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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".

Long-term Arsenic Exposure and Blood Pressure

Overexposure to naturally occurring arsenic in groundwater and soil can cause a variety of cancers and has been associated with developmental effects, neurotoxicity, diabetes, and cardiovascular disease.

Globally, 200 million people are estimated to drink water which exposes them to arsenic at concentrations above the World Health Organization's recommended limit of 10 µg/L. More than 50 million of these people reside in Bangladesh alone, where nearly all rural households rely on groundwater for drinking water.

There is a strong and direct relationship between high blood pressure (BP) and cardiovascular disease (CVD) mortality. Rapid increases in the prevalence of high BP in low-income countries has likely contributed to the rising epidemic of CVD in these populations.

Previous cross-sectional studies have indicated associations between exposure to inorganic arsenic and prevalence of high BP. Prospective cohort studies that can better characterize the association between arsenic and high BP are lacking.

Longitudinal studies with repeated measurements of BP, which provide a powerful tool to evaluate health outcomes that change over time, are needed to assess whether arsenic is associated with increasing BP over time.

The Health Effects of Arsenic Longitudinal Study (HEALS), a long-term prospective cohort study, was conducted to assess the association of baseline arsenic exposure (measured both in water and urine) with longitudinal changes in BP in 10,853 participants in Bangladesh.

The researchers analyzed BP readings taken from each participant four times between October 2000 and March 2009, with the initial measurements serving as a baseline. For arsenic exposure, they tested well-water and urine samples collected at each of the four visits.

This is the first large epidemiologic study to examine the relationship between arsenic exposure from drinking water and longitudinal change in BP.

In the HEALS population, the correlation of well water arsenic concentrations (ranged from 0.1 to 864 µg/L, median = 62 µg/L) and urinary creatinine-adjust arsenic at baseline was high, supporting the validity of self-reported data on well use and exposure to arsenic across the board in this population.

Even when controlling for age, sex, smoking status, educational status, and diabetes history, the researchers found an average annual increase in systolic blood pressure of 0.43 mmHg for participants exposed to medium-low levels of arsenic in groundwater (12–62 µg/L), 0.54 mmHg for those exposed to medium-high levels (62–148 µg/L), and 0.48 mmHg for those exposed to high levels (above 148 µg/L), as compared with the control group, which was exposed to levels below 12 µg/L.

The results reveal a non-monotonic dose response relationship, in which individuals exposed to the highest arsenic levels in the study often had a smaller increase than those exposed to medium-high levels, indicating that baseline blood pressure may have already been affected significantly by past arsenic exposure.

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Long-term Arsenic Exposure and Blood Pressure

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In the present study, exposure to water with arsenic concentrations > 12 µg/L was associated with a greater increase of 0.43–0.54 mmHg/year and 0.39–0.41 mmHg/year for systolic BP (SBP) and diastolic BP (DBP), respectively.

Evidence suggests that the risk of CVD rises continuously as both SBP and DBP increase from 115 mmHg and 75 mmHg, respectively. Arsenic exposure has been related to CVD mortality in the cohort study and high BP is a CVD risk factor.

The results suggest that long-term arsenic exposure may accelerate age-related increases in BP, which might be one mechanism by which arsenic may lead to CVD. Further studies are needed to investigate other preclinical indicators or biomarkers of CVD with multiple measurements.

Source: Environmental Health Perspectives, Vol. 123, No. 8, Pages 806–812, August 2015.

Developmental Effects of Parental Exposure to Metal-contaminated Soil

Studies of the impact of contaminants on human health and the environment are increasingly important, given the higher risk of exposure associated with industrial and technological growth.

Soil is a very complex material. Because of rapid population growth, intense industrial activity and petrochemical development, more earth is being contaminated with substances of various origins.

The effects of contaminants in soil are closely related to human health, particularly to reproduction, and are among the issues prioritized by environmental health programs.

Children are more vulnerable than adults when exposed to contaminants because of their metabolism and their ability to absorb. In critical periods of prenatal as well as postnatal structural and functional development, specific structures or functions may be more sensitive to damage. The effects of exposure may not be evident until a later stage of development.

The fetus is always vulnerable to any toxins in the mother's body. Hence it is of particular interest to evaluate the effects on the psychomotor development of the children born to mothers who were exposed to contaminants.

This study was conducted to investigate the effects of exposure of female Wistar rats to urban soil influenced by the dispersion of air contaminants. Three periods of exposure were specified: before pregnancy (pre-pregnancy), during pregnancy and during lactation. Offspring from exposed rats were evaluated for deleterious effects on development using physiological, behavioral and hematological parameters.

