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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 Sri Lanka's Ministry of Health, Nutrition & Indigenous Medicine to Organize a Training Course on Risk Assessment and Risk Management of Chemicals

in Colombo, Sri Lanka from November 28 - December 2, 2017

Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol, President of the Chulabhorn Research Institute (CRI) and Course Director, led a team of international faculty to conduct in-country training on **"Risk Assessment and Risk Management of Chemicals"**, in collaboration with Sri Lanka's Ministry of Health, Nutrition & Indigenous Medicine and the World Health Organization (WHO), through the WHO South-East Asia Regional Office and the WHO Country Office for Sri Lanka, from November 28 - December 2, 2017 in Colombo, Sri Lanka. The training was supported by the WHO South-East Asia Regional Office and the WHO Country Office for Sri Lanka; the Thailand International Cooperation Agency (TICA), Ministry of Foreign Affairs, Thailand; and CRI.

The CRI team has a vast amount of experience with regard to providing training for participants within the 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 roles as a WHO Collaborating Centre for Capacity Building and Research in Environmental Health Science and Toxicology, and the WHO-designated regional centre for training in chemical safety.

The course was designed to provide basic scientific knowledge on 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

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IPCS, the University of Ottawa (Canada) and Utrecht University (the Netherlands), as tools that participants can use for further guidance on the risk assessment and risk management of chemicals.

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, Risk Characterization – human, Risk Characterization – ecological; Risk Management; and Risk Communication.

The 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.

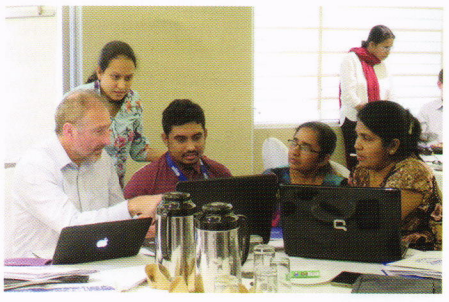
Welcome addresses were delivered by Mr. Janaka Sugathadasa, Secretary of the Ministry of Health, Nutrition & Indigenous Medicine, Dr. Razia Pendse, the WHO Representative to Sri Lanka, and Dr. Rajitha Senaratna, the Honorable Minister of Health, Nutrition & Indigenous Medicine.

The Opening Address and opening keynote lecture entitled, **"Essential Principles of Toxicology for Risk Assessment: Disposition of Chemicals in the Body"** was delivered by Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol, President of CRI and Course Director.

The teaching faculty for this training course included Professor Herman Autrup from the University of Aarhus (Denmark), Professor Leonard Ritter from the University of Guelph (Canada), Professor David Russell from the WHO Collaborating Centre for Chemical Incidents and Public Health England (UK), Professor Martin van den Berg from Utrecht University (the Netherlands), 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.

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

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



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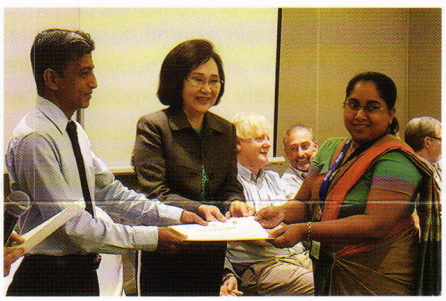
This in-country training course was attended by 35 participants from various offices of the Regional Director of Health Services (RDHS), the Ministry of Health, Nutrition & Indigenous Medicine.

In addition to attending lectures, participants worked on 8 different case studies, including fluoride in drinking water, organotin in seafood, risk assessment and management of a maritime incident, bisphenol A, endosulfan, hair dyes and the risk of breast cancer, risk assessment of the chlorination of water, and assessment of the use of fluoroquinolone in aquaculture.

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 offices 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.

Children's Environmental Health Based on Birth Cohort Studies of Asia

In recent years, public concern about health and the environment in societies across Asia has been stirring in response to news about air pollution, arsenic contamination of groundwater exposure to phthalates through food ingestion, air inhalation, and direct dermal contact, and the long term impact of the Fukushima Daiichi Nuclear Disaster.

In order to assess the consequences of prenatal exposure to environmental pollutants and to identify preventable risk factors, several large-scale birth cohort studies are being conducted worldwide.

