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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".

Prenatal Exposure to Outdoor Air Pollution and Childhood Allergic Diseases

Asthma and allergy are the most common diseases and the major health issues in children worldwide because they lead to a heavy economic burden and poor quality of life.

Although it is largely accepted that gene-environment interactions contribute to the development of asthma and allergies, the changing environment is likely responsible for the rapid increase in the prevalence.

Mounting evidence suggests that exposure to ambient air pollution is associated with the development of childhood allergic diseases. However, the effect of prenatal exposure to air pollution on the risk of childhood asthma and allergy is unclear.

Emerging evidence shows that exposure to air pollution during pregnancy is associated with elevated risk of adverse birth outcomes which may have long-term health implications. These can include the child's increased susceptibility to disease in later life.

Early childhood allergic diseases are even being linked with trimester-specific exposure during pregnancy.

Some recent studies have highlighted that exposure to air pollution during pregnancy is a potential stimulus for the early programming of asthma and allergy. For example, prenatal exposure to air pollution is now seen to be significantly associated with asthma and allergic diseases in preschool children in China.

Despite the importance of early-life exposure in the development of allergy, information on the effects of air pollution exposure, particularly during pregnancy, on allergic diseases early in life has rarely been assessed.

The scarce epidemiological evidence on the effect of prenatal exposure to air pollution on the risk of childhood asthma and allergic diseases does warrant further investigation.

In this study, the researchers hypothesize the fetal origins of childhood allergies and speculate these diseases may originate during a specific trimester of pregnancy, triggered by traffic-related air pollution.

The researchers evaluated the association between maternal exposure to outdoor air pollution during different trimesters of pregnancy and lifetime incidence of asthma, allergic rhinitis, and eczema in 2598 preschool children, aged 3-6 years in China.

The results showed that incidence of asthma, allergic rhinitis, and eczema in children was associated with maternal exposure to traffic-related pollutant NO_2 throughout pregnancy. After adjustment for other pollutants and trimesters, the researchers found the association was significant only in specific trimesters: the first trimester for eczema, the second trimester for asthma, and the third trimester for allergic rhinitis.

Although several recent studies found that early childhood allergic diseases were associated with trimester-specific exposure during pregnancy, this is the first study to systematically investigate the effect on

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Prenatal Exposure to Outdoor Air Pollution and Childhood Allergic Diseases

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childhood allergy of prenatal exposure to outdoor air pollution during different trimesters.

Sensitivity analysis indicated that the association between exposure to traffic-related air pollutant NO_2 during trimesters of pregnancy and childhood allergic diseases was stable.

Parental and residential factors had only significant effects in sensitive trimesters.

It is becoming clear that a family history of allergic diseases, or parental atopy, is associated with an increased risk of atopic diseases in the offspring. This suggests that some genetic markers could impose susceptibility in childhood to environmental factors.

Maternal exposure to traffic-related air pollutant NO_2 during pregnancy, especially in specific trimesters, is associated with an increased risk of developing asthma, rhinitis, and eczema in preschool children.

The results support the hypothesis that childhood allergic diseases originate in fetal life and are triggered by traffic-related air pollution in sensitive trimesters. However, they cannot separate the effects of prenatal and postnatal exposure.

These findings may contribute to the development of more effective prevention and intervention measures to help children avoid allergic diseases. The key components are air pollution and the critical exposure window. However, further investigations are needed to identify the relevant mechanism(s) underlying the association between exposure to air pollution during the specific trimester *in utero* and childhood allergic diseases.

Source: Environmental Research, Vol. 150, Pages 119–127, October 2016.

COFFEE GROUNDS COULD CLEAN UP LEAD AND MERCURY-CONTAMINATED WATERS

Coffee is one of the most commonly consumed beverages in the world, which makes spent coffee grounds one of the most frequently tossed out waste products (worldwide, ~6 million tons yearly) from domestic and restaurant consumption and also from coffee manufacturing industries. Converting this waste into a useful resource could decrease the burden of waste management.

