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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".

Effects of Pesticides on Human Health: Cancer and Other Associated Disorders

Poisoning from pesticides is a global public health problem and accounts for nearly 300,000 deaths worldwide every year.

Pesticides are used extensively in agricultural and in domestic settings. The mode of exposure signifies the concentration of pesticides exposure.

Exposure to pesticides is thought to be linked to numerous health disorders such as Hodgkin's disease, non-Hodgkin lymphoma, Parkinson's disease, endocrine disruption, respiratory and reproductive disorders.

Pesticides are believed to cause cancers in humans. Glyphosate, for example, is associated with breast cancer. Pesticides which contain alkyl ureas and amines are found to be associated with brain tumors. Dieldrin causes tumors in the lung, liver, lymphoid tissue, uterus, thyroid, and mammary glands in test animals at doses as low as 0.1 ppm.

Several pesticides are recognized as endocrine disruptor compounds (EDCs). They interfere endocrine systems and may have adverse effects on physiological processes including development, growth, and reproduction in organisms and/or in their progeny.

Oxidative stress is one of the major mechanisms associated with several diseases and aging. Many pesticides (paraquat, rotenone and maneb) cause reactive oxygen species (ROS)-mediated stress and neurodegenerative diseases.

Oxidative stress is known to cause DNA damage which in turn may cause malignancies and other disorders.

Many pesticides have been shown to modulate gene expression at the level of non-coding RNAs, histone deacetylases, and DNA methylation patterns, suggesting their role in epigenetics.

Research from the past few decades has tried to identify more precisely the mechanisms of action by which pesticides produce their harmful effects.

The present study reviews abnormalities associated with pesticides with special emphasis on cancer, Parkinson's disease, Alzheimer's disease, pesticide-mediated disorders of respiratory and reproductive tracts, as well as epigenetic modifications.

A common concept developing from the large number of genetic, biochemical and cell biological studies is that the oncogenic and other disease-causing potentials of pesticides demonstrate dose and context-dependent effects.

It is new evident that most of the pesticides used worldwide can affect normal cellular metabolism in one way or another.

The major hurdle to such studies is the limitation of low dose chronic exposure to pesticides. Animal models may show similar responses, but the associated repair machinery is different. The life span and dietary patterns of animals and humans are also different. Mice and rats have far fewer repair enzymes compared to humans.

For the sake of study, we cannot ask humans to voluntarily consume pesticides. The questions are challenging and new

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Effects of Pesticides on Human Health: Cancer and Other Associated Disorders

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models must be explored by the scientific community.

Many studies have shown a significant correlation between pesticide poisoning and oncogenic modulations, and there are some studies which support these findings.

At the molecular level, it is well established that pesticides can cause oxidative stress and act as endocrine disruptors. Damage to DNA and other macromolecules linked to pesticide poisoning can modulate gene expression.

Such changes are mostly harmful to cells and play a key role in the pathogenesis of multiple disorders including cancer.

The findings of recent studies related to pesticides and epigenetics indicate that pesticides can cause numerous modulations in DNA methylation patterns, histone modification and also at the level of non-coding RNAs.

Finding alternatives to chemical pesticides is a major area of concern. More advanced research is needed to develop less harmful, more specific, and more efficient pesticides which will be less toxic to humans and the environment.

The best way to decrease the damage caused by these chemicals will be to investigate narrow-spectrum pesticides and natural agents such as bacteria and viruses which have ultimate specificity.

An alternative approach maybe to use insect hormones such as juvenile and molting hormones and their derivatives as insecticides. These could be alternative strategies to avoid resistance in insects and to minimize effects on non-targeted species, including humans.

Source: Environmental Toxicology and Pharmacology, Vol. 63, Pages 103-114, October 2018.

Modeling Parkinson's Disease and Treatment Complications in Rodents

Animal models of neurological deficits are essential to assess new therapeutic options and reduce treatment complications.

Over the last decades, several rodent models of Parkinson's disease (PD) have been developed, and have now become the first-line experimental tool for therapeutic screening purposes.

Parkinson's disease (PD) is a progressive neurodegenerative disorder affecting approximately 1% of the population over 60 years of age.

