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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".

Maternal Phthalate Exposure and Blood Pressure

Phthalates are a class of synthetic organic chemicals, commonly found in consumer products, and recognized as endocrine-disrupting compounds. Human exposure to phthalates can occur through ingestion, inhalation, and skin absorption.

During pregnancy, women undergo major hemodynamic and cardiovascular changes including changes in plasma volume, blood pressure, cardiac output, and vascular resistance. Arterial blood pressure decreases starting as early as 7 weeks of the first trimester, and reaches its lowest point during the second trimester and gradually increase during the third trimester to reach preconception levels postpartum. Interference with these physiological changes may lead to hypertensive disorders of pregnancy, postpartum hypertension, or both.

The present study examined the association of phthalate exposure during pregnancy with maternal blood pressure trajectories from mid-pregnancy through 72 months postpartum, in a cohort of 892 pregnant women from Mexico City. Urinary levels of a mixture of 15 phthalate metabolites were measured during the second and third trimesters. Postpartum trajectories for systolic (SBP) and diastolic blood pressure (DBP) through 72 months were identified and the researchers also tested for the probabilities of the overall and individual phthalates of being on those trajectories.

Two postpartum trajectory classes were identified. The first ("increase–increase") class was characterized by a small transient increase in blood pressure from 1 to 12 months postpartum followed by a long-term steady increase in blood pressure through 72 months postpartum. The second ("decrease–increase") class was charecterized by a sharp decrease in blood pressure from 1 to 18 months postpartum with a gradual increase thereafter.

The results showed that an increase in overall urinary phthalate mixture levels was associated with steeper SBP rise during mid-to-late pregnancy, higher postpartum blood pressure (increase-increase class), and a lower probability of being in a decrease-increase postpartum trajectory for SBP and DBP.

Intriguingly, the relationships with blood pressure differed by metabolites. Higher urinary 2-ethylhexyl phthalate was associated with a trajectory of greater SBP rise during mid-to-late gestation but lower short- and long-term postpartum SBP. Similarly, higher urinary monobenzyl phthalate (MBzP) was associated with higher SBP and DBP during mid-to-late pregnancy, and lower short-term postpartum blood pressure, but it was not associated with blood pressure after 18 months.

The findings indicate that phthalates may interfere with blood pressure regulation and that phthalate metabolites may act on different vasoactive pathways. Therefore, phthalates could have a distinct effect on blood pressure levels.

This suggests that exposure to phthalates at earlier life stages may have lifelong consequences on the blood pressure trajectory, potentially elevating the risk for chronic illnesses later in life, such as hypertension.

The trajectory associated with each phthalate biomarker differed slightly and further follow-up and future studies are necessary to study potential changes to blood pressure trajectories in early pregnancy as well as other long-term cardiometabolic health consequences.

Source: Environmental Health Perspectives, Vol.129, No. 12, Article 127007-1, December 2021.

Fipronil and Human Health Risk

Fipronil is a broad-spectrum phenylpyrazole insecticide for its central neurotoxic action on gamma-aminobutyric acid (GABA) receptors by inhibiting GABA gated chloride channel, resulting in over activation of neurons.

Exposure to a large amount of fipronil in a short time will cause adverse effects on the nervous system in humans and animals. Long-term intake of fipronil may cause damage to liver, thyroid, and kidney.

Fipronil can be degraded into metabolites in the environment, mainly including fipronil desulfinyl, fipronil sulfone, and fipronil sulfide. Fipronil and its metabolites have been found in environmental samples and food samples as well as biological samples, such as urine, serum, and hair.

In addition, the toxicity of metabolites to mammals was reported to be higher than that of parent fipronil. The binding affinity of fipronil sulfone to the GABA receptor of a vertebrate was 6 times that of fipronil.

The World Health Organization classified fipronil as a moderate hazardous pesticide, and further as a group C (possibly human) carcinogen by the U.S. Environmental Protection Agency (USEPA).

