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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".

WHO South-East Asia Regional Meeting Air Quality and Health

June 18-20th, 2018

at the Chulabhorn Convention Center, Bangkok, Thailand

The Chulabhorn Research Institute (CRI), a WHO Collaborating Center for Capacity Building and Research in Environmental Health Science and Toxicology, co-organized the **WHO South-East Asia Regional Meeting on Air Quality and Health** with the World Health Organization South-east Asia Regional Office (SEARO) from June 18-20th, 2018 at the Chulabhorn Convention Center, Bangkok, Thailand.

The meeting was attended by 70 participants, including representatives from governmental agencies related to health, the environment and policy-making from 10 member countries in the South-east Asian region, representatives from international organizations and NGOs, and world-renowned experts in the area of air pollution and health.

The objectives of the meeting were to increase capacity in the region in terms of reduction of health impacts from exposure to air pollution, and the fostering of collaborations among institutions in the region towards a better understanding of issues related to air quality management and sources of supporting data and information available to assist in development of strategic plans and policies related to management of air pollution towards sustainable development.

During the meeting, a representative from the United Nations Environment Programme UNEP) provided information on work that was being conducted in the region and internationally. The participants then provided summaries on the current situations and what was being done in the respective countries, e.g. policies and national strategic

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WHO South-East Asia Regional Meeting on Air Quality and Health
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plans on air quality management, regulations and safety standards, as well as various projects that utilized health impact data to assess air quality.

In addition, break-out groups were formed to discuss and collect views on various aspects of air quality management, e.g. capacity building to increase national capabilities, risk communication, and development and implementation of systems to monitor the effectiveness of national policies on air quality management. The conclusions from this regional meeting will feed into the **"First Global Conference on Air Pollution and Health"**, which will be held at the WHO headquarters in Geneva, Switzerland, from October 30th to November 1st, 2018. For more information, please visit: http://www.who.int/airpollution/events/conference/en/

The Effect of Ambient Particle Matters on Hospital Admissions for Cardiac Arrhythmia in China

Over the past decades, air pollution has been a cause of considerable public concern worldwide, especially in developing countries, where ambient particle matters (PMs) have been perceived as the predominant pollutant.

Worldwide, ambient PMs were the fifth-ranking mortality risk factor in 2015, accounting for 7.6% of global deaths and 4.2% DALYs (disability-adjusted life years or number of years last due to ill health).

In China, ambient PMs were ranked the fourth among contributors to the disease burden, leading to more than 0.9 million premature deaths annually.

Cardiac arrhythmia is a group of complex conditions related to the risk of cardiovascular complications and sudden death.

Increasing evidence is making clear that air pollution is associated with the risk of hospital admission, emergency room visits and mortality from cardiovascular causes.

A number of epidemiological studies have suggested that exposure to ambient PMs might be responsible for cardiac arrhythmia. However, most of the studies assessing this link were carried out in North America, Western Europe and East Asia.

As a result, these findings may not be applicable to Mainland of China, because of the different ambient air pollutant mixtures, weather patterns, health status and population susceptibility.

A time-stratified case-crossover analysis was conducted in 26 large Chinese cities, 175,265 hospital admissions identified for cardiac arrhythmia between January 2014 and December 2015.

The aim of the present study was to evaluate the association between ambient PMs and hospital admissions for cardiac arrhythmia in China and to examine the potential effect modifiers.

Age, gender and prespecified comorbid health conditions including hypertension, diabetes, congestive heart failure and hyperlipidemia were stratified to evaluate susceptibility factors.

This is the first multisite study in China, or even in the Asia, to examine the short-term effects of air pollution on arrhythmia.

The results showed that short-term elevated concentration of $PM_{2.5}$ and PM_{10} were significantly associated with increased risk of cardiac arrhythmia hospital admissions.

The study did not find the evidence of effect modification by gender, but found a stronger and longer lasting effect among aged population (>65 years) and people with diabetes.

Other prespecificed comorbid health conditions including hyper-tension, congestive heart failure and hyperlipidemia did not modify the risk.

Future investigations with tightly controlled exposure level, as well as long-term observational studies, are needed to help elucidate the long-term effects.

Source: Environmental Health, Vol. 17, No. 60, July 2018.

WHO : One Third of Global Air Pollution Deaths in Asia Pacific

Air pollution levels remain dangerously high in many parts of Asia according to new data from the World Health Organization (WHO).

