposure levels pose minimal health risks on average, the persistence of certain compounds and their association with severe health outcomes demand proactive measures. Strengthening integrated pest management, reducing pesticide use, and expanding monitoring infrastructure are critical steps toward mitigating airborne pesticide pollution and safeguarding both environmental and human health.

Source: Environment International, Vol. 187, Article 108653, May 2024.

Ambient Air Pollution and Urological Cancer Risk

(Continued from page 4)

Chronic exposure generates reactive oxygen species, causing oxidative stress and DNA damage, while persistent inflammation promotes tumor initiation and progression. Air pollution also induces epigenetic alterations, including abnormal DNA methylation, which can modify cancer-related pathways. Endothelial dysfunction caused by particulate matter may increase kidney susceptibility due to its high blood flow. Furthermore, PM carries toxic substances that activate oncogenic signaling and impair tumor suppressor genes such as p53 and RB, contributing to carcinogenesis.

This first comprehensive meta-analysis demonstrates that long-term exposure to $PM_{2.5}$ and NO_2 is associated with an increased risk of urological cancers, particularly bladder and kidney cancers. Reducing air pollution could substantially lower the global cancer burden.

Future research should prioritize high-pollution regions, rare urological cancer types, and improved exposure assessment methods.

Source: Nature Communications, Vol. 15, Article 5116, June 2024.

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IARC Monographs Volume 134:
Aspartame, Methyleugenol, and Isoeugenol

Aspartame and isoeugenol are possibly carcinogenic to humans (Group 2B)

Methyleugenol is probably carcinogenic to humans (Group 2A)

This volume of the IARC Monographs provides evaluations of the carcinogenicity of three agents: **aspartame, methyleugenol, and isoeugenol.**

Aspartame is a low-calorie artificial sweetener that has been widely used in foods and beverages since the 1980s. Historically, artificially sweetened beverages have been the major source of exposure to aspartame, but to a lesser extent at present since aspartame is typically used in mixtures with other sweeteners. The highest concentrations of aspartame are found in tabletop sweeteners, chewing gums, and food supplements. Other sources include cosmetics and medicines.

Methyleugenol is a flavour and fragrance compound that occurs naturally in essential oils of various plants. It is used in cosmetics and personal care products and as an insect attractant. Although its use as a flavouring agent is prohibited in the European Union and the USA, it is still present in various foods and consumer products due to its natural occurrence in herbs and spices. The general population is ubiquitously exposed through the ingestion of food or use of personal care products.

Isoeugenol is a fragrance and flavour compound that occurs in many plant species and in wood smoke. It is used in food, cosmetics, household products, animal feed, and veterinary medicines. Firefighters and workers involved in isoeugenol synthesis or handling isoeugenol-containing products may be exposed.

Source: IARC Monographs on the Identification of Carcinogenic Hazards to Humans Volume 134: Aspartame, Methyleugenol, and Isoeugenol, April 2024.

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WHO: Laboratory Biosecurity Guidance

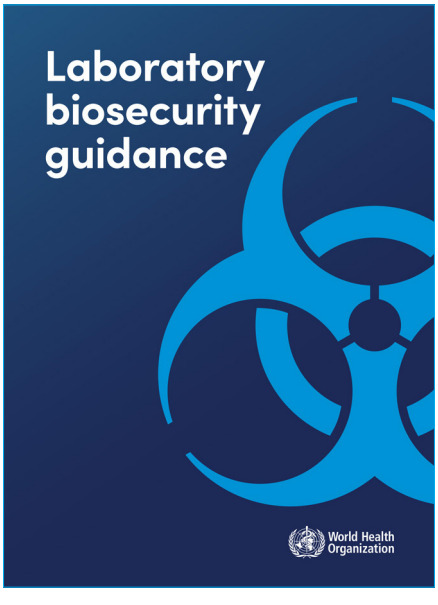
The World Health Organization's **Laboratory Biosecurity Guidance** provides a comprehensive framework to strengthen global capacity for managing biological risks in laboratories. It emphasizes that biosecurity and biosafety are interconnected elements of biological risk management, both essential for preventing accidental or deliberate misuse of high-consequence materials.

This updated guidance adopts a risk- and evidence-based approach, aligning with the principles introduced in the Laboratory Biosafety Manual (LBM4), and reflects lessons learned from the COVID-19 pandemic and rapid advances in life sciences.

The document addresses emerging challenges such as gain-of-function research, synthetic biology, genome editing, artificial intelligence, and do-it-yourself biology, all of which have expanded the biological threat landscape. It introduces practical tools, including decision trees and templates for biosecurity risk assessment, enabling institutions to identify vulnerabilities and implement locally relevant, sustainable risk control measures.