The contaminated urban soil in this study came from the Rio Grande, Brazil. The contaminants were mainly metals, including arsenic, lead, cadmium, chromium, copper, nickel, and zinc.

In a previous study of contaminated urban soil from this area, lead and arsenic had the highest relative concentration in relation to the control. The exposure of male Wistar rats to contaminated soil was found to have deleterious effects on various parameters of health.

This study demonstrated the potential harmful health effects of contaminated soil and raised the possibility that exposure may interfere with human development in its different stages.

Developmental effects of parental exposure to contaminated soil can be evaluated using a number of approaches, including comparison of the mass at birth of offspring and at weaning to assess overall weight gain, and through other variables such as age of detachment of ears (bilateral), the appearance of fuzz, hair appearance, eruption of incisors (the front teeth), opening of ears (bilateral), opening of eyes (bilateral) and testicular descent.

In the present study, a delay in the onset of incisor eruption and ear opening was observed in pups from dams exposed to contaminated soil during pregnancy, indicating a delay in the development of these indirectly exposed animals.

When litter birth outcomes were assessed, the larger numbers of stillborn pups from dams exposed to contaminated soil compared to the control group suggested maternal toxicity.

In behavioral parameters, parental exposure during pregnancy and lactation resulted in no significant differences in the evaluated parameters when compared to the control. In contrast, pups from the pre-pregnancy group displayed decreased locomotor and exploratory activity in addition to increased levels of anxiety.

This suggested that chronic exposure post conception does not disrupt locomotor and exploratory ability of the offspring.

Hematological parameters remained unchanged in pups born to mothers exposed to contaminated soil during lactation. However, significant decreases in the number of lymphocytes and leukocytes were found among pups born to dams exposed to contaminated soil during the pregnancy period and the pups from the pre-pregnancy group had reduced platelet counts.

Exposure was delivered using the aqueous extract of a soil in its raw state, contaminated under the influence of an industrial complex, and reflecting exactly the scenario of real human exposure.

These results clearly showed the negative influence of parental exposure to contaminated urban soil on the general development of these rats during three critical phases (pre-pregnancy, pregnancy and lactation). It is clear that developing organisms are highly sensitive to external factors.

Source: Science of the Total Environment, Vol. 520, Pages 206–212, July 2015.

Personal Lifestyle Patterns related to Persistent Organic Pollutants and Mercury Levels in Pregnant Women in Japan

Polychlorinated biphenyls (PCBs), polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans (PCDDs/PCDFs), perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA), categorized as persistent organic pollutants and mercury are known environmental chemicals that have been detected ubiquitously not only in the environment but also in humans.

Exposure to environmental chemicals during prenatal and neonatal periods may cause various toxicities including carcinogenicity, teratogenicity, endocrine, immune, and reproductive disruption, and neurobehavioral effects.

Epidemiological studies of Asian, European, and US populations have revealed that environmental chemical levels in maternal samples were associated with demographic, behavioral, dietary, and socio-economic characteristics.

Fish and seafood are the main dietary sources of PCBs and PCDDs/PCDFs exposure in Japan, Taiwan, Nordic countries, and Italy; whereas, meat products, dairy products and fish are the main dietary sources of these persistent organic pollutants in the US, the Netherlands, and Germany.

Potential exposure sources of PFOS and PFOA were reported to be fish and marine mammals, red meat, animal fat, tap (drinking) water, and household dust in Spain, Norway, and Denmark.

Many reports to date have also found fish/seafood consumption responsible for bioaccumulated methylmercury in humans.

It is reasonable to suppose that the presence of exposure sources and their contribution to whole body burden levels of environmental chemicals varies according to the specific characteristics of populations in different countries or regions.

The elimination rate of toxic substances as a reflection of internal metabolism is an effective way to detect body burden levels of environmental chemicals.

Tobacco smoking and the consumption of alcoholic beverages are regarded as behavioral factors related to altered elimination rates of environmental chemicals. For example, tobacco smoking induces increased expression of dioxin-metabolizing enzymes, such as cytochrome P450 (CYP) 1A2, leading to enhanced elimination of PCDDs/PCDFs and dioxin-like PCBs (DL-PCBs).

To date, limited epidemiological studies have been conducted in Japan among pregnant women with no history of accidental poisoning.