In recent decades, a large amount of experimental evidence on the toxicity of fipronil has provided extensive background information on the adverse effects of fipronil exposure. However, the consequences of fipronil exposure to human health have been less investigated.

The goal of this review is to comprehensively summarize the current human exposure to fipronil and associated health risks, focus on the degree of human exposure to fipronil in different routes, and point out the possible sources of exposure.

In addition, the knowledge gap and future direction are surveyed to help strengthen understanding the risk of fipronil on human health.

The present review summarized potential human exposures to fipronil through ingestion and inhalation, as well as results of human biomonitoring studies.

Some recent studies have highlighted the importance of dietary intake as a pathway of human exposure to fipronil. However, most exposure assessments are conducted in an individual food category, which makes it difficult to estimate the total human exposure to fipronil.

The total concentration and health risk of fipronil may be underestimated, as its metabolites were not measured in some dietary exposure studies. More importantly, not all sources of fipronil exposure have been identified. New findings on this aspect will help to elaborate the occurrence of fipronil.

Currently, there are still conflicting results about human exposure to fipronil in biomonitoring, which can be attributed to the size of the number of biomonitoring samples, or different sources and routes of fipronil exposure in different regions.

Although several epidemiological studies have reported the health effects of acute fipronil poisoning, the available data are too limited for low-dose fipronil exposure.

In summary, many countries and organizations have formulated restrictions and residue limit management of fipronil, but it is still crucial to carry out long-term monitoring for environmental, food, and biological samples that may be exposed to fipronil.

Additionally, considering that infants and children may be sensitive groups exposed to fipronil, it is necessary to strengthen improvement and prioritization of exposure assessment. To further understand the health effects and risks of fipronil, large-scale population cohort studies and total dietary studies are also needed.

Source: Journal of Agricultural and Food Chemistry, Vol. 70, Issue 1, Pages 63-71, January 2022.

Prenatal Exposure to Phthalates and Its Effects on Cognitive Functions

Phthalates are chemicals widely used in packaging and consumer products, which have been shown to interfere with normal hormonal function and development in some human and animal studies.

Occupational exposure occurs primarily through inhalation and dermal contact, while consumer exposure is primarily via oral and dermal routes.

Several scientific investigations have associated exposure to phthalates with various health problems, primarily due to their effects as hormone disruptors.

The major windows of developmental vulnerability occur *in utero*, during infancy, and in early childhood. As a result of the widespread use of phthalate esters and our subsequent exposure to them, their adverse effects on children's neurocognitive development have become a significant public health concern.

In recent decades, pregnant women's exposure to phthalates has been shown to alter the cognitive outcomes of their babies, and some studies have found delays in motor development.

(Continued on page 3)

Early-life Exposure to Air Toxics and Childhood Asthma: Machine Learning-driven Identification

Air toxics are hazardous air pollutants that cause or may cause serious health effects. Exposure to air toxics early in life predisposes children to asthma. Epidemiological studies have linked prenatal and early-life exposure to air toxics with childhood wheeze, asthma, and altered lung function.

Although individual air toxics have been associated with asthma, only a limited number of studies have specifically examined combinations of air toxics associated with the disease. Additionally, there are limited statistical methods to parse the effects of mixtures where individual air toxics may contribute only slightly to an adverse outcome but have a different impact in combination with other air toxics.

The present study hypothesized that exposure to combinations of air toxics during early life is associated with asthma outcomes in later childhood.

The study geocoded 125 air toxic levels from the US National Air Toxics Assessment (NATA) to residential locations for 151 children with mild to severe persistent asthma of a cohort study to map each child's exposure to air toxics during the first years of life.

The study then applied Data-driven ExposurE Profile extraction (DEEP), a machine learning-based method, to discover combinations of early-life air toxics associated with current use of daily asthma controller medication, lifetime emergency department visit for asthma, and lifetime overnight hospitalization for asthma.

