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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 Collaborates with WHO and Bhutan's Ministry of Health to Organize a Training Course on Risk Assessment and Risk Management of Chemicals in Paro, Bhutan from June 1-4, 2016

A team of international training experts in the field of risk assessment and risk management of chemicals, led by the Chulabhorn Research Institute (CRI), traveled to Paro, Bhutan to conduct in-country training on "**Risk Assessment and Risk Management of Chemicals**", in collaboration with Bhutan's Department of Public Health, Ministry of Health and the World Health Organization (WHO), through the WHO South-East Asia Regional Office and the WHO Country Office for Bhutan from June 1-4, 2016 with support from WHO South-East Asia Regional Office and WHO Country Office for Bhutan; the Thailand International Cooperation Agency (TICA), Ministry of Foreign Affairs, Thailand; and CRI

The course was designed to provide basic scientific knowledge of the principles and concepts of risk assessment and the process involved, to illustrate how risk assessment is conducted and what different issues are involved, and to introduce the WHO IPCS Human Health Risk Assessment Toolkit and the Electronic Distance Learning Tool (eDLT) on Risk Assessment and Risk Management of Chemicals, which was developed by CRI in collaboration with WHO IPCS, the University of Ottawa (Canada) and Utrecht University (the Netherlands).

The eDLT is an interactive, web-based, self-learning tool that is administered through a Learning Management System and hosted through a website at **http://www.chemDLT.com**, where interested persons can find more information about how to access and use it.

There are a total of 8 modules: Introduction, Problem Formulation, Hazard Assessment, Exposure Assessment,

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Risk Characterization – Human, Risk Characterization – Ecological; Risk Management; and Risk Communication.

The Bhutanese participants were given access to the eDLT two weeks prior to the start of the face-to-face training, went through one module and quiz in the face-to-face training for an opportunity to ask questions, and were given an additional week of access after the end of the face-to-face training to review the training material.

The Opening Address for this training course was delivered by Dr. Ornella Lincetto (WHO Bhutan Country Office). Welcome statements were also made by Ms. Lesley Onyon (WHO SEARO), and Mr. Rinchen Wangdi (Chief Engineer, Department of Public Health, Bhutan Ministry of Health).

The teaching faculty for this training course included Professor Martin van den Berg from Utrecht University (the Netherlands), Professor David MacIntosh, an adjunct professor at the Harvard School of Public Health (USA), Ms. Lesley Onyon from WHO SEARO, as well as Professor Mathuros Ruchirawat, CRI Vice President for Research and Academic Affairs, Dr. Jutamaad Satayavivad, CRI Associate Vice-president for Scientific Affairs, and Dr. Daam Settachan, research scientist from CRI's Laboratory of Environmental Toxicology.

The teaching team has vast experience in providing training for participants within the South-East Asian (SEA) region, having trained participants from Brunei Darussalam, Cambodia, India, Indonesia, Laos, Malaysia, the Maldives, Myanmar, Nepal, the Philippines, Sri Lanka, Thailand, Timor Leste and Vietnam, amongst others.

This training course was conducted as part of CRI's role as a WHO-SEARO-designated Regional Training Center for Chemical Safety in the SEA Region.

This training course on Risk Assessment and Management of Chemicals is an important step in the bilateral cooperation with Bhutan, reflecting the shared commitment of the two countries to improve the quality of life for their people. Chemicals Management and Chemical Safety are increasingly important issues for Bhutan.

The management of chemicals and the assessment of the risks involved in their effective and beneficial use in industry and agriculture is an area of specialization in which CRI has developed considerable expertise, both through education and research programs and through close collaborations with eminent scientists and researchers from renowned universities in other countries, as well as from international organizations such as the WHO.

This in-country training course was attended by 34 participants from various

governmental agencies and academic institutions, including: Bhutan Chamber of Commerce and Industry, Bhutan National Environmental Commission, Khesar Gyalpo University of Medical Sciences of Bhutan, Ministry of Agriculture and Forests, Ministry of Economic Affairs, Ministry of Education, Ministry of Health, and Ministry of Labour and Human Resources.

In addition to attending lectures, participants worked on several different case studies, including occupational and environmental exposures to benzene, health risks of polychlorinated biphenyls in indoor air from construction material, and the health impacts of arsenic in drinking water, and gave presentations on the final day of training to demonstrate their understanding of the risk assessment process.