It is clinically characterized by typical motor symptoms including akinesia, bradykinesia and tremor at rest. These symptoms are attributed to the progressive degeneration of dopaminergic neurons in the substantia nigra pars compacta (SNc) and their axonal projections to the caudate-putamen.

PD is a multisystem neuro-degenerative disorder in which non-motor symptoms (autonomic, psychiatric and cognitive ones) also play an important role and significantly affect patients' quality of life.

Animal models are essential to investigate molecular mechanisms of neurodegeneration as well as network dysfunctions causing specific symptoms and treatment-related complications. Furthermore, animal models are needed to test new potential therapies.

The present review focuses on the different rodent models currently available to replicate behavioral phenotypes relevant to PD, discussing their advantages and disadvantages in relation to different criteria for model validation.

The models currently available can be grouped into 3 main categories, based on the method used to induce specific features of the human disease: i) pharmacological models; ii) toxin models; iii) genetic models.

None of the models so far available is able to recapitulate all the features of the human disease, but several well-characterized models with com-plementary features currently provide a valuable repertoire of tools to address specific scientific hypotheses.

There is in nature a high degree of genetic homology and neuro-anatomical similarity among mammalian species, including mice, rats and primates.

Despite the pitfalls inherent in the animal model selected, each model is a valuable tool to address specific scientific question, and test potential new therapeutic approaches.

Which is the most predictive model for identifying the efficacy of symptomatic or potential disease-modifying interventions? This is still an open question and requires successful translation of therapeutic principles from the lab to the clinic in order to be resolved.

In the meantime, animal models can be used to address fundamental questions about the biological effects of particular treatments. However, only the translation of the results into clinical studies would make it possible to observe which category of PD symptoms or aspects of human pathology can be improved by a certain therapy.

One possible future development may consist in combining the features of different models, creating "mixed-models", for example models presenting at the same time a sustained dopamine nigrostriatal degeneration and alpha-synuclein accumulation or other pathologies in specific pathways.

Such improvements may aid in the assessment of new therapies attempting, for example, to simultaneously intervene in both the symptoms and the progression of a disease.

Source: Behavioural Brain Research, Vol. 352, Pages 142-150, October 2018.

Hearing Loss in Children with E-waste Lead and Cadmium Exposure

Hearing loss is one of the most common chronic disabling conditions. Roughly 360 million people around the world suffer from moderate to profound hearing loss due to various causes.

The incidence of neonatal hearing loss in the U.S. is nearly 1.1 per 1000 infants and the average prevalence of mild or worse unilateral or bilateral hearing impairment in children and adolescents exceeds 3%.

For decades, most efforts have attempted to link occupational noise or ototoxic drugs (e.g., antibiotics) with hearing impairment. Exposure to either can initiate a series of pathological changes which can result in damages to spiral ganglion neurons within the cochlea. Some damage can occur, even when the hair cells in the inner ear are not sufficiently injured to undergo degeneration.

Recent studies have shown that heavy metal exposure may also be considered a risk factor for hearing loss in humans.

Lead (Pb) is a ubiquitous environmental toxicant already associated with many potential health problems. An early comparative study investigated the changes in Pb-induced neurobehavior, and found that the brainstems auditory evoked potential, both in animals and school-age children, was diminished.

Cadmium (Cd) is a typical nephrotoxicant that may produce a series of physiological disorders, even cancer. A recent *in vivo* study finds that high Cd exposure may cause an increase in auditory threshold and a decrease in response latency.

Research on the ototoxic effects of lead (Pb) and cadmium (Cd) has not been conclusive. However, experimental data does reveal damage to cochlea or vestibular function through ROS generation and apoptosis, leading to disorders in auditory nerve conduction and finally to significant hearing loss.

Guiyu, a town in China's Gyangdong province, is a typical electrical waste (e-waste) recycling area. Guiyu has attracted much attention among researchers over the last few decades due to local practices of informal dismantling of toxic waste without proper environmental protection measures. In previous studies, the levels of heavy metals in children and neonates of Guiyu were found to be much higher compared to other areas.

In terms of the ototoxic effect of heavy metals, there are only limited epidemiologic studies available regarding the prevalence of hearing loss in children, especially in an e-waste-polluted area.

This study, which focused on the association between blood Pb and urinary Cd exposure and hearing loss in early childhood, attempted to determine to what extent exposure to these environmental chemical pollutants affect the hearing of preschool children living in Guiyu.

