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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".

THE INTERNATIONAL CONFERENCE ON LIVER AND LUNG CANCER: CURRENT AND FUTURE RESEARCH

Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol presides over Opening Ceremony of important conference on cancer research

On January 8th, 2019, Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol, President of the Chulabhorn Research Institute (CRI), presided over the opening ceremony of the **International Conference on Liver and Lung Cancer: Current and Future Research**, which was co-organized by the Chulabhorn Research Institute (CRI) and the US National Cancer Institute (NCI) at the Chulabhorn Convention Center, Bangkok, Thailand.

Cancer is presently one of the leading causes of mortality globally and in Thailand it is now the number one cause of death. Through the leadership and vision of Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol, cancer has been the basis for much of the research conducted at CRI, whether it be anti-cancer properties of natural and synthetic compounds, etiological factors of key cancers in the region, such as liver cancer, or molecular changes and markers associated with different types of cancer. Liver cancer is an important cancer in the northeastern part of Thailand and is the subject of collaborations between researchers in Thailand, including at CRI, the Chulabhorn Hospital, the Thai National Cancer Institute, Chiang Mai University and Khon Kaen University, and the US NCI, in a project entitled, **"Thailand Initiative in Genomics and Expression Research for Liver Cancer (TIGER-LC)"**.

The International Conference on Liver and Lung Cancer: Current and Future Research addressed various aspects of cancer research, from biology and etiology to approaches to early detection and diagnosis, as well as targets for therapeutic intervention and treatment. It brought together more than 400 participants, and eminent scientists as invited international speakers from

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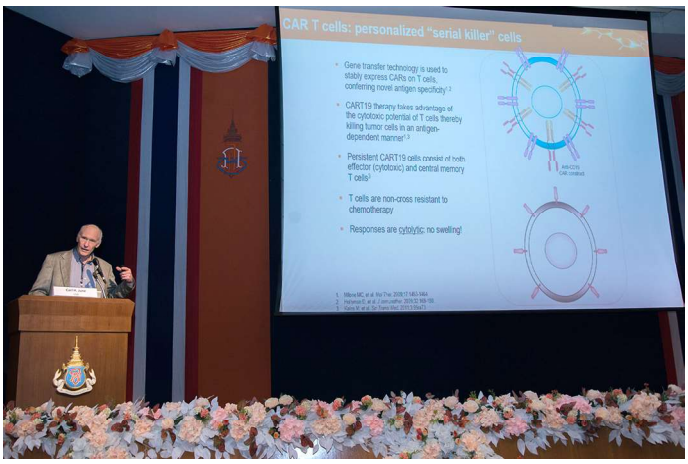

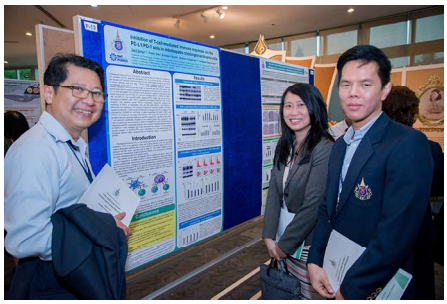

8 countries, with the keynote lecture entitled, "CAR-T-cell Therapy and Advances in Cancer Immunotherapy" being given by Professor Carl June from the University of Pennsylvania, USA. The scientific programme comprised 25 lectures in 3 major sessions, on (1) population studies on liver and lung cancer; (2) cancer drivers, biomarkers of risk, diagnosis, prognosis and responses to therapy; and (3) translational studies.

This international conference was organized as a tribute to Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol for Her tireless efforts to promote cancer research and treatment in Thailand through CRI and the Chulabhorn Hospital, which, together, will be a center for research and technology transfer that will lead to effective diagnosis and treatment of cancer in Thailand.

Air Pollution as a Determinant of Rheumatoid Arthritis

Air pollution has long been implicated in many cardiovascular and respiratory diseases. Recent studies have been evaluating the potential role for particulate pollutants associated as well with autoimmune diseases, including rheumatoid arthritis (RA).

