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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: TIGER-LC Study Identified Common Molecular Subtypes Between Two Types of Liver Cancers Among Asian Populations

From left: CCR investigator Curtis Harris, M.D., Director of CCR Robert Wiltrout, Ph.D., President of CRI Professor Dr. HRH Princess Chulabhorn Mahidol, Ph.D. and and CCR investigator Xin Wei Wang, Ph.D.

Liver cancer represents one of the main causes of cancer-related death and is considered as a major public health problem in Thailand. Due to the limited treatment options and poor prognosis for patients, researchers at the Chulabhorn Research Institute and the Center for Cancer Research (CCR), National Cancer Institute USA have established the **Thailand Initiative in Genomics and Expression Research for Liver Cancer (TIGER-LC)** to study two major types of liver cancer in Thailand: hepatocellular carcinoma (HCC) and intrahepatic cholangiocarcinoma (ICC).

Since 2008, the consortium that consists of researchers and clinicians from five cancer hospitals in Thailand, has been collecting tissue samples and clinical data from individuals with and without liver cancer to create a comprehensive biorepository for large scale studies.

In research reported in the journal Cancer Cell (July 2017), the TIGER-LC research team analyzed the genetic sequences, gene activity and metabolic profiles of 199 tumor samples from patients in Thailand, including both HCCs and ICCs. The team used the data to identify subtypes of each disease with common molecular features. Surprisingly, the molecular features that defined certain subsets of ICCs closely resembled subsets of HCCs with similar gene expression matrices, tumor biology and clinical outcomes.

(Continued on page 2)

CRI: TIGER-LC Study Identified Common Molecular Subtypes Between Two Types of Liver Cancers Among Asian Populations

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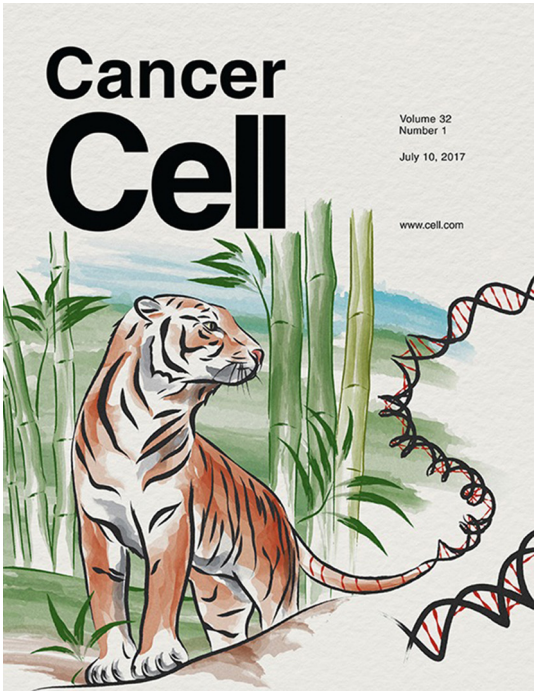


Examining additional tumor samples from other countries, the team found that several of the subtypes they had identified in the original analysis were represented among patients of all ethnicities, but some occurred only among Asians.

Recognizing the molecular nature of liver cancer's common subtypes could lead to more effective treatment of liver cancers, regardless of diagnosis. The findings could help researchers identify patients with particularly aggressive liver cancers and recommend appropriate treatments. The team has also identified genetic mutations as possible drivers of specific subtypes. These findings may point researchers toward new strategies for drug development.

Source: Cancer Cell, Vol. 32, Issue 1, Pages 57-70, July 2017.

On the cover: **The Thailand Initiative in Genomics and Expression Research for Liver Cancer (TIGER-LC) Consortium** (depicted as a tiger) emerges from foliage, representing molecular, clinical, and epidemiological studies from teams in the United States, Thailand, and Japan, to generate a multilayered genomic and genetic liver cancer data ecosystem (represented by the tiger's tail). Although common molecular subtypes (depicted as bamboo stalks) are observed among liver cancer types, there are differences observed between Asian and Caucasian populations (depicted by different bamboo colors). Cover art by Ethan Tyler.

