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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".

The 8th Princess Chulabhorn International Science Congress

"Environmental Health: Inter-linkages among the Environment, Chemicals and Infectious Agents"

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

The 8th Princess Chulabhorn International Science Congress was held during the week of November 13-17, 2016 to commemorate the seventieth anniversary of the accession to the throne of His Majesty King Bhumibol Adulyadej and the seventh cycle (84 years) birthday of Her Majesty Queen Sirikit. The theme of the Congress, "**Environmental Health: Inter-linkages among the Environment, Chemicals and Infectious Agents**", is in line with the views of Her Royal Highness Princess Chulabhorn, who has consistently emphasized the importance of research and development in environmental health, which can integrate the fields of environmental science, chemical safety and infectious agents.

The Congress included a keynote lecture, a Nobel laureate lecture, plenary sessions, symposia, and a roundtable discussion. There were also 16 platform presentations and 159 poster presentations displaying research conducted by congress participants. The Congress involved a total of 75 invited speakers from 20 countries and over 700 participants from 29 countries.

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Prenatal Exposure to Outdoor Air Pollution and Childhood Allergic Diseases

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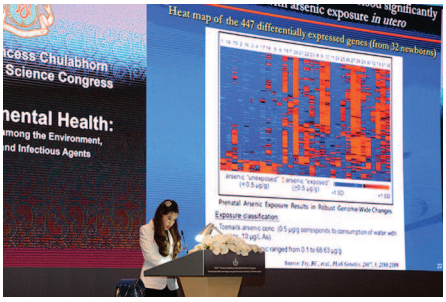
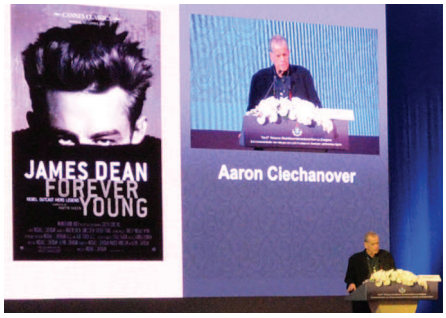


The Opening Keynote Lecture was given by Her Royal Highness Princess Chulabhorn on the topic of, "**Environmental Health: The Significance of Early Life Chemical Exposures**".

Her Royal Highness provided a general overview of the public health problems facing the developing world that are a major obstacle to sustainable development because they impact not only the health of the present generations but also future ones. She provided a specific example on arsenic to illustrate the extent of the potential impacts and the importance of early life exposures to environmental toxicants, showing that prenatal exposures can lead to a series of biochemical and biological changes with lasting effects and implications for future manifestation of adverse health effects. The collective evidence presented contributes to our understanding of how arsenic affects biological systems and suggests plausible mechanisms by which prenatal arsenic exposure and continued exposure during early childhood may lead to increased risk for development of cancer and other diseases later in life.

Her Royal Highness also touched upon the fact that our attempt to gain a good understanding of exposures and resultant effects is complicated by a multitude of modifying factors. Susceptible populations, such as children, the elderly and people with pre-existing diseases, offer a layer of complexity in terms of exposure and manifestation of resultant health effects.

Therefore, in order to effectively address health risks and impacts of exposure to chemicals and infectious agents, much research is needed to gain a better understanding of these complex factors that impact on human health, and to define the inter-linkages, such that policy decisions and interventions can be taken to control and ultimately reduce the global burden of disease attributed to them, thereby improving the quality of life for all.

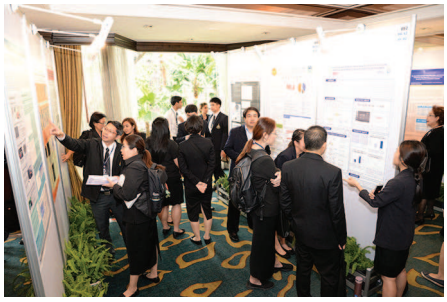


The Nobel laureate lecture was given by Distinguished University Professor **Aaron Ciechanover**, Faculty of Medicine Technion–Israel Institute of Technology, Haifa, Israel, on the topic of **"The Revolution of Personalized Medicine - Are We Going to Cure all Diseases and at What Price"**. Professor Ciechanover's discovery of the ubiquitin pathway for cellular protein degradation won him the Nobel Prize in Chemistry in 2004.