Due to its functional chemical composition (contains fatty acids, lignin, cellulose, hemicellulose, polyphenols, etc.), spent coffee has been reutilized as fertilizer, animal feedstuff, biodiesel fuel, and metal ion adsorbent. In the latter case, researchers are focused on the appropriate functionalization and utilization of spent coffee grounds in water remediation, mostly for the removal of heavy metal ions.

Although these studies show the efficient use of spent coffee grounds for water remediation, collection of the grounds after the remediation process is costly and cumbersome, requiring further post-processing such as centrifugation and filtration of the segregated particles.

These researchers report a novel approach to utilize spent coffee grounds as active filler for the formation of bioelastomeric composite foams, composed of 60 wt % of spent coffee grounds and 40 wt % of silicone elastomer using sugar leaching technique.

The researchers prove that the prepared foam is able to efficiently remove lead (Pb^{2+}) and mercury (Hg^{2+}) from water, even in the presence of other metal ions. In still water, the foam removed up to 99 percent of lead and mercury ions over a period of 30 hours. But people can't always wait for 30 hours. In a more practical test in which lead-contaminated water flowed through the foam, the water was made free of up to 67 percent of any lead ions.

According to studies published for spent coffee grounds, such bioelastomeric foams have improved their performance. Of critical importance is the fact that they can be easily handled and disposed of. One of the proposed applications is to use these grounds as thin filters for a continuous-flow process to clean contaminated waters.

In conclusion, the present study demonstrates the formation of spent coffee based bioelastomeric foams by a straightforward method, and their successful use for the removal of heavy metal ions from water. The coffee loading is performed during the fabrication of the foam, without specific reaction conditions.

The materials showed high adsorption capacities of Pb^{2+} and Hg^{2+} ions. The adsorption mechanisms of the Pb^{2+} ions in the bioelastomeric foam were identified, revealing the interaction of the metal ions with the carboxylate groups of the coffee filler.

In addition, the study demonstrates the removal of the above-mentioned metal ions from wastewater with six metal ions present. The present foams demonstrate improved performance, compared to earlier studies, with a remarkable adsorption capacity.

Furtheremore, these materials can be easily handled and disposed of, and can be employed as filters for continuous-flow metal ions adsorption, enabling new methods for the reutilization and valorization of this particular waste.

Source: ACS Sustainable Chemistry & Engineering, Vol. 4, No. 10, Pages 5495–5502, September 2016.

ASSOCIATION OF FOOD CONSUMPTION DURING PREGNANCY WITH MERCURY AND LEAD LEVELS IN CORD BLOOD

Mercury and lead are two widespread environmental contaminants. In utero exposure to these toxic metals has been linked to various adverse health effects related to growth and development. These effects include preterm delivery, low birth weight, growth delay, and negative impacts on neurodevelopment, with problems in areas of cognition, behavior, and school performance.

The fetus may be vulnerable to the adverse effects of these metals because mercury and lead in maternal body can easily enter the placenta.

Because of rapid cell division and differentiation in the fetus, even relatively low levels of *in utero* exposures to mercury and lead may cause serious influence on fetuses or infants, even at levels that wouldn't harm their mothers.

Food consumption is known to be a major exposure source for mercury and lead. Mercury is particularly pervasive, entering the food chain from both natural and anthropogenic sources.

However, there has been no evidence as to the relationship between food consumption during pregnancy and levels of mercury or lead in cord blood.

Safe thresholds for levels of mercury or lead during pregnancy period have never been established, although recommendations have been made for adults.

The present study was conducted to measure levels of mercury and lead in the blood and urine of pregnant Korean women and in cord blood, and to evaluate the relationship between food consumption and levels of mercury and lead in bio-specimens.

Researchers also tried to identify dietary sources which pregnant women might be exposed to these toxic metals.

In this study, the mercury level detected in the blood and urine of pregnant women and in cord blood was generally higher than those reported in western countries and the US, and lower than in Japan and Taiwan, which are characterized by their longer coastlines.

These results show the importance of tight management of mercury where pregnant women are concerned.