Evidence suggests that, instead of a mere interface between air and the bloodstream, the airway tissues may be capable of transforming air particles and presenting them as antigens. Thus, the particles in ambient air can become precursors and generators of autoimmunity.

RA is an autoimmune disease that is mediated by both genetic and environmental factors. Although the joints are the chief targets of RA, other tissues – most prominently in the lungs – can be involved.

Lung disease is common in RA, notably during the phase leading up to the disease, when biological markers are found despite the absence of symptoms.

Although all the anatomic structures of the lung may be affected, studies suggest predominant involvement of the airways in early asymptomatic RA.

The incidence of RA has been found to be higher in urban areas. The risk of developing RA and of producing RA-specific autoantibodies is seen to increase among populations residing in heavily polluted areas.

As yet, however, no strong epidemiological evidence has clearly linked RA with any specific air pollution particles.

Abnormalities such as diminished vital capacity, bronchiectasis, and infiltrations of lymphocytes and plasma cells indicate chronic inflammation. These abnormalities are strongly associated with the presence of anti-citrullinated peptide antibodies (ACPAs) in serum, alveolar, and bronchial biopsy specimens.

While known to exert direct pathogenic effects on joints, ACPAs have been detected in the lungs up to 10 years before the onset of RA symptoms. Thus, the lung may be an autoimmunity initiation site for RA.

Inducible bronchus-associated lymphoid tissue (iBALT) is a typical tertiary lymphoid structure. It is absent

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The Role of Endocrine Disruptors in Ocular Surface Diseases

Endocrine disruptors (EDs) are a group of compounds that occur in increasing amounts in the environment. These compounds change the hormone homeostasis of the target organs, mostly by binding to their receptors and affecting their signaling pathways.

Among the hormones altered by endocrine disruptors are sex hormones, thyroid hormones, and insulin. Studies have documented abnormalities in the reproductive and metabolic systems of various species of animal exposed to endocrine disruptors.

Endocrine disruptors can play a significant role in ocular diseases when hormone deficiency or excess is involved in the mechanism of that disease.

Dry eye disease (DED) is a multifactorial diruption of the ocular surface (OS) characterized by the loss of lacrimal film homeostasis and accompanied by ocular symptoms caused by factors such tear film instability, tear hyperosmolarity, inflammation and damage to the OS.

Hormonal dysfunctions are part of the pathophysiology in the development of disease, mainly androgen, estrogen and progesterone deficiencies.

Cataracts, dry eye disease and retinal diseases, such as macular hole and diabetic retinopathy, are some of the frequent problems in which hormones have been implicated.

The increased prevalence of DED in women has been associated with changes in estrogen and androgen levels in ocular tissues, where these hormones modulate the expression of several genes and the function and secretion of the lacrimal gland, Meibomian glands and other tissues.

Sexual hormone imbalance may lead to dysfunctions of the glands, including a reduction in acinar cell size and number, changes in DNA expression and increased inflammation.

Estrogenic and androgenic receptors are present in the lacrimal gland, Meibomian glands, cornea and other ocular tissues. The disruption of sex hormone signaling may therefore directly interfere with the normal function of these glands, and may contribute to DED.

According to the WHO, EDs are mostly man-made and can be found in many daily products, including plastic bottles, food cans, detergents, flame-retardants, toys, cosmetics, toiletries, pesticides, and pharmaceuticals.

The compounds classified as EDs have different chemical configurations. These structures are capable of simulating or altering the hormonal system of humans and other animals, thereby impairing the normal functioning of the immune, nervous and endocrine systems.

Two basic ED action mechanisms are known as the genomic pathway, which involves the transcription of target genes, and the nongenomic pathway, which transduces signals mediated by membrane-bound ERs or other receptors through cross talk and/or bypass. EDs can also act through epigenetic mechanisms or via several other signaling-independent mechanisms simultaneously.

Once in contact with living organisms, including humans, EDs are able to deregulate the production of hormones affecting different tissues and organs. They can induce the reproductive, neurological and metabolic disorders that may take place in the mechanisms of DED.

