CRI/ICEIT NEWSLETTER

VOL. 36 NO. 2 – April 2026
ISSN 0858-2793
BANGKOK, THAILAND

Chulabhorn Research Institute

INTERNATIONAL CENTRE FOR ENVIRONMENTAL AND INDUSTRIAL TOXICOLOGY (ICEIT)

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

Air Pollution and Long COVID

Long COVID, also known as post-COVID-19 condition, refers to the persistence or emergence of symptoms beyond three months after initial SARS-CoV-2 infection, without an alternative explanation. It affects approximately 10-20% of individuals recovering from COVID-19 and presents with a wide spectrum of symptoms, including dyspnea, fatigue, and sleep disturbances.

Beyond subjective symptoms, increasing evidence indicates that long COVID may involve objective physiological and structural lung abnormalities, such as impaired pulmonary function and radiological changes detectable on chest computed tomography (CT) scans. Previous studies have shown that many patients exhibit reduced diffusion capacity of the lungs for carbon monoxide (DLCO) and structural abnormalities like ground-glass opacities, fibrotic changes, and airway disease.

Air pollution ($PM_{2.5}$, PM_{10}, NO_2) is a known respiratory risk factor associated with severe acute COVID-19, but its role in long COVID remains underexplored and largely limited to self-reported outcomes.

Most prior studies use ambient or residential air pollution estimates, which may not reflect true individual exposure influenced by daily activities and environments. This study addresses this gap by examining how ambient, residential, and personal exposure measures relate to lung health in long COVID patients.

A cohort of 95 patients was assessed 3-6 months post-infection using spirometry (FEV1, FVC, FEV1/FVC), DLCO, and chest CT scans, with follow-up in 38 patients after about nine months. Air pollution exposure included modeled ambient $PM_{2.5}$, PM_{10}, NO_2, and O_3, along with residential and personal $PM_{2.5}$ measurements.

No significant associations were found between ambient or residential pollution and lung outcomes at baseline, suggesting these measures may not capture individual risk in long COVID.

Lung function (FEV1, FVC, and DLCO) generally improved over time, but higher pollution exposure was linked to less recovery, suggesting a subtle negative effect.

The key finding was a significant link between personal $PM_{2.5}$ exposure and persistent radiological abnormalities, especially airway disease. Higher personal exposure increased the likelihood of follow-up abnormalities such as mosaic attenuation, suggesting ongoing small airway dysfunction after COVID-19.

Notably, personal $PM_{2.5}$ levels were higher and only weakly correlated with residential estimates, highlighting that the importance of individual behaviors and microenvironments (e.g., indoor or occupational exposure) may play a key role in determining health outcomes.

Overall, the findings suggest that personal-level exposure provides a more accurate indicator of the impact of air pollution on long COVID lung pathology. These results also reinforce the importance of environmental factors in the persistence of post-COVID complications.

Future research should focus on personal exposure assessment, long-term follow-up, and biological pathways, while public health efforts should prioritize air quality improvement to help mitigate long COVID outcomes.

Source: Environmental Research, Vol. 286, Article 122707, December 2025.

Rice Cadmium Exposure and Liver Cancer

Primary liver cancer is a major global health problem and is particularly prevalent in China, where it ranks among the leading causes of cancer-related death. Guangxi Province is a high-risk region, showing incidence rates far above the national average. While established risk factors such as hepatitis B virus infection, alcohol intake, metabolic diseases, and aflatoxin exposure are well documented, increasing attention has been given to environmental carcinogens.

Cadmium (Cd), classified as a Group I human carcinogen, can enter the human body through inhalation and dietary intake, accumulate in the liver, and induce chronic liver injury. Rice, as a staple food in southern China, represents a major source of dietary cadmium exposure. Although experimental studies suggest a link between cadmium and liver toxicity, there is still a lack of large-scale epidemiological evidence demonstrating the relationship between long-term, low-level cadmium exposure from rice and liver cancer at the population level.

The present study aimed to clarify the relationship between cadmium exposure and liver cancer in Guangxi. Specifically, it sought to investigate the spatial association between rice cadmium levels and liver cancer incidence, to compare internal cadmium exposure between liver cancer patients and healthy individuals, and to determine whether rice consumption is the primary pathway for cadmium exposure in the local population. The study also aimed to provide evidence for understanding the potential role of environmental cadmium in shaping the geographic distribution of liver cancer.