Regarding lead, the levels in maternal and cord blood in the present study were much lower when compared to 50 μg/L, the reference value proposed by the Centers for Disease Control and Prevention (2013) and also lower than in other studies, including a study in Arctic Canada.

However, lead exposure at the current level *in utero* was found to be associated with some negative health outcomes of children after birth, and thus warrants further investigation.

The biological half-lives of mercury and lead range from 1.5 to 2.5 months, both in blood and urine. However, mercury and lead levels in urine cannot be used well as exposure markers because of great variability. Also, mercury accumulates in the kidneys and has greater risk of external lead contamination during sampling.

In spite of unstable urinary levels of mercury and lead, mercury and lead levels in the present study were highly correlated among media, particularly for mercury.

This indicates that both mercury and lead in the maternal body can be easily transferred to cord blood, leading to adverse health outcomes for the fetus.

Because of the small number of the maternal blood samples and the instability of urinary levels of mercury and lead as exposure markers, researchers estimate the relationship between food consumption and mercury and lead levels in cord blood.

Fish consumption was positively associated with mercury level in cord blood, while cereal and vegetable consumption was positively associated with lead levels in cord blood.

Since fish consumption has been a major contributor for exposure to mercury for most human populations, proper fish consumption should be recommended for a balancing between harmful effects of mercury and health benefits of the omega-3 polyunsaturated fatty acids and essential minerals contained in fishes for the development of the central nervous system of fetus during pregnancy.

Furthermore, the consumption of tea restrained increases of lead levels in cord blood. The decline in lead levels in cord blood associted with tea consumption in this study was consistent with observed protective effects of green tea on blood lead levels in rats.

In summary, perinatal mercury and lead levels were increased with fish consumption, and cereals or vegetable consumption, respectively. However, the consumption of tea restrained increases of lead.

Mercury and lead exposures in the early stages of life warrant further investigation. Intervention is warranted to protect susceptible populations such as pregnant women and their unborn children.

Future studies using different time windows or other media such as hair can present cumulative exposures with the large sample sizes needed to confirm this study.

Source: Science of the Total Environment, Vol. 563–564, Pages 118–124, September 2016.

Environmental Chemicals Impact Dog Semen Quality *In Vitro*

Dogs and humans may both be impacted by hormone disrupting chemicals in the environment.

A significant decline in human semen quality over the last 70 years has been widely reported. Declining sperm counts linked with epidemiological data on increased incidences of testicular cancer and genital tract abnormalities, are indicative of an adverse environmental effect on male reproduction.

Since these reproductive problems, termed as "testicular dysgenesis syndrome" (TDS), cluster in geographical areas, they are thought to have a common aetiology and have been associated with endocrine perturbations in early life.

Exposure of developing males to environmental chemicals (ECs), particularly those with endocrine disrupting activity, is thought to be the initiator.

As 'man's best friend' and closest animal companion, the dog shares the same environment, exhibits the same range of diseases, many with the same frequency, and responds in a similar way to therapeutic treatments.

There is evidence that over the last 40 years, the incidence rate of canine testicular cancer has increased in parallel with changes seen in humans.

In addition, histological signs which characterise human TDS have recently been described in the dog. These include seminiferous tubule abnormalities and testicular germ cell neoplasia *in situ* (GCNIS) cells which are known precursors of seminomas in the human.

Since human TDS includes a reduction in sperm count and an increased incidence of cryptorchidism, the researchers hypothesised that the dog may exhibit similar manifestations of TDS and that this may be associated with exposure to endocrine disrupting chemicals.

The study therefore monitored canine semen quality over a period of 26 years and interrogated an extensive database of electronic health records for evidence of a temporal change in the incidence of cryptorchidism reported in male puppies.

To explore a possible environmental aetiology, concentrations of chemical pollutants were measured in canine testes and semen collected from the same geographical area in which the temporal study had been carried out. The effects of chemicals, at testicular concentrations, were tested on endocrine and sperm function *in vitro.*

The results showed that a population of breeding dogs exhibit a 26 year (1988–2014) decline in sperm quality and a concurrently increased incidence of cryptorchidism in male offspring (1995–2014).