Birth cohort studies begin at or before the birth of their subjects, and continue the same individuals at later ages, on more than one occasion. Cohorts are a type of observational study in which there is no randomization of exposure groups and no attempt to manipulate exposures.

Hypotheses of the Developmental Origins of Health and Disease (DOHaD) propose that prenatal threats including environmental exposures are the origin of all disease and may impact human development.

The Birth Cohort Consortium of Asia (BiCCA) was established in 2011 to address these issues. BiCCA is the working group for the coordination of birth cohorts in Asia. Through an enlarged set of data and extended diversity of participants and of environmental exposure levels, this international cooperation will provides deeper insights into child environmental health, especially the regional concerns in Asia. For more information, please visit – http://www.bicca.org/

The BiCCA includes 23 birth cohorts, totaling approximately 70,000 study subjects. The consortium has conducted studies in 10 Asian countries.

Research published by groups not part of BiCCA was included in the studies to present a more comprehensive investigation of the association between exposure to environmental pollutants and related effects on the health of children.

Environmental pollutants include mercury, environmental tobacco smoke (ETS), polychlorinated biphenyls (PCB), perfluoroalkyl substances (PFAS) and phthalates.

This study sought to classify the effects of such compounds on fetal growth and pregnancy outcomes, neuro-development and behavioral problems, allergic disease and immune function, and the endocrine system and puberty.

Accumulating evidence has shown that exposure to environmental pollutants including ETS, mercury, PCB, phthalates, and PFAS may impact pregnant women and children in Asia.

A spread of negative effects has been identified. Mercury and PCB have been shown to affect children's neurodevelopment; ETS has been associated with infant birth weight, children's neurodevelopment and allergy disease; phthalate impacts on endocrine function; PFAS alters children's neurodevelopment, endocrine system, and allergic response.

According to the evidence summarized in the present study, environmental pollutants are related to food consumption (e.g., seafood and mercury measurements). In order to completely evaluate pediatric health,

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Arsenic in Seafood is Associated with Increased Thyroid-stimulating Hormone in Healthy Volunteers

Exposure to exogenous elements like arsenic (As) may influence thyroid enzymes, thyroid-stimulating hormone (TSH), and the two principal thyroid hormones, free thyroxine (FT4) and free triiodothyronine (FT3). Little is known about how this is related to organic arsenicals, the main form of As in seafood.

Fish and seafood naturally contain relatively high amounts of As. Together with rice and rice products, these foods represent the main contributors to total As intake for humans, particularly where there is relatively low exposure from drinking-water or occupation.

As exposure from fish intake may have an impact on thyroid hormones. Possible effects of As on thyroid metabolism may contribute to, or modify, the beneficial health effects of fish and seafood consumption. Since various seafood species contain different spectrums of arsenicals, it is important to assess the possible impact of seafood As on thyroid hormone metabolism.

Although little studied, exposure to As may also interact with the metabolism of selenium (Se) and compromise the supply of Se to Se-dependent iodothyronine deiodinases. In this way As may also indirectly affect thyroid metabolism via T4 conversion to T3. As has been shown to enhance the excretion of Se.

The content of iAs in most seafood is low, in finfish usually <0.2 mg/kg dry weight. Exceptions are some shellfish and edible algae (e.g. hijiki), that may constitute up to 4.5 mg/kg dry weight and >60 mg/kg dry weight, respectively.

Hence, previous observations on changes in thyroid hormones following exposure to relative high levels of iAs are not comparable with As exposure from fish and seafood, since the organic species dominate in seafood.

The purpose of the present study was to investigate whether three different controlled seafood diets (cod, salmon or blue mussels), naturally rich in various forms of As, were associated with changes in TSH, FT4 and FT3 and the FT4:FT3 ratio in plasma in healthy volunteers.

Plasma concentrations of TSH increased significantly in all seafood groups. Plasma Se and iodine were negative and positive factors, respectively.

The increase in TSH was proportional to the increase in plasma As, but with different constants, depending on the type of seafood ingested. This may be interpreted as reflecting differences between the seafoods in the content of certain arsenicals.

There were also indications of changes in FT4, FT3 and the FT4:FT3 ratio consistent with a net inhibiting effect of As on FT4 to FT3 conversion.