Liver cancer incidence data and rice cadmium concentrations were collected from 44 counties and districts across Guangxi. Blood samples were obtained from 105 diagnosed liver cancer patients and 105 healthy controls to assess internal cadmium exposure.

To further explore exposure pathways, paired sampling of blood and rice was conducted in five representative areas, yielding 316 blood samples and 216 rice samples.

The results revealed a clear spatial clustering of liver cancer incidence across Guangxi, with high-incidence areas largely overlapping regions with elevated cadmium concentrations in rice.

Statistical analysis demonstrated a significant positive correlation between rice cadmium levels and liver cancer incidence, particularly after accounting for regional variation.

At the individual level, liver cancer patients showed markedly higher blood cadmium concentrations compared with healthy controls, with average levels approximately two times greater. A substantial proportion of patients had blood cadmium levels exceeding the average observed in the general population.

Further analysis in the five representative areas showed a strong positive relationship between cadmium concentrations in rice and those in human blood. Areas with higher rice cadmium levels consistently exhibited higher blood cadmium concentrations, indicating strong agreement between external and internal exposure. This pattern supports the conclusion that rice consumption is the dominant pathway for cadmium exposure in the Guangxi population.

Although the overall trend was positive, some regional inconsistencies were observed, particularly in southwestern Guangxi, where high liver cancer incidence occurred despite relatively low rice cadmium levels. This suggests the influence of additional factors such as hepatitis B infection, aflatoxin exposure, and environmental toxins.

The findings of this study provide integrated evidence linking environmental cadmium exposure from rice to liver cancer risk in Guangxi. The spatial overlap between high cadmium levels in rice and high liver cancer incidence suggests that dietary cadmium may contribute to the geographic pattern of the disease. The elevated blood cadmium levels observed in liver cancer patients further support the role of internal cadmium exposure in disease development.

The strong correlation between rice cadmium and blood cadmium indicates that rice is a key exposure pathway, reinforcing the importance of dietary intake as a source of environmental contamination. These findings are consistent with previous toxicological and epidemiological studies showing that cadmium accumulates in the liver and contributes to carcinogenesis.

However, liver cancer is a multifactorial disease, and the observed relationships cannot be attributed solely to cadmium exposure. Other risk factors, including viral infections, mycotoxins, and lifestyle factors, likely interact with cadmium to influence disease risk. The observed regional differences highlight the complexity of these interactions and the need for comprehensive analysis.

Despite providing valuable insights, the study has several limitations that indicate directions for future research. Larger and more comprehensive cohort studies with long-term follow-up are needed to confirm causal relationships between cadmium exposure and liver cancer. Future studies should include detailed adjustment for confounding factors such as hepatitis virus infection, aflatoxin exposure, age, gender, and lifestyle variables.

In addition, mechanistic studies using cellular and animal models are required to better understand how cadmium contributes to liver carcinogenesis at the molecular level. Improved spatial matching between environmental samples and individual exposure data would also strengthen conclusions. Expanding the number of study regions and increasing sample sizes will enhance statistical power and allow more robust dose–response analysis.

Ultimately, these efforts will support the development of effective public health strategies, particularly those aimed at reducing cadmium contamination in rice and minimizing dietary exposure in high-risk regions.

Source: Environmental Geochemistry and Health, Vol. 48, Article 183, February 2026.

PFAS Epidemiology: From Exposure to Mitigation

Per- and polyfluoroalkyl substances (PFAS) are synthetic chemicals that have been widely used since the 1940s because of their unique properties, such as resistance to heat, water, and oil. These characteristics make them useful in industrial processes and everyday products, including non-stick cookware and waterproof materials. However, the same stability that makes PFAS useful also makes them highly persistent in the environment. Over time, they have accumulated in water, soil, air, and living organisms worldwide.

PFAS can be divided into long-chain and short-chain compounds. Both types can remain in the human body for long periods and accumulate in different organs. Exposure occurs mainly through contaminated food, drinking water, and air.

Increasing evidence shows that PFAS are associated with various health problems, including liver disease, cancer, reproductive issues, immune dysfunction, and effects on the nervous system.