Some studies found that PCBs and PCDDs/PCDFs in maternal samples increased with maternal age, alanine aminotransferase levels and alcohol intake. Levels decreased with maternal history of delivery and smoking.

However, no study has assessed maternal smoking and alcohol habits prior to pregnancy, which is considered an important period, because chemicals that have a long half-life could be influenced by lifestyle factors during the entire perinatal period.

There have also been no current studies which evaluate associations between background exposure levels of environmental chemicals, even though certain chemical levels could be correlated with the presence of other chemicals in the human body. This information could help in estimating the magnitude of body burden levels after exposure to various chemicals.

The purpose of this study was to evaluate the association between personal lifestyle characteristics and environmental chemical levels during the perinatal period in the general Japanese population.

322 pregnant women were enrolled in the Hokkaido Study on Environment and Children's Health. Each participant completed a self-administered questionnaire and a food-frequency questionnaire to obtain relevant information on parental demographic, behavioral, dietary, and socioeconomic characteristics.

In total, 58 non-dioxin-like PCBs (NDL-PCBs), 17 PCDDs/PCDFs and 12 DL-PCBs congeners, PFOS, PFOA and mercury were measured in maternal samples taken during the perinatal period.

Most concentrations of environmental chemicals were correlated with the presence of other environmental chemicals, especially in the case of NDL-PCBs, PCDDs/PCDFs, and DL-PCBs which had similar exposure sources and persistence in the body.

Maternal smoking and alcohol consumption, fish and beef intake, and household income were significantly associated with concentrations of environmental chemicals.

PCDDs/PCDFs and DL-PCBs and PFOS decreased with maternal smoking history. NDL-PCBs, PCDDs/PCDFs and DL-PCBs increased with maternal alcohol consumption during pregnancy.

Total hair mercury increased with household income indicating that high socio-economic status is related to increased fish consumption, dental amalgams and vaccines, which are all associated with increased exposure to mercury.

Beef and fish/seafood intake may be important exposure sources of NDL-PCBs.

These results may reflect various lifestyle patterns associated with exposure sources and elimination rates of these environmental chemicals, which may also influence the level of fetal exposure to environmental chemicals through effects on maternal exposure levels.

Source: Chemosphere, Vol. 133, Pages 13–21, August 2015.

Chronic Ultra-low Dose Roundup Exposure and Damage to Liver and Kidneys in Rats

Glyphosate-based herbicides (GBH), such as Roundup, are major pesticides used worldwide.

Glyphosate's primary perceived mode of herbicidal action is to inhibit 5-enolpyruvylshikimate-3-phosphate synthase (EPSPS) of the shikimate aromatic amino acid biosynthesis pathway present in plants and in some bacteria. Since this pathway is absent in vertebrates, it has generally been assumed that glyphosate poses minimal health risks to mammals, including humans.

However, converging evidence suggests that GBH residues pose a particular risk to kidney and liver function.

Most results from previous GBH toxicity studies in animal models were obtained at doses far greater than what the human population is generally exposed to.

Doses tested typically exceeded the glyphosate acceptable daily intake (ADI), which is currently set at 0.3 mg/kg bw/day in the European Union and 1.75 mg/kg bw/day in the USA, based on hepatorenal toxicity measurements in rats. GBH toxicity was not investigated in life-long experiments.

A 2-year study was conducted in rats administering 0.1 ppb Roundup (50 ng/L glyphosate equivalent) via drinking water (giving a daily intake of 4 ng/kg bw/day of glyphosate). The results showed a clearly increased incidence of anatomorphological and blood/urine biochemical changes indicative of structural and functional pathology of liver and kidneys.

In order to confirm these findings the researchers conducted a transcriptome microarray analysis of the liver and kidneys from these same animals.

The results showed that the expression of 4,224 and 4,447 transcript clusters (a group of probes corresponding to a known or putative gene) were altered respectively in liver and kidneys.

These alterations in gene expression profiles are typical of disturbances measured in cases of fibrosis, necrosis, phospholipidosis, mitochondrial membrane dysfunction and ischemia.

Therefore these results confirm the ultra-low dose Roundup-induced increased incidence of hepatorenal pathologies suggested by observations at anatomical, histological and biochemical levels.

An important consideration is that Roundup is not a single compound, but combines an active ingredient (glyphosate) with various adjuvants which are required in order to stabilize and allow penetration of glyphosate into plants.

In short term acute exposures, some adjuvants can be considered responsible for Roundup toxicity. However, as the adjuvant composition is proprietary and not fully disclosed, it is not possible to attribute the toxicity of the whole agricultural herbicide formulation to a given component.