Dietary As from seafood appeared to influence thyroid hormones, particularly by stimulating an increase in plasma TSH levels. The association with change in TSH levels varied with the type of seafood consumed indicating that the chemical forms of As are important.

Further studies are needed to elucidate the mechanisms by which different organic As from seafood may impact thyroid function and thyroid hormone metabolism.

Source: Journal of Trace Elements in Medicine and Biology, Vol. 44, Pages 1-7, December 2017.

Air Pollution and Arterial Hypertension

According to the World Health Organization, urban air pollution is an important cause of global mortality and is responsible for approximately 800,000 premature deaths each year.

Outdoor contamination has been recognized as the 13th leading cause of mortality globally, especially in low- and middle-income countries where air pollution concentrations continuously rise.

The adverse effects of air pollution on cardiovascular health have become the focus of a series of major epidemiologic and observational studies.

While economic expansion, industrial growth, and climate change contribute to increased levels of outdoor contamination in developing nations, the composition and sources of air pollutants vary across different regions.

Short- and long-term exposure to air pollution is associated with high blood pressure through inflammation, oxidative stress, and arterial remodeling.

Recent studies suggest that even modest rises in airborne pollutants can trigger an increase in arterial BP within hours.

Inhalation of air pollutants affects heart rate, heart rate variability, vascular tone and blood coagulability, and promotes atherosclerosis.

Although short-term exposures to air pollution have been associated with increased cardiovascular morbidity and deaths from myocardial ischemia, arrhythmia, and heart failure, the exact relationship between air pollution exposure and arterial hypertension remains unclear.

In this context, both the American Heart Association (2010) and the European Society of Cardiology (2015) have issued official statements discussing the impact of air pollution exposure to cardiovascular health.

The aim of this review was to analyze the biological mechanisms responsible for air pollution-induced cardiovascular toxicity and to describe the consequences of short- and long-term exposures to the cardiovascular system, focusing on hypertension.

The results link air pollution to cardiovascular toxicity. Polluted air is a triggering factor which can induce hypertension through imbalance in the autonomic nervous system and subsequent vasoconstriction.

Individual susceptibility may play an important role in determining the exact hemodynamic responses. The major strategy in decreasing the harmful effects of air pollution is the reduction of air pollutants themselves.

Source: Journal of the American Society of Hypertension, Vol. 11, Issue 11, Pages 709-715, November 2017.

The Role of Cadmium in Obesity and Diabetes

Multiple studies have shown an association between environmental exposure to hazardous chemicals, including toxic metals, and obesity, diabetes, and metabolic syndrome.

Obesity and diabetes mellitus type 2 (DM2) have reached epidemic proportions since the turn of the 20th and 21st centuries. In 2013, every third person worldwide had excessive bodyweight (overweight or obesity). In that same year, there were nearly 382 million diabetics, and that number is expected to increase more than 1.5 fold, to 592 million by 2035.

Obesity is associated with increased adipose tissue mass and adipocyte dysfunction, increased production of proinflammatory adipokines, oxidative stress, endoplasmic reticulum stress, and insulin resistance.

This very tight interaction between obesity and diabetes has led to the introduction of a new term: "diabesity". The complex of pathologies including obesity, hyperglycemia, arterial hypertension and dyslipoproteinemia are clustered and are termed "metabolic syndrome".

The impact of environmental pollution on the incidence of diabetes and metabolic syndrome has already been well demonstrated. Moreover, it has been proposed that a differential response of the human organism to environmental pollution, even in terms of similar exposure patterns, indicates the presence of gene-environment interaction that may promote the development of metabolic syndrome.

Toxic metals and metalloids also seem to be involved in metabolic syndrome pathophysiology. Association between this syndrome and exposure to mercury, lead, or arsenic has already been demonstrated.

Cadmium (Cd) is a heavy metal that, like endocrine disrupting chemicals, has a particular impact on the functioning of reproductive organs, including testes, placenta, and ovaries.

The mechanisms of toxicity of Cd include induction of oxidative and endoplasmic reticulum stress, inflammatory response, genotoxicity, and interference with essential metals (especially zinc).