The present study discovered 20 multi-air toxic combinations and 18 single air toxics associated with at least 1 of the 3 asthma outcomes.

The multi-air toxic combinations included those containing acrylic acid, ethylidene dichloride, and hydroquinone, and they were significantly associated with asthma outcomes.

Acrylic acid was not only the individual air toxic most strongly associated with daily controller medication, but was also the primary member of the 7 multi-air toxic combinations associated with this outcome. Acrylic acid also appeared in 3 of the other 11 combinations associated with emergency room visit and overnight hospitalization for asthma, indicating that it was a major contributor to adverse asthma outcomes among children.

Both ethylidene dichloride and hydroquinone appeared in 3 of these 8 combinations, indicating that these 2 chemicals may play a role in the development of poor asthma outcomes among children. Most other air toxics in these combinations were largely not individually associated with this outcome,

These findings highlight the main strength of DEEP, namely its ability to identify significant multi-air toxic combinations, whose constituent air toxics may not be individually associated with the health outcome of interest.

Due to the unique ability of DEEP to examine air toxic combinations, we identified 16 air toxics that were found to be significantly associated with childhood asthma outcomes only in combination with other air toxics.

In conclusion, this study demonstrated innovative use of data science methods and data sources to identify specific combinations of early-life air toxic exposures associated with later childhood asthma outcomes.

The study suggests that chemical pollutants should be closely monitored together in combination, especially in locations with vulnerable populations.

The results do not provide evidence of a causal effect of any chemical on adverse childhood asthma outcomes, which will need further investigation.

Source: Journal of Clinical Investigation, Vol. 131, Issue 22, Article e152088, November 2021.

Prenatal Exposure to Phthalates and Its Effects on Cognitive Functions

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Determining how gestational exposure to these chemicals may affect the development of cognitive and motor functions in childhood and beyond is of significant interest.

The analysis of the published literature reveals a negative association between prenatal exposure to some phthalate congeners and cognitive functions in children.

Most studies find statistically significant inverse relationships between maternal urinary phthalate concentration during pregnancy and subsequent outcomes in children's cognitive and motor scales, especially in boys rather than girls. However, many associations are not significant, and there were even positive associations, especially in the third trimester.

In conclusion, the relationship between exposure to phthalates during pregnancy and results on neurocognitive scales is sufficiently clear.

This review underscores the urgency of policies aimed at reducing phthalate exposure among pregnant women. Further studies are needed to confirm whether low and high molecular weight phthalates have different or sex-specific effects when investigating the association between phthalates and neurocognitive development.

Source: Toxicology, Vol. 463, Article 152980, November 2021.

Gender Difference in the Associations among Heavy Metals with Hemogram

Investigating heavy metal pollution is critical for early diagnosis and treatment to avoid consequent comorbidities. Hemogram parameters, on the other hand, provide information regarding human health.

Several studies showed the importance of hemogram parameters as predictors or indicators of dementia, primary ovarian insufficiency and type 2 diabetes mellitus. However, few studies have investigated the association between heavy metal pollution and hemogram parameters.

A health survey of 2447 participants was conducted in southern Taiwan between June 2016 and September 2018.

The present study aimed to investigate gender differences in the association between heavy metals and hemograms including hemoglobin (Hgb), mean corpuscular volume (MCV) and mean corpuscular hemoglobin concentration (MCHC). Seven heavy metals were analyzed, including blood lead (Pb), urine nickel (Ni), urine chromium (Cr), urine manganese (Mn), urine arsenic (As), urine copper (Cu) and urine cadmium (Cd).

The results show that in females, Pb and Ni were negatively associated with Hgb, MCV and MCHC, and Cr was also negatively associated with MCHC. In males, As and Cd were positively associated with MCV, and Pb was negatively associated with MCHC.

Female hemogram parameters were more susceptible to heavy metal poisoning such as Pb, Ni and Cr, but As affected males more than females. This sex dimorphism could relate to intrinsic sex-specific epigenetic mechanisms, hormone-regulated metabolism of heavy metals and iron-regulated metabolism.