Clinical Effects of Air Pollution on the Central Nervous System

The adverse effects of ambient air pollution on respiratory and cardiovascular health are firmly established. However, impact on the central nervous system (CNS) was first described only a decade ago.

Recent clinical and epidemiological studies indicate adverse associations between air pollution and neurological disease, but mechanistic pathways remain elusive.

Ambient air pollution is a mixture of gases and particulate matter released into the surrounding air by stationary (industrial and household etc.) and mobile (vehicles, trains, ships etc.) sources.

These gaseous mixtures are primarily composed of Carbon monoxide (CO), Ozone (O_3), Nitrogen dioxide (NO_2) and Sulfur dioxide (SO_2).

Air pollution and particulate matter (PM) are associated with neuro-inflammation and reactive oxygen species (ROS), processes strongly related to the pathogenesis of CNS diseases.

This review examines the clinical and epidemiological effects of air pollution and their association with CNS pathologies in order to better set the stage for further investigation.

The conceptual framework focuses on adverse effects of air pollution with respect to neurocognition, white matter disease, stroke, and carotid artery disease.

Both children and older individuals exposed to air pollution exhibit signs of cognitive dysfunction and enhanced progression of neurodegenerative processes underlying Alzheimer's (AD) and Parkinson's diseases (PD). However, evidence on middle-aged cohorts is lacking.

Discrete components of air pollution and their functional effects on cognition have been examined. A study has demonstrated that PM is associated with lower verbal learning performance, NO_2 is inversely associated with logical memory abilities, and that O_3 exposure is associated with lower executive functioning.

Furthermore, studies demonstrate evidence of structural brain effects, such as white matter injury secondary to air pollution exposure, which can manifest as neurocognitive dysfunction.

Exposure is also associated with adverse vascular effects. The relationship between stroke and air pollution has been established over the past decade. The Global Burden of Disease Study 2013 found 29.2% of the global stroke burden was attributble to air pollution. Large populations living in highly polluted environments are at risk.

Findings suggest increased risk for hospitalization and subsequent stroke mortality following transient increases in the followng constituents: $PM_{2.5}$, PM_{10}, SO_2, CO and NO_2.

Increases in stroke incidences and mortalities are seen in the setting of air pollution exposure, and CNS pathology is robust.

(Continued on page 4)

The Global Burden of Disease Study 2015: AMBIENT AIR POLLUTION

Long-term exposure to ambient air pollution increases mortality and morbidity from cardiovascular and respiratory disease and lung cancer and shortens life expectancy.

Based on this evidence, the Global Burden of Diseases, Injuries, and Risk Factors Study 2015 (GBD 2015) has estimated the burden of disease attributable to 79 risk factors, including ambient air pollution, in 195 countries and territories from 1990 to 2015.

The GBD 2015 study identified air pollution as a leading cause of global disease burden, especially in low- and middle-income countries.

The researchers estimated global population-weighted mean concentrations of ozone and particulate matter with an aerodynamic diameter of less than 2.5 μm ($PM_{2.5}$), using satellite-based estimates, chemical transport models, and ground-level measurements.

Based on integrated exposure-response functions for each cause of death, the relative risk of mortality was estimated.

Deaths due to ischaemic heart disease, cerebrovascular disease, chronic obstructive pulmonary disease, lung cancer, and lower respiratory infections were identified in epidemiological studies using non-linear exposure-response functions spanning the global range of exposure.

Ambient $PM_{2.5}$ was the fifth-ranking mortality risk factor in 2015. Exposure to $PM_{2.5}$ caused 4.2 million deaths and 103.1 million disability-adjusted life-years (DALYs) in 2015, representing 7.6% of total global deaths and 4.2% of global DALYs, 59% of which were in east and south Asia.

Deaths attributable to ambient $PM_{2.5}$ increased from 3.5 million in 1990 to 4.2 million in 2015. Exposure to ozone caused an additional 254,000 deaths and a loss of 4.1 million DALYs from chronic obstructive pulmonary disease in the same year.

