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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".

CRI's WHO Collaborating Centre on Capacity Building & Research in Environmental Health Science & Toxicology

Official Visit of Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol to the World Health Organization's Regional Office for South-East Asia

On February 10th, 2016, Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol, President of the Chulabhorn Research Institute (CRI), paid an official visit to the World Health Organization's Regional Office for South-East Asia (WHO SEARO) in New Delhi, India, for the annual review of progress on collaborative activities previously agreed upon between WHO SEARO and CRI, carried out by CRI's International Centre for Environmental Health and Toxicology (ICEHT), a WHO Collaborating Centre for Capacity Building and Research in Environmental Health Science and Toxicology since 2005.

Discussions including technical discussions attended by a team of senior researchers from CRI on February 8th and 9th, covered the following areas: (1) CRI's capacity building/training programmes in Chemical Safety and Occupational and Environmental Health/Medicine, including in-country training, for which interest has been expressedby Bhutan and Sri Lanka; (2) further development of training modules/ courseware for both face-to-face and web-based training in chemical safety, including the use of the developed electronic distance learning tool on risk assessment and risk management of chemicals for train-the-trainers courses; (3) enhancing and widening information dissemination networks, e.g. for

(Continued on page 2)

Official Visit of Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol to the World Health Organization's Regional Office for South-East Asia

(Continued from page 1)

raising awareness and disseminating information related to chemical safety and risk assessment, including upcoming training opportunities at CRI; and (4) leadership in research in the area of environmental health and chemical safety.

In terms of the capacity building/ training programmes in Chemical Safety and Occupational and Environmental Health/Medicine, CRI organized 3 international training courses in 2015, and 7 have been tentatively planned for 2016.

These courses are open to participants, primarily from the Asia Pacific region, and are taught by international experts from world-renowned academic and research institutions with a wealth of teaching experience in the region.

The organized courses in 2015 included:

	Training Course	Date	Participating Countries
1.	Environmental Toxicology	7-15 May 2015	12 (Bhutan, Brunei, Cambodia, India, Jordan, Laos, Mongolia, Nepal, Philippines, Pakistan, Thailand, Vietnam)
2.	Occupational and Environmental Health (Advanced)	17-22 August 2015	8 (Brunei, Cambodia, Laos, Mongolia, Myanmar, Nepal, Sri Lanka, Vietnam)
3.	Environmental and Health Risk Assessment and Management of Toxic Chemicals	5-18 December 2015	15 (Bhutan, Botswana, Brazil, Brunei Darussalam, Cambodia, India, Laos, Morocco, Nepal, Philippines, Sri Lanka, Tanzania, Thailand, Tunisia, Vietnam)

Courses tentatively scheduled for 2016 include:

	Training Course	Estimated time frame
1.	Detection of Environmental Pollutants and Monitoring of Health Effects	15-26 February 2016
2.	Risk Assessment and Risk Management of Chemicals	1-4 June 2016 (in Bhutan)
3.	Environmental Toxicology	2-10 June 2016
4.	Occupational and Environmental Health	July 2016
5.	Environmental Immunotoxicology and Reproductive Toxicology	September 2016
6.	Risk Assessment and Risk Management of Chemicals	November 2016 (in Sri Lanka)
7.	Environmental and Health Risk Assessment and Management of Toxic Chemicals	November/December 2016

Those who are interested in applying for a fellowship to attend such training courses can check the calendar of events on CRI's website at http://www.cri.or.th/en/ac_actcalendar.php.

Prenatal Exposure to Chlorpyrifos and Childhood Tremor

Chlorpyrifos (CPF), a broad-spectrum chlorinated organophosphate (OP) insecticide, has been banned for indoor residential use by the US EPA since 2001. However, it is currently used for agricultural purposes across the United States, with the heaviest applications in California. In fact, CPF is one of the most widely used insecticides in the world.

CPF has been linked to neurodevelopmental deficits in human and animal studies. In animal studies, exposure leads to disruption of neuronal development, neurotransmitter systems, and synaptic formation in different brain regions that are highly involved in the control of movement and known to be associated with tremor.

In humans, OPs and CPF are known to cross the placenta, reaching the fetus during a period of rapid brain development.