Key recommendations include establishing robust institutional biosecurity programs, integrating them with biosafety policies, and strengthening the role of Institutional Biosafety Committees (IBCs) alongside biosafety officers. The guidance also outlines measures for personnel reliability, physical security, inventory control, information security, and emergency preparedness.

At the national level, WHO advocates a two-tier oversight system combining institutional review with regulatory authority to ensure accountability and transparency. Ethical considerations, codes of conduct, and fostering a culture of responsibility are highlighted as critical components for mitigating risks. By providing clear strategies and practical templates, this guidance aims to safeguard scientific progress while minimizing biosecurity threats, ultimately supporting global health security and resilience.



Source: WHO Guidance, Laboratory Biosecurity Guidance, June 2024.

International Conference on

Environmental Pollutants and Toxicants Affecting Health: Collaborative Efforts for Improving Quality of Life

June 19 - 21, 2024

at Convention Center, Chulabhorn Research Institute, 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)

Collaborating Institutions:
Hub of Talents on Air pollution and Climate, Thailand
Health and Environmental Sciences Institute, U.S.A.

The international conference titled "**Environmental Pollutants and Health Impacts: Collaborations for Better Quality of Life**" was organized by the Center of Excellence on Environmental Health and Toxicology (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). The conference was held on June 19-21, 2024, at the Chulabhorn Research Institute Convention Center, Bangkok, Thailand.

Besides the Hub of Talents in Environmental Health, the event was also supported by the **Hub of Talents on Air Pollution and Climate** and the **U.S. Health and Environmental Sciences Institute (HESI)**, which provided funding for international participants, particularly from developing countries, to attend the conference and participate in the Environmental Toxicology Training Program held at the Chulabhorn Research Institute from June 24-28, 2024.

A total of 250 registered participants from 21 countries attended the conference, including 32 invited speakers from 13 countries with representative from governmental agencies, academic and research institutions, as well as graduate students (Master's and Ph.D. levels), who are expected to play key roles in environmental health in the future—whether in academia, research, or policy-making at national or international levels.

The conference aimed to foster collaboration within the Asia-Pacific region among countries facing similar environmental health challenges. It served as a platform for representatives from various countries to exchange views with environmental health experts on key topics, promoting cooperation. One tangible outcome of the conference was the development of a database of environmental health experts, categorized by key environmental health issues. This database will facilitate expert searches and support current and future collaborations.

The final session focused on establishing a **Collaborative Network in Environmental Health**, aiming to agree on methods for building and sustaining the network. Discussions included strategies for recruiting interested members, especially from the region. Two main topics were addressed: 1. Development of an Environmental Health Expert Database – Participants shared insights and experiences from similar database projects and discussed ways to link related databases. 2. Identifying Regional Coordinators – Representatives from relevant agencies in the region were sought to act as coordinators, providing information and connecting with other organizations.

CALENDAR OF EVENTS

International Training Courses on Environmental Health Risk Assessment and Management of Toxic Chemicals

	Training Course	Date	Duration	Closing Date
1	Environmental Health Risk Assessment and Management of Toxic Chemicals	December 9-14, 2024	6 work days	September 30, 2024

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

Course Description:

The course is an integration of science and policy, covering the fundamental basis of environmental and health risk assessment and management from exposure assessment and risk characterization; mode of action and human relevance framework; the relationship between risk assessment and risk management; and the need for open, transparent and participatory acceptance procedures and credible communication methods. Emphasis will be placed on human health risk assessment, 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, e.g. hazardous waste site release, through the use of case studies relevant to problems faced in developing countries, and describes the policy context in which decisions to manage environmental health risks are made. Teaching and learning aids, such as an electronic distance learning tool on risk assessment and risk management of chemicals and the WHO IPCS Human Health Risk Assessment toolkit will be introduced.

Requirement: Participants should have jobs/responsibilities related to the assessment of risk from the use of chemicals.

This year, the course will be held back-to-back with the **9th Princess Chulabhorn International Science Congress (PC IX) on "The Challenges of One Health: The Roles of Biosciences and Chemistry",** to be held at the Shangri-La Hotel, Bangkok, Thailand from December 15-18, 2024. More information, please visit the Congress website at https://pc9.cri.or.th.

Participants interested in attending both the training course and international science congress will be given special consideration.

Fellowships:

A limited number of fellowships are available that will cover roundtrip airfare, accommodation (on site) and meals, 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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