Around one third, or 2.2 million of the world's 7 million premature deaths each year from household (indoor) and ambient (outdoor) air pollution are in the WHO Western Pacific Region, home to one quarter of the world's population.

WHO's new estimates show that 9 out of 10 people in the world breathe air containing high levels of pollutants. Polluted air penetrates deep into their lungs and cardiovascular system.

In 2016, there were 2.2 million air pollution-related deaths in this Region, 29% due to heart disease, 27% to stroke, 22% to chronic obstructive pulmonary disease, 14% to lung cancer and 8% to pneumonia.

Ambient air pollution

Ambient air pollution, which effects urban and rural areas alike, is made up of fine particulate matter (PM_{10} and $PM_{2.5}$). $PM_{2.5}$ includes pollutants such as sulfate, nitrates and black carbon. These pose the greatest risks to human health.

Major sources of this fine particulate matter include inefficient energy use in households, and industry, in the agriculture and transport sectors, and in coal-fired power plants. In some areas, sand and desert dust, waste burning and deforestation also contributed to air pollution. Air quality can also be influenced by these natural elements and by other geographical, meteorological and seasonal factors.

Household air pollution

Most household air pollution is caused by the burning of kerosene and solid fuels such as wood in stoves, open fires and lamps.

More than 40% of the world's population still do not have access to clean cooking fuels and respiratory-friendly technologies in their homes. Women and children are, as a result, most at risk from household air pollution.

More countries taking action

More than 4,300 cities in 108 countries are now included in WHO's ambient air quality database, making it the world's most comprehensive database on ambient air pollution.

Since 2016, more than 1000 additional cities have been added to WHO's database which shows that more countries are measuring and taking action to reduce air pollution than ever before.

Key findings:

- WHO estimates that around 90% of people worldwide breathe polluted air. Over the past 6 years, ambient air pollution levels have remained stubbornly high, , but with declining concentrations in some parts of Europe and the Americas.

- The highest ambient air pollution levels are in the Eastern Mediterranean Region and in South-East Asia, where annual mean levels often exceed more than 5 times WHO limits. Low and middle-income cities in Africa and the Western Pacific rank second in serious air pollution.

- Africa and some areas of the Western Pacific have a serious lack of data about the air pollution they face. However, the database for Africa now contains PM measurements for more than twice as many cities as previous versions. Even so, data is available for only 8 of 47 countries in the region.

- Europe has the highest number of places reporting data.

- In general, ambient air pollution levels are lowest in high-income countries, particularly in Europe, the Americas and the Western Pacific. In the cities of the high-income countries of Europe, air pollution has been shown to lower average life expectancy by anywhere from 2 to 24 months, depending on pollution levels.

While the latest data show ambient air pollution still at dangerously high levels in the WHO Western Pacific Region, some progress is also apparent.

The estimated number of air pollution deaths in the Region has come down from 2.8 million in 2012 to 2.2 million in 2016.

WHO air quality recommendations call for countries to reduce annual mean values of air pollution to 20 $\mu g/m^3$ for PM_{10} and 10 $\mu g/m^3$ for $PM_{2.5.}$

Measures are being taken to reduce air pollution from particulate matter. Air pollution does not recognize borders. Improving air quality demands sustained, coordinated government action at all levels. Countries need to work together on solutions for sustainable transport, more efficient and renewable energy production and use, and waste management.

NOTE: **WHO's ambient air quality database:** The database builds mainly on well-established, public air quality monitoring systems as a source of reliable data in different parts of the world. The primary source of data includes official reporting from governments. Other sources include Clean Air Asia and the European Environment Agency for Europe's Air Quality e-Reporting database, ground measurements compiled for the Global Burden of Disease project, and peer-reviewed journal articles.

The database together with the summary of results, methodology used for compiling the data and WHO country groupings can be found at - https://www.who.int/phe/health_topics/outdoorair/databases/cities/en/

Source: WHO News Release, May 2018. (http://www.who.int/news-room/detail/02-05-2018-9-out-of-10-people-worldwide-breathe-polluted-air-but-more-countries-are-taking-action)

Prenatal Exposure to Bisphenol A and Hyperactivity in Children

Increased rates of certain neurodevelopmental disorders in children such as autism, learning disabilities, and attention-deficit hyperactivity disorder (ADHD) have raised concerns over the possible causes of these disorders, and whether they might be due to early environmental influences.