Plenary Lectures included: "**Signatures of Mutational Processes in Human Cancer**" by *Sir* Michael R. Stratton (Wellcome Trust Sanger Institute, U.K.); "**Air Pollution and Smoking: Leading Global Causes of Cancer**" by Jonathan M. Samet (University of Southern California, U.S.A.); "**The Role of the Environment - Precision Medicine; Liver and Lung Cancer**" by Curtis C. Harris (NCI, U.S.A.); "**Cancer Genomic Heterogeneity and Hepatocarcinogenesis**" by Xin Wei Wang (NCI, U.S.A.); "**The Complex Pathway to Noncommunicable Disease: Opportunities for Prevention**" by William A. Suk (NIEHS, U.S.A.); "**Using Exposomics to Assess Cumulative Risks from Multiple Environmental Stressors**" by Martyn T. Smith (University of California, Berkeley, U.S.A.); "**Protein Research in Relation to Human Health and Disease**" by *M.R.* Jisnuson Svasti (Chulabhorn Research Institute, Thailand); "**Susceptibility to Infectious Diseases: Does Exposure to Environmental Chemicals Play a Role?**" By Gwen W. Collman (NIEHS, U.S.A.); "**Health and Environment in South-East Asia – Challenges Ahead**" by Poonam Khetrapal Singh (WHO, SEARO); and "**Collaboration Across Sectors is the Key to Inventing New Medicines and Bringing Them to Patients**" by Michael Rosenblatt (Flagship Ventures, U.S.A.)

In addition, there were also 13 symposia and a roundtable discussion on "**Regional Environmental Health**", which was jointly organized by the UNEP Regional Office for Asia-Pacific and the WHO Regional Office for South-East Asia.

The 8th Princess Chulabhorn International Science Congress provided an important platform for showcasing research findings in the areas of the environment, chemicals and infectious agents, and the interplay among the three in impacts on human health, which are important in the prevention and mitigation of environmental health problems.



WHO Workshop on Human Biomonitoring (HBM) to Support Chemical Risk Assessment, Bangkok, Thailand, 17-19 November 2016

The Chulabhorn Research Institute (CRI) and the World Health Organization (WHO) co-hosted the WHO Workshop on Human Biomonitoring (HBM) to Support Chemical Risk Assessment at the CRI Convention Center, Bangkok, from 17-19 November 2016.

This meeting was attended by 28 participants from 11 countries (Bhutan, Canada, Costa Rica, Ghana, India, Indonesia, Israel, Malaysia, Myanmar, Spain, Thailand) and institutions participating in the WHO Chemical Risk Assessment Network, and also included 7 experts from the European Commission, MAK Commission, Public Health England UK, WHO HQ, WHO SEARO, and UNEP.

The sessions organized for the meeting included those to introduce Human Biomonitoring and Human Health Risk Assessment, including examples from Spain, Costa Rica, Canada, Israel and Thailand; provided information on currently available resources, including Biological Tolerance Values, the OECD Biomonitoring Database, and the UK HBM Programme and harmonization methodologies in Europe; and discussed the unique needs of developing countries with regards to implementing HBM.

Conclusions and recommendations from the workshop included: (i) information about the areas of work being undertaken, protocols being used and results obtained in the area of HBM should be shared among members of the network – this includes sharing of protocols by the MAK Commission, OECD and European authorities; (ii) documenting, sharing and learning from case studies of HBM in various countries would be extremely valuable, particularly on the issues of data quality and inter-comparability; (iii) standardized protocols for sample collection, analysis and data recording are prerequisites for multi-country projects; (iv) laboratory proficiency and standard control material availability are important challenges, particularly for developing countries; and (v) reference values for use and interpretation of HBM data, particularly in public health and for assessing health impacts of environmental contaminants, are needed.

Prenatal Exposure to Perfluorocarboxylic Acids and Childhood Growth

Perfluorocarboxylic acids (PFCAs) are synthetic organic chemicals consisting of chains of fluorinated carbons which vary in chain length. They have been used widely since the 1950s in consumer products such as lubricants, polishes, water-repellant coatings for paper and textiles, fire-fighting foams, and cookware.