Blood Pb and urinary Cd were collected from 234 preschool children, aged 3-7 years, from an electronic waste (e-waste) recycling area and a reference area matched in Shantou, in southern China.

Pure-tone air conduction (PTA) was used to test child hearing thresholds at frequencies of 0.25, 0.5, 1, 2, 4 and 8 kHz. A PTA ≥ 25 dB was defined as hearing loss.

Researchers found that children living in the e-waste area showed reduced hearing ability, compared to children in the reference area. Because Pb exerts a significant risk for child hearing loss,. Numbers of such susceptible populations living in e-waste recycling areas bear a higher risk of hearing loss due to long-term environmental chemical insults.

A higher median blood Pb level was found in the exposed group. Compared with the reference group, the exposed group also had a greater prevalence of hearing loss.

No statistical association was found between urinary Cd levels and child hearing loss in this study. Because the specimen source, study size and body burden of exposure may have partially influenced the final results in this aspect of the study, further research is warranted to verify these findings.

The PTA in the left and right and in both ears, and hearing thresholds at average low and high frequencies, as well as single frequencies of 0.5, 1 and 2 kHz, were all increased in the exposed group.

The results show that Pb exposure correlated with the age of the child and with nail biting habits. Decreased Pb and Cd levels were related to higher levels of the parents' education and whether or not a child washed her hands before dinner.

This suggests that long-term exposure, increasing age, and poor hygiene may contribute to the accumulation of Pb in the body. Washing hands and other good hygiene practice in the family are protective factors which reduce opportunities for contamination.

This study is basically a first attempt to determine hearing loss in 3- to 7-year-olds who live in an informal e-waste recycling area.

The researchers found a high proportion of hearing loss and higher average hearing thresholds among children living in the e-waste area, compared to the reference area. Their results showed that low-frequency hearing is affected and associated with blood Pb level, but not with Cd exposure.

To conclude, the present study suggests that early childhood exposure to Pb from e-waste recycling areas may affect hearing development.

These results point to the need to pay more attention to the developmental auditory system of children and to reduce children's exposure to environmental chemical pollutants in e-waste recycling areas.

Source: Science of The Total Environment, Volume 624, Pages 621-627, May 2018.

HEI's Report: One in Three Households Worldwide Exposed to Household Air Pollution

In 2016, a total of 2.5 billion people, a third of the global population, were exposed to household air polluted by the use of solid fuels for cooking and heating, according to a new report, **Household Air Pollution and Noncommunicable Disease (Communication 18)**, issued on July 31, 2018 by the Health Effects Institute (HEI).

This enormous population faces a significant health risk. Exposed in daily life to high levels of particulate matter from household air pollution (HAP).

The latest scientific evidence from a growing number of epidemiological studies and systematic science reviews point to the fact that HAP exposures increase the risk of many noncommunicable diseases, including cataracts, heart disease, and respiratory diseases including lung cancer.

In 2016, HAP exposures contributed substantially to the global disease burden, translating into an estimated 2.5 million deaths. The economic consequences of HAP health impacts are also large. The best available estimate from the World Bank dates from 2013 and suggests an annual global welfare loss in 2011 of about $1.5 trillion U.S. dollars from HAP exposures.

Most of the people affected live in low and middle-income countries in Asia and Africa. These populations, especially women and children, who spent more time at home, face a double burden. The air they breathe indoors contributes to the toxicity of the air outdoors, with its already full range of pollutants from industry, transport, and other sources.

Clean energy solutions are necessary to substantially reduce disease burdens.

The report found that traditional interventions to reduce exposure, for example, by introducing improved solid fuel cookstoves, have had mixed effects. Some reductions in exposure are seen, but with relatively few health benefits.

The cost of alternative fuel, cultural attachment to older stoves, and the challenges of operating new stoves are all likely contribute to lower than expected improvements in exposure and health.

These findings suggest that more extensive clean energy solutions, such as bringing natural gas and electricity to rural homes, are needed to significantly improve health outcomes.

