CRI/ICEIT
NEWSLETTER

VOL. 29 NO. 4 – October 2019
ISSN 0858-2793
BANGKOK, THAILAND

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".

Organochlorine Pesticide Exposure as a Risk Factor for Breast Cancer in Young Indian Women

Breast cancer incidence in India has been increasing steadily during the last 30 years. Currently, it is the most common cancer among Indian women, with an age-adjusted incidence rate of 25.8/100,000 women.

Younger women (≤40 years) constitute about 11%-31% of all breast cancer cases in India. Higher rates of breast cancer in younger women have also been reported in other South Asian countries.

The risk factor profile for younger Indian women is different from their Western counterparts because early first childbirth, multiparity, and breastfeeding are social norms in India, and the use of oral contraceptives and hormone replacement therapy is low.

Inherited/familial breast cancer accounts for 5%-10% of cases. Rising rates of breast cancer in India are being attributed to westernization of lifestyles. At the same time, however, the Western population with the same risk factors do not have similar onset at younger ages. Hence, environmental factors may have a role which is yet to be explored.

Environmental pollution due to the use of pesticides reportly plays a role in increasing the risk for various cancers. Among these, estrogenic-organochlorine compound pesticides have been evaluated for their potential role as a risk factor for breast cancer. However, the possible role of these pesticides in increasing breast cancer risk in young women has not been explored.

Organochlorine pesticides (OCPs) are synthetic pesticides widely used worldwide. They are known for their high toxicity, slow degradation, and bioaccumulation. Even though many of these compounds have been banned in developed countries since 1970s, insecticides such as DDT, HCH, aldrin, and dieldrin are still in use in developing countries in Asia.

These pesticides cause neurological damage and endocrine disorders and are reported to increase the risk of hormone-related cancers including breast, prostate, stomach, and lung cancers. Furthermore, individuals in younger age groups are reported to face higher risks of health hazards due to pesticides than those in higher age groups.

The present study found statistically significant association between exposure to OCPs and risk of breast carcinoma in young women. Significantly higher blood levels of α-, β-, γ-HCH; heptachlor; DDE; endosulfan-1; dieldrin; and endosulfan-2 were found in these breast cancer patients.

However, the odds ratio for breast cancer were found to be significant only with β-HCH, heptachlor, DDE, and dieldrin.

This is probably because exposure to OCPs is low in these populations overall, and hence can not be confirmed as a significant breast cancer risk.

However, exposure to OCPs should be monitored as possibly contributing to an increasing incidence of breast cancer in younger women in India.

Source: South Asian Journal of Cancer. Vol. 8, Issue 4, Pages 212-214, October-December 2019.

Early-life Exposome and Lung Function in Children in Europe

The developmental period of a human, in both prenatal and early postnatal life, is likely to be particularly susceptible to environmental hazards. Exposures during this period could permanently affect body structure, physiology, and metabolism, leading to long-term health effects.

There is a large body of epidemiological literature on the effects of early-life exposures on respiratory health in children, but few studies have investigated several families of exposure, or applied an exposome approach.

The exposome, a concept defined as encompassing all environmental exposures from conception onwards, offers a new paradigm in environmental health research.

By simultaneously considering a large set of exposures, the exposome approach can overcome the limitations of focusing on a single exposure or family of exposures in the environmental studies done so far.

Specifically, exposome studies limit the risk of selective reporting (i.e, testing many exposures and only reporting the most significant associations). By contrast, exposome studies allow explicit reporting of multiple testing as well as correction for possible confounding by co-exposures.

The present study aimed to evaluate the association between a broad range of prenatal and postnatal lifestyle and environmental exposures and lung function in children in the large European Human Early-Life Exposome (HELIX) cohort (consisting of six existing longitudinal birth cohorts in France, Greece, Lithuania, Norway, Spain, and the UK of children born between 2003 and 2009) for whom a valid spirometry test was recorded for each child.

This is the first study to address the effect of the exposome on lung function in children by considering a broad range of prenatal and postnatal environmental factors.