This review aims to provide a comprehensive understanding of PFAS and their impacts on both human health and the environment. Specifically, it seeks to critically evaluate the health effects associated with PFAS exposure by integrating evidence from epidemiological and experimental studies. In addition, the review compares legacy PFAS compounds with emerging alternatives to assess differences in persistence, bioaccumulation, and toxicity. It also examines current global regulatory frameworks and policy measures implemented to mitigate PFAS exposure. Through these objectives, the study intends to inform and support evidence-based decision-making for the effective management and control of these persistent environmental contaminants.

Although economic and technological development has improved human life, it has also increased environmental pollution, especially from emerging contaminants like PFAS.

The review explains the sources, environmental behavior, and health risks of PFAS in a systematic way. It also compares older PFAS compounds with newer alternatives and evaluates their safety, while reviewing control policies worldwide to support the "One Health" approach, which integrates environmental, animal, and human health.

The results show that PFAS are widely distributed in the environment and can easily move through water, air, and food chains, eventually reaching humans. Short-chain PFAS tend to spread more easily in water, while long-chain PFAS accumulate more in sediments and living organisms. Because of this, humans are continuously exposed to these substances.

Many epidemiological studies have linked PFAS exposure to liver disease, particularly non-alcoholic fatty liver disease, often showing a clear dose-response relationship. PFAS also affect the reproductive system by reducing fertility, altering hormone levels, delaying puberty, and impairing fetal development. In addition, they are associated with neurodevelopmental problems such as attention disorders and behavioral changes, although findings are sometimes inconsistent depending on study design and population.

PFAS exposure also weakens the immune system, leading to reduced vaccine response and increased susceptibility to infections. Furthermore, strong evidence links PFAS to several types of cancer, including liver, thyroid, kidney, and breast cancer. Some compounds, such as PFOA (perfluorooctanoic acid), have been classified as carcinogenic to humans.

At the biological level, PFAS affect multiple pathways, including lipid metabolism, hormone regulation, immune signaling, oxidative stress, and gene expression. Although newer PFAS alternatives have been introduced to replace older compounds, recent evidence suggests that these substitutes may also persist in the environment and may not be as safe as initially believed.

Global regulations have helped reduce some PFAS levels, especially for well-known compounds like PFOS (perfluorooctane sulfonate) and PFOA. However, the growing use of newer PFAS compounds continues to pose challenges for monitoring and risk assessment.

Future research should focus on long-term studies that can better establish cause-and-effect relationships between PFAS exposure and disease. There is a need to study newer PFAS compounds and mixtures of chemicals, since real-world exposure usually involves multiple substances. Improved monitoring systems and standardized testing methods are also necessary to better assess exposure levels.

Special attention should be given to vulnerable populations such as pregnant women, children, and workers with high exposure risks. Researchers should integrate epidemiological data with mechanistic and molecular studies to better understand how PFAS cause disease. In addition, stronger international collaboration and stricter regulations are needed. Finally, developing safer chemical alternatives through green chemistry is essential to prevent repeating the cycle of replacing harmful chemicals with equally risky substitutes.

Source: Ecotoxicology and Environmental Safety, Vol. 314, Article 120050, April 2026.

Chlorpyrifos Increases the Risk of Parkinson's Disease

Parkinson's disease (PD) is a progressive neurodegenerative disorder marked by the loss of dopaminergic neurons in the substantia nigra, leading to motor impairment and accumulation of pathological α synuclein.

Although a small proportion of cases are caused by genetic mutations, most cases are believed to result from interactions between genetic susceptibility and environmental factors. Among these, pesticide exposure has been consistently linked to increased PD risk, but the specific agents and mechanisms involved are still not fully understood.

Chlorpyrifos (CPF), an organo-phosphate pesticide widely used in agriculture, has been linked to neuro-developmental toxicity, but its relationship to PD and the underlying biological mechanisms has remained insufficiently defined.

The present study aimed to determine whether long-term environmental exposure to CPF is associated with an increased risk of developing PD and to evaluate the biological plausibility of this association using human epidemiological data and experimental animal models.

The Parkinson's Environment and Genes (PEG) study, a large population-based case–control study conducted by the University of California, Los Angeles (UCLA) including 829 PD patients and 824 controls was used to assess CPF exposure through geocoded residential and workplace proximity to pesticide application.

Experimental validation was performed using mice exposed to CPF via inhalation for 11 weeks, followed by behavioral and neuropathological analyses.

Mechanistic studies were conducted using transgenic zebrafish to investigate CPF-induced neurotoxicity and underlying molecular pathways.