The results of the study indicate that even when levels of consumption of a GBH formulation are far lower, and even when glyphosate-equivalent concentrations are at admissible levels, there are associated wide-scale alterations of the liver and kidney transcriptome that correlate with observed signs of hepatic and kidney anatomorphological and biochemical pathological changes in these organs.

In addition, as the investigated dose of Roundup is environmentally relevant in terms of levels of exposure to humans, domesticated animals and wildlife, the results potentially have significant health implications for all these populations.

Furthermore, data also suggests that new studies incorporating testing principles from endocrinology and developmental epigenetics, in particular to evaluate the endocrine disruptive capability of GBH/glyphosate, should be performed to investigate potential consequences of low dose exposure early in life as well as in adults.

Source: Environmental Health, Vol. 14, No. 70, 14 Pages, August 2015. (Open Access)

The Neurological Effects of Chlorpyrifos in Rodent Brain

Organophosphates (OPs) are used for a wide variety of important applications. The toxicity of this group of pesticides is most commonly attributed to the inhibition of the enzyme acetylcholinesterase (AChE).

However, there is also significant evidence in the human epidemiological literature that OP exposures not associated with acute toxicity may also result in prolonged neurological and neurobehavioral deficits, including impairments of cognition.

Moreover, as an etiological mechanism, AChE inhibition may not account for all of the symptoms associated with acutely toxic or lower level OP exposures.

Previous experiments in animals indicated that axonal transport is negatively affected by OPs, a potentially notable finding, given the fundamental importance of axonal transport to neuronal health and brain function.

The new study was conducted to evaluate the effects of the commonly used OP pesticide, chlorpyrifos, on axonal transport in the brains of living rats using manganese (Mn^{2+})-enhanced magnetic resonance imaging (MEMRI) of the optic nerve projections from the retina to the superior colliculus.

MEMRI is a non-invasive imaging method that has been successfully used to detect impairments of axonal transport in the brains of aged rats, mouse models of Alzheimer's disease, frontotemporal dementia, and mice homozygous for a deletion in the amyloid precursor protein gene.

Axonal transport is an essential process in neurons that is responsible for the movement of a variety of important

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Disposition of Inorganic Mercury in Pregnant Rats and their Offspring

Exposure to environmental toxicants such as methylmercury and/or inorganic mercury (Hg^{2+}) can lead to serious toxicological consequences in the renal, hepatic, cardiovascular, nervous and reproductive systems. The effect of mercuric species on the reproductive system and the developing fetus is of particular concern.

Numerous studies have shown that methylmercury can cross the placenta and accumulate in the fetus, with negative impact on fetal health.

Despite the prevalence of Hg^{2+} in the environment and the ability of methylmercury to biotransform into Hg^{2+}, either in plasma or target cells, little is known about the ability of Hg^{2+} to cross the placenta into fetal tissues.

Considering the placental accumulation of Hg^{2+}, it seems possible that Hg^{2+} may also gain access to fetal tissues and organs. Because the biotransformation of methylmercury–Hg^{2+} probably occurs primarily in maternal blood and organs, it is important to understand how Hg^{2+} is handled in maternal organs, as well as in those of the fetus.

The current study on Wistar rats was conducted to assess the disposition and transport of Hg^{2+} in placental and fetal tissues, and to test the hypothesis that acute renal injury in pregnant dams can alter the accumulation of Hg^{2+} in fetal tissues.

The pregnant rats were injected intravenously with either a non-nephrotoxic (0.5 mmol/kg) or a nephrotoxic dose (2.5 mmol/kg) of $HgCl_2$ and the disposition and toxicity of mercuric ions assessed not only in placental tissues, but also in fetal organs, at 6 or 48 hours after exposure to Hg^{2+}.

The results showed that accumulation of Hg^{2+} in the placenta was rapid and dose-dependent.

Following exposure to non-nephrotoxic dose, the placental burden of Hg^{2+} 6 hours after exposure was similar to 48 hours after exposure. This finding suggests that placental accumulation of Hg^{2+} is rapid. The placenta does not have efficient mechanisms to eliminate mercuric ions, indicating that at least a fraction of the Hg^{2+} that gains access to the placenta subsequently enters fetal tissues.

Hg^{2+} has been shown previously to be retained in the placenta, which may be due, in part, to the binding of Hg^{2+} in blood to the endothelium of placental blood vessels. The Hg^{2+} that is transported into fetal tissues likely exists as a conjugate of a thiol-containing biomolecule, which may utilize a number of various transport proteins to cross the placenta and access fetal organs/tissues.