Blood Pb was negatively associated with Hgb, MCV and MCHC in the female participants, and MCV in the males and the researchers hypothesize that the Pb level increased, as the estrogen level decreased, disrupted the RBC membrane and inhibited heme biosynthesis, which, in turn, led to microcytic anemia in females.

Urine Ni was negatively associated with Hgb, MCV and MCHC in the females. Furthermore, increased exposure to Ni was significantly related to a decreased MCV level in females and the researchers hypothesize that Ni-induced oxidative stress and disruption of iron hemostasis may have caused microcytic anemia in this study.

Another important findings was that urine As and Cd were positively associated with MCV in the male participants. In addition, the positive association between Cd and MCV was more pronounced in the male than in the female participants. Further investigations are warranted to investigate if the gender dimorphism is affected by hormone or gender epigenetics.

In conclusion, heavy metal poisoning was found to disrupt hemogram parameters and was associated with gender differences. Further research is needed to discuss the mechanism behind these associations.

Source: International Journal of Environmental Research and Public Health, Vol. 19, No. 1, Article 189, January 2022.

Long-term Air Pollution Exposure and Dementia in Older Adults

Dementia is a major public health issue, affecting more than 47 million people worldwide. Alzheimer's disease (AD) contributes to about two-thirds of dementia cases and is the sixth leading cause of death in the United States.

As there are no disease-modifying treatments for the most common types of dementia, it is a top research priority to identify modifiable risk factors for dementia that can be used for intervention at the population level.

There is growing evidence associating air pollution with neuro-degenerative disease, especially Alzheimer's disease and related dementias (ADRD). In addition, neuropathologic changes are known to occur many years prior to the diagnosis, and the relevant time window in which air pollution might increase the risk of dementia or AD is unclear.

The two national U.S. population-based cohorts of those aged more than 65 years old from the Medicare Chronic Conditions Warehouse (2000-2018), combined with high-resolution air pollution datasets, was conducted to investigate the association of long-term exposure to ambient fine particulate matter ($PM_{2.5}$), nitrogen dioxide (NO_2), and ozone (O_3) with dementia and AD incidence, respectively.

This is the first nationwide, population-based cohort study that focuses on the simultaneous health effects of $PM_{2.5}$, NO_2, and O_3 on dementia and AD.

A majority of the study population had comorbidity at some point during follow-up, in which 16.6% developed dementia, and 6.5% developed AD. The average annual level of $PM_{2.5}$ was 9.3 $\mu g/m^3$, below the US EPA standard of 12 $\mu g/m^3$. The average NO_2 level was 17.1 ppb, considerably below the EPA annual standard of NO_2 of 53 ppb. The annual warm-season average O_3 was 42.6 ppb, while the EPA standard for daily maximum of 8-hour average O_3 is 70 ppb.

The study showed elevated hazard ratios (HRs) for both dementia and AD in relation to $PM_{2.5}$, and less markedly to NO_2, while HRs for warm-season O_3 were not elevated.

The researchers found a larger effect of both $PM_{2.5}$ and NO_2 on AD compared to dementia, which may reflect the fact that dementia includes a wide range of diseases with distinct etiologies, some of which may be unrelated to air pollution, while AD is a subset of dementia and a single disease, for which a stronger association was found.

(Continued on page 8)

Chronic Exposure to Low Concentrations of Chlorpyrifos Affects Reproduction in Rats

Chlorpyrifos (CPF) is a very effective, low-cost, and easily accessible organophosphate insecticide, acaricide, and miticide used to control foliage and soil-borne insect pests on a variety of food and feed crops. The main mechanism of action of CPF is the inhibition of the enzyme acetylcholinesterase (AChE) in exposed organisms inducing disorders of the central and peripheral nervous system.