The study strengthened the evidence for the contribution of chemical exposures (phenols, phthalate metabolites, and perfluoroalkyl substances) to the impairment of lung function development and highlights the need for larger prospective studies.

The results suggest that prenatal exposure to PFASs (perfluorononanoate and perfluorooctanoate) and postnatal exposure to copper, ethyl-paraben, DEHP (di-ethylhexylphthalate) and DINP (di-iso-nonylphthalate) metabolites, and house crowding are associated with a lower FEV_1 (forced expiratory volume in 1 second) in childhood.

Regarding chemical exposures, the findings showed associations between lung function in childhood and prenatal exposures to perfluorooctanoate and perfluorononanoate, which are ubiquitous in the synthetic fluorinated compounds typically used as stain repellents because of their surfactant properties and hydrophobicity.

The majority of exposure to PFASs can be attributed to diet, *in utero* exposure through placental transfer, and breastfeeding.

The findings are in line with experimental studies reporting the immunosuppressive effects of PFASs, and animal models showing impaired lung development associated with exposure to PFASs, more specifically, induced airway inflammation and altered airway function, possibly through oxidative stress mechanisms.

The strongest exposure-FEV1% associations identified in the present study were with postnatal exposure to DEHP metabolites (MECPP, MEHHP, MEOHP, and sum of DEHP metabolites), and one DINP metabolite (OXOMiNP), which are mainly used as plasticisers and can be either ingested, inhaled, or absorbed through dermal contact.

Several of these phthalates have been highlighted in the literature for their negative association with FEV_1 in adults and in children, and with other respiratory outcomes in children, including when exposed prenatally.

DEHP metabolites are suspected to affect the respiratory system, especially in the paediatric age group, possibly by promoting immunological and inflammatory mediators.

The findings for DINP are of particular public health importance because the use of DINP is increasing in Europe as a substitute to DEHP, and it is now one of the most commonly used plasticisers.

Regarding phenols, the study showed an inverse association with postnatal ethyl-paraben exposure, a compound used as a preservative in cosmetics; this is in line with a previous observation of an association between ethyl-paraben and FEV_1 at age 5 years in boys from the previous cohort, although for prenatal instead of postnatal exposure.

This study covered many more exposures related to the urban and outdoor environment than most previous epidemiological studies. Of these new indicators, only facility density around schools was associated with a lower FEV_1.

Greater facility density or diversity is expected to create a more walkable environment, but could also be related to higher exposure to hazardous factors in the urban environment, such as air pollution, noise, and reduced green space, which could explain our association with a lower FEV_1.

In conclusion, this study is the first to use a comprehensive exposome approach to report that specific prenatal and postnatal chemical exposures (phenols, phthalate metabolites, perfluoroalkyl substances) might be associated with the impairment of lung function development.

These findings have important public health implications because they suggest that preventive measures aimed at lowering exposure to the identified ubiquitous chemicals, through stricter regulation and informing the public by labelling these chemicals in consumer products, could help to prevent lung function impairment, which in turn should prevent the development of chronic respiratory disease in adulthood.

Source: The Lancet Planetary Health, February 2019. (https://doi.org/10.1016/S2542-5196(19)30010-5)

In Utero Exposure to Bisphenol-A Disrupts Key Elements of Retinoid System in Male Mice Offspring

Bisphenol-A (BPA), an endocrine disrupting chemical (EDC), is used in the production of polycarbonate plastic and epoxy resins. Consequently, it is present in many consumer products including food containers, water bottles, children toys, medical devices, dental sealants as well as the thermal paper of cash register receipts.

Humans are continuously exposed to BPA from different sources, diet and dermal contact being the main routes of exposure. BPA has been measured in different human biological samples, including blood, urine, human milk and placenta. Although BPA bans on products for children were associated with decreasing intake trends, those for adults were increasing and varied greatly among continents.

According to the European Chemical Agency (ECHA), BPA exposure has been related to alterations in reproduction, mammary gland development, cognitive function and metabolism.