Increasing over the past 25 years, ambient air pollution contributed substantially to the global burden of disease in 2015, due to the ageing of population, changes in non-communicable disease rates, and increasing air pollution in low- and middle-income countries.

Only modest reductions in these burdens will occur in the most polluted countries unless $PM_{2.5}$ values are decreased substantially. However, if exposure could be reduced, the potential health benefits would be substantial.

Although global rates of mortality due to $PM_{2.5}$ exposure decreased from 1990 to 2015, the absolute numbers of attributable deaths and DALYs actually increased. This was due to rising levels of pollution and increasing numbers of deaths from non-communicable diseases in the largest low- and middle-income countries in east and south Asia, where populations are growing and ageing.

This research places the burden of disease as a result of ambient air pollution. Within the context of other common potentially modifiable risk factors at a national level, helping to prioritise air pollution from a population health perspective.

In the analysis of trends in the burden of disease caused by ambient air pollution, researchers show where, and the extent to which, ambient air pollution is changing as a contributor to disease burden, and the extent to which these trends in the attributable burden reflect progress, or absence of it, in reducing exposure versus changes in demographic factors.

This helped them to see more clearly the challenges that must be overcome in order to reduce the public health effects of exposure to air pollution.

Trends in attributable deaths reflect demographic and epidemiological trends as well as increasing levels of air pollution in low- and middle-income countries.

If major reductions are not made in pollution levels, these trends will lead to increasing burdens.

Unless $PM_{2.5}$ levels markedly decline, non-linear exposure-response functions suggest only modest reductions in the disease burdens of the most polluted countries.

As a result, the challenges for future reductions in the burden of disease attributable to air pollution are substantial.

International experience has shown that exposure to ambient air pollution and its associated burden of disease can be lowered for entire populations when policy action occurs at the national and subnational levels via aggressive air quality management programmes which focus on major sources of air pollution.

Source: The Lancet, Vol. 389, Pages 1907-1918, May 2017.

The Global Burden of Disease Study (GBD) is the most comprehensive worldwide observational epidemiological study to date. It describes mortality and morbidity from major diseases, injuries and risk factors to health at global, national and regional levels. Examining trends from 1990 to the present and making comparisons across populations enables understanding of the changing health challenges facing people across the world in the 21st century.

Mercury Exposure and Alzheimer's Disease in India - An Imminent Threat?

In 2008, the World Health Organization (WHO) declared dementia as a priority condition through the Mental Health Gap Action Program. As of 2015, there were an estimated 46.8 million people with dementia worldwide. This number will increase to an estimated 75.6 million in 2030 and 135.5 million in 2050.

According to the latest World Alzheimer's Report (2015), India has the third-largest population of dementia patients in the world with 4.1 million in India, 4.2 million in the USA, and more than 9 million in China.

India is the third highest energy consumer after China and the USA with 5.3% global share in 2015. It is also the fastest growing country in the world in terms of both population and economy. Increasing energy production brings the greater burden of more potential emissions of mercury (Hg). India already ranks second in the world for high Hg emissions.

Hg has been shown to increase the risk of getting Alzheimer's disease (AD). Long-term exposure to Hg has been reported to produce Alzheimer's like symptoms. Some autopsies have found increased Hg concentration in brain tissues of AD patients. There are increasing reports of AD and dementia in different age groups in India.

Inorganic Hg damages the nervous system and produces pathological changes similar to AD. Hg exposure helps to form "neurofibrillar tangles" inside brain cells that are one of the two diagnostic markers for AD.

Low level inorganic Hg exposure damages nerve cells (like AD), but other metals (zinc, aluminium, copper, cadmium, manganese, iron, and chromium) aggravate the toxic effects of Hg inside nerve cell. Increasing Hg exposure can generate free radical and produce oxidative stress in brains.

The increasing rate of Hg emission, deposition and regular exposure, especially in developing countries like China and India may increase neurodegenerative disorders that lead to dementia and death.

Global assessments made by UNEP in 2013 on Hg show a continuous increase in anthropogenic Hg production in developing countries. Coal burning for power generation and for industrial purposes continues to increase, especially in developing countries.

