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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".

Endocrine-Disrupting Chemicals and Congenital Heart Diseases

Endocrine-disrupting chemicals (EDCs) are environmental contaminants that interfere with hormonal regulation, affecting multiple physiological systems. They are widely present in pesticides, plastics, plasticizers, and heavy metals. EDCs have been linked to chronic conditions such as obesity, diabetes, hypertension, and cardiovascular disease. Congenital heart disease (CHD), the most common congenital malformation, affects nearly 1% of live births globally and remains a major cause of infant morbidity and mortality. While genetic predisposition plays a role, environmental factors during pregnancy are increasingly recognized as critical determinants of fetal cardiac development.

Animal studies have demonstrated that EDCs can disrupt embryonic cardiovascular development through oxidative stress and hormonal imbalance. However, human evidence has been limited, often relying on self-reported exposure or occupational data rather than direct measurement of chemical concentrations.

This gap in knowledge underscores the need for robust epidemiological studies using biospecimen-based exposure assessment to clarify whether maternal EDC exposure contributes to CHD risk. Understanding this relationship is essential for developing preventive strategies and informing public health policies.

The systematic review and meta-analysis focused on determining whether maternal exposure to endocrine-disrupting chemicals, measured directly in human biospecimens during pregnancy, is linked to congenital heart disease in offspring. Seventeen case-control studies were included, primarily from China and one from Iran, that measured EDC concentrations in maternal samples such as blood, urine, and hair.

This is the first study that establishes associations between maternal EDC exposure and CHD based on epidemiological evidence of human biospecimens.

The analysis of seventeen case-control studies revealed a consistent link between maternal exposure to endocrine-disrupting chemicals and congenital heart disease in offspring. Mothers with higher levels of EDCs in biospecimens such as blood, urine, or hair were more likely to have children with heart defects compared to those with lower exposure. These defects included septal abnormalities, conotruncal malformations, right and left ventricular outflow tract obstructions, and anomalous pulmonary venous return.

Heavy metals such as lead, cadmium, mercury, and manganese emerged as the most concerning group of EDCs, showing strong associations with severe forms of CHD. Other chemicals, including polycyclic aromatic hydrocarbons, polyfluorinated alkyl substances, and triclosan, were also linked to increased risk, though their impact appeared less pronounced than that of metals.

In summary, maternal exposure to EDCs, particularly heavy metals, significantly increases the risk of CHD in offspring. This meta-analysis strengthens the evidence by using biospecimen-based measurements, but limitations such as heterogeneity and geographic concentration warrant caution.

Large-scale prospective studies with standardized exposure assessment and inclusion of diverse EDC classes are urgently needed to confirm causality and guide preventive strategies.

Source: Pediatric Cardiology, Vol. 46, Pages 628-638, April 2024.

Neurotoxic Effects of Heavy Metals: Epigenetic Mechanisms

Heavy metal pollution has become one of the most pressing environmental challenges worldwide, not only because of its persistence and widespread distribution but also due to its profound impact on human health. Among the most concerning consequences is its effect on the nervous system.

Metals such as manganese, mercury, lead, cobalt, cadmium, nickel, and silver, along with metalloids like arsenic, have been implicated in a range of neurodevelopmental disorders and neurodegenerative diseases, including Alzheimer's disease, Parkinson's disease, and attention deficit/hyperactivity disorder.

Traditionally, these effects have been attributed to mechanisms such as oxidative stress, mitochondrial dysfunction, and neuroinflammation. However, recent research has revealed that epigenetic alterations play a central role in mediating heavy metal-induced neurotoxicity.

Epigenetics refers to heritable changes in gene expression that occur without altering the underlying DNA sequence. These changes include DNA and RNA methylation, histone modifications, and the regulation of non-coding RNAs. They influence critical processes such as neuronal development, synaptic plasticity, and cellular stress responses.

Unlike genetic mutations, epigenetic modifications are reversible, which opens exciting possibilities for therapeutic intervention through epigenome editing, pharmacological agents, and even dietary strategies.

Studies have shown that heavy metals disrupt the epigenome through multiple pathways.