Confronted with the increasing frequency of DED and OS diseases, a comparative analysis of the growing effects of many chemical products that act as EDs in the metabolism of body organs and systems indicates the need to better investigate the potential relationships beyond the rarely documented case reports of isolated associations mentioned by patients.

Evidence from case-control studies and experimental assays can provide the information necessary to confirm hypotheses about the impact of certain chemicals on the endocrine system and the pathophysiology of the DED and OS diseases.

Source: Medical Hypotheses, Vol.122, Pages 157-164, January 2019.

Air Pollution as a Determinant of Rheumatoid Arthritis

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from the normal lung and develops only in response to antigenic stimuli. iBALT seems to develop in response to chronic environmental proinflammatory stimuli (silica or particulate matter) and is associated with RA-specific auto-antibodies (ACPAs) and RA-related lung disease.

The induction of diesel exhaust particles in polluted air can lead to an inflammatory environment with high citrullination levels in the lungs. Thus, in turn, may induce iBALT formation, causing a transition toward a more specific immune response via the production of anti-citrullinated peptide antibodies.

Air pollution not only triggers innate immune responses at the molecular level, increasing the levels of proinflammatory cytokines and reactive oxygen species, but is also involved in adaptive immune responses.

Via the aryl hydrocarbon receptor (AHR), diesel exhaust particles can trigger a T-cell switch to the Th17 profile. In the murine collagen-induced arthritis model, animals whose lymphocytes lack the AHR will develop milder arthritis.

In conclusion, findings from cellular and molecular studies add to the mounting epidemiological evidence that particulate matter is involved in the development of iBALT. The role for iBALT in initiating RA highlights the central part played by the lung in the pathophysiology of this disease and the consequences of prolonged inflammation.

No evidence is available to date for hierarching the effects of intrapulmonary antigen presentations on the initiation or perpetuation of RA.

Source: Joint Bone Spine, Vol. 86, Issue 1, Pages 37-42, January 2019.

Exposure to Environmental Lead and Diabetes

The increased incidence of type 2 diabetes since the 1950s is thought to be primarily due to coincident alterations in lifestyle factors, including increased caloric intake, altered nutrient quality, and a general reduction in physical activity. Another potential contributing factor in industrialized countries is the exposure of the population to environmental pollutants and industrial chemicals.

Exposure levels of many environmental toxicants have risen concurrently with the disease incidence. Of particular interest in this regard is the metal, lead (Pb).

Although overall Pb exposure levels have diminished in recent decades, there is an under-recognized but persistent occurrence of Pb exposure in poor underserved urban populations. In particular, individuals living in poor urban communities with older housing stock are at significant risk for childhood Pb contamination.

Do low levels of Pb exposure increase susceptibility to diabetes? The question is complicated by the fact that the most exposed populations are often coping, as well, with additional metabolic, nutritional and environmental stressors.

The present review considers the human, animal and *in vitro* studies which have examined the effects of Pb exposure on the development of diabetes and related metabolic conditions.

Although relatively limited in scope and conducted at higher levels of blood Pb than typically seen in human exposures, a number of animal studies support the idea that at some level of exposure, perhaps in combination with other metabolic stresses, Pb promotes the development of diabetes.

Even very low doses of Pb are likely to have harmful effects, and that there remains a significant segment of the population is regularly exposed to environmental Pb. The effects of such exposure on the metabolic health much therefore continue to be an important research topic.

The possibility that early-life Pb exposure might later have physiological effects is supported by observations in humans and in mice that Pb exposure leads to specific epigenetic imprints in the genome that persist throughout life and even across generations.

While it is not yet possible to directly link specific patterns of epigenetic imprinting to specific physiological or pathophysiological parameters, it is likely that such links exist. A clear mechanistic pathway may emerge which shows how limited exposure to lead might have long-lasting health effects.

Human exposures, even at low levels are likely to continue into the foreseeable future, but the solution to the public health problem is clear.

If lead is removed from the built environment, the risk of exposure falls. In the meantime, we still need to understand the mechanisms by which lead affects biological systems. The exact contribution that Pb exposures play in determining diabetes risk must be determined.

In addition, understanding how xenobiotics affect cellular and organismal physiology can provide valuable clues to how normal physiological systems function and how changes in those systems can lead to diseases like diabetes.