A decline in the number of males born relative to the number of females was also observed.

ECs, including diethylhexyl phthalate (DEHP) and polychlorinated biphenyl 153 (PCB153), were detected in adult dog testes and commercial dog foods at concentrations reported to perturb reproductive function in other species.

Testicular concentrations of DEHP and PCB153 perturbed sperm viability, motility and DNA integrity *in vitro* but did not affect luteinizing hormone (LH) stimulated testosterone secretion from adult testis explants.

Although the mechanism remains to be determined, the researchers have shown that chemicals present in testis and ejaculate directly affect sperm function and viability.

Since the increased incidence of cryptorchidism coupled with declining sperm quality in males is indicative of canine "testicular dysgenesis syndrome (TDS)", the domestic dog may be a useful sentinel for the study of environmental influences on human male fertility.

Source: Scientific Reports, Vol. 6, Article Number 31281, August 2016.

Long-Term Exposure to $PM_{2.5}$ and Kidney Function in Older Men

Chronic exposure to ambient fine particulate matter ($PM_{2.5}$) is a well-known risk factor for cardiovascular-related morbidity and mortality. There is evidence suggesting that pathways at the molecular level, including inflammation and oxidative stress, and at the function level, including arterial blood pressure (BP) and vascular/endothelial function, may have a role in $PM_{2.5}$-related cardiovascular morbidity and mortality.

It is also hypothesized that renal function impairment may be a mediating factor in the cardiovascular effects of long-term $PM_{2.5}$ exposure because the kidney is a vascularized organ susceptible to large-vessel atherosclerotic disease and microvascular dysfunction.

Impaired renal function, as determined from the estimated glomerular filtration rate (eGFR), is also associated with cardiovascular events and mortality.

There is limited experimental evidence suggesting that particle exposure affects the kidney; *in vivo* studies in rats have shown controlled exposure to urban or diesel exhaust particles to be associated with increased cytokine expression in the kidney and to aggravate acute renal failure.

A recent cross-sectional study of stroke patients in the Boston metropolitan area found that living near a major roadway was associated with reduced eGFR.

To date, no longitudinal population study has directly evaluated whether long-term $PM_{2.5}$ exposure is associated with reduced renal function or with increased age-related decline in renal function.

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Neurodegenerative and Neurological Disorders by Small Inhaled Particles

The exposure to ambient particulate matter (PM) has been associated with lung inflammation, asthma, and fibrosis and cardiovascular disorders.

Observations that exposure to PM is linked with neuroinflammation indicate that the brain is among the extra-pulmonary targets of PM.

During the last decade several research groups have postulated that exposure to air pollution, especially PM, could be an important environmental risk factor for neurotoxicity and more specifically may potentiate the risk of neurodevelopmental and neuro-degenerative disorders.

The world's population is steadily aging. As a result, age related diseases, such as dementia, have become a major public health concern in the first world countries. In 2001, it was estimated that there were almost 5 million Europeans suffering from Alzheimer's disease (AD), and this figure is projected to almost double by the year 2040.

AD and Parkinson's disease (PD) represent the two most common progressive neurodegenerative diseases. Age is considered to be the major risk factor for both diseases and with the continuous rise in life expectancy, the number of patients is expected to increase steeply in coming years.

About 40% of people over the age of 85 suffer from AD and another 10% from PD. The vast majority of AD and PD cases are sporadic in nature with no clear involvement of inheritable genetic mutations.

Pesticides, metals, and ambient PM are among the environmental factors that have been seen as possible factors in the degenerative conditions.

This comprehensive review aims to assess the biological plausibility of the relationship between neurological effects and exposure to inhaled ambient particles.

When assessing the biological effects of environmental factors, it is important to consider possible exposure routes and evidence for actual exposure. This is particularly important in cases where compounds affect multiple systems and induce a variety of effects.

Inhalable ambient PM is a complex air-suspended mixture of particles which vary in particle number, size, surface area and chemical composition. The mixture comprises substances that are directly emitted into the air (automotive exhaust, industrial emissions, etc.) or are generated in chemical reactions.