Risks associated with BPA were considered negligible, compared with the EFSA's temporal-Tolerable Daily Intake (TDI) of 4 μg/kg bw/day. However, the EFSA also stated that the risk to humans could not be ignored, because 15.7-19.8% of pregnant women exceeded the exposure of 0.08 μg/kg bw/day which produced adverse effects on the mammary gland in rats.

The retinoid system plays an essential role in the homeostasis of physiological processes such as tissue differentiation and development, cell proliferation and apoptosis, immune response, development and organogenesis of the foetus.

The retinoid system consists of: 1) chemical molecules derived from vitamin A, known as retinoids; 2) proteins involved in their transport, biosynthesis and biotransformation and 3) nuclear receptors involved in their signalling.

Retinoids are not synthetized *de novo*, yet they are essential nutrients obtained from the diet, either from vegetables as β-carotene, or from animal sources such as retinol (REOH) and retinyl esters, including retinyl palmitate (REPA).

In addition, some retinoids have been tested as candidates for therapeutic use in diseases such as cancer, acne and metabolic alterations. The biological activity of functional retinoids, such as all-trans-retinoic acid (ATRA) and 9-cis-4-oxo-13,14-dihydro-retinoic acid (9C4O13,14DHRA), is mediated by binding to retinoic acid receptors (RARs) which are involved in the regulation of gene expression. RARs form heterodimers with retinoid X receptors (RXRs), which in turn are able to dimerise with many other nuclear receptors, including PPAR.

Alteration of key elements of the retinoid system is associated with severe, sometimes lethal, adverse developmental effects as well as a wide array of abnormalities in different organ systems.

Outcomes of interaction between BPA exposure and the retinoid system have revealed changes at the level of retinoid receptors, as well as toxic effects in the liver.

There is a data gap regarding the lasting effects on the retinoid system resulting from BPA exposure in utero. The present work has been conducted to quantify retinoid concentrations as well as gene expression of target genes of enzymes, transport proteins, receptors and signalling hormones related to the retinoid system, in the liver of mice exposed to BPA *in utero*, several weeks after they were born.

Male mice were exposed *in utero* to BPA following maternal subcutaneous doses of 0, 10 and 100 μg/kg bw/day from gestational day 9-16 and were then sacrificed at post-natal day 30 (PND30). Retinoid concentrations and gene expression of key elements involved in the retinoid system were determined in liver.

BPA increased all-trans-retinoic acid concentration and expression of *Adh1*, *Aox1* and *Cyp1a2* (biosynthesis of retinoic acid), but reduced *Mrp3* (efflux from hepatocyte to blood), increased *Bcrp* expression (biliary excretion) and changed the retinoid-dependent signalling system after reducing expression of *Rxrβ* and increasing that of *Fgf21*.

Such a modulation on the gene expression of transporters of glucuronide metabolites might be also involved in the regulation of BPA levels. Extrapolation of those results from animal studies to humans should be made with caution since location of transporters might differ between species.

Taken together, the current work showed modulations on the retinoid system at PND30 following *in utero* exposure to environmentally relevant doses of BPA, supporting its role as a biomarker for the identification of EDCs. Remarkably, the experimental animal model used in the current study exhibited metabolic abnormalities in the long-term.

Those findings occurred in animals which showed altered pancreatic function and impaired glucose metabolism during adulthood. The present information should be useful for enhancing testing methods for the identification of EDCs.

Source: Food and Chemical Toxicology, Vol. 126, Pages 142-151, April 2019.

Persistent Organic Pollutants and the Incidence of Type 2 Diabetes in 2 Cohort Studies

Associations between several persistent organic pollutants (POPs) and type 2 diabetes have been found in humans, but the relationship has rarely been investigated in the general population.

Attempts to identify novel risk factors beyond the traditional ones (such as physical inactivity, smoking and obesity) have provided increasing evidence that environmental contaminants are contributing to a rapid rise in the incidence of type 2 diabetes.

POPs have been implicated in the development of type 2 diabetes for over ten years. Typical suspects include chlorinated compounds such as polychlorinated biphenyls (PCBs), organochlorine pesticides and dioxins.