As part of its capacity building programme, CRI regularly conducts in-country training in developing countries in the Asia-Pacific region in the areas of chemical safety, environmental health, toxicology and risk assessment in response to requests made from the respective country, either directly through an agency/institution with existing collaborations with CRI, or through an international organization such as the WHO, e.g. through the respective regional office at SEARO or WPRO. For more information on CRI's capacity building programme, including a calendar of training events, **please visit http://www.cri.or.th/en/envtox/default.htm.**

THE DISTRIBUTION OF MERCURY IN THE ENVIRONMENT AND ITS HUMAN HEALTH IMPACTS: A REVIEW

Mercury compounds are classified in different chemical types such as elemental, inorganic, and organic forms.

Human activity is the main cause of mercury release, particularly coal fired power stations, residential coal burning for heating and cooking, industrial processes, waste incinerators, and mining activities for gold and other metals.

The most significant source of ingestion-related mercury exposure in humans and animals is the consumption of fish.

Adverse health effects result from the speciation of mercury accumulated in the body, the quantity, frequency, and duration of exposure, and the age of the subject.

The presence of mercury compounds in many different sources (e.g., water, food, soil, and air) can lead, in the long term, to toxic effects on the skin, cardiovascular,pulmonary, urinary, gastrointestinal, and neurological systems of exposed persons.

Mercury toxicity is found to pose more significant health hazards to certain occupational groups, e.g., small-scale gold miners and dental personnel.

Many studies of neurological diseases have found evidence that amalgam fillings may play a major role in the development of conditions such as depression, schizophrenia, bipolar disorder, and memory problems.

This paper reviews the route of mercury exposure to humans, its human health impacts, the associated risk assessment, and treatment based on recent findings from various studies.

Low-dose mercury toxicity is relatively difficult to define, and safety precautions are generally insufficient at the personal level.

Because continuous exposure to mercury can be dangerous, it is desirable to re-evaluate the current reference (risk-free) values.

New technologies have been implemented in the chlorine alkali industry in a stepwise manner. In some fields, therefore, mercury should already have been phased out, as for example, in the health care sector, in mercury free measuring products, and in disinfectants.

Efforts have to be made to intensively mitigate the global mercury burden. In many developing countries, mercury is still a big problem requiring urgent action for proper control.

The main focus of such efforts should be the removal of anthropogenic sources of mercury and prevention of exposure.

Source: Journal of Hazardous Materials, Vol. 306, Pages 376-385, April 2016.

High Levels of Heavy Metals Increase the Prevalence of Sarcopenia in the Elderly Population

Despite increasing concern regarding health problems associated with environmental pollutants, no link between toxic heavy metals and sarcopenia has been identified in the general population.

There is growing concern worldwide regarding health problems due to exposure to heavy metals such as lead, mercury and cadmium, which are widely dispersed in the environment. The general population, not only those communities known to live in contaminated areas, are exposed to low doses of heavy metals during their lifetime.

Long-term exposure to heavy metals is already associated with the development and progression of bone disease. Several studies have demonstrated that osteoporosis and sarcopenia have similar pathophysiology and risk factors. Therefore, long-term accumulation of these heavy metals may be related to increased bone and muscle weakness.

Sarcopenia in the elderly is a disease resulting in certain muscular change. It is also a risk factor for impairment of physical function, limitation of mobility, falls, osteoporosis, and hospitalization.

As a pathophysiologic mechanism, sarcopenia generally results from a complex bone-muscle interaction in relation to chronic disease and aging. However, there have been no reports studying the relationship between heavy metals and sarcopenia.

The purpose of this cross sectional study was to assess the relationship between levels of lead, mercury and cadmium in the blood with sarcopenia and the cumulative effects on skeletal muscles in elderly populations (older than 65 years). Data was obtained from the Korea National Health and Nutritional Examination Survey.

The results show that elderly populations with sarcopenia have high serum levels of heavy metals in both genders. Males showed higher concentrations of blood lead, mercury and cadmium than females.

In 704 elderly persons (344 males and 360 females), sarcopenia was detected in male (26.7%) and in female (7.5%).

The accumulation of three heavy metals in the human body affects muscular mechanisms and results in sarcopenia. Although there has been no direct study of the link between toxic heavy metals and sarcopenia, the findings of this cross sectional study might offer one explanation of the relationship.