PM is roughly classified, based on aerodynamic diameter, into PM_{10} (median particle diameter 10 μm), $PM_{2.5}$ (median particlediameter 2.5 μm) and $PM_{0.1}$ or ultrafine PM (UFPM; median particle diameter 0.1 μm).

Epidemiological research has identified airborne PM as one of the environmental factors potentially involved in AD and PD pathogenesis. Also, there is mounting evidence that the smallest inhalable ambient particulate matter, the ultrafine or nano-sized particles, are capable of inducing effects beyond the respiratory system.

Based on epidemiological, as well as *in vitro* and *in vivo* studies it is becoming increasingly clear that inhalable PM may pose a threat to the brain. The threats are to the developing brain and to the ageing brain, as well. Some of the neurodegenerative effects may be seen in AD.

Translocation of very small particles via the olfactory epithelium in the nose or via uptake into the circulation has been demonstrated through experimental rodent studies with engineered nanoparticles.

Outdoor air pollution has been linked to several health effects, including oxidative stress and neuroinflammation that may ultimately result in neurodegeneration and cognitive impairment.

Accumulating evidence indicates that it is plausible that (chronic) exposure to inhalable PM plays a role in the pathogenesis/pathophysiology of neurodegenerative diseases.

As transport of inhalable material to the brain parenchyma has been demonstrated. Hence, direct effects on the brain are plausible.

However, considering exposure levels, the current paucity on particle specific translocation kinetics, and the plethora of systemic effects that are already known to influence the central nervous system pathology, it is hard to tell which route provides the strongest link.

Moreover, species differences in respiratory tract morphology and physiology require careful consideration when assessing the potential effects of inhalable particles on the central nervous system.

Direct and indirect mechanisms could act together in an additive or even synergistic manner.

Future experiments will be needed to unravel the mechanism(s) of particle-induced neurotoxicity and to identify which components of inhalable PM are the major contributors to the central nervous system pathology.

Source: NeuroToxicology, Vol. 56, Pages 94–106, September 2016..

WHO Releases Country Estimates on Air Pollution Exposure and Health Impact

Air pollution has become a growing concern in the past few years, with an increasing number of acute air pollution episodes in many cities worldwide. As a result, data on air quality is becoming increasingly available and the science underlying related health impacts is also evolving rapidly.

To date, air pollution [both ambient (outdoor) and household (indoor)] is the biggest environmental risk to health, carrying responsibility for about one in every nine deaths annually.

Ambient (outdoor) air pollution alone kills around 3 million people each year, mainly from noncommunicable diseases. Only one person in ten lives in a city that complies with the WHO Air quality guidelines. Air pollution continues to rise at an alarming rate. Economies and people's quality of life are seriously affected, it is a public health emergency.

A new **WHO air quality model** confirms that 92% of the world's population lives in places where the amount of particulate "WHO's Ambient Air quality guidelines". WHO guideline limits for annual mean of $PM_{2.5}$ are 10 µg/m^3 annual mean.

$PM_{2.5}$ includes pollutants such as sulfate, nitrates and black carbon, which penetrate deep into the lungs and the cardiovascular system, posing the greatest risks to human health.

Information in the WHO air quality model is presented via **interactive maps (http://maps.who.int/airpollution/)**, highlighting areas within countries where PM pollution exceed WHO limits.

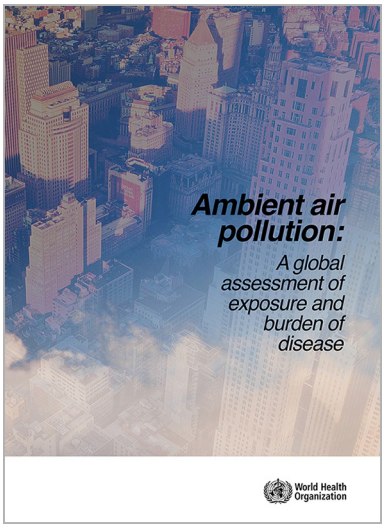
It also represents the most detailed outdoor (or ambient) air pollution-related health data, by country, ever reported by WHO. The model is based on data derived from satellite measurements, air transport models and ground station monitors for more than 3,000 locations, both rural and urban. It was developed by WHO in collaboration with the University of Bath, United Kingdom.