Because they are resistant to any kind of environmental degradation, POPs persist in the environment and tend to accumulate in the food chain. In many countries, most POPs were banned decades ago.

Though the mechanisms that link POP exposure to diabetes are still not fully understood, several pathways have been proposed, including endocrine-disrupting processes and mechanisms related to mitochondrial dysfunction.

The current nested case-control study examined internal exposure to polychlorinated biphenyls (PCB) and pesticides and the incidence of type 2 diabetes among participants of two population-based German cohort studies.

This study assessed the association between baseline POP concentrations and incidence of diabetes by conditional logistic regression adjusted for cohort, BMI, cholesterol, alcohol, smoking, physical activity, and parental diabetes. Effects associted with sex, obesity, parental diabetes and cohort were also examined.

In both cohorts, diabetes cases showed a notably higher BMI, a higher levels of POPs, and a higher frequency of parental diabetes.

The results also showed that PCB-138 and PCB-153 were positively associated with incident type 2 diabetes. In addition, explorative results suggested a higher odds ratio for women and for non-obese participants.

In conclusion, the results support a positive association between POP exposure and development of type 2 diabetes. However, more comprehensive longitudinal studies are needed to gain further insights into the dose-dependent effect of POPs on diabetes.

Studies using real-world populations are required that not only consider exposure to a broad range of POPs, but also to other types of diabetogenic environmental pollutants and chemicals in order to investigate the additive or even multiplicative interactions between these various substances.

In addition, there is a clear need for several agencies to become more aware of and responsive to available evidence on the environmental causes of type 2 diabetes.

Policy-makers in public institutions and private companies, citizens, clinicians, and the media need to work together more effectively to reduce the environmental burden of disease.

Source: Environment International, Vol. 129, Pages 221-228, August 2019.

Maternal Exposure to Ambient Air Pollution and the Effects on Newborn Telomere Length

In the 2016 report on Global Burden of Disease (GBD), ambient air pollution accounted for an estimated 1.6 million deaths in China and ranked fourth in terms of attributable disability-adjusted life-years.

Extensive studies are suggesting that chronic inflammation and oxidative stress experienced by pregnant women who are exposed to air pollution may be related to adverse pregnancy outcomes such as stillbirth, low birth weight, restricted fetal growth, and preterm birth.

Telomeres are non-coding repetitive sequences of DNA (TTAGGG) located at the end of chromosomes. Telomeres protect chromosomes from degradation, recombination, and end-to-end fusion.

Telomere length (TL) reflects biological aging and has been related to aging-related diseases, such as type 2 diabetes, cancer, cardiovascular disease, and all-cause mortality. Evidence indicates that telomere attrition may be accelerated by the inflammation and oxidative stress induced by environmental pollutants such as air pollution.

The initial setting of newborn TL has important implications for telomere dynamics in adulthood, and is affected by the intrauterine environment.

There are extensive reports in the literature which associate air pollution exposures and TL in adults. However, studies on the effects on newborn TL of exposure to air pollution have been limited.

Newborn TL is highly variable, and telomere attrition rates are higher during the first four years of life. Newborn TL is an important predictor for TL in adulthood. Telomeres may serve as a possible mechanism relating fetal programming to health consequences in later life.

The birth cohort study in Wuhan, China is the first to assess the trimester-specific relationships between newborn TL and maternal exposure to air pollution.

The results showed that maternal exposures to $PM_{2.5}$, PM_{10}, CO, and SO_2 during the third trimester were inversely related to newborn TL in the single-pollutant models, and the associations were more pronounced among male infants.

(Continued on page 5)

Effect of Prenatal Exposure to Nanoscale Diesel Exhaust Particles on Adult Male Mice Offspring

Prenatal exposure to air pollution is associated with mood dysregulation and cognitive difficulties in early childhood. Exposure to air pollution in the form of particulate matter (PM) causes blood-brain barrier damage, increased oxidative stress response and amyloid-β deposition in brain tissue, which suggests a causal link between PM exposure and acceleration of the pathogenesis of neurodegenerative diseases. PM exposure is also associated with impaired cognitive function.