Despite the well-documented role of endocrine disrupting chemicals, oxidative and endoplasmic reticulum stress, and inflammation in the pathogenesis of obesity, diabetes and metabolic syndrome, the impact of Cd exposure and the underlying mechanisms are still unclear.

Therefore, the aim of the present work was to review the impact of Cd exposure and status on the risk and potential etiologic mechanisms of obesity and diabetes.

Although laboratory studies have confirmed the complex influence of Cd exposure on adipose tissue physiology and obesity pathogenesis, data from human studies on the interaction between Cd exposure and obesity are contradictory.

In contrast to obesity, the association between Cd exposure and DM2 has been more extensively studied. Additionally, an adverse effect of Cd exposure on carbohydrate metabolism and its regulation in experimental models were revealed more than 40 years.

Several epidemiological studies have suggested that Cd exposure affects the incidence of diabetes mellitus as well as insulin resistance. This research carried out a meta-analysis of all studies assessing risk of prevalence and incidence of diabetes.

Comparing the highest versus the lowest cadmium exposure categories, the researchers found that studies using urine to assess exposure revealed greater incidence of diabetes.

Results of epidemiologic studies linking cadmium exposure and overweight or obesity are far less consistent and even conflicting, depending on differences in exposure levels and the specific marker of exposure (blood, urine, hair, nails).

Laboratory studies have demonstrated that Cd adversely affects adipose tissue physiopathology through several mechanisms, thus contributing to increased insulin resistance and enhancing diabetes.

The existing clinical and experimental data also demonstrate that the amount of Cd exposure associated with diabetes and insulin resistance may be associted as well, with a pro-inflammatory effect of the Cd ion.

The direct effect of Cd on pancreatic β-cells is associted with cellular degeneration and decreased viability, as well as impaired insulin secretion in response to metabolic stimuli.

Although, intimate biological mechanisms linking Cd exposure with obesity and diabetes are still to be adequately investigated, and despite some inconsistencies, the existing human and experimental data demonstrate that Cd plays a significant role in the etiology of diabetes and insulin resistance, even at moderate to low levels of exposure.

Cd has been shown to adversely affect adipose tissue physiology, resulting in impaired adipogenesis, altered lipid and carbohydrate metabolism in adipocytes, as well as in adipose tissue endocrine dysfunction.

The results of this meta-analysis show an elevated risk of diabetes in subjects in the highest category of cadmium exposure.

Despite these findings, however, the evidence supporting the role of Cd in inducing obesity remains unclear and inconsistent.

Overall, the existing data demonstrate that Cd-induced changes of adipose tissue physiology, even without the presence of clinical obesity (increased adipose tissue mass), may at least partially predispose to the insulin resistance and subsequent DM2 that seem to be characteristic for chronic Cd exposure.

Source: Science of the Total Environment, Vols. 601-602, Pages 741-755, December 2017.

Parental Occupational Exposure to Benzene and the Risk of Childhood Cancer

Although benzene has been widely used as a solvent in paints and adhesives, we now know that it causes acute myeloid leukemia (AML) in occupationally exposed adults. Other blood cancers such as acute lymphoid leukemia (ALL), chronic lymphoid leukemia (CLL), multiple myeloma (MM) and non-Hodgkin lymphoma (NHL) also have suspected links with benzene exposure.

The children of parents regularly exposed to mutagenic substances, such as benzene may then have an increased risk of developing childhood cancer.

Proposed causal pathways include genetic alterations in parental germ cells, particularly of the father's sperm, trans placental exposure of the fetus during pregnancy, or postnatal exposure of the child to substances brought home from the workplace.

However, the major source of benzene exposure in children is ambient air pollution because benzene concentrations tend to be higher indoors where children spend most of their time.

Women exposed to benzene where they work are at risk of transmitting the pollutant to their children prenatally through transplacental migration or in infancy through breastfeeding.

Cord blood has been found to contain benzene in proportions similar to maternal blood, and benzene has been found in breastmilk.

While few studies have focused specifically on benzene, previous research on occupational exposure in parents and cancer risk in their children tends to link solvents and paints with childhood leukemia.

This nation-wide, census-based, cohort study in Switzerland investigated the association between parents' occupational exposure to benzene and cancer in their offspring .

By retrieving parental occupations reported at census and assessing exposure to benzene using a job exposure matrix researchers were able to identify cancer cases through record linkage with the Swiss Childhood Cancer Registry.