Introducing such new solutions could have substantial public health benefits. HEI's Global Burden of Disease from Major Air Pollution Sources (GBD MAPS) project estimates that, in China and India alone, policies that shift to reliance on clean fuels could decrease the future burden of disease from ambient air pollution attributable to the residential burning of solid fuels by at least 30% and possibly by more than 95%, depending on the policy.

In India, for example, a policy that would virtually eliminate the use of biomass cookstoves by 2050 could avoid 500,000 early deaths from outdoor air pollution annually, compared to a business-as usual scenario. Eliminating older, more traditional stoves would have substantial benefits for indoor air quality as well.

Communication 18 provides a critical assessment of the state of the science examining the linkages between household air pollution formed by the burning of solid fuels and noncommunicable diseases. The report updates previous systematic reviews with the most recent studies. It answers fundamental questions on the scientific basis for estimating health burden and what the evidence suggests about the exposure reductions necessary to achieve improved health outcomes.

For further information, please visit - https://www.healtheffects.org.

Source: Health Effects Institute (HEI), Communication 18, July 2018.

Lead Exposure Induces Alzheimers's Disease-like Pathology and Disturbes Cholesterol Metabolism in the Young Rat Brain

Developmental lead exposure induced neurodevelopmental disorders including abnormality of synapse structure, irregularity of neurotransmitter activity and dysfunction of neuronal energy and glycogen metabolism. However, the molecular mechanisms by which lead exerts its neurotoxic effects still need to be clarified.

Alzheimer's disease (AD) is a neurodegenerative disease and is the major cause of dementia worldwide. A characteristic hallmark of AD pathology is the deposition of amyloid-beta (Aβ) peptides, which are cleaved from amyloid precursor protein (APP) following processing by β-secretase (BACE1) and γ-secretase. Aβ accumulation in the brain induced neuroinflammation, neurofibrillary tangle formation, neurite dystrophy and synaptic deficits that ultimately lead to neuronal death.

Overwhelming evidence has demonstrated that dysregulation of cholesterol homeostasis in the brain, especially during development, may lead to learning deficits and cognitive impairment and has even been implicated in a growing number of neurodegenerative diseases such as AD.

Brain cholesterol homeostasis plays a direct role in the regulation of Aβ production and disruption of intracellular cholesterol signaling molecules induced AD-like progression.

The present study focused on the role of cholesterol metabolism in lead induced premature AD-like pathology. Weaning rats were treated with lead at different concentrations for 4 weeks.

The results found that developmental lead exposure increased

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Exposure to Volatile Organic Compounds and Airway Inflammation

Indoor air pollution is attracting attention as people spend more time inside. Volatile organic compounds (VOCs) are important indoor air pollutants. These compounds are produced by evaporation at room temperature from diverse sources, such as building materials, paints, cleaning agents, furnishings, adhesives, combustible materials, floor, and wall coverings.

Greater use of VOC-containing products, more effective insulation, and less external ventilation of modern buildings has contributed to increased VOC exposure.

Respiratory symptoms have already been associated with VOCs in industrial or occupational environments, but the effects of exposure to low concentrations, generally, in the indoor environments of daily life are not clear.

Sick building syndrome (SBS) is associated with various health problems that patients may report. The causes of the problems are typically unclear, but there are possible connections with the indoor environment.

In fact, the causal relationship between SBS and the indoor environment was a topic of much debate until a study confirmed the relationship between exposure to VOCs, SBS symptoms, and oxidative stress among office workers.

Another study showed that levels of VOC metabolites in urine correlated with oxidative stress and decreased lung function. However, the mechanisms by which VOCs and oxidative stress contribute to decreased lung function remain unclear.

No studies have been performed on airway inflammation induced by exposure in ordinary life to VOCs, and longitudinal studies about SBS are rare, although there have been a large number of cross-sectional studies.

The present study hypothesized that exposure to VOCs promotes airway inflammation.

Researchers evaluated the levels of VOC metabolites in urine, as well as oxidative stress markers, lung function, and inflammatory markers, including fractional exhaled nitric oxide (FeNO) and the urine leukotriene E4 (uLTE4) of subjects, before and after they moved into a new building with a relatively high level of ambient VOCs.

This study showed that the level of indoor VOCs in the new building was higher than in the old building.

Symptoms included eye dryness and eye irritation, as well as increased levels of a xylene metabolite (o-methylhippuric acid) after subjects moved into the new building.