It has been estimated that up to 85% of PM in cities is related to traffic. Diesel exhaust (DE) is a complex mixture of gaseous-phase compounds and diesel exhaust particles (DEPs).

DEPs, particularly nanoscale PM (<100 nm in aerodynamic diameter), may penetrate into brain tissue by passing through the blood-brain barrier. These particles can also carry on their surfaces large amounts of toxic and hazardous compounds, such as heavy metals and hydrocarbons. This suggests that nanoscale DEPs may cause neurotoxic effects.

The International Agency for Research on Cancer (IARC) designated diesel exhaust (DE) as carcinogenic to humans (Group 1) in 2015.

Maternal exposure to DE affects the morphology of the hippocampus and the cerebral cortex, where accumulation of nanoscale DEPs has been observed. Hippocampus transmission is mainly mediated by glutamate receptors which are crucial for spatial learning and memory.

Previous studies show that prenatal exposure to DE can affect the central nervous system (CNS). However, it remains uncertain if these effects were caused by gaseous compounds, DEPs, or both. A limited number of studies in rodent models have shown that exposure to DEPs can affect the central nervous system.

The present study hypothesized that maternal DEP exposure would cause neurotoxic effects and neuroinflammation by inducing pro-inflammatory cytokines in mice. It aimed to determine if maternal exposure to DEPs affected cognitive functions in adult male mice offspring. To study the effects of prenatal exposure to DEPs on cognitive function, procedures such as prenatal inhalation of DEP are useful.

This study focused on the effects of maternal DEP exposure in terms of anxiety, learning and memory in adult male offspring. Behavioral tests were used, followed by measurement of pro-inflammatory cytokines and N-methyl-D-aspartate (NMDA) receptor gene expression in the hippocampus.

Mice exposed to DEPs *in utero* showed deficits in the Elevated plus maze and Morris water maze test. In addition, mice exposed to DEPs exhibited decreased hippocampal NR2A and NR3B expression.

Taken together, the data suggest that maternal exposure to DEPs not only increased the expression of pro-inflammatory cytokines and reduced expression of NR2A and NR3B in the hippocampus, but also led to anxiety and the impairment of spatial learning and memory in adult male mice offspring.

These results highlight the need to identify means of preventing maternal exposure to DEPs and to control developmental effects on cognitive function. During the perinatal term, the living environment is of interest in terms of preventing the developmental effects of DEPs.

Certainly, environmental enrichment also prevents impairment of hippocampal function. Much more study will be essential to recognizing what further preventive actions can be taken to protect against DEP exposure.

Source: Ecotoxicology and Environmental Safety, Vol. 176, Pages 34-41, July 2019.

Maternal Exposure to Ambient Air Pollution and the Effects on Newborn Telomere Length

(Continued from page 4)

The associations between newborn TL and $PM_{2.5}$ and PM_{10} exposures during the third trimester remained significant in multi-pollutant models, but not SO_2 and CO.

Notably, the inverse relationships between newborn TL and maternal exposures to $PM_{2.5}$, PM_{10}, CO, and SO_2 during the third trimester were more evident in male than in female infants.

Although the underlying mechanisms by which maternal air pollution exposure may lead to shorter newborn TL are yet to be elucidated, these findings are biologically plausible.

The findings suggest that the third trimester is the sensitive window for maternal exposure to $PM_{2.5}$, PM_{10}, CO, and SO_2, and shorter newborn TL. The possible explanation may be that the third trimester is the fastest growth period *in utero* or that there is an accumulation of TL shortening over the course of pregnancy through maternal air pollution exposure.

In conclusion, the findings suggest that maternal exposures to $PM_{2.5}$, PM_{10}, CO, and SO_2 during the third trimester are related to shorter newborn TL. Newborn TL is highly variable and is an important predictor for TL in adulthood.