ADHD usually has an onset in early school-aged children and can possibly persist into adulthood, although some evidence suggests that adult ADHD is not associated with diagnosis in childhood.

ADHD is a heterogeneous disorder, consisting of many symptoms and co-morbidities that are present to varying degrees. The disorder can affect many behavioral aspects, such as attention (e.g., alertness and vigilance), executive function (e.g., working memory, response inhibition, cognitive flexibility, and planning), and reduced response habituation.

Studies attempting to identify a specific genetic component have not been able to account for much of the heritability of ADHD, indicating there may be gene-environment interactions underlying the disorder, including early exposure to environmental chemicals.

Based on several relevant studies, bisphenol A (BPA) was examined as a possible contributor to ADHD in humans. BPA is a widespread environmental chemical that has been shown to disrupt neurodevelopment in rodents and humans.

From a mechanistic standpoint, BPA has been shown to disrupt the catecholaminergic and serotonergic signaling systems, *in vitro* and *in vivo*; these signaling systems are implicated in the manifestation of ADHD.

The adverse health and environmental impacts of BPA, apparent even at very low doses, are already very significant. Due to BPA's endocrine disrupting properties for human health and the environment, the European Chemical Agency recently listed it as a substance of very high concern (SVHC).

Using the National Institute of Environmental Health Sciences, Office of Health Assessment and Translation (OHAT) framework, a systematic review and meta-analysis has been designed to determine the relationship between early life exposure to BPA and hyperactivity, a key diagnostic criterion of ADHD.

The OHAT framework standardizes the review process by providing transparent procedures for collecting evidence, evaluating the validity of study designs and methods, rating confidence in the body of evidence, and integrating human and animal evidence for a final health effect conclusion.

The systematic review of literature found that early BPA exposure is associated with a presumed hazard of hyperactivity in humans, based on "moderate" levels of evidence for the human and "high" levels of evidence for animals.

The study concludes that early life exposure to BPA is a presumed human hazard for hyperactivity, confirming the effect of the known endocrine disrupting chemical on the development of the brain.

This conclusion is based on the integration of information regarding animal and humans derived from reviews of the relevent literature. A rigorous analysis of the body of evidence included evaluation of study quality, meta-analysis, and the consideration of other factors such as publication bias and dose-response.

This review also identified data gaps and the need for additional risk assessments, including assessments of timing of exposure and a thorough dose-response analysis.

This is the first systematic review to date that assesses links between bisphenol A and hyperactivity using both animal and human data.

Systematic reviews and meta-analyses are valuable tools that should also be used in translating science into policy for regulatory purposes, partly because they allow for the assessment of endpoints that might not be evaluated in traditional health regulatory assessments (e.g., behavioral or non-cancer disease endpoints).

Current efforts to devise appropriate methods of assessing harmful chemicals should adopt transparent, standardized and comprehensive procedures of systematic review as valid means of hazard identification.

Dose-response and exposure analyses are also warranted in order to characterize the risks and to more effectively manage BPA exposure.

Given the widespread exposure of BPA and increasing diagnoses of ADHD, the researchers recommend immediate actions to complete such risk analyses. The next steps for the protection of human health should then be taken, especially for the most vulnerable populations, including pregnant women, infants and children.

Source: Environment International, Vol. 114, Pages 343-356, May 2018.

Early-life Triclosan Exposure and Child Neurodevelopment

Triclosan is an antimicrobial compound used in personal care and household products including some deodorants, soaps, toothpastes, mouthwashes, cosmetics, shower gels, cleaning products, and kitchen utensils.

Exposure to triclosan occurs primarily through oral intake (e.g., toothpaste and mouthwash) and dermal absorption (e.g., soaps and cosmetics). Triclosan exposure is ubiquitous among persons in the United States, including pregnant women and children.

After ingestion or absorption, triclosan is primarily excreted in the urine and has an estimated biological half-life of about 21 h.

Results from epidemiological and animal studies suggest that triclosan could adversely affect neurodevelopment by disrupting thyroid hormone homeostasis; however, few studies have

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Long-term Air Pollution Exposure Induced Metabolic Adaptations in Traffic Policemen

Fine particle matter with an aerodynamic diameter of ≤2.5 μm ($PM_{2.5}$) has been well documented as a major air pollutant harmful to human health. It is the leading risk factor for various diseases including cancer, coronary and peripheral vascular disease, diabetes, stroke and respiratory disease.