CPF is considered a moderately toxic compound and is classified in the toxicity category II by the World Health Organization (WHO) (WHO, 2009). No Observed Adverse Effect Level (NOAEL), were established to protect the human population, which stated at 1 mg/kg body weight (BW) per day for AChE activity inhibition in rat brains. On the basis of NOAEL using a 100-fold safety factor, the derivative limit Admitted Daily Intake (ADI) is 0.01 mg/kg BW (WHO, 2015).

It is well known that endocrine disruptors (EDs) mimic the action of estrogen by interfering with the ability of the endocrine system to maintain homeostasis. These alterations lead to the appearance of various endocrine pathologies. EDs may act as agonist, antagonist, or altering endogenous steroid levels by changing the synthesis, metabolism and/or their action on the expression of their receptors.

There is strong evidence that EDs can affect women's reproductive health by inducing alterations in the function of the reproductive organs, infertility and/or cancer.

CPF can act as an endocrine disruptor at low doses. The previous studies also described that chronic exposure to low doses of chlorpyrifos induces endocrine disruption *in vivo.* Experimental studies demonstrated that CPF at doses reported to be safe induces cell proliferation through the estrogen receptor alpha (ERα) in hormone-dependent MCF-7 breast cancer cells.

CPF exposure also induces diminution of circulating levels of estradiol (E2), progesterone (Pg) and luteinizing hormone (LH) and increases proliferation of the ductal tree leading to the appearance of hyperplasia and adenosis in adult rats.

The present study was conducted to evaluate the effects of low concentrations of CPF (0.01 and 1 mg/kg/day) on the reproductive system of virgin adult rats. CPF serum levels and its bioaccumulation in the abdominal adipose tissue after chronic exposure to the pesticide were evaluated.

In the ovary, the researchers studied the effect of CPF on steroid hormone levels and on the expression of gonadal steroido-genesis enzymes, such as cytochrome P450scc (Cyp11) and P450 aromatase (Cyp19), key targets of many EDs.

The effects of CPF on estrous cycle, the histological characteristics and the proliferative state of the uterus were analyzed together with the expression of estrogen receptor alpha (ERα) and progesterone receptor (PR), since it is a very sensitive organ to the action of steroid hormones.

There is evidence that the assessment of the estrous cycle can be a useful measure of the integrity of the hypothalamus-pituitary-ovary reproductive axis. In addition, the determination of the cyclic state of a female accompanied by other experiments can be useful for the safety assessment of toxic compounds or drugs that affect the reproductive system.

The results showed CPF bioaccumulation in adipose tissue during chronic exposure in a dose-dependent relationship. Animals exposed to CPF 1 mg/kg/day showed a 40-fold higher concentration in fat than animals exposed to CPF 0.01 mg/kg/day.

CPF affects ovarian steroid hormones synthesis. The results showed that testosterone and estradiol content significantly decreased in the animals exposed to CPF 1 mg/kg/day with respect to the control group, however, progesterone content did not significantly change among the different experimental groups.

In addition, Cyp11 mRNA expression significantly decreased after CPF exposure, whereas Cyp19 mRNA expression was increased in the ovaries of animals exposed to both doses of CPF.

The findings also demonstrated that CPF exposure induces estrous cycle dysregulation and also induces histological and proliferative changes in endometrial tissue.

In conclusion, the results show that chronic exposure to CPF, at similar doses to the ADI and NOAEL, affects ovarian steroid synthesis, causing alterations in the normal cyclicity of the animals. In addition, CPF induced proliferative changes in the uterus, suggesting that it could affect reproduction or act as a risk factor in the development of uterine proliferative pathologies.

Source: Food and Chemical Toxicology, Vol. 156, Article 112515, October 2021.

Exposure Risk Assessment of Organic Micropollutants in Indoor Dust

Indoor dust is an important source of human exposure to hazardous organic micropollutants (OMPs) because humans spend about 90 % of their time in the indoor environments.