Long-term exposure to CPF was associated with more than a 2.5-fold increased risk of developing PD. In mice, CPF exposure caused motor deficits, dopaminergic neuron loss, microglial activation, and increased pathological α-synuclein accumulation.

Zebrafish studies demonstrated that CPF-induced neurotoxicity involved impaired autophagic flux and accumulation of synuclein proteins. Disruption of autophagy contributed to neuronal vulnerability, while restoration of autophagic function or reduction of synuclein mitigated neuronal damage.

These findings support both the epidemiological association and the biological plausibility of CPF as a causal environmental risk factor for PD, highlighting autophagy and synuclein pathways as key mechanisms and potential therapeutic targets.

Further studies are needed to better characterize long-term human exposure levels, clarify the role of CPF among mixed pesticide exposures, and investigate therapeutic strategies targeting autophagy and synuclein accumulation. Additional research in human populations and chronic exposure models will help establish causality more definitively and guide public health interventions.

Source: Molecular Neurodegeneration, Vol. 21, Article 3, January 2026.

Preterm Birth Risk from Prenatal $PM_{2.5}$ and Heavy Metal Exposure

Preterm birth (PTB), defined as delivery before 37 weeks of gestation, is a major global public health problem and a leading cause of mortality among children under five years old. Its burden is particularly high in developing countries such as China.

The etiology of PTB is complex and involves both genetic and environmental factors. Increasing evidence suggests that prenatal exposure to environmental pollutants, including fine particulate matter ($PM_{2.5}$) and heavy metals, may contribute to PTB risk through mechanisms such as inflammation, oxidative stress, and impaired placental function.

However, most previous studies have focused on single exposures, while real-world exposures occur as mixtures of multiple pollutants that may interact synergistically or antagonistically.

The study aimed to evaluate the joint and interactive effects of prenatal exposure to $PM_{2.5}$ and multiple trace elements on the risk of preterm birth. It also sought to identify key contributors within exposure mixtures and explore nonlinear and interaction effects using advanced mixture modeling approaches.

A nested case–control study was conducted within a birth cohort in Chongqing, China, including 111 PTB cases and 124 matched controls. Maternal blood samples collected in the second trimester were analyzed for concentrations of 18 trace elements using inductively coupled plasma mass spectrometry. $PM_{2.5}$ exposure was estimated using high-resolution spatiotemporal models based on residential addresses and categorized by pregnancy windows, including the first trimester, second trimester, and early-to-mid pregnancy.

The results showed that higher exposure to $PM_{2.5}$, particularly during the second trimester and early-to-mid pregnancy, was significantly associated with an increased risk of PTB. Among trace elements, cadmium (Cd), selenium (Se), and zinc (Zn) were positively associated with PTB risk, whereas lithium (Li) and nickel (Ni) showed inverse associations.

Mixture analyses revealed that combined exposure to $PM_{2.5}$ and metals significantly increased PTB risk. $PM_{2.5}$ contributed the largest weight to the risk-increasing mixture, while Li and Ni contributed most strongly to protective associations. Grouped analyses showed that $PM_{2.5}$ and essential trace elements, such as Zn, were positively associated with PTB, while conditionally essential trace elements, including Li, were inversely associated.

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Prenatal Metal Mixtures and Allergic Symptoms in Childhood

Allergic diseases, including atopic dermatitis and allergic rhinitis, have increased markedly among children worldwide and represent an important public health concern because they are linked to later development of asthma and reduced quality of life.

Prenatal development is a particularly vulnerable period, as exposure to environmental metals can occur through maternal circulation, cross the placenta, and interfere with immune system maturation via oxidative stress, inflammation, and dysregulation of T cell differentiation.

Although epidemiological studies have associated individual prenatal metal exposures with childhood allergic outcomes, humans are exposed to multiple metals simultaneously. Evidence on the health effects of prenatal metal mixtures, particularly in Latin American populations and across different stages of childhood, remains limited.

This study aimed to evaluate whether prenatal exposure to a mixture of arsenic, cadmium, cobalt, copper, manganese, nickel, and lead during the second and third trimesters of pregnancy is associated with allergic symptoms in early and mid childhood. An additional objective was to identify which metals contributed most strongly to any observed mixture effects.