However, most epidemiological and clinical studies have focused only on the association of $PM_{2.5}$ concentrations with disease mortality and morbidity. Exposure duration, physiological changes and underlying mechanisms have rarely been addressed. Moreover, current evidence on physiological response and adverse physiological effects induced by $PM_{2.5}$ exposure remains largely unexplored.

$PM_{2.5}$ refers to a complex mixture of heavy metals, microorganisms and numerous known carcinogens. Because the source, composition and concentration of $PM_{2.5}$ components vary vastly, there are substantial context-dependent variations in its adverse physiological changes.

It has been widely recognized that $PM_{2.5}$ concentrations in China persisted at levels much higher than in most of Europe and North America. Also, the sources and composition of $PM_{2.5}$ in China are quite different.

Thus, conclusions drawn by most previous studies about health effects and underlying physiological mechanisms cannot be simply applied to the Chinese population which is exposed to higher $PM_{2.5}$ pollution with a distinct composition.

Traffic policemen in China comprised a special population. For professional reasons, they are subjected to the greatest exposure to urban air pollution. Due to the high population density and rapid increase of vehicles in China, policemen are more vulnerable to air pollution due to continuous exposure to vehicular emissions as they stand for long hours at the intersections of main roads.

Clinical conventional laboratory tests including hematology, fasting blood glucose (FBG), blood lipids, liver, kidney, immunity and tumor-related biomarkers are less expensive and helpful in fully understanding the adverse physiological changes.

The present study uses conventional laboratory tests to characterize the health outcomes and specific adverse physiological changes caused by exposure to air pollution, which would provide deep insights into the biological basis and pathological mechanisms of air pollution-associated diseases.

A total of 183 traffic policemen with 88 office policemen as the control group, were enrolled in the study. The concentration of $PM_{2.5}$ in both working places, i.e. in traffic and in office, were obtained.

Both groups provided detailed personal questionnaires. All underwent a battery of conventional laboratory tests, including hematology, fasting blood glucose, blood lipids, liver, kidney, immunity and tumor-related markers to assess the adverse physiological changes induced by long-term exposure to $PM_{2.5}$.

The results demonstrated that the values of FBG, high-density lipoprotein cholesterol (HDL-c) and carcinoembryonic antigen (CEA) shared a dose-response relationship with the duration of exposure. Long-term air pollution exposure induced increases of FBG and CEA, and decrease of HDL-c level.

Multivariate analysis confirmed that one hour on duty outdoors per day for one year was associated with an increase in FBG, CEA, and a decrease in HDL-c.

The observed strong association between air pollution and metabolic syndrome suggested that the cardio-metabolic effects of air pollution may be mostly driven by the impairment of glucose homeostasis.

Decreased HDL-c level is one of the major risks for cardiovascular disease. The association between CEA level and air pollution exposure duration also suggested that $PM_{2.5}$ exposure may be involved in the progression of cardiovascular disease.

The study also found supporting evidence that cigarette smoking coupled with air pollution exposure has direct effects on FBG, HDL-c and CEA levels.

Although the levels of RBC count, hemoglobin, creatinine and IgG were higher in traffic policemen group compared to the control group, multiple linear regression analyses showed no significant association between air pollution exposure duration and the Cr, RBC, Hb and IgG levels.

In conclusion, the study of traffic policemen from a Chinese city with severe air pollution clearly demonstrated that $PM_{2.5}$ exposure caused remarkable alterations in physiological biomarkers as revealed by common laboratory tests such as FBG, HDL-c, and CEA.

These findings provide evidence for the association between long-term $PM_{2.5}$ exposure and the type 2 diabetes and cardiovascular disease.

Source: Environmental Toxicology and Pharmacology, Vol. 58, Pages 156-162, March 2018.

STATE OF GLOBAL AIR 2018

One in Three Globally Face a "Double Burden": Exposed to Household Burning and Outdoor Air Pollution

Seven billion people, more than 95% of the world's population, live in areas of unhealthy air, according to a new global study.

The State of Global Air 2018, the annual report and interactive website published at www.stateofglobalair.org by the Health Effects Institute (HEI), identifies air pollution as the leading environmental cause of death worldwide.