Manganese exposure, for example, leads to hypermethylation of promoters involved in neurogenesis, while lead exposure reduces the activity of DNA methyltransferases, impairing cognition and synaptic function. Histone modifications are another key mechanism: manganese and cobalt decrease histone acetylation by increasing the activity of histone deacetylases, which suppresses antioxidant defense pathways.

Mercury exposure, on the other hand, enhances histone methylation and deacetylation, repressing genes essential for neuronal survival. RNA methylation, particularly N6-methyladenosine (m6A), is also affected by cobalt and cadmium, influencing apoptosis and mitochondrial function. Non-coding RNAs, including microRNAs, circular RNAs, and long non-coding RNAs, further contribute to these effects by regulating autophagy, apoptosis, and neuroinflammation.

Maternal and early-life exposure to heavy metals adds another layer of complexity, as it can induce transgenerational epigenetic inheritance. This means that fetal programming may be altered in ways that affect neurobehavioral outcomes throughout life. Epidemiological studies have linked prenatal exposure to arsenic and lead with sex-specific changes in DNA methylation and histone profiles, correlating with cognitive deficits later in life. Emerging contaminants such as nanometallic materials, including silver and zinc oxide nanoparticles, also exhibit epigenetic neurotoxicity, raising concerns about their growing use in medicine and industry.

Recent research has expanded our understanding of these mechanisms and highlighted several critical areas for future investigation. Combined exposure to metals and other pollutants, such as microplastics, appears to amplify neurotoxic risks, yet remains poorly studied. Gender-specific epigenetic responses are increasingly recognized as a factor in differential susceptibility to neurological disorders. Therapeutic strategies targeting epigenetic modifications, such as DNA demethylation, histone deacetylase inhibitors, and microRNA modulators, show promise for reversing heavy metal-induced neuronal damage. Novel mechanisms, including lactate-driven histone modifications and the interplay between multiple epigenetic marks, represent cutting-edge directions for research. Despite these advances, significant gaps remain, particularly regarding essential trace elements like zinc, copper, and aluminum, and their epigenetic roles in neurotoxicity.

In conclusion, heavy metal pollutants exert profound and long-lasting effects on the nervous system through complex epigenetic disruptions. These changes influence gene expression, neuronal survival, and cognitive function, offering both challenges and opportunities.

A deeper understanding of these mechanisms could transform approaches to biomarker discovery, risk assessment, and the development of targeted interventions for neurodegenerative and neurodevelopmental disorders linked to environmental exposure.

Source: Environmental Pollution, Vol. 345, Article 123563, March 2024.

Human Serum Exposome and Chronic Disease Risk in Chinese Population

Chronic diseases such as cardiovascular disorders, diabetes, and metabolic syndrome are leading causes of morbidity and mortality worldwide. While genetic predisposition plays a role, mounting evidence suggests that environmental exposures exert an even greater influence on disease onset and progression. The concept of the "exposome," introduced as a complement to the genome, encompasses all environmental factors encountered throughout life and their cumulative impact on health. Persistent organic pollutants (POPs), pesticides, and perfluoroalkyl substances (PFASs) are widely distributed and implicated in metabolic and neurological disorders. However, most studies focus on single chemicals, neglecting real-world mixtures.

To address these gaps, this study constructed a comprehensive serum exposome atlas for the Chinese population. By analyzing 5,696 individuals across 15 provinces, including both healthy participants and patients with twelve chronic diseases, the research provides unprecedented insights into the distribution of environmental chemicals in human serum and their associations with

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Air Pollution and Risk of Chronic Kidney Disease in Diabetic Patients

Diabetes mellitus is a major global health challenge, ranking among the leading causes of disability-adjusted life years (DALYs) in older populations. Chronic kidney disease (CKD) is one of the most severe and costly complications of diabetes, affecting over 40% of diabetic patients and frequently progressing to end-stage renal disease (ESRD), which requires dialysis or transplantation. CKD not only increases morbidity and mortality but also amplifies cardiovascular risk, including ischemic heart disease and heart failure, thereby imposing a substantial burden on healthcare systems worldwide.

In recent decades, environmental factors such as air pollution have emerged as critical contributors to non-communicable diseases. Epidemiological evidence links exposure to particulate matter ($PM_{2.5}$, PM_{10}) and gaseous pollutants (NO_2, NO_x) with increased incidence of diabetes and impaired kidney function.