The analysis was conducted within the Programming Research in Obesity, Growth, Environment and Social Stressors (PROGRESS) prospective birth cohort in Mexico City and included 542 mother–child dyads with complete exposure, outcome, and covariate data.

Maternal whole blood samples collected during the second and third trimesters were analyzed for seven metals using inductively coupled plasma mass spectrometry.

Childhood allergic outcomes, including atopic dermatitis and allergic rhinitis symptoms, were assessed at ages 4-6 years and 6-8 years using the validated International Study of Asthma and Allergies in Childhood questionnaire.

Metal mixture effects were estimated using weighted quantile sum regression with repeated holdout validation, adjusting for relevant maternal and child characteristics.

Prenatal exposure to metal mixtures was consistently associated with an increased risk of allergic rhinitis symptoms, particularly in mid childhood. Second trimester metal mixtures were associated with higher odds of ever and current allergic rhinitis at ages 6-8 years, with manganese, copper, and lead contributing most strongly to the mixture effect. Third trimester mixtures were also associated with increased allergic rhinitis symptoms at both childhood assessments, with arsenic and nickel emerging as important contributors alongside copper and manganese. In contrast, no consistent associations were observed between prenatal metal mixtures and atopic dermatitis.

These findings are biologically plausible because the second and third trimesters coincide with critical stages of immune development, including T cell maturation, lymphoid organ formation, and maternal antibody transfer. Essential metals such as copper and manganese can become prooxidant at elevated levels, while toxic metals such as arsenic, lead, and nickel are known to alter cytokine production and skew immune responses toward Th2 dominant, allergy prone profiles.

Additional studies should determine whether the observed associations persist into adolescence and adulthood and should incorporate clinical diagnoses, immune biomarkers, and detailed metal speciation. Research examining interactions between metal mixtures and other environmental exposures, such as air pollution or endocrine disrupting chemicals, is also warranted. These findings highlight the need for public health and regulatory strategies that address combined prenatal metal exposures to reduce the burden of childhood allergic disease.

Source: Science of the Total Environment, Vol. 1006, Article 180896, December 2025.

Preterm Birth Risk from Prenatal $PM_{2.5}$ and Heavy Metal Exposure

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Interaction analyses suggested a synergistic effect between $PM_{2.5}$ and essential trace elements, substantially increasing PTB risk, whereas interactions with conditionally essential elements appeared to mitigate risk. Bayesian kernel machine regression (BKMR) further demonstrated nonlinear exposure-response relationships, including a non-monotonic effect of Li, suggesting threshold or saturation effects.

The discussion highlights that environmental exposures during pregnancy act in complex mixtures rather than in isolation. The findings emphasize both harmful and potentially protective roles of specific elements, with biological plausibility linked to oxidative stress, inflammation, and nutrient-related mechanisms.

The study concludes that prenatal exposure to $PM_{2.5}$ and heavy metal mixtures is significantly associated with preterm birth risk, with evidence of complex, nonlinear, and interactive effects. $PM_{2.5}$ and certain metals such as Cd, Zn, and Se were identified as risk-enhancing factors, while Li and Ni showed potential protective associations.

Future research should focus on large-scale, multicenter cohort studies with repeated exposure measurements and broader pollutant coverage to strengthen causal inference. Mechanistic and experimental studies are also needed to clarify biological pathways and dose-specific effects. Improved exposure assessment is recommended to enhance accuracy and inform targeted public health interventions.

Source: Frontiers in Public Health, Vol. 13, Article 1701012, January 2026.

Modern Human Physiologically Based Toxicokinetic Model of Cadmium

Cadmium (Cd) is a widespread environmental heavy metal pollutant that accumulates in the human body through dietary intake, inhalation, and cigarette smoking, leading to serious health effects such as kidney damage, cardiovascular disease, and cancer.

Traditional physiologically based toxicokinetic (PBTK) models for cadmium, largely derived from the Kjellström and Nordberg (1978) framework, have been widely applied for risk assessment. However, these models contain several structural and mechanistic limitations, including inadequate representation of physiological blood flow, simplified assumptions about cadmium transport and excretion, and inconsistencies in modeling blood cadmium fractions. These limitations reduce their accuracy and restrict their application in modern risk assessment and population-based modeling.

The present study aimed to modernize the human cadmium PBTK model by improving physiological realism and addressing limitations of earlier models. It sought to enhance predictions of cadmium distribution, accumulation, and excretion across tissues over the human lifespan, supporting more robust risk assessment.