Evidence is still limited that low to moderate prenatal exposures to CPF and/or OP chemicals, in general, may disrupt motor processes. To date, there are no studies evaluating the longer term consequences of prenatal exposure to CPF on motor development or movement disturbance.

Tremor is a condition that is highly prevalent in human populations, particularly among the elderly. In adults, tremor is associated with exposure to several environmental chemicals,

(Continued on page 3)

THE ORGANOCHLORINE PESTICIDE RESIDUES IN THE INVASIVE DUCTAL BREAST CANCER PATIENTS

Breast cancer is the most prevalent cancer worldwide. It is also the second most common cancer-related cause of death among women. Breast cancer is related to genetic, reproductive, and lifestyle factors, but environmental factors are also important.

Organochlorine pesticides (OCPs) are semivolatile organic compounds of global concern. During the last decades, their sources, distribution, transformation, toxicity and accumulation in terrestrial and aquatic ecosystems have gained significant attention.

Even at very low doses, OCPs have been associated with endocrine-disrupting effects in several mammals, including humans. As environmental estrogens, OCPs could play a critical role in the carcinogenesis of some endocrine related cancers, life breast cancer.

Invasive breast carcinoma (IDC) may be classified into two groups, ductal and lobular types, with different clinical and biological characteristics.

IDC is the most common histologic type comprising 72-80% of all invasive breast cancers. It is classified in three degrees according to the advance of malignancy, i.e. poorly differentiated, moderately differentiated and well-differentiated.

In the present study, OCP residues were measured in morning fasting blood specimens and adipose tissue specimens to investigate their roles in breast cancer patients.

Seventy-five IDC patients were enrolled, with controls of 79 benign breast disease patients and 80 healthy women.

Only β-hexachlorocyclohexane (β-HCH) and pentachlorothioanisole (PCTA) were detectable in blood specimens, and only β-HCH, PCTA and *pp'*-dichlorodiphenyldichloroethylene (*pp'*-DDE) were detectable in adipose tissue specimens.

The results showed that the levels of both β-HCH and PCTA in blood specimens and all levels of β-HCH, PCTA and *pp'*-DDE in adipose tissue specimens were higher in the IDCs than in the controls and were increasingly higher among the IDC patients, whatever the degree of differentiation.

Due to their lipophilicity and persistence, OCPs frequently accumulate in human adipose and breast tissues, resulting in higher levels of OCP residues in the adipose tissue than in the blood.

The levels of β-HCH, PCTA in both blood and adipose tissue specimens were higher in estrogen receptor (ER) positive IDCs, than in ER negative IDCs, implying that OCPs, as endocrine disruptors, play some part in upsetting normal estrogen-progesterone balance, thereby contributing to the risk of breast cancer.

In conclusion, the higher level of OCP residues in the IDC blood and adipose tissue specimens in this study suggests some link with IDC, but the details remain uncertain.

Source: Environmental Toxicology and Pharmacology, Vol. 40, Issue 3, Pages 698–703, November 2015.

Prenatal Exposure to Chlorpyrifos and Childhood Tremor

(Continued from page 2)

including lead, pesticides, tobacco smoke, and harmane, a beta-carboline alkaloid found in coffee. Less attention has been devoted to the prevalence, clinical features and correlates of tremor among children.

As part of a larger prospective cohort research initiated in 1997, this study aimed to identify the longer-term motoric consequences of prenatal exposure to CPF in a sample of New York City children.

Prenatal exposure was measured in umbilical cord blood. Later, at approximately 11 years of age, children were followed-up to receive a comprehensive neurodevelopmental assessment.

The results show that children with high prenatal exposure to CPF were significantly more likely to show mild or mild to moderate tremor in one or both arms.

This is the first report of tremor resulting from early life exposure to this widely used pesticide.

The researchers recently reported that tremor was associated with poorer motor hand function, indicating either that the tremor itself resulted in some reduction in dexterity or that both the tremor and the loss of dexterity were a result of an underlying perturbed motor state.

The possible links between CPF exposure, tremor, and additional measures of motor function, remain to be studied.

Results suggest that early life exposure to CPF has increased the likelihood of mild or mild to moderate tremor in this sample of inner-city children.

Such disabilities could have adverse effects on daily motor tasks such as handwriting and writing-based school performance-outcomes that need to be assessed in future studies.

To conclude, tremor in middle childhood, may be a sign of the effects of prenatal CPF exposure on nervous system function.