Some examples of these chemicals include perfluorooctanoic acid (PFOA), containing 8 carbon atoms (C8), and long-chain PFCAs, such as perfluorononanoic acid (PFNA; C9), perfluorodecanoic acid (PFDeA; C10), perfluoroundecanoic acid (PFUnDA; C11), and perfluorododecanoic acid (PFDoDA; C12).

PFCAs persist in the environment and have been widely detected in both wildlife and humans. Human exposure routes include inhalation and ingestion via dust, water, and food.

Although PFOA has a half-life of approximately 3.5 years in humans, long-chain PFCAs have longer half-lives.

PFCAs are suspected developmental toxicants. Animal studies suggest that PFCAs induce adverse effects on fetal/neonatal mortality and growth, particularly in response to high-dose exposures.

No epidemiologic studies to date have assessed childhood growth in relation to prenatal exposure to long-chain PFCAs.

The present study of a birth cohort in Taiwan examined whether serum concentrations of PFOA and four long-chain PFCAs (PFNA, PFDeA, PFUnDA, and PFDoDA) during pregnancy are associated with fetal and childhood growth.

For 223 Taiwanese mothers and their term infants, researchers measured PFOA and four long-chain PFCAs in third-trimester maternal serum; infant weight, length and head circumference at birth; and childhood weight and height at approximately 2, 5, 8, and 11 years of age.

The results showed that prenatal exposures to long-chain PFCAs were inversely associated with girls' size at birth and with boys' and girls' height across childhood.

Specifically, in girls, prenatal exposures to PFNA, PFDeA, PFUnDA, and PFDoDA were linked to decreased birth weight. Prenatal PFDeA and PFUnDA were associated with increased odds ratio of being small for gestational age. Also, prenatal exposure to PFDoDA was associated with smaller head circumference at birth.

In boys, prenatal exposures to PFNA, and PFDoDA were associated with reductions in height at certain ages in childhood, but not with size at birth.

It is possible that the observed associations in this study population reflect prenatal effects of PFCAs on postnatal growth.

Source: Environmental Health Perspectives, Vol. 124, No. 11, Pages 1794–1800, November 2016.

Workshop on South-East Asian Children's Environmental Health (CEH)

Following the 8th Princess Chulabhorn International Science Congress, which was held at the Shangri-La Hotel from November 13-17, 2016, the Workshop on South-East Asian Children's Environmental Health (CEH) was organized by the Chulahorn Research Institute (CRI) at the CRI Convention Center on November 18 with funding support from the U.S. National Institute of Environmental Health Sciences (NIEHS).

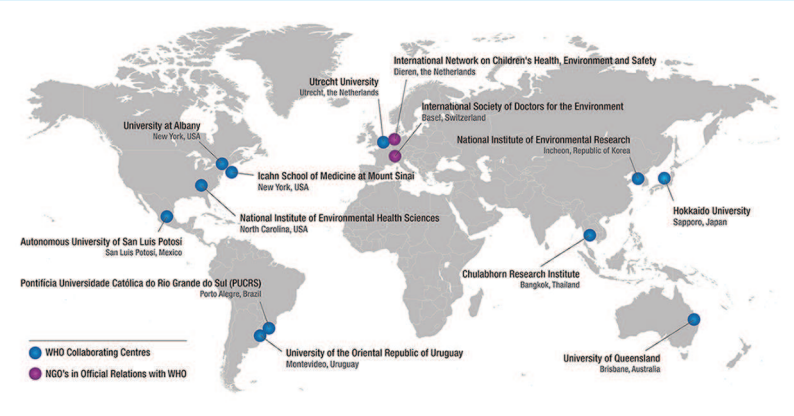
The objective of the workshop was to form a network of institutions in the Asia-Pacific region that will conduct work in the area of CEH, and to increase coordination with the NIEHS World Health Organization (WHO) Collaborating Centers, focusing on a Global Children's Environmental Health Network (GCEH).

Children are especially vulnerable to physical, chemical, and biological threats in their environment. According to WHO, environmental factors account for one-third of the global disease burden in children. Furthermore, in low- and middle-income countries, children disproportionately have the highest environment-related death rates. In fact, the infant death rate from environmental causes is 12 times higher in low-income countries than in high-income countries.

These statistics highlight the need to raise awareness, and to strengthen global capacity for environmental health research and training to protect children's health around the world.