All told, long-term exposure to outdoor and indoor air pollution contributed to 6.1 million premature deaths from stroke, heart attack, lung cancer, and chronic lung disease. That makes air pollution the 4th highest cause of death among all health risks, exceeded only by high blood pressure, poor diet, and heavy smoking.

This year's report and website include for the first time worldwide estimates of exposures to and health risks due to the burning of solid fuels in the home. In 2016 a total of 2.5 billion people, one in three global citizens, were exposed to household air pollution from the domestic use of solid fuels (for example, coal, wood, charcoal, dung, or other biomass) for cooking and heating. Most of this population live in low- and middle-income countries in Asia and Africa. Furthermore, they face a double burden: exposure to both indoor and outdoor air pollution.

Household air pollution can also have major impact on the air outside. In india, emissions of indoor pollution out into the larger environment have been identified as the largest cause of health impacts from among all other sources. The home fires actually contribute to 1 in 4 air pollution-related deaths in India, and are implicated in nearly 1 in 5 such deaths in China.

The analysis found that China and India together are responsible globally for over half of such attributable deaths. Exposures continue to increase in India even as the population grows and ages. This means that India now rivals China for the highest number of air pollution health burdens in the world. Both countries faced some 1.1 million early deaths from outdoor air pollution in 2016. China has made initial progress and is beginning to achieve air pollution declines By contrast, however, Pakistan, Bangladesh, and India have experienced the steepest increases in air pollution levels since 2010.

The State of Global Air 2018 annual report and accompanying interactive website were designed and implemented by the Health Effects Institute in cooperation with the Institute of Health Metrics and Evaluation (IHME) at the University of Washington and the University of British Columbia.

IHME is an independent population health research center that coordinates the annual Global Burden of Disease (GBD) study, a systematic scientific effort to quantify the magnitude of health loss from all major diseases, injuries, and risk factors in populations across the world.

HEI provides leadership for the air pollution portion of the GBD; HEI's www.stateofglobalair.org is the only report and website where all of the estimates of exposure to air pollution and their burden of disease included in the GBD air pollution analyses are made available for full public access.

Source: Health Effects Institute, State of the Global Air 2018, April 2018. (https://www.stateofglobalair.org/whats-new/press-release)

Early-life Triclosan Exposure and Child Neurodevelopment

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examined the association between early-life triclosan exposure and neuro-development in either animals or humans.

In rodents, triclosan exposure decreases circulating thyroxine levels in both the pregnant damn and her fetus during gestation and may interfere with other hormonal pathways. During gestation, thyroid hormones transferred from the mother to the embryo and fetus are critical for proper neurodevelopment. Even small deficiencies in thyroid hormone concentrations during pregnancy have been associated with lower IQ scores in children.

In humans, a previously published study and a prospective birth cohort study in Shanghai, China, both reported that increasing urinary triclosan concentrations during gestation or at delivery were associated with decreased maternal or neonatal thyroid hormone concentrations.

In the present study, the researchers hypothesized that early-life triclosan exposure may reduce circulating thyroid hormone concentrations, which may in turn adversely affect fetal, infant, or childhood cognitive outcomes.

Using a longitudinal pregnancy and birth cohort, the researchers investigated associations between triclosan exposures during different time windows, and cognitive test scores at 8 years of age in 198 children from the Health Outcomes and Measures of the Environment (HOME) Study. The HOME study is a prospective pregnancy and birth control in Cincinnati, Ohio, designed

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IARC: Carcinogenicity of Quinoline, Styrene and Styrene-7,8-oxide

In March 2018, a Working Group of 23 scientists from 12 countries met at the International Agency for Research on Cancer (IARC) in Lyon, France, to finalise their evaluation of the carcinogenicity of **quinoline, styrene, and styrene-7,8-oxide.**

Quinoline is an azaarene present in tobacco smoke and air pollution. Quinoline occurs in petroleum and shale oil processing, and is found in groundwater and soil at coal tar and creosote-contaminated sites. A high production volume chemical, quinoline is used to produce a variety of drugs and dyes.

Quinoline has now been classified as "possibly carcinogenic to humans (Group 2B)", based on sufficient evidence of carcinogenicity in experimental animals. In mice and rats, quinoline induced rare tumours of various embryological origins. Malignant tumours were induced with a high incidence at the lowest dose tested, occurred with short latency, and caused early deaths.