Source: NeuroToxicology, Vol. 51, Pages 80–86, December 2015.

IMPACT OF MERCURY EXPOSURE ON IMMUNE STATUS IN CHILDREN

Studies have concluded that *in utero* exposure to environmental pollutants may cause changes in reproductive, metabolic and immune systems.

This is of particular concern because it suggests that even low levels of environmental pollutants can impact the developing fetus and affect the developmental trajectory and function of various systems.

Mercury exposure has been shown to affect immune status in animals, as reflected by cytokine expression. Mercury may also play a role in T-helper cells 1 (Th1)/ Th2 imbalance, susceptibility to autoimmune diseases and dys-regulated immune response to infections.

However, data in the literature does not present a clear picture regarding the impact of low-dose mercury exposure and long-term immune status in humans.

This may be due to the interacting effects of factors such as selenium body load, mercury species and pattern of exposure, and individual susceptibility to disturbances in immunological response that may have impact on the action of mercury and/or the cytokine profile.

The present study tested the hypothesis that fetal and childhood mercury exposure is associated with childhood cytokine profiles.

Researchers investigated whether childhood selenium levels interacted with any of the associations found.

Studying a group of children who would have a higher risk of mercury toxicity, the researchers aimed to compare the associations of fetal and current mercury exposure with a panel of cytokines to test whether the effect was stronger in individuals with a lower selenium body load, and whether the association between current mercury level and cytokine levels differed by cord blood mercury level.

A previous study of cord blood mercury concentrations in a cohort of 1057 newborns in Hong Kong found that cord blood mercury concentrations exceeded the US.EPA recommended level of 29 nmol/L in almost 80% of the babies tested.

Although such exposure levels may be considered "low-dose", compared with mercury levels in other countries, both maternal and cord blood mercury were at the high end of the "low-dose exposure" groups, probably due to high dietary fish and seafood consumption in the local community.

Children were recruited from a previously established birth cohort between the ages of 6-9 years for assessment and measurement of blood mercury, selenium and cytokine levels including interleukin (IL)-4, IL-6, IL-8, IL-10, IL-13 and tumor necrosis factors (TNF)-alpha.

This is the first birth cohort study to investigate the associations between *in utero* mercury exposure and markers of subsequent immune status.

It was found that childhood and cord blood mercury concentrations were not associated with levels of most of the cytokines tested.

The exception was for IL-10, where concentrations were negatively associated with current mercury exposure, but not fetal exposure. This was particularly true among children with lower current selenium concentrations and lower fetal mercury exposure.

None of the other cytokine levels were associated with either cord blood or current blood mercury concentrations, except that cord blood mercury was negatively associated with IL-6.

The differential impact of mercury exposure on different cytokines by time period of exposure may also indicate that *in utero* and postnatal/childhood mercury exposures affect immune status via different mechanisms.

As the adverse effects of mercury have been known to be alleviated by increased selenium levels, it is plausible that the stronger negative association between current mercury exposure and IL-10 concentrations in subjects with low selenium concentrations is due to a reduction in the protective effects of selenium.

The results suggested that differential associations between current mercury exposure and IL-10 by fetal mercury exposure are speculative and require replication, especially with methylmercury, the species of mercury most relevant to populations with high fish consumption.

Further studies on molecular changes induced by *in utero* methylmercury exposure and its impact on later associations between immune status and methylmercury exposure will improve the understanding of its immunotoxicity.

Moreover, further investigation will be needed to discover whether small reduction in IL-10 concentrations are associated with increased pro-inflammatory potential or development of autoimmunity.

Source: Environmental Research, Vol. 144, Pages 66-72, January 2016.

Dietary Exposure to Cadmium and Risk of Breast Cancer in Post-menopausal Women

Cadmium (Cd) is an extremely toxic metal commonly found in industrial workplaces. Soil contamination by cadmium represents a threat to health because grains, leafy and root vegetables bioconcentrate Cd, resulting in significant sources of Cd exposure for the general population through diet and tobacco smoking.

Low levels of Cd are so ubiquitous in the environment that concerns are being raised about adverse health effects.

Cd has been classified as a Group 1 human carcinogen with sufficient evidence for the lung and limited evidence for prostate and kidney (IARC, 2012).