Dr. William Suk, director of both the Center for Risk & Integrated Sciences (CRIS), and the Superfund Research Program, and the chief of the Hazardous Substances Research Branch in the NIEHS Division of Extramural Research and Training, introduced the historical development of the WHO Global Strategy on CEH to make clear why a Global Children's Environmental Health Network is needed.

The Global Network of WHO Collaborating Centres in Children's Environmental Health is comprised of research institutes around the world (see picture).



Each Collaborating Centre acts as a hub to strengthen national or regional capacity to create healthier environments for children. Such collaboration and the sharing of services and expertise among Centres in the network will build global CEH capacity.

Representatives from Bhutan, India, Myanmar, Vietnam and Thailand joined and presented their country reports, which provided information on the current situation and country needs related to children's environmental health, and the danger of disease and exposure that children face in their own environments.

For more information on the Network of WHO Collaborating Centres for Children's Environmental Health, please visit the website: https://www.niehs.nih.gov/research/programs/geh/partnerships/network/index.cfm.

Exposure to Pyrethroid Pesticides and the Risk of Childhood Brain Tumors in East China

Brain tumors are the most common solid tumors in children and the leading cause of childhood cancer-related mortality. Childhood brain tumors (CBT) can be categorized into a variety of histological types, of which gliomas account for approximately 60%. The remaining 40% consist of embryonal tumors, cranyopharyngiomas, pineal tumors, meningiomas, and others.

Global incidence of CBT has increased over the past several decades. Little is known about the etiology of CBT, but pesticide exposure is suspected. Many epidemiological studies have revealed a positive association between increased risk of CBT and parental or childhood pesticide exposure. Some studies have shown no such association.

In the 1970s after the use of organochlorine insecticides was banned Pyrethroids, a group of synthetic less biodegradable pesticides, were manufactured.

Pyrethroids such as bifenthrin, permethrin, and tetramethrin are categorized by the US Environmental Protection Agency as potential human carcinogens at relatively high exposure levels. Pyrethroids are thought to disrupt the endocrine system and to exert a suppressive effect on the immune system. There is some evidence indicating that pesticides may contribute to chromosomal aberrations, oxidative stress, and cell signaling disturbances in the human body. All of these could be associated with increased cancer risk.

In China, pyrethroid pesticides have been extensively used in agriculture, forestry, horticulture, industry, and home applications, with a huge demand of 3700 tons annually.

Although the acute toxicity of pyrethroids is well documented, few data on health impairment of chronic lower-level exposure are available. Even at relatively low exposure levels, pyrethroids could result in endocrine disruption, immune system suppression, and carcinogenesis.

An epidemiologic study has reported that increased urinary levels of pyrethroid metabolites might be associated with an elevated risk of childhood acute lymphocytic leukemia.

Pyrethroids are metabolized by esterases in the human body. The metabolites are excreted in urine with the excretion half time of 4-13 h. Detection of pyrethroid metabolites in urine by gas chromatography-mass spectrometry method is a reliable way to evaluate pyrethroid exposure levels in individuals.

The present study hypothesized that pesticide exposure is one of the risk factors for the development of CBT.

This hospital-based case control study evaluated the association of pyrethroid pesticide exposure with the risk for CBT in a population of children in East China. In total, 161 CBT cases and 170 controls were recruited from 2 children's medical centers in Shanghai between September 2012 and June 2015.

Pyrethroid pesticide exposure was evaluated by urinalysis of 3 nonspecific metabolites of pyrethroids (cis-DCCA, trans-DCCA, and 3-PBA) and by administering a questionnaire.

Childhood and adult brain tumors have different causes. The CBTs seem to be the result of aberrant growth of germ cells resembling the developing organs. Exposing children or fetuses to pesticides may lead to gene mutations in embryonal or somatic cells, thus further contributing to the initiation of cancerous tumours.

Children are likely to be more sensitive to the carcinogenic effects of pesticides than adults are. Their increased sensitivity is attributed to their quickly dividing cells, and their common characteristics such as hand-to-mouth contact, spending more time on the floor playing, greater intake of food or fluids per pound of body weight than adults, and incomplete enzymatic detoxification function.