In conclusion, this study demonstrates that high levels of blood lead, mercury and cadmium increase the prevalence of sarcopenia in both genders of elderly populations.

Source: Journal of Bone Metabolism, Vol. 23, Pages 101-109, May 2016.

Recent Concerns: Environmental Pollutants and Child Health

Fetuses and infants have long been recognised as especially vulnerable to the effects of environmental agents that disrupt developmental processes with possible lifelong consequences.

In recent years, many new studies have evaluated associations between environmental pollutants and child health.

This study review aims to provide a broad summary of this literature, comparing the epidemiological evidence for the effects of a wide range of environmental contaminants on child health outcomes.

Environmental contaminants include air pollutants, heavy metals, organochlorine compounds, perfluorolkyl substances, polybrominated diphenyl ethers, pesticides, phthalates and bisphenol A.

The review addresses effects on fetal growth and prematurity, neuro-development, respiratory and immune health, and childhood growth and obesity.

As noted previously, much of the new evidence on chemicals and child health comes from the prospective birth cohort studies. The cohorts are characterised by biological sample collection at multiple time points during pregnancy, at birth, and after birth, with repeated follow-up examinations during childhood.

Findings of recent prospective studies and meta-analyses have corroborated previous good evidence, often at lower exposure levels, concerning effects on fetal growth of air pollution and polychlorinated biphenyls (PCBs), neurotoxic effects of lead, methylmercury, PCBs and organophosphate pesticides, and respiratory health effects of air pollution.

Moderate evidence has suggested the possible role of environmental pollutants in attention deficit hyperactivity disorder and autism (lead, PCBs, air pollution), respiratory and immune disorders (dichlorodiphenyldichloro-ethylene: DDE and PCBs), and obesity (DDE).

In addition, there is now moderate evidence that certain chemicals of relatively recent interest may be associated with adverse child health outcomes, specifically perfluorooctanoate and fetal growth, and the role of polybrominated diphenyl ethers in neurodevelopmental problems.

For other chemicals of recent concern, such as phthalates and bisphenol A, the literature is characterised by large inconsistencies which preclude strong conclusions.

Significant potential for collaborative analyses is indicated. Future research efforts should focus on harmonisation of exposure measurements and outcome assessments.

In conclusion, most of the recent literature evaluates common, and not particularly high exposure situations in general population. However, there is an accumulating body of evidence which suggests that the unborn and young child require more protection than is currently provided.

Large, coordinated research efforts are needed to improve understanding of long-term effects of complex chemical mixtures.

Source: International Journal of Hygiene and Environmental Health, Vol. 219, Pages 331–342, July 2016..

Potential Role of Organochlorine Pesticides in the Pathogenesis of Neuropsychological Development

Organochlorine pesticides were widely used chemicals for agricultural and environmental targets in the 20th century. Although the consumption of organochlorines has been banned in most countries, these compounds can still be detected in the environment, and in animal and human tissues.

Organochlorines induced neuro-logical diseases create major concerns for the health of newborns and adults. Although some scientists have evaluated the mechanisms by which organo-chlorines interfere with neuroendocrine systems and contribute to the development of chronic diseases, reviews of neurological disorders induced by organochlorines and the possible underlying mechanisms are lacking.

Epidemiological studies have indicated that the exposure of pregnant mothers to organochlorine pesticides results in impaired neurodevelopment and postnatal neuropsychological defects, including poor cognitive development, impaired motor functions, inattention, altered activity, and autism, as well as an increased risk of major chronic diseases such as cancers and endocrine system dysfunction later in life.

Moreover, commonly used organo-chlorine pesticides may have lasting effects on the central and peripheral nervous systems.

Growing evidence has indicated that exposure to organochlorines is linked to greater enhanced risk of dementia and Alzheimer's disease and other neurodegenerative disorders such as Parkinson's disease.

Parkinson's disease is a neurological disorder, which has been associated with exposure to organochlorines, leading to α-synuclein accumulation and the depletion of dopaminergic neurons.

This study reviews the potential association between pre- and post-natal exposure to organochlorines and impaired neurodevelopmental processes during pregnancy and neuropsychological diseases such as Parkinson's disease, behavioral alterations, seizures and autism.

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PRENATAL PHTHALATE EXPOSURES AND CHILDHOOD FAT MASS: NO EVIDENCE OF OBESOGENICITY

Childhood obesity has become substantially more prevalent in the past several decades. A growing body of evidence suggests that environmental obesogens, chemicals that alter metabolism, leading to increased fat production and deposition, may be partly responsible.