Cd exerts estrogenic activities on the proliferation of breast cancer cells. Because Cd activates and increases expression of estrogen-regulated genes and activates the estrogen receptor (ER)-alpha, this metal appears to have the potential to induce the development of hormone-dependent tumors in humans, including breast, uterus and prostate cancers.

These carcinogenic and estrogenic activities make Cd a contaminant of potential concern for hormone dependent cancers, including breast cancer.

Breast cancer is the most frequently diagnosed cancer and the leading cause of cancer death among women worldwide. Post-menopausal women represent the most appropriate population to investigate the possible impact of exogenous factors with potential estrogenic activity on breast cancer because after menopause, their estrogenic influence is predominant.

The potential estrogenic influence of Cd should, therefore, be more closely monitored in post-menopausal women.

This article reviews available studies between 2012 and 2014 focusing on post-menopausal women and the association between dietary exposure to Cd and breast cancer.

Meta-analyses were performed on the whole set of data, and separate analyses were conducted after stratification for study design, geographic location, use of hormone replacement therapy (HRT), tumor estrogen receptor status (ER+ or ER−), progesterone receptor status (PGR+ or PGR−), body mass index (BMI), smoker status and zinc or iron intake.

When all the studies were combined, the results revealed no statistically significant increase in the risk of breast cancer.

Subgroup analyses made it possible, however, to identify several sources of inconsistency between these studies, including geographical location, tumor ER, PGR and smoker status, HRT use, BMI, and zinc and iron absorption.

This review does not therefore, support the hypothesis that dietary exposure to Cd increases the risk of breast cancer in post-menopausal women. However, misclassification in dietary Cd assessments in primary studies could have biased the results towards a finding of no association.

As studies estimating the dietary Cd intake in relation to breast cancer have only been performed during the last few years, the number of available studies is limited, and stratifications have led to restricted statistical persuasiveness and less precise risk estimates.

Further investigation of the increased risk of breast cancer caused by the estrogenic activity of Cd should focus on improved accuracy of individual dietary Cd intakes and differentiation of breast cancers by pathologic features.

This is the first comprehensive meta-analysis focusing exclusively on post-menopausal women and combining data on dietary Cd exposure and breast cancer risk.

Source: Environment International, Vol. 86, Pages 1–13, January 2016.

The Effect of Ventilation on the Indoor Air Concentration of PCBs

Exposure to polychlorinated biphenyls (PCBs) is known to cause adverse immunological, reproductive, and dermatological effects. PCBs also increase the risk of obesity and type-2 diabetes, and affect cardiac and neuropsychological functioning.

In 2013, the International Agency for Research on Cancer (IARC) categorized all PCBs as Group 1 carcinogenic to humans.

The Danish Health and Medicines Authority (DHMA) has stated that, air concentrations of PCB_{total} above 300 ng/m^3 may pose a health risk, and has suggested interventions for Denmark reducing concentration below this action level.

PCBs are categorised as semivolatile organic compounds (SVOCs). They are known to redistribute from the building materials in which they are originally introduced (i.e. the primary sources), finding their way to indoor surfaces (secondary sources).

These compounds are absorbed or emitted, depending on the surrounding concentration in the air. They migrate from primary sources to adjacent material (secondary sources).

Remediation is problematic since simple removal of the original source is often insufficient. However, ventilation can dilute the concentration of indoor contaminants if emissions are constant and independent of ventilation.

The transport mechanisms of PBCs depend on a range of factors like source/sink capacity, concentration, air concentrations, presence of airborne particles, ventilation and indoor and outdoor temperatures

The present intervention study was conducted to determine the impact of increased ventilation on PCB air concentration by installation of balanced mechanical ventilation units.

An elementary school classroom and two small bedrooms in a residential

(Continued on page 6)

Low-level Arsenic Exposure and Developmental Neurotoxicity in Children

Risk assessments of arsenic have for some time been focused on skin, bladder, and lung cancers, and skin lesions as sensitive cancer and non-cancer health endpoints, respectively.

A previous review of the literature on short-term health effects of inorganic arsenic in children did conclude that skin lesions were the most sensitive endpoint reported in arsenic-exposed populations. However, an increasing number of epidemiologic studies informing risk assessment have been examining neurodevelopmental effects in children.