The model retained a multi-compartment structure but introduced key improvements, including tissue-specific blood flow, separation of tissue blood and compartments, and physiologically accurate gastrointestinal absorption via the liver. A kidney filtrate compartment was added to better represent renal processes. Cadmium kinetics were refined by incorporating reversible binding to plasma proteins and interactions with metallothionein in liver and kidney.

Calibration used U.S. population data, including biomonitoring data from the National Health and Nutrition Examination Survey (NHANES) 2013–2014, autopsy-derived tissue concentrations, and exposure estimates.

The updated model demonstrated improved performance over existing cadmium PBTK models, accurately reproducing cadmium concentrations in blood, urine, liver, and kidney across ages, sexes, and smoking statuses, generally within two-fold of observed values. Unlike earlier models, it avoided overestimation and unrealistic spikes in blood cadmium at young ages.

It also provided a more physiologically consistent representation of cadmium in blood, correctly predicting that most cadmium resides in red blood cells, with smaller fractions bound to plasma proteins and minimal free cadmium. Tissue distributions aligned with known biology, showing significant accumulation in the kidney and liver and increasing body burden over time.

The model captured lifetime dynamics, indicating that cadmium is largely retained in tissues at younger ages, while excretion via urine and feces increases with aging and may exceed daily absorption. Long biological half-lives, particularly in the kidney, were also predicted. Sensitivity analysis identified key drivers of cadmium kinetics, including gastrointestinal absorption and blood–tissue transport.

Collectively, the modernized model improves accuracy, enhances mechanistic understanding, and supports more reliable risk assessment. Future work should address exposure variability, particularly in smokers, and expand to population-based and longitudinal modeling frameworks.

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

Exposure to 1,3-Butadiene and Toluene and Its Link to Kidney Function in School-Aged Children

Rapid industrialization has increased environmental contamination, particularly from volatile organic compounds (VOCs) such as 1,3-butadiene and toluene, which are widely emitted from petrochemical activities, traffic, and fuel combustion. These compounds are readily inhaled and metabolized into reactive intermediates that can cause systemic toxicity, including kidney injury.

Children are especially vulnerable due to immature metabolic and excretory systems, which prolong toxicant retention, as well as higher exposure from behavioral factors such as increased outdoor activity and respiration rate.

1,3-butadiene is classified as a human carcinogen due to its genotoxic metabolites, while toluene, although non-carcinogenic, has been linked to renal dysfunction. Both compounds share metabolic pathways that can generate oxidative stress and organ damage.

The present study aimed to investigate whether exposure to 1,3-butadiene and toluene, measured through urinary metabolites, is associated with kidney function in school-aged children. It also sought to evaluate whether living closer to a petrochemical complex increases exposure and whether these exposures are related to early signs of renal impairment.

The study included 312 children aged 6 to 12 years living at varying distances from a petrochemical complex in Taiwan. Urine and blood samples were collected to measure metabolites of 1,3-butadiene and toluene using advanced analytical techniques. Kidney function was assessed using multiple indicators, including blood urea nitrogen, estimated glomerular filtration rate (eGFR), neutrophil gelatinase-associated lipocalin (NGAL), and cystatin C.

Statistical analyses, including regression and correlation methods, were used to examine relationships between exposure levels and kidney function while controlling for potential confounding factors such as age, sex, body mass index, and environmental exposures.

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WHO's Report:
Throwing Away Our Health: The Impact of Solid Waste on Human Health – Evidence, Knowledge Gaps, and Health Sector Responses

The WHO's report **"Throwing Away Our Health: The Impact of Solid Waste on Human Health – Evidence, Knowledge Gaps, and Health Sector Responses"** summarizes evidence on the links between municipal solid waste and human health, including waste types, management practices, hazards, exposure pathways, and affected populations.

The report reviews existing epidemiological evidence linking mismanaged waste to respiratory illness, vector borne disease, injuries, and chronic health risks, while emphasizing significant knowledge gaps that hinder effective policymaking. It stresses that sustainable, integrated waste management systems, clear governance, stronger enforcement, and health sector engagement are urgently needed to prevent harm.