Fine particulate matter ($PM_{2.5}$), due to its small size, can penetrate deep into the respiratory tract and enter systemic circulation, triggering oxidative stress, systemic inflammation, endothelial dysfunction, and vascular injury, all mechanisms implicated in renal damage. Despite these findings, the specific relationship between air pollution and CKD onset in diabetic populations remains insufficiently studied and inconsistent across regions, particularly in areas with low pollution levels.

The present study addresses this gap by analyzing data from the UK Biobank, a large prospective cohort, to quantify the exposure-response relationship between key ambient air pollutants ($PM_{2.5}$, PM_{10}, $PM_{2.5-10}$, NO_2, and NO_x) and incident CKD in diabetic patients.

Furthermore, it extends the analysis globally for $PM_{2.5}$ exposure, providing insights into public health implications and mechanistic pathways underlying pollutant-induced renal injury.

The study demonstrated clear positive associations between exposure to fine particulate matter ($PM_{2.5}$), coarse particles (PM_{10}), and gaseous pollutants (NO_2 and NO_x) with the onset of chronic kidney disease (CKD) among individuals with diabetes. Participants exposed to higher concentrations of $PM_{2.5}$ and PM_{10} consistently exhibited greater CKD incidence compared to those in lower exposure categories, indicating a dose-response relationship.

In contrast, $PM_{2.5-10}$ showed no significant association, suggesting that smaller particles are more biologically active and capable of penetrating systemic circulation.

Exposure-response analyses revealed near-linear trends for $PM_{2.5}$, PM_{10}, NO_2, and NO_x within the UK cohort, while global modeling for $PM_{2.5}$ indicated a non-linear increase in CKD risk at higher concentrations. Geographic mapping highlighted urban regions such as London and Birmingham as hotspots for air pollution–related CKD burden, underscoring the public health relevance of these findings.

Air pollutants may contribute to kidney injury through multiple biological pathways. Fine particles and gaseous pollutants can induce oxidative stress and systemic inflammation, disrupt endothelial function, and cause DNA damage, ultimately impairing glomerular and tubular integrity. Additionally, particulate components interfere with the renin-angiotensin system and elevate markers of kidney injury. These mechanistic links provide strong biological plausibility for pollutant-driven CKD progression in vulnerable populations.

This study provides compelling evidence that ambient air pollution, particularly fine particulate matter and nitrogen oxides, significantly elevates the risk of chronic kidney disease among individuals with diabetes.

The findings highlight that even relatively low pollutant concentrations, as observed in the UK, can contribute to CKD onset, reinforcing the need for stringent air quality standards and targeted interventions for high-risk populations.

Source: Ecotoxicology and Environmental Safety, Vol. 270, Article 115829, January 2024.

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disease risk. Advanced analytical platforms enabled the quantification of 267 chemicals, with 74 high-frequency exposures selected for detailed risk modeling.

The study aimed to identify high-frequency exposures, assess regional and demographic determinants, and explore mixture effects on disease risk.

Geographical region was the most influential factor, explaining over fifteen percent of exposure variance, with industrialized coastal provinces showing the highest chemical burdens.

Age was the second major determinant, correlating with higher levels of POPs and PFASs. Serum concentrations of several chemicals exceeded international safety benchmarks, and emerging compounds like 6:2 chlorinated polyfluoroalkyl ether sulfonate were detected.

The study established robust associations between chemical exposures and chronic diseases, particularly hyperlipidemia, metabolic syndrome, and hyperuricemia. Hyperlipidemia was linked to multiple classes of pollutants, including pesticides, PFASs, and phthalates, with novel risk factors such as fipronil sulfone and PFHpS identified.

Metabolic syndrome, a cluster of conditions involving obesity, hypertension, and dyslipidemia, showed strong associations with mixtures of persistent pollutants and endocrine-disrupting chemicals like Monoethyl and Monocyclohexyl Phthalates.

Hyperuricemia, a precursor to gout and cardiovascular disease, was newly associated with indole-3-butyric acid and

(Continued on page 4)

Application of Human-Derived Cell Lines in Neurotoxicity Studies

Environmental pollution from industrial, agricultural, and domestic sources has become a major global concern, particularly due to its neurotoxic effects on humans.