Neurotoxicity has not been as clearly associated with low levels of exposures to arsenic, as it has for metals such as lead and mercury. Neurotoxic effects of arsenic may result through its more general toxic effects at higher exposure levels, or through interactions with nutrition, one-carbon metabolism, and methylation which are important for proper growth and neurodevelopment, particularly in nutritionally deficient populations.

This systemic review of 24 cross-sectional, case control and cohort studies evaluates current evidence on the impact of lower arsenic exposure levels i.e., largely <100 μg/L of arsenic in drinking water, on the central nervous system of children.

In particular, the available studies are evaluated for evidence supporting potential points of departure for developing a health-protective U.S. Environmental Protection Agency (EPA) reference level (RfD) for assessing health risks of non-cancer effects based on this endpoint.

An EPA RfD is a daily level of exposure at and below which no non-cancer health effects are expected, even in sensitive subpopulations, for up to lifetime exposure in the case of repeated, regular exposures.

In fact, however, the overall evidence does not consistently show a causal exposure-response relationship at low doses.

The most rigorously conducted studies from Bangladesh indicate possible inverse associations with cognitive function, predominantly involving concurrent arsenic exposure as measured by biomarkers (i.e., arsenic in urine or blood) and raw verbal test scores at ages 5-11 years.

This observation from Bangladesh could in part be related to the predominance of arsenic sources in that country. The slope of the relationship between total arsenic concentration in urine and water was much greater at higher arsenic water concentrations above 60 μg/ml.

A number of issues limit the possible applications of these findings to US populations: Non-comparability of outcome measures across studies; inaccuracies of biomarkers and other measures of inorganic arsenic exposure; potential effect modification by cultural practices; insufficient adjustment for nutritional deficiencies, maternal IQ, and other important confounders, as well as the presence of other neurotoxicants in foreign populations.

Of the few U.S. studies available, the most rigorously conducted did not find a consistent dose-response relationship between arsenic concentrations in tap water or toenails and decrements in IQ scores.

If the strongest dose-response relationship identified in the most rigorous evidence from Bangladesh could be generalized to U.S. populations, possible RfD for neurological effects in children would be estimated in the range of 0.4–1 μg/kg-day.

These doses are higher than the U.S. EPA's RfD for chronic lifetime exposure (0.3 μg/kg-day), thus indicating protectiveness of the existing value for potential neurotoxicity in children.

This value is being scrutinized under the EPA's ongoing assessments of both non-cancer and cancer toxicity values for arsenic, based on a number of potential health endpoints.

The EPA has also been advised to consider shorter-term exposures associated with neurodevelopmental effects in children, along with other possible health endpoints, in its reassessment of inorganic arsenic health risks.

Source: Toxicology, Vol. 337, Pages 91–107, November 2015.

The Effect of Ventilation on the Indoor Air Concentration of PCBs

(Continued from page 5)

apartment building were investigated. Three highly contaminated rooms were found to have secondary sources with large surface areas. The primary sources (PCB caulks) in both buildings were encapsulated.

The concentrations of PCB in the air as well as the air exchange rate were measured.

During the intervention, the air exchange rate increased during a two-month period. The intervention caused a significant decrease in the indoor air concentration of PCB_{total}, indicating an increase in the overall emission rate.

After the two-month period, the ventilation was turned off in the classroom. Concentrations of PCBs soon returned to the same levels measured prior to the intervention, after adjustment for indoor temperatures.

The results clearly demonstrate the ability of secondary contaminations to change from sink to sources, thereby confirming the difficulties in remediating SVOC contamination.

In future studies, the effect of increased ventilation on air concentration in cases with primary sources should be investigated as well.

In addition the effect of air velocity on mass transfer should be investigated as this alone may affect the emission and concentration of PCBs in indoor air.

Source: Building and Environment, Vol. 94, Pages 305-312, December 2015.

OCCUPATIONAL LEAD EXPOSURE AND ASSOCIATIONS WITH SELECTED CANCERS

Lead is a major environmental pollutant closely linked to industrial settings. The occurrence of lead in the environment has decreased greatly in recent decades because of the elimination of most leaded gasoline; however, occupational exposures to lead are primarily in the storage battery industry and lead pigments in paints.

In many developed countries, strict controls have reduced some of these environmental and occupational dangers. However, rapid industrialization in developing countries such as China, means that exposure to lead continues to be an issue worldwide.