Source: Toxics. Vol. 6, Issue 3, Article No. 54, September 2018.

Welding Fume Exposure and Inflammation

Increasing evidence suggests that welding fume exposure is associated with systemic inflammation. Although cellular metabolites may be associated with inflammation, there is limited information concerning the metabolomic changes which occur during welding fume exposure.

Such changes may play an important role in the occurrence, development, and prevention of metal-associated diseases.

Metabolomics has been increasingly recognized as a powerful functional tool for understanding complex biological machinery and for developing new biomarkers for environmental biomonitoring to improve prevention and treatment.

They represent a fast and reproducible approach that directly reflects biological events related to exposure. Monitoring disturbances of the metabolome is now more sensitive, easily accessible, less expensive, and more accurate.

The present study investigates human metabolomics changes pre- and post-welding fume exposure and further explored the potential biological functions in boilermakers.

The results revealed some different metabolite classes between exposure groups, mainly in lipid pathways (steroid hormones, acylcarnitine class, and 12,13-Di-hydroxyoctadec-9-enoic acid species); amino acid utilization (isoleucine, proline and phenylalanine); and S-(3-hydroxypropyl) mercapturic acid (3-HPMA).

These altered compounds showed a close relationship with inflammation, which may indicate an inflammatory mechanism of welding fume exposed boilermakers at the metabolomics level.

In the metabolite set enrichment analysis (MSEA) for diseases, the top two disease-associated metabolite pathways were systemic inflammation-related, including rheumatoid arthritis and systemic lupus erythematosus; the interaction was related to decreased cortisol and cortisone from steroid hormones.

Further, C-reactive protein (CRP) is considered a strong marker for systemic inflammation, and positive correlations are found within CRP and cortisol. These results provide new evidence for an association between welding fumes and systemic inflammation at the metabolomics level.

In summary, this global metabolomics study provides evidence that metabolite changes during welding fume exposure are closely associated with systemic inflammation. The altered metabolites detected may be potential health monitoring biomarkers for boilermakers, especially for the prevention of inflammation-related disease.

Source: Environmental Health, Vol. 17, Article No. 68, August 2018.

The Link of Organophosphorus Pesticides with Neurodegenerative and Neurodevelopmental Diseases

Organophosphorus (OP) compounds have been the most widely used pesticides during the past half century.

The neurotoxicity of OP has been under consideration because their main mechanism of action is through the nervous system and the disruption of neural transmission.

The importance of this property has been highlighted in acute exposures leading to severe intoxication. Most studies have focuses on treatment of OP poisoning.

On the other hand, many studies carried out in the field of environmental toxicology have investigated other cellular or sub-cellular mechanisms of these chemicals, particularly in chronic exposures at low levels.

These mechanistic pathways, for example, oxidative stress, inflammation and genetic damage have been found to play a fundamental role in the development of chronic disorders such as neurodegeneration, cancer and diabetes. Not surprising, concern over these characteristics of OP compounds has been growing rapidly.

Most importantly, OPs are initially neurotoxic and they act by increasing an important neurotransmitter (acetylcholine) in the synaptic cleft of neurons in the nervous system.

In this study, the association of OPs with chronic diseases of central nervous system like neurodegenerative diseases including Alzheimer's disease (AD), Parkinson's disease (PD), and amyotrophic lateral sclerosis (ALS) as well as neurodevelopmental disorders including attention deficit hyperactivity disorder (ADHD), autism, and neurodevelopmental delays along with their main mechanism of toxicity, inhibition of cholinesterase enzymes, as well as the other newly proposed ones are discussed.

These neurodegenerative and neurodevelopmental disorders are among the afflicting neurological diseases which overshadow human life. Their higher risk in relation to OP exposures has been uncovered in epidemiological studies.

Experimental studies exploring the underlying mechanisms have also provided some evidence for the involvement of cholinergic deficit, oxidative stress, neuro-inflammation, and epigenetic modifications as processes which are common in the toxicity of OPs and the pathophysiology of the aforementioned diseases.