Phthalates are used in many consumer products, including toys, food packaging, cosmetics, and pharmaceuticals. Exposures to phthalates during gestation are hypothesized to affect production and accumulation of body fat by modifying peroxisome proliferator-activated receptors, associated with adipogenesis, lipid and carbohydrate metabolism, and androgen levels, or by disrupting thyroid hormone function.

Experimental animal studies and limited epidemiologic evidence suggest that prenatal exposure to phthalates may be obesogenic, with potential sex-specific effects of phthalates having anti-androgenic activity.

The current longitudinal, prospective cohort study was conducted to assess associations between prenatal phthalate exposures and childhood fat mass.

Phthalate metabolite concentrations were analysed in third-trimester maternal urine in a cohort of women enrolled at Mount Sinai Medical Center in New York City between 1998 and 2002. Fat mass was measured in the children during multiple follow-up visits between the ages 4 and 9 years.

The researchers did not observe associations between maternal urinary phthalate concentrations and percent body fat in models examining continuous exposures.

Fat mass was lower among children in the highest exposure group with the highest summed di(2-ethylhexyl) phthalate (ΣDEHP) concentration in maternal urine.

No notable modification of associations by the child's sex was observed although there was limited ability to detect heterogeneity in this small sample.

In conclusion, prenatal phthalate exposures were not associated with increased body fat among children 4-9 years of age, although high prenatal DEHP exposure may be associated with lower fat mass in childhood.

The finding that high prenatal DEHP exposure was associated with lower body fat in children runs counter to the hypothesis that phthalates are environmental obesogens. This hypothesis is based in part on evidence that phthalates interact with peroxisome proliferator-activated receptors, which are involved in metabolism.

However, some animal studies of relatively high dose postnatal DEHP exposure report lower body fat. Most animal and human studies of phthalates and adiposity have assessed postnatal exposures.

The researchers focused on prenatal exposures because susceptibility to obesity is thought to be 'programmed' during fetal development, making the fetus particularly sensitive to environmental exposures that affect fat development and accumulation.

The statistically significant association for DEHP and lower body fat was seen only in the highest exposure group.

There is still no agreement on what, if any, associations to expect in relation to prenatal phthalate exposure and child growth.

Larger prospective studies examining prenatal exposures are needed to replicate these findings. In addition, future studies will need to examine phthalates in terms of cumulative exposure.

Source: Environmental Health Perspectives, Vol. 124, No. 4, Pages 507-513, April 2016.

Potential Role of Organochlorine Pesticides in the Pathogenesis of Neuropsychological Development

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The hypothesis is consistent with recent studies emphasizing the correlation between environmental and genetic factors with the pathophysiology of neurodevelopmental and neurobehavioral defects.

It has been suggested that maternal exposure to organochlorine pesticides can result in impaired motor and cognitive development in newborns and infants. Moreover, *in utero* exposure to these compounds contributes to the etiology of autism. Although impaired neurodevelopment occurs through prenatal exposure to organochlorines, breastfeeding causes postnatal toxicity in infants.

Due to occupational exposure to pesticides, farmers and chemical workers are at increased risk of developing long-term brain damage, cognitive dysfunction and some serious behavioral and neurodegenerative disorders.

As serious neurological complications are among the most critical global life-threatening problems, there is an urgent need to adopt a preventive approach by restricting the use of industrial and agricultural pesticides and to find efficient biocompatible alternatives to replace hazardous ones.

Source: Life Sciences, Vol. 145, Pages 255–264, January 2016.

Exposure to Air Pollution and Cognitive Functioning Across the Life Course

Ambient outdoor air pollution is recognized as a global public health issue, both in developed and developing countries. Exposure to air pollution, whether in the long or short term, has been shown to be associated with increased risk of respiratory, cardiovascular and cerebrovascular disease. However, its association with cognitive functioning and impairment is unclear.

The relationships of air pollution to cardiovascular and cerebrovascular disease suggests a harmful impact on the brain and on cognitive processes through vascular and inflammatory mechanisms.

Studies of this pathology have begun to soften the distinction between Alzheimer's and vascular dementia.

However, no systematic reviews have as yet addressed the association between air pollution exposure and cognition across the full life course, including exposure *in utero*.