Previous studies have reported the occurrence of a variety of OMPs such as polychlorinated biphenyls, polycyclic aromatic hydrocarbons, organophosphate esters (OPEs), personal care products (PCPs), pharmaceuticals, and pesticides in the indoor dust.

Some OMPs may induce a number of adverse effects on human health including respiratory diseases, neuropsychological disorders, and cancer due to the chronic exposure. Therefore, it is important to characterize the chemical composition of OMPs in the home dust and estimate human health risks.

Chemicals present in the house dust can enter the body by means of ingestion after hand-to-mouth contact, direct skin absorption, or resuspended dust inhalation. Comparatively, ingestion is reported as the primary pathway for dust exposure. Toddlers are more vulnerable to OMPs due to frequent hand-to-mouth and object-to-mouth activities.

The health risk assessment of OMPs recommended by the U.S. Environmental Protection Agency (EPA) has been extensively adopted. However, many previous studies only conducted health risk assessments for a few detected OMPs due to the lack of chronic reference dose (RfD) and cancer slope factor (CSF) for most of the chemicals.

Conditional toxicity value (CTV) software was analyzed to predict toxicity threshold values such as RfD and CSF. This software has been employed to conduct health risk assessment of OMPs in the samples from groundwater.

The present study conducted a comprehensive survey and health risk assessment of 508 OMPs in the dust samples from different micro-environments of Malaysia for the first time.

A total of 57 OMPs were detected using Automated Identification and Quantification System (AIQS) with LC-MS databases, and assigned to 7 chemical classes in this study.

The daily intakes of OMPs were determined through the ingestion pathway for diverse age groups and the human noncarcinogenic and carcinogenic risk of exposure to these detected substances were assessed.

The concentrations and compositions of influential OMPs varied in different microenvironments, suggesting different sources and usage patterns in the house.

Based on the median concentration, the predominant OMPs in this study were attributed to PCPs, followed by OPEs, pesticides, consumable products, pharmaceuticals, corrosion inhibitors and others. The dust samples in the children's room of apartment showed the highest concentration of OMPs.

Five PCPs were detected, of which octocrylene, an ingredient in sunscreens and cosmetics, was the most abundant PCP.

Twelve OPEs were observed, of which 2,2-Bis(chloromethyl)-propane-1,3-diyltetrakis(2-chloroethyl)bisphosphate (V6) was the dominant.

In the study, 8 insecticides, 8 fungicides, and 3 herbicides were observed among the pesticides.

Carbendazim was the most common insecticide. Thiabendazole accounted for more than 95% of total concentration of fungicides and Diuron and terbutryn were the frequently detected herbicides.

Consumable products in the study consisted of 3 sweeteners, 1 surfactant, and 3 preservation/additives. N,N-Dimethyldodecylamine N-oxide and sucralose were the most frequently observed consumable products in the study. N,N-dimethyldodecylamine N-oxide is an important non-ionic surfactant of PCPs. Sucralose is widely used as an artificial sweetener in food, which is nontoxic to humans.

Theophylline (bronchodilator) was the most commonly observed pharmaceuticals.

Two corrosion inhibitors including 1H-benzotriazole and 4-5-methyl-1H-benzotriazole were discovered widely in home dust. 1H-Benzotriazole is commonly used as an anticorrosive and complexing agent for metals and vulcanizing accelerator for rubber materials.

For the other OMPs, theobromine was detected in every sample. Theobromine is the most known methylxanthine besides theophylline. They are present in coffee, tea and/or chocolate, which are very prevalent in Malaysia. Theobromine and theophylline are listed as group 3 carcinogens according to the International Agency for Research on Cancer (IARC, 2020).

Then, the noncarcinogenic and carcinogenic risks of exposure to these substances for diverse age groups were assessed based on the median concentration.

Noncarcinogenic risk of OMPs via ingestion pathway was in an acceptable scope among all the sampling sites.