The occupational exposure limits (OEL) for lead and for inorganic compounds of lead were set in China in 1979, based on maximum allowable concentrations of 0.05 mg/m^3 for lead dust and 0.03 mg/m^3 for lead fume.

Lead is a suspected carcinogen. Inorganic lead compounds are currently designated by the International Agency for Research on Cancer (IARC) as probably carcinogenic (Group 2A), based on limited evidence in humans and sufficient evidence in animals.

However, owing to inadequate evidence, organic lead compounds have been designated by the IARC as not classifiable with regard to carcinogenicity (Group 3). (IARC, 2006).

Epidemiologic studies of occupational lead exposure have suggested increased risks of cancers of the stomach, lung, kidney, brain, and meninges. However, the totality of the evidence is inconsistent.

Very few previous studies have evaluated occupational lead exposure among women, although differences between the sexes have been observed for lead exposure and metabolism.

There is thus a need for additional well-designed epidemiologic studies including both men and women to resolve the question of whether lead is a carcinogen.

The present study investigated the association between occupational lead exposure and risk of cancers of the stomach, lung, kidney, brain, and meninges in two large prospective cohort studies of women and men in Shanghai, China.

The results showed evidence of an association between exposure to lead dust or lead fume and an increased subsequent risk of meningioma in the female cohort, with higher cumulative exposure associated with higher risk.

The association of high lead exposure with brain cancer appeared to be limited to the female cohort. By contrast, in the male cohort, elevated risks of lung and stomach cancer were observed.

No significant associations between lead exposure and cancers of the kidney and brain were observed overall.

In 2006, when the IARC classified lead as a probable carcinogen, the epidemiological evidence was the most consistent for stomach cancer, with elevated kidney and lung cancer risks observed in some studies.

Evidence from human studies was considered limited. Since the publication of that monograph, several studies have attempted to further evaluate these risks.

The mechanisms by which lead may increase cancer risk remain unclear. Inhalation and oral ingestion are the two primary routes through which lead first enters the body.

The lungs and the stomach come into initial contact, but exposure then continues through the bloodstream, affecting other organs.

The brain and nervous system are especially sensitive to the potential toxic effects of lead as it passes through the blood-brain barrier. The high reabsorptive activity of the renal proximal tubules also lends itself to the accumulation and uptake of lead in the kidneys.

Some studies have shown very little or no mutagenicity for the main forms of lead. Therefore, it has been suggested that lead may act through indirect mechanisms to facilitate the carcinogenic effects of other DNA-damaging agents.

It has been proposed that lead may play a role in carcinogenesis through mechanisms that involve oxidative damage, induction of apoptosis, altered cell-signaling pathways, inhibition of DNA synthesis and repair, and interaction with DNA-binding proteins.

The different patterns of lead-exposed occupations reported for males and females may result in differences by cohort in the intensity and duration of lead exposure in the workplace. By occupation, males appear to be more vulnerable.

These combined results of two large cohort studies, are still too limited by the small number of exposed cases for some cancer sites, most notably brain cancer and meningioma, and by the relatively short follow-up period for the male cohort.

In the face of these constraints, any interpretation overall or sex-specific associations should be made with caution.

In conclusion, though limited by the small number of cases, it appears that lead exposure is associated with an increased risk of several cancers, in particular, meningioma, brain cancer, and kidney cancer in males and females.

The associations observed in women between lead and meningioma and between lead and brain cancer in the present study underscore the importance of including women in future studies attempting to evaluate the carcinogenicity of lead.

Source: Environmental Health Perspectives, Vol. 124, No. 1, Pages 97–103, January 2016.

ANNOUNCEMENT AND CALL FOR ABSTRACTS

The 8th Princess Chulabhorn International Science Congress

Congress Theme: ENVIRONMENTAL HEALTH: INTER-LINKAGES AMONG THE ENVIRONMENT, CHEMICALS AND INFECTIOUS AGENTS

November 13-17, 2016 at Shangri-La Hotel, Bangkok, Thailand

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

Nobel Laureate Lecture: The Critical Role of the Ubiquitin Pathway in the Development of Human Disease, ***Aaron Ciechanover (Nobel Laureate, Israel)***

The Congress will be held **to commemorate the seventieth anniversary celebrations of His Majesty King Bhumibol's accession to the throne, His Majesty's upcoming ninetieth birthday, and the seventh cycle (84 years) birthday of Her Majesty Queen Sirikit,** auspicious occasions for the people of Thailand to celebrate and pay tribute to Their Majesties the King and Queen. The program will feature a Nobel Laureate Lecture, Keynote Lecture, Plenary Lectures, Symposia, Platform and Poster Presentations.