Other experimental studies examining those pathophysiological mechanisms have provided evidence of the AD-inducing effects of OPs.

Increased levels of Amyloid β in the brains of AD mice model eight months after acute exposure to chlorpyrifos, along with some behavioral alterations, have been reported.

In fact, the function of cholinergic neurons in the nervous system, especially in the frontal and temporal cortex, is crucial for cognitive and memorial behavior.

Accumulation of amyloid β in this neuron can be promoted by multifactorial pathways, especially cholinergic exitotoxicity, oxidative stress, genetic and epigenetic modifications, and sus-ceptibility due to polymorphism of paraoxonase 1 (PON1) which protects neurons against acetylcholinesterase inhibitors.

This review includes epidemiological evidence of the association of PD with exposure to OPs, where as the experimental evidence extracted from the common mechanisms between the pathophysiology of AD and toxicity of OP has more weight.

The various elements of oxidative stress have been widely studied in both the toxicity of OP and the pathophysiology of PD.

Regarding ALS, in addition to the mechanisms, the similarity extends to the phenotype of the disease and OP toxicity appears as OP-induced delayed neuropathy (OPIDN). OPIDN is characterized by distal axonal degeneration followed by demyelination of central and peripheral axons which can lead to paralysis.

Evidence mostly from epidemiological studies during the last two decades has associated OP exposure with neurodevelopmental diseases, including ADHD, autism, and some other neurodevelopmetal disorders such as cognitive problems and decreased IQ. Since the exact etiology of these health problems is still unclear, more mechanistic evidence is required in order to explore their relation to the OPs.

Taken together, exploring the link between OPs and neurodegenerative and neurodevelopmental diseases has provided a large volume of literature on the evidence, mechanisms, experimental modeling, and therapeutic strategies.

Applying risk assessment approaches should be helpful on clarifying the exact role of environmental or occupational exposure to OPs in the initiation and progression of these neurological diseases.

Source: Toxicology, Vol. 409, Pages 44-52, November 2018.

Exposure to Arsenic Species in Wheat and Rice and Associated Health Implications

Arsenic, a naturally occurring metalloid, is widely present as an environmental contaminant. It enters the food chain mainly via contaminated water and several widely consumed foodstuffs.

Most toxicological assessments have focused on inorganic arsenic (iAs) in drinking water. Whether exposure to arsenic in the most frequently consumed foods (e.g. rice and wheat) has the same implications for health as exposure through drinking water is still unclear.

Previous studies have indicated that rice is the most common arsenic exposure source in food stuffs. Rice which is cultivated in submerged soil conditions does have a comparatively higher tendency to take up iAs. Populations not exposed to iAs via drinking water may therefore still be at risk if their diet depends significantly on rice.

Wheat is another important staple food with a worldwide consumption of 730.9 million tonnes, greater than the 506.5 million tonnes of rice consumed annually (Food and Agriculture Organization, 2017). Although past studies have reported lower arsenic levels in wheat than rice, health risks due to consumption of wheat grown in arsenic affected regions.

iAs is a recognized carcinogen and its chronic exposure has been reported to result in increased risk of bladder, lung, and skin cancer, type 2 diabetes, and cardiovascular disease.

Organic arsenic compounds are considered less toxic than iAs but should still be included in exposure assessments. Since toxicity depends on the chemical forms, arsenic speciation in rice and wheat can provide useful information for risk assessment and management.

In 2014, the Joint Food and Agriculture Organization and the World Health Organization (FAO/WHO) Expert Committee on Food Additives established an advisory levels of 200 μg/kg iAs for polished rice grains (Codex Alimentarius Commission, 2014).

Several countries have still not implemented this limit and are in the process of setting regulatory limits for rice based products.

Adoption of this advisory limit in different geographical regions requires an exposure assessment via rice.

The present study was conducted in six villages of Pakistan where arsenic concentrations above 10 μg/L have previously been found in 89% of the local ground water resources.

The concentrations of total arsenic (tAs) and arsenic species in wheat and in raw and cooked rice were determined to assess the relative contribution of dietary arsenic to aggregate daily exposure.