Increase in atmospheric Hg release from different point sources may increase Hg exposure in many industrially developed cities in India. Mercury content of marine surface waters has also been reported to increase (~ three times) compared to pre-anthropogenic conditions.

Hg evasion processes in coastal ocean waters are major non-point sources. About 2600 tons of Hg emission are estimated to take place annually from evasion processes.

Conversion of elemental Hg (released by evasion processes) into oxidized forms at ocean-atmosphere interface may increase the bioavailability of Hg in coastal environments. Thus, people who live in the coastal areas of India may also be exposed to reactive Hg from this non-point source.

In addition, oxygen-depleted zones in the seas around India, both the Bay of Bengal and the Arabian Sea, may facilitate microbial methylation of inorganic Hg to form monomethylmercury, a highly neurotoxicant.

Formation of monomethylmercury is expected to increase Hg accumulation in marine food webs, which increases the likehood of Hg concentration in seafood (including fish). Increasing fish catch and consumption per capita in the coastal states of India may therefore increase potential health risks associated with chronic exposure to Hg in India.

Long-term exposures to Hg, both from point and non-point sources, can be major sources of health risk in India. However, the relationship between increasing Hg exposure and increasing neurodegenerative disorder in India is still not clear.

This commentary points to the need for better understanding of the relationship between Hg release and AD in India and in other countries, and the need to protect human health and the environment from the adverse effects of Hg.

Source: Science of The Total Environment, Vol. 589, Pages 232–235, July 2017.

Clinical Effects of Air Pollution on the Central Nervous System

(Continued from page 2)

Growing evidence supports a role in facilitating the process of atherosclerosis, particularly carotid artery disease.

Carotid artery disease is a significant risk factor for stroke and cognitive impairment. Studies suggested that residential proximity to a major roadway is associated with increased carotid intima-media thickness (CIMT). Results have demonstrated that $PM_{2.5}$ is an independent risk factor for carotid artery stiffness.

Air pollution is also linked to intracranial vascular disease. Among seniors, $PM_{2.5}$ increases resting cerebrovascular resistance and decreases cerebral blood flow velocity. Prenatal $PM_{2.5}$ and O_3 exposure are also associated with carotid artery stiffness and increased CIMT, respectively.

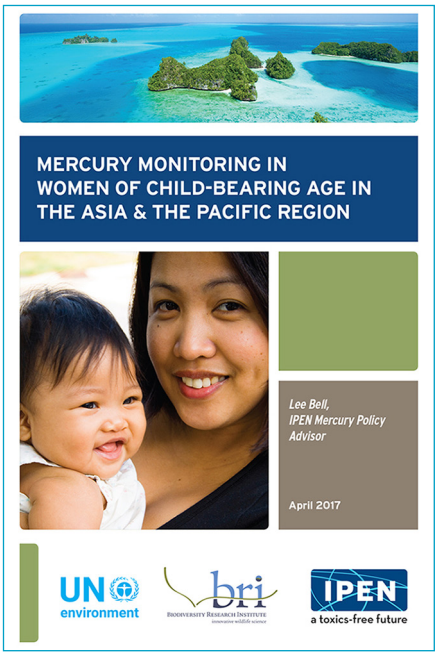
In conclusion, air pollution from urban environments affects the central nervous system in a multitude of ways. Exposures impact cognitive function, stroke risk, and carotid artery disease.

Pediatric and elderly populations are highly vulnerable due to CNS development and aging processes.

Compiling and contextualizing current evidence should provoke thought and future investigations focused on improving health outcomes.

Source: Journal of Clinical Neuroscience, Published online in May 2017.

MERCURY MONITORING IN WOMEN OF CHILD-BEARING AGE IN THE ASIA & THE PACIFIC REGION



The report describes a pilot study undertaken between 2015 and 2016 by IPEN in collaboration with the Biodiversity Research Institute (BRI) and UN Environment (United Nations Environment Programme: UNEP) to assess mercury concentration in the hair of selected participants in the Asia and the Pacific region.

The data obtained will provide a sampling of mercury levels in small selected populations which may contribute to national information on mercury concerns.