The model has carefully calibrated data from satellite and ground stations to maximize reliability. National air pollution exposures were analysed against population and air pollution levels at a grid resolution of about 10 km x 10 km.

This new model is a big step forward towards even more confident estimates of the huge global burden of more than 6 million deaths (1 in 9 of total global deaths) from exposure to indoor and outdoor air pollution. More and more cities are monitoring air pollution now, satellite data is more comprehensive, and we are getting better at refining the related health estimates.

The full report on "**Ambient air pollution: A global assessment of exposure and burden of disease**"can be downloaded from WHO Website at http://apps.who.int/phe/publications/air-pollution-global-assessment/en/index.html.

The report presents a summary of methods and results of the latest World Health Organization (WHO) global assessment of ambient air pollution exposure and the resulting burden of disease.



Source: News Release from WHO on September 27, 2016 at http://www.who.int/mediacentre/news/releases/2016/air-pollution-estimates/en/

Long-Term Exposure to $PM_{2.5}$ and Kidney Function in Older Men

(Continued from page 4)

The present study investigated whether exposure of 1-year averaged $PM_{2.5}$ concentrations was associated with reduced renal function and with renal function decline over time in an ongoing prospective cohort study of older men living in the Boston metropolitan area.

The subjects included 669 participants from the Veterans Administration Normative Aging Study with up to four visits between 2000 and 2011.

Serum creatinine was measured at each visit, and eGFR was calculated as a measure of renal function. One-year's exposure to $PM_{2.5}$ prior to each visit was assessed using a validated spatio-temporal model that utilized satellite remote-sensing aerosol optical depth data.

Participants' median 1-year $PM_{2.5}$ exposure levels ranged from approximately 7.5 to 12.5 µg/m^3. By comparison, the primary National Ambient Air Quality Standard for 1 year is 12 µg/m^3 averaged over 3 years. This standard is designed to be protective of all groups of people, including the elderly.

For this particular population, the researchers estimated that a 2.1-µg/m^3 increase in $PM_{2.5}$ over a 1-year period was associated with a reduction in eGFR comparable to that seen with a 2-year increase in age in the same men.

In summary, the researchers found that long-term $PM_{2.5}$ exposure was associated with reduced eGFR and increased age-related eGFR decline in this longitudinal sample of predominantly older White men.

These findings support the hypothesis that long-term $PM_{2.5}$ exposure negatively affects renal function and renal function decline. These findings should be verified in other study populations with longitudinal follow-up.

Source: Environmental Health Perspectives, Vol. 124, No. 9, Pages 1353-1360, September 2016.

The Chem HelpDesk

"Strengthening capabilities for sound chemicals management"

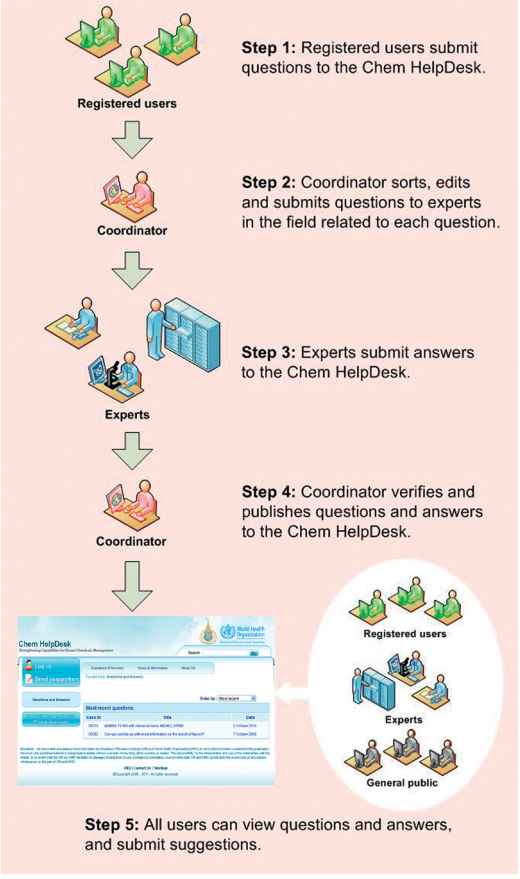
The Regional HelpDesk for Chemical Safety, or **Chem HelpDesk** was established as a joint-initiative between WHO SEARO and CRI, through the WHO Collaborating Center for Capacity Building and Research in Environmental Health Science and Toxicology. The aims of the Chem HelpDesk are to address the issue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