This study showed changes in inflammatory markers before and after a move into a new building where higher levels of ambient VOCs and formaldehyde were present.

Decreased levels of FeNO and an increase in uLTE4 after the move suggested changes in the inflammatory environment of the subjects' airways, and possibly increased non-Th2 inflammation.

The FeNO measurement is used to assess the degree of Th2 allergic inflammation in the airway. Decreased levels of FeNO after the move suggested a decreased level of Th2 inflammation in the airway, and may suggest a relative increase in non-Th2 inflammation in the airway with increased uLTE4.

In addition, a high ratio of uLTE4 to FeNO (uLTE4/FeNO) seems to indicate predominantly non-Th2 airway inflammation. Thus, increases in uLTE4 and uLTE4/FeNO in the current study suggest an increase in non-Th2 inflammation in the airway of the study population after they moved into the new building.

Some patients with asthma insisted that their respiratory symptoms became aggravated after they moved into a new house. They pointed to the new indoor environment as a trigger for their symptoms.

This longitudinal study is the first to show that 7 days of VOC exposure in ordinary life can affect airway inflammation, possibly non-Th2 inflammation, and the lung mechanics of the general population.

Source: Environmental Health, Vol. 17, No. 65, August 2018.

Lead Exposure Induces Alzheimers's Disease-like Pathology and Disturbes Cholesterol Metabolism in the Young Rat Brain

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Aβ accumulation and amyloid plaque deposition in the cortex and hippocampus.

The study demonstrated that lead exposure significantly induced early AD-like pathology and disturbed cholesterol metabolism in young growing rat brains.

Lead exposure induced early AD-like progression in young rats by aggravated Aβ42 accumulation and amyloid plaque deposition, increased APP and BACE1 protein expression.

Then, lead exposure dysregulated cholesterol metabolism by enhancing the expression of sterol regulatory element binding protein 2 (SREBP2), liver X receptor-α (LXR-α) and ATP-binding cassette transporter protein family member A1 (ABCA1) and decreasing the expression of 3-hydroxy-3-methylglutaryl-CoA reductase (HMG-CR) and low density lipoprotein receptor (LDL-R).

With the dose increased, the adverse effects of lead exposure got worse. All of the above results suggested that disturbed cholesterol homeostasis in young rats may underlie the deleterious effects of lead-induced early AD-related pathology.

Source: Toxicology Letters, Vol. 296, Pages 173-183, October 2018.

The Chem HelpDesk

"Strengthening capabilities for sound chemicals management"

The Regional HelpDesk for Chemical Safety, or **Chem HelpDesk** was established as a joint-initiative between WHO SEARO and Chulabhorn Research Institute (CRI), through the WHO Collaborating Center for Capacity Building and Research in Environmental Health Science and Toxicology. The aims of the Chem HelpDesk are to address the issue of the widening gap in the fields of chemical safety and chemicals management between developed and developing countries, and to empower countries in the South-East Asia Region to manage the import, manufacture and processing, storage, distribution, transport, use, recycling and disposal of chemicals, in ways that minimize significant adverse impacts on health and the environment.

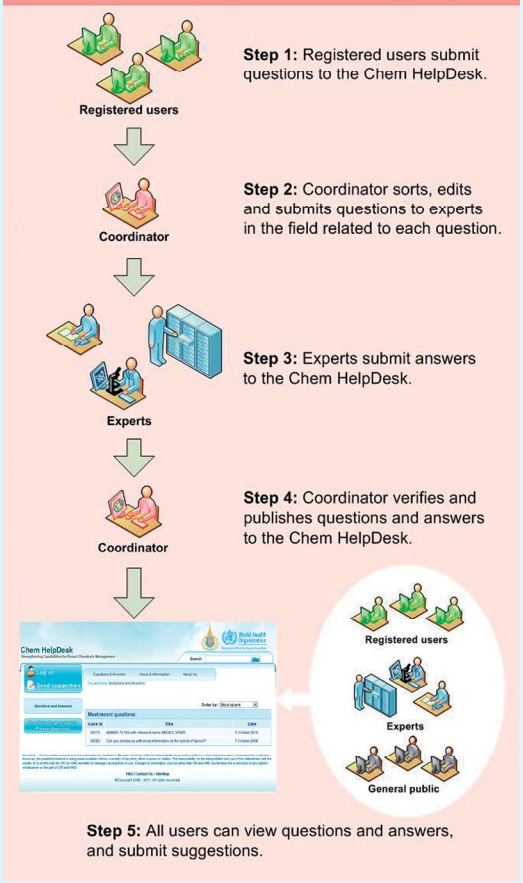
The Chem HelpDesk is not-for-profit, and through its website provides cost-free answers to questions submitted by registered users. These answers are provided by experts in the respective fields, who supply technical and scientific advice as part of a Community of Practice (CoP). It is the aim of the Chem HelpDesk to benefit users and to help countries in areas of most need to protect human health and the environment through the safe use and management of chemicals.