Women from 1-2 locations in 6 countries participated in the study, including Cook Islands, Kiribati, Marshall Islands, Nepal, Tajikistan, and Tuvalu.

The women who participated constitute part of the vulnerable sub-population groups at risk from mercury, a potent neurotoxicant which can affect both the health of the mother and impact on a range of developmental endpoints in the developing foetus with lifelong consequences.

Participants were then required to provide a small sample of hair and to complete a questionnaire. The samples of hair were shipped to BRI laboratories in the United States for analysis.

The results of the sampling varied greatly between locations, but some consistent trends were observed.

Women from Small Island Developing States (SIDS) in the Pacific were found to have very high levels of mercury body burden, compared to most other locations.

This is consistent with data from the study questionnaires and prior studies indicating that most of these women have a diet rich in seafood. Large predatory fish that feature in the diet of women in the Pacific SIDS are commonly cited in the literature as having high methylmercury (MeHg) concentrations in their flesh. Consumption of these fish results in the transfer of mercury to humans and subsequent elevated mercury body burden.

Of the 234 women who participated, 163 (69.2%) had mercury body burdens which exceeded the reference level of 1 ppm total mercury in hair.

The reference level corresponds closely with the U.S. EPA's reference dose (RfD) of 0.1 µg/kg bw/day and a blood mercury concentration of 4-5 µg/L.

Current scientific literature suggests that adverse effects on sampled individuals begin to occur at or above the reference level of 1 ppm.

Recent studies also conclude that negative developmental effects may occur at even lower levels. Of the 150 participants located in Pacific Island States, 144 (96%) exceeded the 1 ppm reference level. Among participants who lived in areas other than the Pacific Island States 21.4% exceeded the 1 ppm reference level.

The majority of participants located in countries other than Pacific Island states live near some source of mercury pollution such as a waste disposal site, industrial emissions or polluted waterway yet reported a much lower mercury body burdens.

The participating women from Tajikistan all lived within a few kilometres of Minamata Convention Annex D mercury emission point sources (cement plant and thermal power station).

Women of the Jalari community in Nepal (location 1) consume fish from a waterway contaminated with effluent from medical waste and dental facilities which are a potential mercury pollution source.

The exception to this was a group of women from location 2 in Nepal who live in an urban environment but work in the manufacture of gold plated idols for religious purposes. The method of gold plating known as mercury gilding involves the use of mercury. This could be a potential cause of the higher body burden of mercury among some members of this group of women and is discussed more completely in the report.

The results from location 1 in Nepal (fisherfolk) are much lower, with most samples below the 1 ppm reference level, despite the fact that these women, as fisherfolk, have a relatively high fish diet derived from polluted waterways.

Note:

IPEN is a network of non-governmental organizations working in more than 100 countries to reduce and eliminate the harm to human health and the environment from toxic chemicals.

Biodiversity Research Institute (BRI) is a nonprofit ecological research group whose mission is to assess emerging threats to wildlife and ecosystems through collaborative research, and to use scientific findings to advance environmental awareness and inform decision makers. BRI is the leading international institute supporting the global mercury monitoring efforts for the Minamata Convention on Mercury.

Source: Mercury Monitoring in Women of Child-Bearing Age in the Asia and the Pacific Region, 36 Pages, April 2017. (Available at http://ipen.org/Mercury-Monitoring-in-Women)

MOBILE PHONE USE AND GLIOMA RISK

The use of mobile phones has rapidly expanded and increased over the past decade to a total of 7 billion users worldwide in 2014.

Mobile phone users are regularly exposed to radiofrequency electromagnetic fields (RF-EMFs). The potential carcinogenic effects of exposure to such emissions from cellular phones in parallel to increased usage has caused growing concern, particularly with regards to the potential increased risk of brain tumors.

In May 2011, RF-EMFs were classified as carcinogenic to humans (group 2B) by the International Agency for Research on Cancer (IARC). This classification was made in the setting of limited epidemiologic evidence, mechanistic support or animal modelling supporting a potential association between exposure to wireless phones and increased risk of glioma and acoustic neuroma.