Neurotoxicity refers to structural and functional impairment of the nervous system caused by exogenous compounds, which, during development, can lead to disorders such as autism, attention deficit, and intellectual disability.

Over 200 substances, including metals, pesticides, persistent organic pollutants (POPs), and solvents, are recognized neurotoxicants.

Traditional rodent models have been widely used for toxicity screening, but ethical issues and interspecies variability limit their relevance to human health risk assessment. Consequently, human-derived neural cell lines have emerged as promising *in vitro* models for studying neurotoxicity and developmental neural toxicity of environmental pollutants.

This review aims to summarize the application of human-derived cell lines in neurotoxicity research, focusing on immortalized neural cell lines such as SH-SY5Y and neural cells derived from human pluripotent stem cells. It discusses commonly used biological endpoints and assays, evaluates pollutant-induced neurotoxic effects, and explores future directions for improving *in vitro* models and detection methods.

The study synthesizes findings from multiple experimental approaches using human neural cell lines exposed to environmental pollutants. Immortalized cell lines, particularly SH-SY5Y neuroblastoma cells, were cultured under standard conditions and differentiated using agents like retinoic acid.

Biological endpoints assessed include cell viability, oxidative stress, cell cycle alterations, and apoptosis. Additional studies employed human pluripotent stem cell-derived neurons and primary neural cells to evaluate developmental toxicity.

Human-derived cell lines have proven effective in elucidating neurotoxic mechanisms of diverse pollutants. SH-SY5Y cells, expressing both immature and mature neuronal markers, are widely used to study pesticides, flame retardants, perfluoroalkyl and polyfluoroalkyl substances (PFASs) phenols, polycyclic aromatic hydrocarbons (PAHs), heavy metals, nanoparticles, and microplastics.

These pollutants commonly reduce cell viability, induce oxidative stress, disrupt mitochondrial function, and trigger apoptosis via caspase activation and autophagy pathways.

For instance, organochlorine and organophosphorus pesticides impair neuronal signaling by reducing acetylcholinesterase activity and dopaminergic markers, while flame retardants and PFASs alter neurotransmitter pathways and promote Alzheimer's disease-related protein expression.

Heavy metals such as lead and cadmium induce oxidative stress and DNA damage, whereas nanoparticles and microplastics cause mitochondrial dysfunction and autophagy.

Neural cells derived from pluripotent stem cells provide insights into developmental toxicity, revealing that compounds like bisphenol A and brominated flame retardants impair differentiation and axonal growth.

Despite their utility, current models are largely two-dimensional and fail to replicate complex *in vivo* microenvironments, limiting predictive accuracy.

Human-derived neural cell lines, particularly SH-SY5Y, are indispensable tools for assessing neurotoxicity of environmental pollutants. They enable mechanistic studies of cellular damage, oxidative stress, apoptosis, and disrupted neurotransmission. Stem cell-derived models offer advantages for developmental toxicity studies but require standardized differentiation protocols.

Advancing neurotoxicity research demands development of three-dimensional models, including brain organoids and bioprinted neural tissues, to better mimic human neural architecture. Incorporating multi-pollutant exposure scenarios and natural environmental samples will improve ecological relevance.

Source: Science of the Total Environment, Vol. 912, Article 168839, February 2025.

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phthalates, expanding the scope of environmental contributors to metabolic disorders.

Beyond single-exposure analyses, the study employed multi-exposure models to capture the synergistic effects of chemical mixtures. These models revealed that combined exposures exert non-negligible risk-enhancing effects, often stronger than those of individual chemicals. Dose-response curves demonstrated nonlinear relationships, with some chemicals showing rapid risk escalation even at low concentrations, emphasizing the complexity of real-world exposure scenarios.

Population susceptibility varied markedly. Elderly individuals and men exhibited heightened vulnerability to metabolic syndrome and hyperlipidemia, likely due to slower excretion rates and sex-specific metabolic differences. Alcohol consumption correlated with higher PFAS levels, suggesting that lifestyle factors can amplify chemical burdens.