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

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

For more information, please visit: http://www.chemhelpdesk.org
or e-mail: coordinator@chemhelpdesk.org

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



IARC Announced the Carcinogenicity of Some Industrial Chemicals

In February 2016, the International Agency for Research on Cancer (IARC) brought together 24 experts from 8 countries to evaluate the carcinogenicity of ***seven industrial chemicals.***

The following chemicals are classified as ***probably carcinogenic to humans (Group 2A)***, based on "limited evidence in humans" and "sufficient evidence of carcinogenicity in experimental animals"

1. ***N,N*-Dimethylformamide** (a solvent) causes testicular cancer
2. **2-Mercaptobenzothiazole** (found in rubber products) causes urinary bladder cancer
3. **Hydrazine** (used in rocket propellant and aircraft fuel) causes lung cancer
4. **Tetrabromobisphenol A** (a flame retardant) with strong mechanistic evidence

The following chemicals are classified as ***possibly carcinogenic to humans (Group 2B)***, based on "sufficient evidence of carcinogenicity in experimental animals"

1. **1-Bromopropane** (a solvent used in chemical manufacturing, spray adhesives, vapour degreasing, and dry cleaning)
2. **3-Chloro-2-methylpropene** (used in chemical industry and as a seed fumigant)
3. ***N,N*-Dimethyl-*p*-toluidine** (used in dental materials, bone cement and nail polish)

These assessments will be published as volume 115 of the IARC Monographs *(in preparation)*.

Source: The Lancet Oncology, Vol. 17, Pages 419-420, April 2016.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute

Year 2016-2017

	Training Course	Date	Duration	Closing Date
1.	Environmental and Health Risk Assessment and Management of Toxic Chemicals	November 26 - December 13, 2016	2 weeks	October 15, 2016
2	Principles of Toxicology, Toxicity Testing and Safety Evaluation	February 7-22, 2017	2 weeks	December 15, 2016
3	Environmental Toxicology	April 2017	1+ weeks	February 15, 2017
4	Occupational and Environmental Health	August 2017	2 weeks	June 15, 2017

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

Course Description:

1. **Environmental and Health Risk Assessment and Management of Toxic Chemicals (November 26 - December 13, 2016)**

The course is an integration of science and policy, covering the fundamental basis of environmental and health risk assessment and management from exposure assessment and risk characterization; mode of action and human relevance framework; the relationship between risk assessment and risk management; and the need for open, transparent and participatory acceptance procedures and credible communication methods. Emphasis will be placed on human health risk assessment, although the principles of ecological risk assessment will also be covered. Importantly, 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 an electronic distance learning tool on risk assessment and risk management of chemicals and the WHO IPCS Human Health Risk Assessment toolkit, will be introduced.

Requirement: Participants should have jobs/responsibilities related to assessment of risk from the use of chemicals.

2. **Principles of Toxicology, Toxicity Testing and Safety Evaluation (February 7-22, 2017)**

This course presents the fundamental and basic concepts of toxicology, including dose-response relationships; types of harmful effects; mechanisms involved in chemical actions from the entrance of chemicals into the body until excretion; toxicokinetics; activation and detoxification mechanisms; biologic and chemical factors that influence toxicity and the principles of testing for toxic effects.

Requirement: Participants should have basic knowledge in chemistry and biological or 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

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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