WHO highlights the central role of the health sector in reducing health care waste at the source, improving safe treatment and disposal systems, advocating for health protective policies, and supporting climate resilient technologies. Ultimately, the report calls for coordinated multisectoral action, guided by the waste hierarchy of prevention, reduction, reuse, recycling, and safe disposal, to safeguard health, reduce inequity, and transform waste from a public health burden into an opportunity for cleaner environments and greener economies.

Source: WHO Publication. Throwing Away Our Health: The Impact of Solid Waste on Human Health – Evidence, Knowledge Gaps, and Health Sector Responses. 16 December 2025.

Exposure to 1,3-Butadiene and Toluene and Its Link to Kidney Function in School-Aged Children

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Children residing closer to the petrochemical complex exhibited significantly elevated levels of renal injury biomarkers, particularly NGAL and cystatin C, indicating increased kidney stress and early tubular damage.

Urinary metabolites of 1,3-butadiene and toluene were significantly associated with multiple kidney function indices. Specifically, trihydroxybutyl mercapturic acid (THBMA), a metabolite of 1,3-butadiene, and benzylmercapturic acid-2 (BMA-2), a metabolite of toluene, showed positive associations with blood urea nitrogen (BUN), suggesting altered nitrogen metabolism and early renal stress. In addition, dihydroxybutyl mercapturic acid (DHBMA), a biomarker of exposure to 1,3-butadiene, was positively associated with eGFR and creatinine clearance, indicating the presence of glomerular hyperfiltration.

These findings suggest the presence of glomerular hyperfiltration, an early response of the kidney to stress, which may precede long-term damage. Additionally, higher exposure levels were associated with increased risk of abnormal kidney function markers, indicating a potential health risk even at relatively low environmental exposure levels.

The study provides evidence that environmental exposure to volatile organic compounds can affect kidney function in children. The associations between exposure biomarkers and renal indicators suggest that even low-level, chronic exposure may lead to early kidney changes, such as hyperfiltration. Although hyperfiltration may initially act as a compensatory mechanism, it can contribute to long-term nephron damage and a subsequent decline in kidney function.

The findings further indicate that, although proximity to petrochemical complexes contributes to exposure, other sources such as traffic emissions and solvent use may also play important roles.

In conclusion, the results demonstrate that environmental exposure to volatile organic compounds is associated with early alterations in kidney function in children, increasing the risk of long-term renal impairment. These findings underscore the need for improved environmental monitoring and preventive strategies.

Further research is needed to confirm these findings through larger and long-term studies that track kidney function over time. Future work should include more precise environmental measurements of pollutant levels and explore additional biological markers to improve assessment of kidney injury.

Studies incorporating genetic and mechanistic analyses would help clarify how these chemicals affect renal health. Expanding sample size and improving exposure assessment will strengthen conclusions and support the development of effective public health strategies to reduce environmental risks for children.

Source: Environmental Pollution, Vol. 389, Article 127400, April 2026.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute Scheduled for Year 2026

	Training Course	Date	Closing Date
1	Environmental Health and Toxicology	May 18-22, 2026	April 22, 2026
2	Metabolomics for Drugs Discovery and Development	September 21-25, 2026	July 21, 2026
3	Environmental Health Risk Assessment and Management of Toxic Chemicals	November - December 2026	October 2026

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

Environmental Health and Toxicology (May 18-22, 2026)

The Chulabhorn Research Institute (CRI) is aware of the importance of providing a training program to assist developing countries with human resource development in the fields of environmental toxicology and environmental health. In May 2026, CRI is organizing a training course on “Environmental Toxicology and Health” for participants from developing countries in the Asia Pacific region.

Course Description:

This course provides students and participants with a background of the major groups of toxic substances encountered through food and the environment, as well as through exposures in the workplace. These toxicants include toxic substances in air, water, soil and food; pesticides; persistent environmental pollutants; and hazardous wastes. It also covers emerging threats, such as climate change and antimicrobial resistance and their impacts on human health. The course focuses on the chemistry, fate and distribution in the environment, mechanisms of action, and toxicity. The course also provides information on the latest technologies used to study changes and effects in biological systems, e.g., biomarkers, omics technologies, gene-environment interactions, epigenetics and transgenic models. The course combines the use of traditional lectures with discussion of articles published in scientific journals.

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

Requirement:

Applicants must fulfill the following requirements:

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

Fellowships:

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

Submission:

Interested persons should apply through the Royal Thai Embassy in your country.

Contact: Chulabhorn Research Institute (CRI)
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Calendar

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

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

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