This exposome atlas provides a benchmark for biomonitoring and highlights environmental determinants of chronic disease in China. The findings advocate for stricter regulation of persistent pollutants, targeted interventions for high-risk groups, and integration of mixture models in epidemiological research. By mapping serum chemical residues and linking them to disease risk, this study advances the exposome paradigm and lays the foundation for precision prevention strategies and future toxicological investigations.

Source: Nature Communications, Vol. 15, Article 2268, March 2024.

Urinary Cadmium Concentration and COVID-19 Severity

Coronavirus disease 2019 (COVID-19)., caused by severe acute respiratory syndrome corona-virus 2 (SARS-CoV-2), has led to a global health crisis with millions of deaths and severe socioeconomic disruption. Despite advances in treatment and vaccination, a significant proportion of patients still develop severe complications such as acute respiratory distress syndrome (ARDS), multi-organ failure, and death.

Identifying risk factors that predispose individuals to severe disease is essential for early intervention and improved outcomes. While age, comorbidities, and inflammatory markers have been widely studied, the role of environmental toxicants such as heavy metals remains poorly understood.

Cadmium and nickel are common environmental pollutants known to induce oxidative stress, inflammation, and immune dysfunction, mechanisms that overlap with the pathophysiology of severe COVID-19.

Cadmium, in particular, has a long biological half-life and accumulates in the body over decades, primarily in the kidneys, making urinary cadmium a reliable marker of chronic exposure.

Nickel, in contrast, reflects recent exposure due to its short half-life. Given the scarcity of evidence on how these metals influence COVID-19 severity, this research addresses a critical knowledge gap by exploring whether cadmium and nickel exposure correlates with worse clinical outcomes.

Understanding this relationship could help identify novel biomarkers for risk stratification and inform public health strategies to mitigate environmental risk factors during pandemics.

The objective of this study was to assess whether cadmium and nickel exposure, as reflected by blood and urinary concentrations, is associated with the severity and clinical outcomes of patients with COVID-19.

This retrospective multicenter cohort study was conducted in Taiwan between June 2022 and July 2023. A total of 574 patients with confirmed SARS-CoV-2 infection were enrolled and classified into severe and non-severe groups based on NIH criteria.

Blood and urine samples were collected within three days of diagnosis to measure cadmium and nickel concentrations using inductively coupled plasma mass spectrometry. Clinical data, including demographics, comorbidities, inflam-matory markers, and severity scores, were recorded.

Severe COVID-19 patients were older, had more comorbidities, and exhibited higher inflammatory markers compared to non-severe cases. Both blood and urinary cadmium concentrations were significantly higher in severe cases, while urinary nickel showed a modest increase and blood nickel did not differ significantly.

Urinary cadmium demonstrated a strong dose-response relationship with disease severity and outcomes. Patients in the highest quartile of urinary cadmium had markedly worse physiological scores, higher risks of hypoxemia, ARDS, shock, and invasive ventilation, and significantly higher mortality rates, reaching over forty percent.

Multivariable analysis confirmed urinary cadmium as an independent predictor of severe COVID-19, with a threshold of 2.05 μg/g creatinine offering the highest predictive value. Nickel showed weaker associations.

The biological plausibility of these findings lies in the shared pathways of oxidative stress and immune dysregulation triggered by both cadmium exposure and SARS-CoV-2 infection. Cadmium promotes the generation of reactive oxygen species (ROS), disrupts mitochondrial electron transport, and induces endoplasmic reticulum stress, leading to apoptosis and tissue injury. It also upregulates inflammatory cytokines such as IL-6 and TNF-α, contributing to a cytokine storm.

Chronic cadmium accumulation impairs both innate and adaptive immunity, reducing the body's ability to control viral replication and repair tissue damage. Nickel, although less cumulative, can also induce ROS and DNA damage, suppress antioxidant defenses, and activate pro-inflammatory signaling pathways.

SARS-CoV-2 infection itself causes endothelial dysfunction, oxidative stress, and hyper-inflammation. When combined with pre-existing cadmium burden, these effects may synergize, amplifying oxidative injury, promoting micro-vascular thrombosis, and accelerating multi-organ failure. The cumulative nature of cadmium, especially in older individuals, likely predisposes them to severe disease compared to nickel, which reflects only recent exposure.