Many studies have investigated the potential association between mobile phone use and the risk of glioma. Glioma is the most common malignant tumor of the central nervous system. Glioma may originate from a variety of different aetiological pathways relative to other non-glioma cerebral tumors.

However, the exact pathogenesis of glioma remains unclear. Whilst an underlying biological mechanism linking mobile phone use with glioma risk has not been established, the issue remains controversial and topical.

The long latency period between radiation exposure and subsequent tumor development mean that many studies are of insufficient follow-up length to actually identify potential associations and long-term risk.

More studies have recently reported an association between long-term (≥ 10 years) mobile phone use and an increased risk of glioma, but the absolute number of such studies is small. A minimum observation period of at least 10 years is required to study the long-term carcinogenic risks of RF field exposure during use of mobile phone.

The tendency for users to favour one side of the head when using their phone means that the ipsilateral brain is especially exposed to radiation, whereas the contralateral side is much less exposed.

Therefore, an important part of researching potential links between mobile phone use and tumor risk is to study potential correlations between the side of the head predominantly used during phone calls and the position of the glioma within the cerebrum.

The researchers performed a systematic review of studies on mobile phone use and glioma published by the end of 2015. The objective of this meta-analysis was to explore the potential association between mobile phone use and glioma based on time, partial laterality and glioma grade. This meta-analysis eventually included 11 studies comprising a total 6028 cases and 11488 controls.

The results showed a significant positive association between long-term mobile phone use (minimum, 10 years) and glioma risk, suggesting a possible dose-response relationship. There was also an association between mobile phone use and low-grade glioma in subgroups with regular or long-term use.

A significant positive association was found between long-term ipsilateral mobile phone use and the risk of glioma. Contralateral mobile phone use was not associated with glioma, regardless of the duration of use.

These findings suggest a possible role of cumulative exposure and regional localization in a potential underlying biological mechanism linking mobile phone use with glioma.

Limited data supports ipsi/contralateral mobile phone use with glioma. Larger and longer studies are required to better characterize this possible link.

However, current evidence is of poor quality and limited quantity. It is therefore necessary to conduct high quality research with large samples in order to better characterize any potential association between long-term ipsilateral mobile phone use and glioma risk.

There is no evidence to suggest that long-term mobile phone use is associated with high-grade glioma, mainly because of the biological differences between low-grade glioma and high grade-glioma.

Low-grade glioma (WHO grade II tumor) is associated with a long latency period. Users are thus potentially more vulnerable to the radiation from mobile phones which in itself may be a chronic carcinogenic agent.

Cases affected by radiation are more likely to be detected. Relative to low-grade glioma, **high-grade disease (WHO grade III tumor)** has a short latency period. However, most secondary gliomas initially progress from low-grade ones.

Primary glioblastoma progresses quickly and has both a short latency period and disease course. These factors may affect analysis for any association between mobile phone use and high-grade glioma.

In any case, these results supporting a potential association between mobile phone use and glioma risk need to be interpreted with caution secondary to marked between-trial heterogeneity. The observed correlation between long-term use of mobile phone and glioma risk may be artificial, and secondary to systematic differences in response rates between cases and controls.

In conclusion, using meta-analysis, there was no association between any duration of mobile phone use and an increased risk of glioma, but there was an association among long-term users of ≥10 years.

Furthermore, researchers observed a correlation between the risk of mobile phone use and low-grade glioma in particular, especially among long-term users. However, the findings require confirmation with prospective, longer-term studies.

Source: PLoS ONE, Vol. 12, Issue 5, e0175136, May 2017.

EU Recognizes Bisphenol A as an Endocrine Disruptor

The European Chemicals Agency (ECHA) has classified Bisphenol A as an endocrine disruptor and a 'substance of very high concern (SVHC)'.

On 16 June 2017, ECHA's member state committee, made up of representatives from all 28 EU countries, unanimously agreed to additionally identify Bisphenol A as a substance of very high concern because of its endocrine disrupting properties which cause "probable serious effects to human health which give rise to an equivalent level of concern to carcinogenic, mutagenic, toxic to reproduction substances".