The aim of this systematic review was to examine whether a relationship exists between air pollution and cognitive outcomes in children and adults, and to identify areas for future research.

Because of variations in exposure assessment and outcome measures, meta-analysis was impossible. However, many studies showed weak but quantified relationships between various air pollutants and cognitive function. Pollution exposure *in utero* has been associated with increased risk of neuro-developmental delay.

Exposure in childhood has been inversely associated with neuro-developmental outcomes in younger children and with academic achievement and neurocognitive performance in older children. In older adults, air pollution has been associated with accelerated cognitive decline.

The evidence to date is coherent in that exposure to a range of largely traffic-related pollutants has been associated with quantifiable impairment of brain development in the young and cognitive decline in the elderly.

There is insufficient evidence at present to comment on consistency, in view of the different indices of pollution and end-points measured, the limited number of studies, and the probability at this stage of publication bias.

The burden of air pollution is expected to increase over the coming years if no counter measures are used to prevent it.

There is now accepted evidence that air pollution is responsible for a huge burden of cardio-respiratory disease. This review suggests that it may be playing a part also in increasing burdens of cognitive impairment, thus putting more pressure on governments worldwide to reduce levels of air pollution, in keeping with the imperative to reduce reliance on fossil fuels.

Source: Environmental Research, Vol. 147, Pages 383-398, May 2016.

Mercury as a Possible Link between Maternal Obesity and Autism Spectrum Disorder

Obesity is a metabolic disorder considered to be a worldwide epidemic, especially in developed high-income countries. Close connections have been shown between maternal overweight and obesity before and during pregnancy, and cognitive and neuropsychiatric disorders in offspring.

Autism spectrum disorder (ASD) is a behaviorally defined neuro-developmental disorder usually diagnosed in children during the first 3 years of life.

In recent decades there has been a significant increase in both ASD and obesity which suggests a link between increased rates of ASD with increased incidence of obesity and associated metabolic disorders like diabetes. However, the mechanisms of association between ASD and maternal obesity are unknown.

Taking into account the existing data indicating the association between mercury exposure, development of obesity, and ASD, the researchers hypothesize that mercury may serve as an additional link between maternal obesity and ASD.

The mechanisms of the influence of mercury on obesity may involve mercury-induced alteration of various signaling pathways in adipocytes, insulin production and signaling.

Obesity has been associated with excessive accumulation of mercury in the maternal organism. After conception, the fetus will be predisposed to develop ASD due to conditions of mercury overload within the obese body of the mother.

Previous studies have demonstrated that the overweight and the obese are characterized by a significantly higher level of mercury in hair, blood and urine than the non-obese. That is why the elevated mercury burden of an obese mother may be transferred to the fetus during pregnancy. Multiple studies have indeed suggested the placental transfer of mercury *in utero.*

Close associations have been demonstrated repeatedly between maternal and child mercury status. Cord blood mercury levels have been closely linked with maternal mercury blood levels during pregnancy, in agreement with earlier studies.

Finally, a growing body of data indicates the influence of mercury exposure and mercury status as an ASD risk for children. However, additional experimental and clinical studies are required to prove the hypothesis and to provide novel data on the role of mercury in maternal-obesity-associated ASD development.

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CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute
Schedule for 2016-2017

	Training Course	Date	Duration	Closing Date
1.	Environmental Immunotoxicology and Reproductive Toxicology	September 19 - 30, 2016	2 weeks	July 31, 2016
2	Environmental and Health Risk Assessment and Management of Toxic Chemicals	November 26 - December 13, 2016	2 weeks	October 10, 2016

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

Course Description:

1. Environmental Immunotoxicology and Reproductive Toxicology (September 19 - 30, 2016)

This course consists of 2 parts. The first part provides an overview of blood cells and the mammalian immune system, including a detailed description of all three arms of the immune response, how toxic chemicals can impact normal immune system homeostasis to result in adverse health outcomes, cellular/functional methodologies for determining the potential immunotoxicity of a given chemical, and how immunotoxicology relates to other scientific fields such as drug development and risk assessment. The second part provides an introduction to hormonally active agents and their mechanisms of action; routes of exposure, bioaccumulation, distribution and metabolism; effects on reproduction and development in humans and animals; and methods for studying changes in gene expression.

Requirement: Participants should have some basic knowledge on the biology of the immune and reproductive systems, as well as methodologies for assessing the functioning of these systems.