Toddler was the most affected group for cancer risk among all the age groups, regardless of the micro-environments. The carcinogenic risks of these OMPs were higher than 10^{-6}.

Theobromine accounted for more than 89 % of the cumulative cancer risk, implying that the carcinogenic risk of theobromine needs further monitoring in the future.

The application of the CTV software was a feasible way to assess health risk of OMPs in dust. These outcomes may provide a benchmark for future efforts to monitor dust sample quality and ensure the safety of residents in Malaysia.

The situation also indicates urgent needs for further efforts to develop models with larger applicability domains.

Source: Chemosphere, Vol. 287, Part 3, Article 132340, January 2022.

WHO Human Health Risk Assessment Toolkit: Chemical Hazards Second Edition

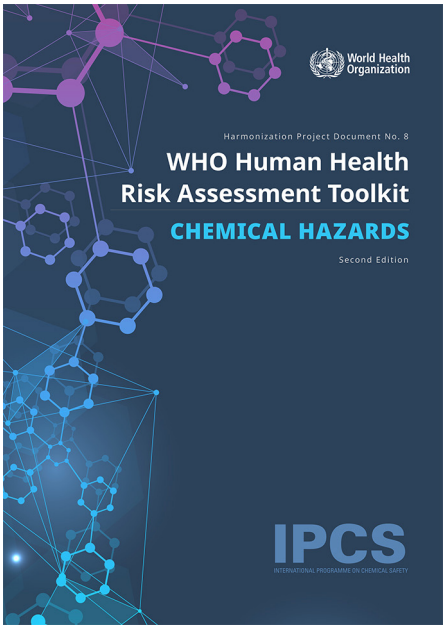
The **WHO human health risk assessment toolkit: chemical hazards** is to provide guidance to identify, acquire and use the information needed to assess chemical hazards, exposures and the corresponding health risks in their given health risk assessment contexts at local and/or national levels.

The Toolkit provides road maps for conducting a human health risk assessment, identifies information that must be gathered to complete an assessment and provides electronic links to international resources from which the user can obtain information and methods essential for conducting the human health risk assessment.

The Toolkit has been developed for public health and environmental professionals, regulators, industrial managers and other decision-makers with at least some training in the principles of risk assessment who are responsible for conducting human health risk assessments and making decisions on whether to take action to manage human health risks associated with exposure to chemicals.

Since the publication of the first edition in 2010, there have been a number of new developments in chemical risk assessment methodologies, new tools and new WHO publications. This revised edition of the Toolkit is intended to incorporate information about these new developments in methodologies, and to keep references and links to the information sources up to date.

It is hoped that, in all countries, the identification of human health risks related to chemicals as well as related management decisions and mitigation measures will be based on best evidence through the application of best risk assessment methodology and use of available authoritative risk assessment information developed by international organizations in combination with locally relevant information.



Source: WHO Human Health Risk Assessment Toolkit: Chemical Hazards, Second Edition. (IPCS harmonization project document, no. 8), December 2021.

US EPA'S CLIMATE ADAPTATION PLAN

The U.S. Environmental Protection Agency (EPA) released its **Climate Adaptation Action Plan** in October 2021, which describes steps EPA will take to address the impacts of climate change on communities across the Nation.

EPA also launched a new Climate Adaptation web page (https://www.epa.gov/climate-adaptation) that will act as a hub for climate adaptation resources from across EPA.