The substance 4,4'-isopropylidene-diphenol (**Bisphenol A**) has a harmonised classification for the hazard class Reproductive toxicity category 1B ('may damage fertility'), based on its effects on reproductive function.

Bisphenol A has been identified recently as SVHC, according to Article 57(c) of REACH [(c) substances meeting the criteria for classification as toxic for reproduction category 1 or 2 in accordance with Dangerous Substances Directive (67/548/EEC)], and was included in the Candidate List by decision of ECHA ED/01/2017 of 4 January 2017.

In itself, this decision is nothing new. France banned Bisphenol A in baby bottles in 2010 and in food containers in 2012. The substance is widely used in the plastic coatings inside aluminium food cans, it is also present in plastic straws, many plastic toys and on the coatings of paper till receipts.

This is a double first: to begin with, it is the first time a substance has been declared highly concerning for its endocrine disrupting properties and their negative effects on human health. Secondly, it is the first time the label 'endocrine disruptor' has been placed on Bisphenol A.

Note: **The European Chemicals Agency (ECHA)** is the driving force among regulatory authorities in implementing the EU's groundbreaking chemicals legislation for the benefit of human health and the environment as well as for innovation and competitiveness. ECHA helps companies to comply with the legislation, advances the safe use of chemicals, provides information on chemicals and addresses chemicals of concern.

Source: ECHA News, 16 June 2017 (ECHA/PR/17/12) - https://echa.europa.eu/-/msc-unanimously-agrees-that-bisphenol-a-is-an-endocrine-disruptor.

Associations between Ambient Fine Particulate Air Pollution and Hypertension in China

High blood pressure (BP) is a well-established risk factor for cardiovascular morbidity and mortality. It was evidenced that high BP was a leading single risk factor for the global burden of diseases. Two-thirds of adults with hypertension live in developing countries.

Epidemiological studies have reported that short-term exposure to air pollution in the form of fine particulate matter ($PM_{2.5}$) is significantly associated with hypertension and BP variations. However, limited evidence is available regarding its long-term effects on hypertension in developing countries.

China is a developing country with severe air pollution problems. Several national surveys have revealed that hypertension prevalence was rapidly increasing and the number of hypertension patients was estimated to exceed 300 million by 2025.

Thus, it is of great public health importance to explore the long-term health effects of $PM_{2.5}$ on BP in China.

This cross-sectional, nationally representative survey (13,975 participants) was conducted in China from June 2011 to March 2012 to study the association between long-term exposure to $PM_{2.5}$, hypertension prevalence and blood pressure (BP).

The survey identified 5,715 cases of hypertension, about 40.9% of the study population.

The annual mean exposure to $PM_{2.5}$ varied greatly among study participants with the mean of residential $PM_{2.5}$ exposure as 72.8 $\mu g/m^3$, which is much higher than 10 $\mu g/m^3$ of the Air Quality Guidelines issued by the World Health Organization (WHO, 2006).

This study demonstrated that long-term exposure to ambient $PM_{2.5}$ was significantly associated with an increased prevalence of hypertension and slightly elevated levels of systolic BP in China.

In addition, the effects of $PM_{2.5}$ on hypertension were found to be particularly strong among middle-aged, obese and urban residents.

This is the first nationwide study in China to explore the long-term effects of air pollution on hypertension and BP.

Source: Science of The Total Environment, Vol. 584–585, Pages 869-874, April 2017.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute
Schedule for 2017 - 2018

	Training Course	Date	Duration	Closing Date
1.	Environmental and Health Risk Assessment and Management of Toxic Chemicals	December 5-19, 2017	2 weeks	October 20, 2017
2.	Detection of Environmental Pollutants, Testing and Screening of Toxicity	February 2018	9 work days	December 15, 2017
3	Environmental Toxicology	May 2018	10 work days	February 28, 2018

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

Course Description:

Environmental and Health Risk Assessment and Management of Toxic Chemicals (December 5-19, 2017)

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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please visit - http://www.cri.or.th/en/ac_actcalendar.php

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