In addition to the **"Questions & Answers"** service for registered users, the website also provides information on the safe use of chemicals, as well as a comprehensive list of links to other important websites related to chemicals management in the region. General users have access to the database of questions and answers, as well as all other information on the website.

For more information, please visit: http://www.chemhelpdesk.org

or e-mail: coordinator@chemhelpdesk.org

The functional workflow of the Chem HelpDesk is divided into 5 steps:



Long-Term Exposure to Ambient $PM_{2.5}$ and Chronic Kidney Disease

Chronic kidney disease (CKD) represents a serious global public health challenge and is increasingly prevalent in both developed and developing countries.

The Global Burden of Disease Study 2015 estimated that deaths from CKD had increased by 31.7% from 0.9 million in 2005 to 1.2 million in 2015 and ranked as the 17th leading cause of death worldwide.

The most severe stage of CKD, end-stage renal disease, requires costly dialysis or transplant, seriously affects patients' quality of life, and results in an enormous economic burden. Besides itself posing a direct threat, CKD is also closely associated with other forms of morbidity, especially cardiovascular disease, the leading global cause of death.

The cardiovascular mortality rate is about two to three times higher in patients with stage 3 or 4 CKD than in those with normal kidney function. The traditional cardiovascular risk factors, such as obesity, hypertension and diabetes, are also CKD risk factors.

Air pollution has been regarded as a novel risk factor for cardiovascular diseases. Exposure to PM with an aerodynamic diameter of less than 2.5 μm ($PM_{2.5}$) is causally associated with an increased risk of cardiovascular diseases. However, there is limited information about CKD and air pollution.

The present study conducted a large cohort study to investigate the association between long-term exposure to $PM_{2.5}$ and the development of CKD in 100,629 adults in Taiwan.

Ambient $PM_{2.5}$ concentration was estimated at each participant's address using a satellite-based spatiotemporal model.

Incident CKD cases were identified by an estimated glomerular filtration rate (eGFR) of less than 60mL/min/1.73 m^2. A wide range of potential confounders/ modifiers during the medical examinations were collected.

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CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute
Schedule for 2018 - 2019

	Training Course	Date	Duration	Closing Date
1.	Environmental and Health Risk Assessment and Management of Toxic Chemicals	December 3-15, 2018	2 weeks	October 20, 2018
2.	Environmental Toxicology	April 2019	10 working days	February 28, 2019
3.	Occupational and Environmental Health	May 2019	1 week	March 29, 2019

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

Course Description:

Environmental Toxicology (April 2019)

The course provides students and participants with a background of the major groups of toxic substances encountered by man and animals through food and the environment, and also through exposure at the workplace. These toxicants include mycotoxins, naturally occurring plant and animal toxins, toxic substances in air, water and soil, N-nitroso compounds, solvents, plastics, pesticides and pollutants. The course focuses on the chemistry, fate and distribution in the environment, mechanisms of their action, toxic manifestation in living organisms, as well as toxic syndrome in human beings.

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

Fellowships:

A limited number of fellowships are available that will cover roundtrip airfare, accommodation (on site) and meals, training materials, and health insurance.

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

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

Long-Term Exposure to Ambient $PM_{2.5}$ and Chronic Kidney Disease

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The study shows that long-term exposure to ambient $PM_{2.5}$ was associated with an increased risk of incident CKD (based on eGFR <60mL/min/1.73 m^2 at a follow-up visit) among adult residents of Taiwan.