Multiple diagnostic categories including all cancers, leukemia, ALL, AML, lymphoma, NHL, central nervous system (CNS) tumours, and glioma were investigated.

The results from the period of the census showed an increased risk of leukemia among children whose mothers were occupationally exposed to benzene.

Adjusting for a range of socio-economic factors and residential exposures, the association between exposure to benzene and childhood leukemia was strongest among children born in Switzerland.

There was no evidence of an increased risk for other childhood cancers associated with maternal benzene exposure, and no evidence of increased risks among children whose fathers were exposed to benzene at work.

This nationwide cohort study provides new evidence suggesting that maternal occupational exposure to benzene is associated with an increased risk of childhood leukemia.

The distinguishing features of the study include the nationwide cohort design, the sole reliance on routine data, and the specific focus on exposure to benzene.

This makes it unlikely that the same observation made in previous case-control studies was due to selection or recall bias, which are common in these studies, or to chance findings as a consequence of testing numerous occupational exposures.

A plausible explanation for the findings is that maternal exposure to benzene during pregnancy can initiate leukemia in the developing child.

Source: Environment International, Vol. 108, Pages 84-91, November 2017.

Children's Environmental Health Based on Birth Cohort Studies of Asia

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further birth cohort studies must be conducted in Asia.

Whatever the exposure scenario or outcome, measurements are pivotal elements in the evaluation of children's environmental health. Most studies focus on a single association between exposure and health outcomes.

Exposure sources and routes are all interconnected. Exposures involve multiple pollutants. Multivariate statistical approaches are needed for further studies.

It is necessary to identify causes, effects, pathways and mechanisms, and to realize the complexity of investigating environmental hazards which overlab and mingle together in the real world.

Then researchers can establish more comprehensive exposure models of risk and assignment for children in Asian countries to help governments manage these health challenges more effectively.

In addition to environmental exposure, the genetic susceptibilities of mothers, infants and children from womb to tomb will be the subject of ongoing studies in genomics, transcriptomics, proteomics and metabolomics. As key factors are elucidated, the new data can be compared with western countries to identify significant similarities and differences.

Validation, harmonization, and international collaboration are needed in Asia. Using state of the art technologies, researchers can help to discover the underlying mechanisms.

Source: Science of The Total Environment, Vol. 609, Pages 396-409, December 2017.

IARC: Carcinogenicity of Benzene

In October 2017, a Working Group of 27 scientists from 13 countries met at the International Agency for Research on Cancer (IARC) in Lyon, France, to finalise their evaluation of the carcinogenicity of benzene.

The current Working Group **confirmed the carcinogenicity of benzene on the basis of sufficient evidence in humans, sufficient evidence in experimental animals, and strong mechanistic evidence.**

Benzene has been classified as carcinogenic to humans (IARC group 1) since 1979, on the basis of sufficient evidence that it causes leukaemia. This evaluation was reaffirmed specifically for acute myeloid leukaemia (AML) and acute non-lymphocytic leukaemia in 2009; positive associations with acute lymphocytic leukaemia, chronic lymphocytic leukaemia, multiple myeloma, and non-Hodgkin lymphoma were also recognised at that time.

The present evaluation was undertaken to review new epidemiological and mechanistic evidence as well as to explore the potential to characterise quantitative relationships for cancer risk and for biological endpoints related to cancer mechanisms.

Benzene, an aromatic hydrocarbon, is a ubiquitous air pollutant, arising mostly from anthropogenic sources, notably combustion. It is a component of gasoline, vehicle exhaust, industrial emissions, and tobacco smoke, and was used historically as a solvent in industry and consumer products.

The uses of benzene as a solvent are now restricted in many countries, but it is still produced in high volumes for use primarily as a chemical intermediate.

Occupational exposure to benzene can occur in diverse industries, including petroleum, chemical production, and manufacturing, and in some countries still occurs in industries where high levels were observed historically, such as shoemaking, painting, printing, and rubber manufacturing.

The population at large can be exposed to benzene in polluted air and water and through the use of benzene-containing products. Benzene concentrations in workplace and outdoor air have declined over time; the Working Group noted concentrations less than 3.00 and 0.005 mg/m^3 in workplace and outdoor air, respectively, in high-income countries, but higher levels have been reported in some low-income and middle-income countries.