In China, the annual demand for pyrethroids is up to 8 million pounds, which might cause significant exposure in the population, and also cause potential harm to children's health. By contrast, the estimated annual use of pyrethroids in the United States and Germany is only 1 million pounds and 170 thousand pounds, respectively.

In this study, the researchers found that pyrethroid pesticide exposure was positively associated with an increased risk of CBT, a finding which further verifies the potential carcinogenesis of pyrethroids.

This is the first study to reveal by quantitative evaluation the association between pyrethroid exposure and the risk of CBT.

In East China, the warm and humid climate favors pest propagation, which in turn stimulates the large demand for residential use of pesticides. Studies from East China report that more than half the families queried regularly used pesticides at home. This is a big concern for children's health.

Household pesticide use is considered an important contribution to pyrethroid exposure, and may be associated with the risk of childhood brain tumors, leukemia, and lymphoma.

The household pesticides in the study which significantly increased the risk of CBT were mosquitocides and cockroach killers, according to the questionnaire data. These formulations include pyrethroids.

To conclude, in the present study increased urinary metabolite levels of pyrethroid pesticides were associated with increased risk of CBT. Prospective cohort studies with larger sample sizes are required to confirm these findings.

Because of the substantially higher concentrations of pyrethroid pesticide metabolites in children in East China and the possible carcinogenic characteristic of pyrethroids, efforts should be made generally to protect children from pesticide exposure

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Source: Environmental Pollution, Vol. 218, Pages 1128–1134, November 2016.

Serum Concentrations of Persistent Organic Pollutants, Lactation History, and Maternal Risk of Diabetes

Persistent organic pollutants (POPs) are resistant to degradation in the environment and are highly persistent in the human body. These pollutants include organochlorine pesticides (OCP), polychlorinated dibenzo-*p*-dioxins (PCDD), polychlorinated dibenzofurans (PCDF), polychlorinated biphenyls (PCB), and perfluoroalkyl substances (PFASs).

Breast milk is known to contain environmental chemicals, and lactation is an important excretion route for many of them, especially those with long elimination half-lives in humans. It has been found that lactation lowers the POP burden in mothers.

Although several studies have linked higher POP storage with diabetes risk, it remains unclear whether potential long-term benefits of lactation on chronic disease risk in women could be attributed to the reduction in POP burden.

Among 4,479 parous women participating in the National Health and Nutrition Examination Survey (NHANES) between 1999 and 2006, 3 hypotheses were examined: (1) a lifetime lactation history is associated with lower serum POP concentrations, (2) a lactation history is associated with a lower diabetes risk, whereas higher POP concentrations are associated with a higher diabetes risk, and (3) a lactation history may lower diabetes risk through reducing serum POP concentrations.

The results showed lifetime lactation history to be inversely associated with serum concentrations of 17 out of the 24 OCP, PCBs, and PFASs. The inverse association between lactation and diabetes was slightly attenuated after adjustment for POPs.

Age-stratified analyses showed are inverse association between lactation periods and serum POP concentrations, observed primarily among participants <60 years. However, age did not significantly modify the association between lactation history and diabetes prevalence.

The results confirmed that the number of lactation periods ≥ 1 month was significantly associated with a lower diabetes risk and also lower serum concentrations of 17 selected POPs.

Moreover, the relative reduction of pollutants through lactation is also determined by the mother's age, weight change, and baseline POP. These differences in toxicokinetics of POPs need to be considered in future studies.

Transfer of pollutants from mothers to infants through lactation is likely. Concerns are emerging regarding the health impact on of infants due to exposure to POPs through breastfeeding. However, existing studies have consistently suggested that the long-term beneficial effects of lowering maternal risk of diabetes through breast-feeding outweighs the risk associated with POP exposures to offspring.

In addition, it has been shown that the body burden of many POPs in U.S. populations has steadily decreased in the past decades. Therefore, any adverse impact of POPs transferred through lactation to children may decrease over time. Taken together, these findings remain in line with current recommendations encouraging breastfeeding.

In conclusion, lifetime lactation history has been inversely associated with both serum POP concentrations and the risk of having diabetes.

These findings provide some support to the hypothesis that lactation may lower the subsequent diabetes risk, and that this may at least in part happen through reducing the maternal POP body burden due to the POP elimination through human milk.