During the follow-up, 4,046 incident CKD cases were identified, and the incidence rate was 6.24 per 1,000 person-years.

A significant concentration-response trend was observed. Every 10$\mu g/m^3$ increment in the $PM_{2.5}$ concentration was associated with a 6% higher risk of developing CKD.

The association between $PM_{2.5}$ exposure and CKD development remained robust after adjustment for a wide range of potential confounders and modifiers.

The study used the satellite-based spatiotemporal model with a high spatial resolution to estimate the long-term exposure level to $PM_{2.5}$. This novel technology enables researchers to obtain the individual level of exposure to $PM_{2.5}$ and overcome the spatial coverage and interpolation problems that occur when using only data from monitoring stations.

In conclusion, this large-cohort study found that long-term exposure to $PM_{2.5}$ was associated with an increased risk of CKD. Although the estimated increase in risk was small at the individual level, the relevant public significance could be tremendous, given that exposure to air pollution is ubiquitous.

CKD not only contributes to total mortality, but also seriously affects the patients' quality of life. The findings support the global strategies of air pollution reduction to prevent CKD development.

Source: Environmental Helath Perspectives, Vol. 126, No. 10, October 2018.

International Conference on

"Liver and Lung Cancer : Current and Future Research"

January 8-10, 2019

at Chulabhorn Convention Center, Bangkok, Thailand

Announcement and Call for Abstracts For Poster Presentation

*Abstract Submission Deadline – **November 25, 2018***

https://cancer2019.cri.or.th

Keynote Lectures:

- CAR-T and Advances in Cancer Immunotherapy

Sessions (Tentative):

Session 1: Population Studies of Liver and Lung Cancer

- Etiological Factors for Liver Cancer
- Lung Cancer in Non-smokers
- Diversity of Lung Cancer Risk and Survival
- Liver Fluke and Cholangiocarcinoma

Session 2: Cancer Drivers, Biomarkers of Risk, Diagnosis, Prognosis and Response to Therapy

- Search liver Cancer Drivers
- Drivers of ICC
- Drivers of HCC
- Molecular Subtypes of Liver Cancer
- Hepatobiliary Cancer Genome
- Detection and Localization of Surgically Resectable Cancers with a Multi-analyte Blood Test
- Advances in Improving Low Dose CT Diagnosis of Early Stage Lung Cancer
- Molecular Studies of Fields of Injury and Cancerization
- The Impact of Genomic Changes on the Targeted Treatment of Lung Cancer
- The Metabolome and Microbiome of Lung Cancer

Session 3: Translational Studies

- Cell Immunity and HCC
- Precision Oncology in HCC
- Antibody-based Therapy in Liver Cancer
- The Development and Testing of Novel Therapeutic Approaches to Lung Cancer in Preclinical Models
- Combination of Immuno and Targeted Therapies of Lung Cancer

Confirmed Invited Speakers:

- **Dr. Ludmil B. Alexandrov,** University of California, San Diego, USA
- **Prof. Herman Autrup,** University of Aarhus, Denmark
- **Dr. Vajarabhongsa Bhudhisawasdi,** Khon Kaen University, Thailand
- **Dr. Tim F. Greten,** National Cancer Institute, USA
- **Prof. John Groopman,** Johns Hopkins Bloomberg School of Public Health, USA
- **Dr. Curtis C. Harris,** National Cancer Institute, USA
- **Dr. Mitchell Ho,** National Cancer Institute, USA
- **Prof. Bruce E. Johnson,** Dana-Farber Cancer Institute, USA
- **Prof. Carl H. June,** University of Pennsylvania, USA
- **Prof. Stephen Lam,** University of British Columbia, Canada
- **Dr. Scott W. Lowe,** Memorial Sloan Kettering Cancer Center, USA
- **Prof. Nickolas Papadopoulos,** Johns Hopkins Medicine, USA
- **Dr. Brid M. Ryan,** National Cancer Institute, USA
- **Prof. Bin Tean The,** Duke-NUS Medical School, Singapore
- **Prof. Xin Wei Wang,** National Cancer Institute, USA
- **Prof. Lars Zender,** University Hospital Tübingen, Germany
- **Prof. Andrew X. Zhu,** Massachusetts General Hospital, USA

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

Correspondence should be addressed to:

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

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