EPA's Climate Adaptation Action Plan accelerates and focuses attention on five priority actions the Agency will take over the next four years to increase human and ecosystem resilience as the climate changes and disruptive impacts increase:

1. Integrate climate adaptation into EPA programs, policies, rulemaking processes, and enforcement activities.

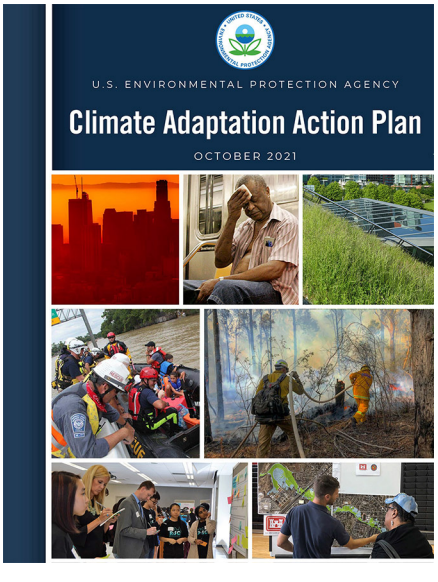
2. Consult and partner with Tribes, states, territories, local governments, environmental justice organizations, community groups, businesses, and other federal agencies to strengthen adaptive capacity and increase the resilience of the nation, with a particular focus on advancing environmental justice.

3. Implement measures to protect the Agency's workforce, facilities, critical infrastructure, supply chains, and procurement processes from the risks posed by climate change.

4. Measure and evaluate performance.

5. Identify and address climate adaptation science needs.

Anticipating and recovering from the impacts of climate change will require all levels of government to work together. EPA's climate adaptation strategies will be informed by the best available science and will deliver co-benefits for mitigation of greenhouse gases and other pollution, public health, economic growth and job creation, national security, and environmental justice—all of which will be central to building a more resilient future.



Source: US EPA News Releases, October 2021.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute, Year 2022

	Training Course	Date	Duration	Closing Date
1	Environmental Toxicology and Health	June 15-20, 2022	7 work days	May 5, 2022
2	Environmental and Health Risk Assessment and Management of Toxic Chemicals	December 2022	10 work days	October 2022

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

Course Description:

Environmental Toxicology and Health (June 13-17, 2022)

This course provides students and participants with a background of the major groups of toxic substances encountered by man and animals through food and the environment, as well as through exposure at the workplace. These toxicants include toxic substances in air, water and soil; solvents; gases; pesticides; hazardous wastes and other pollutants. The course focuses on the chemistry, fate and distribution in the environment, mechanisms of their action, toxic manifestation in living organisms, as well as toxic syndrome in human beings. The course also provides information on the latest technologies used to study changes and effects in biological systems, e.g. biomarkers, the omics technologies, gene-environment interactions, epigenetics and transgenic models, and covers environmental health issues such as climate change, and their adverse health effects in humans.

Requirement: Participants should have some basic knowledge in chemistry and the biological/biomedical sciences.

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)
54 Kamphaeng Phet 6 Rd.,
Lak Si, Bangkok 10210, Thailand
Tel: +66 2 553 8535
Fax: +66 2 553 8536
E-mail: envtox@cri.or.th

More information and application:
Please visit - http://www.cri.or.th/en/ac_actcalendar.php

Long-term Air Pollution Exposure and Dementia in Older Adults

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In summary, the study suggests that exposures to $PM_{2.5}$ and NO_2 are associated with incidence of dementia and AD.

Future studies of air pollution and dementia in other countries, including low-to-middle-income countries in which there are currently few studies, will be important.

Examining the role of specific pollutant components in ADRD may also be important because different components of $PM_{2.5}$ (e.g., metals, elemental carbon, organic carbon, sulfate, and nitrate) and different sources of $PM_{2.5}$ (e.g., traffic, industrial, cooking, and biomass burning) may have different neurotoxicities.

A better understanding of component-specific and source-specific effects of $PM_{2.5}$ on ADRD could potentially inform pollution control policies on specific sources.

Source: Nature Communications, Vol. 12, Article 6754, November 2021.

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.

Correspondence should be addressed to:

ICEIT NEWSLETTER
Chulabhorn Research Institute
Office of Academic Affairs
54 Kamphaeng Phet 6 Road
Lak Si, Bangkok 10210, Thailand
Tel: +66 2 553 8535
Fax: +66 2 553 8536
CRI Homepage: <http://www.cri.or.th>

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