Human health hazards associated with daily consumption of rice, wheat and household groundwater by children (age ≤ 16 years) and adults (age > 16 years) were calculated, based on these exposures, to provide an indication of the hazards of each exposure source.

The present study has ascertained the health risks associated with exposure to total arsenic (tAs) and its species in the most frequently consumed foods.

In this study population, inorganic arsenic exposure from consumption of wheat was higher than from rice, followed by lower levels of dimethylarsinic acid (DMA) from raw and cooked rice.

Raw rice was a moderate source of exposure in the study villages, although cooking in low volumes of arsenic-rich water and higher cooked rice consumption frequency may contribute significantly in producing a potential risk.

The study participants had prolonged arsenic exposure in their total water intake. This would include water taken indirectly through cooking rice and kneading wheat flour, and in raw rice and locally grown wheat. Their total daily intake was 16 ± 40 μg iAs /kg bw/day with relative contributions from food (6%), amd from drinking and cooking water (94%).

This total daily intake of iAs exceeded the provisional tolerable daily intake of 2.1 μg/kg body weight/day in 74% of the study participants.

A significant association between tAs in cooked rice and cooking water resulted in tAs intake which was 43% higher in cooked rice compared to raw rice.

The study suggests that arsenic intake from food, particularly from wheat consumption, has particular significance, where iAs is relatively low in water.

Chronic health risks were in fact found to be significantly higher from wheat intake than from rice. The risk with rice in terms of acute effects was below the USEPA's limit of 1.0.

Chronic non-cancer risks due to aggregated exposure of iAs from wheat and raw rice indicated somewhat higher mean hazard quotient values (2.7 ± 1.1) than the acceptable limit of 1.0 in 100% of participants.

Children were at a significantly higher health risk than adults due to iAs exposure from rice and/or wheat. The dietary exposure of participants to tAs was attributable to staple food intake with ground water iAs <10 μg/L. However the preliminary advisory level (200 μg/kg) was achievable with rice consumption of ≤200 g/day and compliance with ≤10 μg/L iAs in drinking water.

Although the daily iAs intake from food was lower than total water intake, the potential health risk from exposure to arsenic and its species in food still exists and requires exposure control measures.

Dietary exposure to iAs occurs naturally and unavoidably, for example, in raw rice or wheat grains. However irrigating crops, cooking rice and kneading wheat flour in low arsenic water could reduce dietary exposure.

The study findings suggest the need to identify maximum tolerable level of iAs for the most frequently consumed foods, such as rice and wheat, and the need for recommendations on consumption frequency in order to lower exposure risks.

Furthermore, arsenic remediation of water used for drinking, irrigation and food preparation is an immediate requirement for populations in arsenic affected regions.

Source: Science of The Total Environment, Vol. 634, Pages 366-373, September 2018.

Air Pollution and Child Health: Prescribing Clean Air

This report summarizes the latest scientific knowledge about the links between exposure to air pollution and adverse health effects in children. It is intended to inform and motivate individual and collective action by health care professionals to prevent damage to children's health from exposure to air pollution. Air pollution is a major environmental health threat. Exposure to fine particles in both the ambient environment and in the household causes about seven million premature deaths each year. Ambient air pollution (AAP) alone imposes enormous costs on the global economy, amounting to more than US$ 5 trillion in total welfare losses in 2013.

This public health crisis is receiving more attention, but one critical aspect is often overlooked: how air pollution affects children in uniquely damaging ways. Recent data released by the World Health Organization (WHO) show that air pollution has a vast and terrible impact on child health and survival. Globally, 93% of all children live in environments with air pollution levels above the WHO guidelines. More than one in every four deaths of children under 5 years is directly or indirectly related to environmental risks. Both AAP and household air pollution (HAP) contribute to respiratory tract infections that resulted in 543,000 deaths in children under 5 years in 2016.

Although air pollution is a global problem, the burden of disease attributable to particulate matter in the air is heaviest in low- and middle-income countries (LMICs), particularly in the WHO African, South-East Asia, Eastern Mediterranean and Western Pacific regions. LMICs in these regions – especially the African Region – have the highest levels of exposure to HAP due to the widespread use of polluting fuels and technologies for basic daily needs such as cooking, heating and lighting. Poverty is correlated with high exposure to environmental health risks. Poverty can also compound the damaging health effects of air pollution, by limiting access to information, treatment and other health care resources.