The Working Group focussed its review of epidemiological studies on those in which occupational or environmental exposure to benzene was specifically identified. Important new evidence came from several large occupational cohort studies.

New data were also available from several recent studies that showed positive associations between AML in children and environmental exposure to benzene.

In male and female mice, several whole-body inhalation studies reported the induction of tumours of the haematopoietic and lymphoid tissues, Zymbal gland carcinoma, squamous cell carcinoma of the preputial gland, forestomach squamous cell carcinoma, and lung adenoma.

Benzene is easily absorbed, widely distributed, and extensively metabolised, yielding a complexity of reactive electrophiles via multiple metabolic pathways in various tissues, including bone marrow. It exhibits many of the key characteristics of carcinogens.

In particular, strong evidence, including in exposed humans, shows that benzene is metabolically activated, induces oxidative stress, is genotoxic, immunosuppressive, and causes haematotoxicity.

In addition, strong evidence from experimental studies shows that benzene causes genomic instability, inhibiting topoisomerase II; modulates receptor-mediated effects relevant to aryl hydrocarbon receptor, and induces apoptosis.

In benzene-exposed humans, epoxide-protein and benzoquinone-protein adducts are formed in blood. Additionally, benzene induces oxidative stress in exposed humans, human cells, and mouse bone marrow.

In studies of occupationally exposed humans, benzene induces oxidative DNA damage, DNA strand breaks, gene mutations, chromosomal aberrations, and micronuclei. Specific cytogenetic changes induced in exposed humans include aneuploidy, translocations, and various other structural chromosome changes.

In the bone marrow of experimental animals exposed *in vivo*, benzene induces DNA adducts, chromosomal aberrations, and micronuclei. Similarly, in human cells *in vitro*, benzene or its metabolites induce DNA adducts, DNA damage, and chromosomal aberrations.

Many studies in exposed humans have demonstrated haematotoxicity, ranging from decreased white blood cell counts at lower exposures to aplastic anaemia and pancytopenia at higher exposures. Benzene-induced haematotoxicity is associated with future risk of developing haematological malignancy or related disorders.

Although no human studies of benzene exposure directly examined changes in immune function, multiple experimental animal studies demonstrate haematotoxicity and consistent immunosuppressive effects on humoural and cell-mediated functional assays.

The Working Group investigated the shape and slope of the exposure-response function for AML in metaregression analyses of six published occupational cohort studies with suitable data.

The relationship of benzene exposure with the log relative-risk was well described by a linear model.

In the majority of human studies that reported exposure-response information for benzene and endpoints relevant to the key characteristics of carcinogens (ie, micronuclei, chromosomal aberrations, and leukocyte counts), an exposure-response gradient was reported.

This assessment will be published in Volume 120 of the IARC Monographs.

Source: The Lancet Oncology, Vol. 18, Issue 12, Pages 1574-1575, December 2017.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute, Year 2018

	Training Course	Date	Duration	Closing Date
1	Detection of Environmental Pollutants and Monitoring of Health Effects	February 5 - 16, 2018	9 working days	December 15, 2017
2	Environmental Toxicology	April 19 - 27, 2018	10 working days	February 28, 2018
3	Environmental Immunotoxicology and Reproductive Toxicology	October 2018	2 weeks	To be announced
4	Environmental and Health Risk Assessment and Management of Toxic Chemicals	November/December 2018	2 weeks	To be announced

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

Course Description:

1. Detection of Environmental Pollutants, Testing and Screening of Toxicity (February 5 - 16, 2018)

This course covers both theoretical and practical aspects in toxicology relating to the detection of different types of toxicants and their associated toxicity. It presents the different analytical methods in environmental toxicology; toxic compounds in the environment, mechanisms of actions and their effects on man; how to monitor human exposure through the use of biomarkers; and modern techniques instrument analysis. Participants will have an opportunity to conduct hands on experiments and testing.

Requirement: Participants should have jobs/responsibilities related to the detection of toxicity from toxic compounds in the environment and their effects in humans.

2. Environmental Toxicology (April 19 - 27, 2018)

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.

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CRI/ICEIT Newsletter

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