Lectures (Partial list):

- Environmental Chemical Exposure and Infectious Agents
- Liver Cancer
- The Role of the Environment - Precision Medicine; Liver and Lung Cancer
- Emerging and Recurrent Infectious Diseases
- Using Exposomics to Assess Cumulative Risks from Multiple Environmental Stressors
- Immune Checkpoint Blockade in Cancer Therapies: New Insights and Opportunities for Cures
- Interdisciplinary Stratagems to Advance Fundamental Scientific Knowledge are Required to Reduce the Double Burden of Disease

Symposia (To be confirmed):

- Environmental Toxicants / Health Problems
- Pollution: the Single Largest Cause of Death and Disability
- Inflammation / Disease Development
- Environmental Causes of Chronic Diseases Studied in International Cohorts
- Global Epidemic of Obesity
- The Role of Nutrition / Diet to Reduce Disease Risks Associated with Environmental Exposures
- *In Utero* and Early Childhood Exposure and Cancer in Children
- Epigenetic Roadmap of Inflammation-related Disorders and Therapy Options
- Chemical-biological Synergism
- Infectious Diseases and Biotechnology
- New Technologies – Nanoscience
- New Approaches in Risk Assessment

Invited Speakers (Partial list):

Jan Alexander	(Norway)
James P. Allison	(U.S.A.)
William W. Au	(P.R. China)
Herman N. Autrup	(Denmark)
Jennifer Lyn Baker	(Denmark)
Jordana Bell	(U.K.)
Bruce Blumberg	(U.S.A.)
Alan R. Boobis	(U.K.)
Flemming R. Cassee	(The Netherlands)
Chunying Chen	(P.R. China)
Gwen Collman	(U.S.A.)
Daniel R. Dietrich	(Germany)
Tariq Enver	(U.K.)
Susan E. Erdman	(U.S.A.)
John M. Essigmann	(U.S.A.)
Ellen Fritsche	(Germany)
Rebecca Fry	(U.S.A.)
Peter R. Galle	(Germany)
Mary Gamble	(U.S.A.)
Timothy W. Gant	(U.K.)
Joseph Graziano	(U.S.A.)
John D. Groopman	(U.S.A.)
Curtis C. Harris	(U.S.A.)
Bernhard Hennig	(U.S.A.)
Denis L. Henshaw	(U.K.)
Jun Kanno	(Japan)
Philip Landrigan	(U.S.A.)
L.H. Lumey	(U.S.A.)
Susan Preston-Martin	(U.S.A.)
Ram Sasisekharan	(U.S.A.)
Martyn T. Smith	(U.S.A.)
Sir Michael R. Stratton	(U.K.)
William A. Suk	(U.S.A.)
Young-Joon Surh	(South Korea)
Duncan S. Sutherland	(Denmark)
Cathy Vaillancourt	(Canada)
Marco Vinceti	(Italy)
Xin Wei Wang	(U.S.A.)
Victor Wepener	(South Africa)
Kurt S. Zänker	(Germany)

FELLOWSHIPS:

A limited number of fellowships are available to **participants whose abstracts have been selected by the Scientific Program Committee**; this will cover:

1. Registration fee and/or accommodation at the Chulabhorn Research Institute (ONLY for participants from developing countries).

AND/OR

2. Partial or discount airfare by low cost airlines (ONLY for participants from Asian countries).

For further information, please visit the Congress Website: **https://pc8.cri.or.th**

CALL FOR ABSTRACTS:

Topics for Platform and Poster Presentations:

1. Chemical and Infectious Agents
2. Exposure
3. Diseases Resulting from Environmental Exposure
4. Mechanisms and Pathways of Disease Development
5. Modifiers of Susceptibility and Disease Outcomes
6. Tools and Technologies
7. New and Emerging Therapy

Selection of the submissions to be presented as platform or poster presentations will be made by the Scientific Program Committee.

Deadline for Abstract Submission:

September 15, 2016

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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