Children are society's future. But they are also its most vulnerable members. The immense threat posed to their health by air pollution demands that health professionals respond with focused, urgent action. Although more rigorous research into how air pollution affects children's health will continue to be valuable, there is already ample evidence to justify strong, swift action to prevent the damage it clearly produces. Health professionals must come together to address this threat as a priority, through collective, coordinated efforts. For the millions of children exposed to polluted air every day, there is little time to waste and so much to be gained.

Source: Air Pollution and Child Health. WHO Reference Number: WHO/CED/PHE/18.01 Publication Date: October 2018. (http://www.who.int/ceh/publications/air-pollution-child-health/en/)

IUTOX 15th International Congress of Toxicology (ICTXV)

Congress Theme:

Toxicology Solutions for Global Public, Environmental, and Personal Health

July 15–18, 2019,

at the Hawaii Convention Center in Honolulu, Hawaii, USA.

Congress Website: https://www.toxicology.org/events/ict/index.asp

ANNOUNCEMENT

International Training Course on Occupational and Environmental Health, and Regional Meeting on Children's Environmental Health (CEH)

Chulabhorn Research Institute, Bangkok, Thailand, 2019

	Activity	Date	Duration	Closing Date for Application
1	International Training Course on Occupational and Environmental Health	May 21-25, 2019	5 days	March 22, 2019
2	Regional Meeting on Children's Environmental Health (CEH)	May 27-28, 2019	2 days	March 22, 2019

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

1. International Training Courses on Occupational and Environmental Health (May 21-25, 2019)

This course is taught by international lecturers from Boston College, Mount Sinai School of Medicine and the University of Maryland School of Medicine, as well as from the National Institute of Environmental Health Sciences from the United States and from CRI, Thailand, and provides basic scientific knowledge of the principles and concepts of environmental and occupational medicine, knowledge of the basics of toxicology, and of the on-line resources available in occupational and environmental medicine. It also provides physicians with basic knowledge of the principal occupational and environmental diseases and exposures in order to improve diagnosis, treatment and prevention of these conditions. The major occupational and environmental hazards and the associated diseases will be reviewed. Emphasis will be placed on the importance of the occupational/environmental exposure history as a critical tool for proper diagnosis of occupational and environmental disease. There will also be discussion on the multi-disciplinary nature of occupational and environmental medicine, which underscores that treatment, prevention and control of occupational and environmental diseases requires a team approach that includes physicians, nurses, industrial hygienists, environmental scientists, engineers and policy makers.

Applicants should be medical and related personnel, including physicians, nurses, and health-scientists (minimum of B.Sc. or equivalent), who deal with issues related to occupational and environmental health in their line of work.

2. Regional Meeting on Children's Environmental Health (CEH; May 27-28, 2019)

This meeting is being co-organized by the Chulabhorn Research Institute (CRI; WHO Collaborating Center for Capacity Building and Research in Environmental Health Science and Toxicology) and the United States National Institute of Environmental Health Sciences (US NIEHS; WHO Collaborating Center for Environmental Health Sciences) and WHO-SEARO with the following objectives: (a) to discuss cross-cutting issues and commonalities among countries/ regions; (b) to discuss lessons learnt and explore research collaborations; (c) to review availability of and discuss educational tools to help translate research findings into tangible outputs; and (d) to help set the agenda for the International Conference in Children's Environmental Health in collaboration with WHO and the Global Network of WHO Collaborating Centres for CEH (Tentatively scheduled for early 2020)

Participants receiving fellowships from CRI to attend the international training course and regional meeting are requested to prepare a country report on environmental health or environmental risk factors related to children's environmental health in their respective countries, which will be important in initiating collaborative discussions during the meeting.

Fellowships: *A limited number of fellowships are available that will cover roundtrip airfare, accommodation (on site at the CRI Residence) and meals, training materials, and health insurance.*

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

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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