2. Environmental and Health Risk Assessment and Management of Toxic Chemicals (November 26 - December 13, 2016)

The course is an integration of science and policy, covering the fundamental basis of environmental and health risk assessment and management from exposure assessment and risk characterization; mode of action and human relevance framework; the relationship between risk assessment and risk management; and the need for open, transparent and participatory acceptance procedures and credible communication methods. Emphasis will be placed on human health risk assessment, although the principles of ecological risk assessment will also be covered. Importantly, the course teaches the practical application of risk assessment methods to various problems, e.g. hazardous waste site release, through the use of case studies relevant to problems faced in developing countries, and describes the policy context in which decisions to manage environmental health risks are made. Teaching and learning aids, such as an electronic distance learning tool on risk assessment and risk management of chemicals and the WHO IPCS Human Health Risk Assessment toolkit, will be introduced.

Requirement: Participants should have jobs/responsibilities related to assessment of risk from the use of chemicals.

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

Mercury as a Possible Link between Maternal Obesity and Autism Spectrum Disorder

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The contribution of mercury to ASD development in children from obese mothers should be determined. If a significant role of mercury in maternal obesity ASD risk is confirmed, additional perspectives of risk modification will be opened.

Taking into account the universal mechanisms of mercury toxicity, transport, and accumulation, further preventive actions may be undertaken to reduce the risk of mercury toxicity and mercury-associated ASD development.

In particular, it is supposed that the use of mercury chelators (like N,N-bis-(2-mercaptoethyl)isophthalamide, NMBI), antioxidants, and anti-inflammatory compounds prior or during pregnancy may have a beneficial effect.

However, the safety of such actions should repeatedly be tested to avoid adverse health effects in a developing fetus.

Source: Medical Hypotheses, Vol. 91, Pages 90-94, June 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: Why Our Proteins Have to Die, So We Shall Live?
The Ubiquitin Proteolytic System - from Basic Mechanisms thru Human Diseases and on to Drug Developmen,
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):

- Signatures of Mutational Processes in Human Cancer
- Cancer Genomic Heterogeneity and Hepatocarcinogenesis
- The Role of the Environment – Precision Medicine; Liver and Lung Cancer
- Using Exposomics to Assess Cumulative Risks from Multiple Environmental Stressors
- Immune Checkpoint Blockade in Cancer Therapies: New Insights and Opportunities for Cures
- The Complex Pathway to Noncommunicable Diseases: Opportunities for Prevention

Symposia (To be confirmed):

- 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
- Impact of the Epigenetic Road Maps on Cancer and Inflammation-related Disorders
- 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.)
Chunying Chen	(PR China)
Gwen Collman	(U.S.A.)
Stephania A. Cormier	(U.S.A.)
Giannino Del Sal	(Italy)
Daniel R. Dietrich	(Germany)
Paul Elliott	(U.K.)
Tariq Enver	(U.K.)
Susan E. Erdman	(U.S.A.)
John M. Essigmann	(U.S.A.)
Soldano Ferrone	(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.)
Marco Guerrini	(Italy)
Curtis C. Harris	(U.S.A.)
Peter Hecht	(U.S.A.)
Bernhard Hennig	(U.S.A.)
Denis L. Henshaw	(U.K.)
David L. Heymann	(U.K.)
Hans-Peter Huber	(Germany)
Jun Kanno	(Japan)
Michele La Merrill	(U.S.A.)
Qing Lan	(U.S.A.)
Philip Landrigan	(U.S.A.)
Monica Lind	(Sweden)
L.H. Lumey	(U.S.A.)
Roberta McKean-Cowdin	(U.S.A.)
Shoji Nakayama	(Japan)
Eng Eong Ooi	(Singapore)
Michael Rosenblatt	(U.S.A.)
Nathaniel Rothman	(U.S.A.)
Jonathan M. Samet	(U.S.A.)
Ram Sasisekharan	(U.S.A.)
Udo Schumacher	(Germany)
Poonam Singh	(WHO, SEARO)
Rahul Singhvi	(U.S.A.)
Peter Sly	(Australia)
Martyn T. Smith	(U.S.A.)
Avrum Spira	(U.S.A.)
Gary Stoner	(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.

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>

For back issues of our newsletter, please visit:

http://www.cri.or.th/en/envtox/et_newsletter.htm