In conclusion, urinary cadmium concentration measured early in the course of COVID-19 is strongly associated with disease severity and mortality, making it a potential biomarker for risk stratification. Public health strategies to reduce cadmium exposure through diet, smoking cessation, and environmental control are essential.

Further research is needed to confirm causality and explore whether interventions targeting oxidative stress could improve outcomes in patients with high cadmium burden.

Source: Environmental Health, Vol. 23, Article 29, March 2024.

WHO - Ethics and Governance of Artificial Intelligence for Health: Guidance on Large Multi-modal Models

The World Health Organization (WHO) has released new guidance on the ethics and governance of large multi-modal models (LMMs) — a rapidly growing form of generative artificial intelligence (AI) with potential applications across healthcare.

The guidance outlines five broad applications of LMMs for health: (1) Diagnosis and clinical care, such as responding to patients' written queries; (2) Patient-guided use, such as for investigating symptoms and treatment; (3) Clerical and administrative tasks, such as documenting and summarizing patient visits within electronic health records; (4) Medical and nursing education, including providing trainees with simulated patient encounters, and; (5) Scientific research and drug development, including to identify new compounds.

AI refers to algorithms that learn from data to perform tasks without explicit programming. Generative AI, a subset of AI, creates new content such as text, images, or video. LMMs can process multiple data types and generate diverse outputs, making them promising for healthcare, research, public health, and drug development. However, their ability to handle a wide range of tasks is still unproven.

While LMMs offer significant benefits, they also pose risks: misinformation, bias, overreliance on AI, cybersecurity threats, and unequal access. WHO emphasizes collaboration among governments, developers, healthcare professionals, and civil society to ensure safe and equitable use. Governments should invest in public infrastructure, enforce laws protecting rights and privacy, and require independent audits. Developers must involve stakeholders early, ensure transparency, and design models for well-defined tasks with predictable benefits.

Source: WHO Publication, Ethics and governance of artificial intelligence for health: Guidance on large multi-modal models, January 2024.

WHO Technical Advisory Group on Biosafety (TAG-B) : Meeting Report

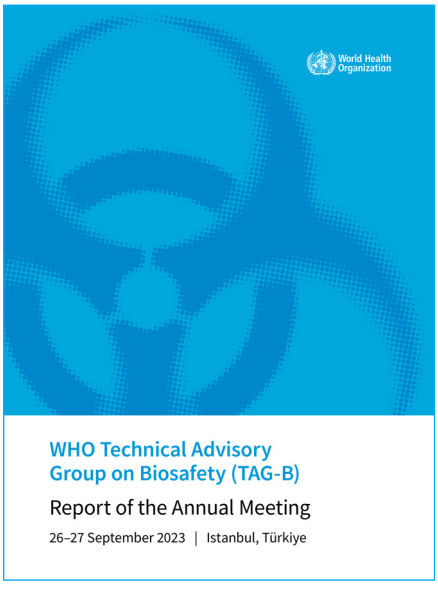
The World Health Organization (WHO) Technical Advisory Group on Biosafety (TAG-B) provides independent advice to WHO, including its strategic priorities and plans of action on specific topics relating to laboratory biosafety and biosecurity.

TAG-B members bring expertise from 16 countries representing all WHO regions and is further strengthened by gender representation. The report summarizes the presentations, discussions and recommendations of the TAG-B at their annual meeting held in Istanbul, Türkiye from 26-27 September 2023.

This meeting was convened to (1) Advance the work of the TAG-B by reviewing the progress of current projects and to facilitate in-depth and extensive discussions on these initiatives; (2) Meet with TAG-B Secretariat from WHO Headquarters, staff of Regional Offices and representatives of WHO Collaborating Centres to discuss perspectives and priorities, and identify Member States' needs related to laboratory biosafety and biosecurity; (3) Discuss and reach consensus on the strategy for the remainder of 2023 and priority actions for the TAG-B; and (4) Provide advice for laboratory biosafety/biosecurity strategies, priority projects, and deliverables for 2024-2025.

Key priorities and deliverables include: enhancing global focus on laboratory biosafety and biosecurity; strengthening capacity building at global, regional, and national levels; and fostering collaborations within the biosafety and biosecurity partner network.

For more information, please visit https://www.who.int/publications/m/item/who-technical-advisory-group-on-biosafety-report-of-the-annual-meeting-2023.