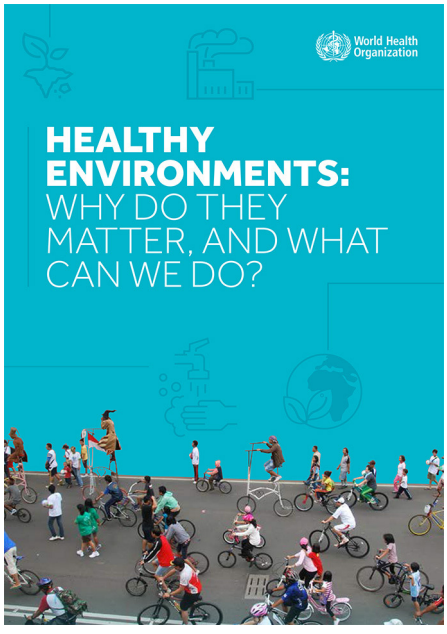
The present study helps us understand the "programming" effect of adverse intrauterine environments on newborn telomere. It provides insights into the role of initial telomere setting on TL-related disorders in later life.

Implications for public health suggest that in order to promote longevity of newborns and improve their health in later life, measures should be taken to reduce air pollution exposure, especially in case of pregnant women.

Source: Environment International, Vol. 128, Pages 254-260, July 2019.

Healthy Environments:
Why do they matter, and what can we do?

The World Health Organization (WHO) has published a new concise guide summarizing key actions which policy-makers can take to create healthier environments for healthier populations (including chemical safety).



Each year, an estimated 12.6 million human deaths are attributable to unhealthy environments (1.6 million deaths due to chemicals).

The unfortunate reality is that the deaths of nearly one in four people on the planet are directly related to the fact that they live or work in an unhealthy environment.

This document presents an overview of sectoral actions that can be taken by various actors and of the support that is being offered by the World Health Organization.

The goal is to create healthier environments, with priority being given to workplaces, cities, dwellings, health care facilities, and emergency settings.

Key risks such as air pollution; water, sanitation and hygiene; chemical safety and radiation; and climate change are addressed.

The actions presented provide an initial overview with reference to policy directions. More detailed information for the next steps urges intersectoral collaboration between a wide range of partners, international organizations, governments, and national and subnational actors, with the goal of creating safe, enabling and equitable environments for better health and a more sustainable future.

Source: WHO reference number (2019): WHO/CED/PHE/DO/19.01 https://www.who.int/phe/publications/healthy-environments/en/.

Seasonal Profiles of Atmospheric PAHs in an E-waste Dismantling Area and their Associated Health Risks

Rapid economic development and the uncontrolled production of electronic waste (e-waste) is a new global problem and e-waste dismantling processes are an important source of air pollution.

Among the pollutants emitted, polycyclic aromatic hydrocarbons (PAHs) are of particular serious concern because of their carcinogenic and mutagenic properties. Moreover, PAHs can be transported via transplacental transfer from the mother to the fetus, thereby exerting adverse impacts on the fetus.

The presence of these hydrocarbons in the environment is mainly attributable to the incomplete combustion of biomass and fossil fuels.

During e-waste dismantling processes, the incomplete combustion of printed circuit boards at relatively low temperature leads to the release of PAHs, which could be important contributors to local environmental pollution.

The emission patterns of atmospheric PAHs from e-waste dismantling processes should be systematically monitored and their sources identified in order to facilitate the control and prevention of health risks.

However, few studies have investigated either the atmospheric PAHs generated by e-waste dismantling in specific regions, especially the PAH levels throughout the year.

Particulates such as $PM_{2.5}$ (particulate matter aerodynamic diameter ≤ 2.5 μm), PM_{10}, and total suspended particulates (TSP) in the air around e-waste dismantling areas are also concerns because various toxic pollutants spread via air particulates. Compared with TSP and PM_{10}, $PM_{2.5}$ has a higher deposition fraction in the human lung and is a greater health risk.

Therefore, in the present study, PAH samples were collected from different PM fractions, i.e., $PM_{2.5}$, PM_{10}, and TSP, as well as the gaseous phase from a typical e-waste industrial park and the surrounding areas in southern China over four seasons.