Well-designed prospective studies with repeat measurements of serum POP concentrations before and after lactation periods and long-term follow-up for incident diabetes are required to help establish causal inferences regarding the role of POP exposure in the relationship between lactation and adverse health conditions.

Source: Environmental Research, Vol. 150, Pages 282–288, October 2016.

Memory Impairment Due to Fipronil Exposure in Rats

Fipronil is a second-generation insecticide which is effective against resistant pest strains. Initially developed to replace organophosphates pesticides.

Studies have confirmed that glutamate-activated chloride channels present in insects, but not in mammals, are fipronil's only targets.

However, fipronil does cause various toxic effects in both target and non-target organisms, including humans. It provokes neurotoxicity, having the antagonism of gamma-aminobutyric acid (GABA) as the primary mechanism for toxic action. GABAergic system appears to be involved in processes related to memory formation and consolidation.

There are no previous studies examining the effects on memory of short-term exposure to low-concentrations of fipronil.

The present work studied the importance of GABA to mechanisms involved in the very early development of fipronil-induced memory impairment in rats. The GABA antagonist picrotoxin was used in the experiments to more clearly identify fipronil's effects.

The impact on cognitive and spatial memory was assessed using new object recognition tasks (ORT) and eight radial arm maze tasks (8-RAM). Locomotor behavior was assessed using open field tasks. Intoxication indicators were weight gain modulation and fipronil in the blood.

Analysis of the animals' weight during the treatment period (15 days) showed that there was a reduction in weight gain in those treated with fipronil and also in those co-exposed with fipronil and picrotoxin.

Fipronil and picrotoxin, as GABA antagonists, can block the appetite stimulation promoted by 2-deoxyglucose. This suggests that GABAergic neurons are part of a regulating mechanism.

The findings indicate that exposure to fipronil, like exposure to picrotoxin, leads to changes in memory in animals. When co-exposed to fipronil and

(Continued on page 8)

Seven New Substances Added to 14th Report on Carcinogens

The US Department of Health and Human Services released its 14th Report on Carcinogens on November 3, 2016. The congressionally-mandated report, which is prepared by the National Toxicology Program (NTP), included seven newly reviewed substances, bringing the total to 248 listings.

Five viruses, a chemical, and a metallic element.

The chemical trichloroethylene (TCE), and the metallic element cobalt and cobalt compounds that release cobalt ions *in vivo*, are being added to the list, as are the following five viruses linked to cancer in humans: **Human immunodeficiency virus type 1 (HIV-1), Human T-cell lymphotropic virus type 1 (HTLV-1), Epstein-Barr virus (EBV), Kaposi sarcoma-associated herpesvirus (KSHV), and Merkel cell polyomavirus (MCV).**

All five viruses are being added to the category of *known to be a human carcinogen.* Collectively, these viruses have been linked to more than 20 different types of cancers. Approximately 12 percent of human cancers worldwide may be attributed to viruses. No vaccines are currently available for these five viruses. Hence, prevention strategies to reduce the infections that can lead to cancer are even more critical.

It's important to note that a listing in the report indicates a cancer hazard, but does not by itself mean that a substance or a virus will cause cancer. Many factors, including an individual's susceptibility to a substance, and the amount and duration of exposure, can affect whether a person will develop cancer. In the case of viruses, a weakened immune system may also be a contributing factor.

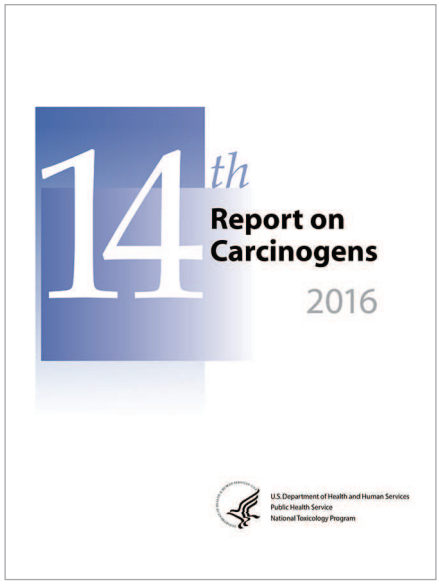
For example, HIV tends to weaken the immune system of the body, increasing the cancer risk. HIV can be transmitted through sexual activity, pregnancy, and infected needles. Individuals suffering from the disease may also develop non-Hodgkin and Hodgkin lymphomas, penile, cervix, vaginal and vulvar cancers, and oral cancers.