Source: WHO, Meeting Report of Technical Advisory Group on Biosafety (TAG-B), March 2024.

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.

Events	Date
International Training Course on Environmental Health Risk Assessment and Management of Toxic Chemicals	**December 9-14, 2024**
The 9th Princess Chulabhorn International Science Congress (PCIX) on "The Challenges of One Health: The Roles of Biosciences and Chemistry"	*December 15-18, 2024*

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.

Contact: Chulabhorn Research Institute (CRI)
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E-mail: envtox@cri.or.th

Calendar

More information and application:

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

The 9th Princess Chulabhorn International Science Congress

THE CHALLENGES OF ONE HEALTH: THE ROLES OF BIOSCIENCES AND CHEMISTRY

December 15-18, 2024 at Shangri-La Hotel, Bangkok, Thailand

Chairperson of the Organizing Committee: **Professor Dr. HRH Princess Chulabhorn Mahidol**

ANNOUNCEMENT AND CALL FOR ABSTRACTS

The Congress will be held **to commemorate the Sixth Cycle (72 years) Birthday Anniversary of His Majesty King Maha Vajiralongkorn Phra Vajiraklaochaoyuhua (Rama X)**, an auspicious occasion for the Thai people to celebrate and pay tribute to His Majesty. The program will feature a Nobel Laureate Lecture, Plenary Lectures, Symposia, Roundtable Discussion, Platform and Poster Presentations.

Invited Speakers (partial list)

Herman Autrup	(Denmark)
Matthew B. Avison	(U.K.)
Yu-Ju Chen	(Taiwan)
Peter Dedon	(U.S.A.)
Hashem B. El-Serag	(U.S.A.)
Tariq Enver	(U.K.)
John M. Essigmann	(U.S.A.)
Suthat Fucharoen	(Thailand)
George Fu Gao	(China)
Tim F. Greten	(U.S.A.)
John D. Groopman	(U.S.A.)
Curtis C. Harris	(U.S.A.)
John A. Hartley	(U.K.)
Paul Hunter	(U.K.)
Ho Jeong Kwon	(Republic of Korea)
Direk Limmathurotsakul	(Thailand)
Teck Yew Low	(Malaysia)
Tom Misteli	(U.S.A.)
Simona Parrinello	(U.K.)
Bradley L. Pentelute	(U.S.A.)
Yong Poovorawan	(Thailand)
Sittiruk Roytrakul	(Thailand)
Ram Sasisekharan	(U.S.A.)
Kwanrawee Sirikanchana	(Thailand)
Motoyuki Sugai	(Japan)
Charles Swanton	(U.K.)
Thipwimol Tim-Aroon	(Thailand)
Greg Towers	(U.K.)
Martin van den Berg	(The Netherlands)
Jennifer van Eyk	(U.S.A.)
Nithiwat Vatanavicharn	(Thailand)
Xin Wei Wang	(U.S.A.)
Pornswan Wasant	(Thailand)
Duangrurdee Wattanasirichaigoon	(Thailand)
John Yates III	(U.S.A.)
Maged Younes	(WHO)
Ari Zimran	(Israel)

Symposia (partial list)

- Antimicrobial Resistance: One Health Perspectives
- Communicable Diseases: One Health Perspectives
- Early Detection and Prevention of Cancer
- Environmental Risk Factors Affecting Health
- Genetic Diseases: From Detection to Therapy
- Protein Changes in Disease
- Relationships between Genetic Mutation and the Environment in Cancer and Infectious Diseases
- Role of Chemical and Biological Sciences for Discovery of Modern Drugs
- Zoonotic Diseases

All participants are invited to submit abstracts for **platform or poster presentations.** Authors should select the appropriate area(s) from the "List of Topics" on the Congress website. Selection of whether the submissions will be presented as platform or poster presentations will be made by the Scientific Program Committee.

Abstract Submission Deadline:
September 15, 2024

Registration

Congress Website:
https://pc9.cri.or.th

Phone: +66 2 553 8535

E-mail: pc@cri.or.th

Nobel Laureate Lecture:

Making New Medicines by Harnessingthe Power of Evolution

Sir Gregory P. Winter
(Nobel Prize in Chemistry 2018, U.K.)

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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