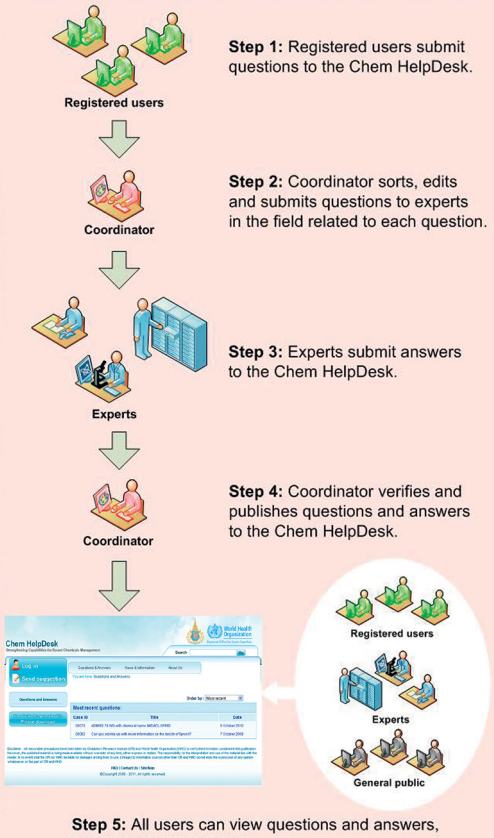
The main objectives of this study were to determine the spatiotemporal profiles of PAHs and the human health risks associated with the bioaccessibility of PAHs in the human lung.

(Continued on page 7)

The Chem HelpDesk

"Strengthening capabilities for sound chemicals management"

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



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

Seasonal Profiles of Atmospheric PAHs in an E-waste Dismantling Area and their Associated Health Risks

(Continued from page 6)

The study findings provide empirical evidence regarding the spatiotemporal profiles of PM-bound and gaseous PAHs in e-waste dismantling areas to facilitate air quality monitoring and management by local governments.

The results show that the concentration of these pollutants was significantly higher at the e-waste dismantling park (EP site) than at the control site.

Most of the atmospheric PAHs were present in the gaseous phase, and most of the particulate-bound PAHs were present in the $PM_{2.5}$ fraction.

Pollutant levels were higher in the spring and winter than in summer and autumn.

E-waste dismantling was identified as the major source of PAH pollution in the area. Approximately 82.4% of the PAHs were attributed to e-waste dismantling at the industrial park (EP site).

Among the sites sampled, pollutant levels and cancer risk were highest at the EP site, and they could pose a cancer risk for humans, although only the bioaccessible PAHs in human lungs were considered.

Infants were at greater health risk than adults, which suggests that air pollution with PAHs is of particular concern in this area.

This study provides clear evidence of the requirement for control measurements of e-waste dismantling process. Greater efforts should be made to decrease pollution with PAHs during e-waste dismantling.

Source: Science of The Total Environment, Vol. 683, Pages 371-379, September 2019.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute
Schedule for 2020

	Training Course	Date	Duration	Closing Date
1.	Principles of Toxicology, Toxicity Testing and Safety Evaluation	January 14 - February 7, 2020	3 weeks	October 31, 2019
2.	Detection of Environmental Pollutants, Testing and Screening of Toxicity	February 24 - March 6, 2020	2 weeks	November 15, 2019
3	Environmental Toxicology	April-May 2020	1 week	January 31, 2020

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

Course Description:

1. Principles of Toxicology, Toxicity Testing and Safety Evaluation (January 14 - February 7, 2020)

The course presents the basic concepts of toxicology, including dose-response relationships; types of harmful effects, mechanisms involved in chemical actions from the entry of chemicals into the body until excretion; toxicokinetics; activation and detoxification of carcinogenesis and mutagenesis; the principles of testing for toxic effects; epidemiology and concepts of risk assessment.

Requirement: Participants should have work experience related to the use of basic knowledge in chemistry, biological sciences, or medicine.

2. Detection of Environmental Pollutants, Testing and Screening of Toxicity (February 24 - March 6, 2020)

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

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

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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