Experimental systems have produced strong evidence that quinoline is genotoxic. Mutations and chromosomal damage in rodents and also in vitro (upon metabolic activation).

No further data were available have been induced on the exposure, absorption, distribution or carcinogenicity of quinoline in humans.

Styrene has been classified as, "probably carcinogenic to humans (Group 2A)" based on limited evidence in humans and sufficient evidence in experimental animals for carcinogenicity. Present in tobacco smoke and air pollution, styrene is a high production volume chemical, styrene is primarily used to produce polystyrene polymers.

Styrene and its principal metabolite in humans styrene-7,8-oxide are found in workplace air, particularly in the reinforced plastics and rubber industries. Styrene-7,8-oxide is primarily used to produce epoxy resins.

The most informative epidemiological studies of cancer have emerged from large occupational cohorts in the reinforced plastics industry, where styrene exposure levels are highest. The largest producers are in Europe, the UK, Denmark, and in the USA, especially in Washington state.

The 2018 Working Group assessed the overall pattern of findings for lymphohaematopoietic malignancies, noting increased incidence or mortality among subtypes of leukaemia and lymphomas in several studies, with greater consistency for leukaemia, and in particular myeloid leukaemia. In this case, however, confounding, bias or chance cannot be ruled out.

Styrene is rapidly absorbed and widely distributed to adipose tissues. It is extensively metabolised in humans and in experimental systems. Approximately 60% of excretion products formed from inhaled styrene come from its metabolism to styrene-7,8-oxide, which is an electrophile that can react directly with DNA.

Strong evidence indicates that styrene and styrene-7,8-oxide are genotoxic.

In human cells in vitro, styrene as well as styrene-7,8-oxide induce DNA damage, gene mutations, chromosomal aberrations, micronucleus formation, and sister-chromatid exchanges. Similar findings has been observed in various experimental systems.

In rodents exposed to styrene or styrene-7,8-oxide, results were equivocal for cytogenetic effects, but positive for DNA damage in multiple tissues.

Although proof of its carcinogenicity in humans is still inadequate, *styrene-7,8-oxide has been classified as a probable cause of cancer in humans (Group 2A)* because there is sufficient evidence in experimental animals to justify the classification.

In B6C3F1 mice, gavage exposure to styrene-7,8-oxide increased the incidences of forestomach squamous cell papilloma and carcinoma in males and females, and hepatocellular adenoma or carcinoma (combined) in males.

This assessment will be published in Volume 121 of the IARC Monographs.

Source: The Lancet Oncology, Vol. 19, Issue 6, June 2018.

Early-life Triclosan Exposure and Child Neurodevelopment

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to examine health effects associated with early-life exposurwe to environmental chemicals.

Full-Scale IQ was not significantly associated with urinary triclosan concentrations during gestation or childhood but was significantly associated with a 10-fold increase in maternal urinary triclosan concentration at delivery.

Perceptual Reasoning Index (PRI) scores were significantly decreased in association with urinary triclosan concentrations at delivery and at 2 years of age.

Associations between repeated triclosan concentrations and cognitive test scores significantly varied among exposure at different time periods for Full-Scale IQ, PRI, Verbal Comprehension Index, and Working Memory.

Maternal urinary triclosan concentrations at delivery, but not during mid to late pregnancy and childhood, were associated with significantly lower children's cognitive test scores at 8 y of age in this cohort of U.S. children.

Future studies to replicate these findings would benefit from using serial urine samples with larger and more diverse cohorts to reduce triclosan exposure misclassification and to enhance the generalizability of these findings, respectively.

Finally, future investigations should examine potential mechanisms underlying the association between early-life triclosan exposure and child neurodevelopment.

Source: Environmental Health Perspectives, Vol. 126, Issue 5, May 2018.

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 work days	January 31, 2019
3	Occupational and Environmental Health	May 2019	1 week	February 28, 2019

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

Course Description:

Environmental and Health Risk Assessment and Management of Toxic Chemicals (December 3-15, 2018)

The course is an integration of science and policy, covering the fundamental basis of environmental and health risk assessment and management, from identification of hazard, assessment methods, the mode of action and human relevance framework, the inherent uncertainties in each step, the relationship between risk assessment and risk management, and the need for open, transparent and participatory acceptance procedures and credible communication methods. Emphasis is placed on human health risk assessment, although the principles of ecological risk assessment will also be covered. 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 electronic distance learning tools and IPCS 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.

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

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