HTLV-1 can be acquired through exposure to biological factors and contaminated cells. It can result in T-cell leukemia lymphoma. EBV is passed through the saliva and affects over 90 percent of adults. Infected individuals typically remain healthy and show no symptoms. MCV is commonly found on the skin and rarely progresses to cancer.

Trichloroethylene (TCE) is an industrial solvent used primarily in producing hydrofluorocarbon chemicals. It is also being listed as a known human carcinogen. Since 2000, TCE had been listed as reasonably anticipated to be a human carcinogen. However, numerous new human studies showing a causal association between TCE exposure and an increased cancer risk have led NTP to reevaluate and reclassify TCE to the category of *known to be a human carcinogen.*

Cobalt and cobalt compounds that release cobalt ions *in vivo* are being listed as *reasonably anticipated to be a human carcinogen*. This listing includes different types of compounds that release cobalt ions into the body. The listing does not include vitamin B-12, which contains cobalt, an essential nutrient bound to protein which does not release cobalt ions. Cobalt is a naturally occurring element used to make metal alloys and other metal compounds, such as military and industrial equipment, and rechargeable batteries. The highest exposure occurs in the workplace and from in failed surgical implants. The listing for this metal and its compounds is based largely on studies in experimental animals.

Source: News Release from NIH on November 3, 2016 at https://ntp.niehs.nih.gov/pubhealth/roc/index-1.html



The 14th Report on Carcinogens is available on the NTP website at http://ntp.niehs.nih.gov/go/roc14

The report identifies many different types of environmental factors, collectively called substances, including chemicals; infectious agents, such as viruses; physical agents, such as X-rays and ultraviolet radiation; mixtures of chemicals; and exposure scenarios in two categories — ***known to be a human carcinogen*** and ***reasonably anticipated to be a human carcinogen.***

Known to be a human carcinogen: This category is used primarily when there is sufficient evidence, from human studies, showing a cause-and-effect relationship between exposure to the substance and human cancer. Occasionally, substances are listed in this category based on human studies showing that the substance causes biological effects known to lead to the development of cancer.

Reasonably anticipated to be a human carcinogen: This category includes substances where there is limited evidence of cancer in humans, or sufficient evidence in experimental animals showing a cause-and-effect relationship between exposure to the substance and cancer. Additionally, a substance can be listed in this category if there is evidence that it is a member of a class of substances already listed in the Report on Carcinogens, or causes biological effects known to lead to the development of cancer.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute, Year 2017

	Training Course	Date	Duration	Closing Date
1	Principles of Toxicology, Toxicity Testing and Safety Evaluation	February 7 - 22, 2017	2 weeks	December 15, 2016
2	Environmental Toxicology	April 24 - May 2, 2017	1+ weeks	February 15, 2017
3	Occupational and Environmental Health	July 17 - 21, 2017	1 week	May 15, 2017
4	Environmental and Health Risk Assessment and Management of Toxic Chemicals	November 2017	2 weeks	To be announced

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

Course Description:

Environmental Toxicology (April 24 - May 2, 2017)

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

Memory Impairment Due to Fipronil Exposure in Rats

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picrotoxin, changes in animal behavior were intensified, suggesting synergistic action.

Additionally, fipronil and picrotoxin co-exposure enhanced effects on memory compared to controls, fipronil exposure, or picrotoxin exposure, suggesting strongly a GABAergic effect.

The locomotor activity of animals in all groups were examined in an open field arena. Researchers observed that none of the treatments, including fipronil exposure, changed their behavior. This indicates that the health or behavior of the animals was not affected by fipronil exposure.

Because the $GABA_A$ receptor is affected by exposure to environmental agents with the same active mechanism as fipronil, it may underlie changes in or the abnormal modulation of processes involved in learning. $GABA_A$ may also affect the formation and consolidation of cognitive and spatial memory.

In conclusion, this study reports that fipronil can have toxic interactions with the central nervous system of mammals, leading to memory impairment due to the modulation of the GABAergic system.

Source: Physiology & Behavior, Vol. 165, Pages 28–34, October 2016.

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

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