When the fetal burden of Hg^{2+} was examined in dams exposed to non-nephrotoxic dose of $HgCl_2$, the total fetal accumulation of Hg^{2+} was greater 48 hours after exposure than 6 hours. This finding suggests that fetal accumulation of Hg^{2+} continues to increase during the initial 48 hours after exposure to $HgCl_2$.

The greatest concentration of Hg^{2+} were localized in the fetal kidneys, followed by the liver and brain. These accumulations also reflect dose-dependence, suggesting that continued maternal exposure may lead to increased fetal exposure.

The inefficient elimination of Hg^{2+} from fetal tissues and the enhanced sensitivity of these tissues to the effects of Hg^{2+} may lead to significant deleterious effects in the fetuses without manifestation of clinical symptoms in the mother.

Collectively, these findings tend to suggest that even acute exposure to $HgCl_2$ can lead to measurable accumulations of Hg^{2+} in fetal tissues.

Taken together, these data indicate that Hg^{2+} is capable of crossing the placenta and gaining access to fetal organs in a dose-dependent manner.

The current study provides novel data suggesting that Hg^{2+}, probably as a thiol-*S*-conjugate, is taken up by the placenta and subsequently gains access to fetal organs.

Additional studies are required to fully characterize the transport and accumulation of Hg^{2+} across the placenta and into fetal tissues.

Source: Toxicology, Vol. 335, Pages 62–71, September 2015.

The Neurological Effects of Chlorpyrifos in Rodent Brain

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macromolecules (e.g., mitochondria, receptor proteins, growth factors) to and from a neuron's cell body.

Further, impairments in axonal transport have been implicated in the pathology of a wide variety of neurological illnesses (e.g., amyotrophic lateral sclerosis, Alzheimer's disease, Huntington's disease, and Parkinson's disease).

It is noteworthy that many of these illnesses are characterized by similar neurobehavioral deficits that have been observed in people who have been exposed to OP-based pesticides.

Here it is also important to note that OP exposure may even represent a potential risk factor for Alzheimer's disease as well as some of the other neurodegenerative disorders mentioned above.

The results of this new study indicate that repeated exposures to a commonly used pesticide, chlorpyrifos, can result in persistent alterations in axonal transport in the living mammalian brain.

Given the fundamental importance of axonal transport to neuronal function, these observations may (at least in part) explain some of the long-term neurological deficits that have been observed in humans who have been repeatedly exposed to doses of OPs not associated with acute toxicity.

These findings may also complement other studies which have identified other deleterious effects of organophosphates that may be additive (or unrelated) to AChE inhibition and include oxidative stress, impairments of mitochondrial function, neuroinflammation, altered neurotrophin responses.

Source: NeuroToxicology, Vol. 47, Pages 17–26, March 2015.

Association of Atmospheric Particulate Matter and Ozone with Gestational Diabetes Mellitus

Gestational diabetes mellitus (GDM) is a common complication of glucose intolerance which typically occurs or is first recognized during pregnancy.

Women with GDM have higher long-term risks of cardiovascular diseases and about one in three will eventually develop type 2 diabetes.

In children, GDM has been associated with both perinatal and long-term adverse health outcomes such as macrosomia, shoulder dystocia, birth injuries, sustained glucose tolerance impairment, obesity, and impaired intellectual abilities.

Evidence associating air pollution with birth defects and pregnancy complications such as gestational hypertension has been widely reported in the last decade. However, links between ambient air pollution and GDM in particular are still unclear.

In this study, the researcher analyzed vital statistics records for 410,267 women in Florida who gave birth during 2004–2005 to examine the association between the risk of GDM and two ambient air pollutants, particulate matter with diameter less than 2.5 μm ($PM_{2.5}$) and ozone (O_3).

Individual air pollution exposure was assessed at each woman's home address at the time of her delivery. Associations between exposure to air pollution and GDM during different gestational periods (trimesters and full pregnancy) were also investigated.

When assessed in single-pollutant models, GDM was significantly associated with per 5-unit increases in both $PM_{2.5}$ and O_3 during the first and second trimesters and the full pregnancy.

The associations were also found in co-pollutant models for $PM_{2.5}$ exposure during the first trimester and for O_3 exposure during all pregnancy periods examined.

The results of this study add to the emerging evidence linking air pollution exposure during pregnancy to pregnancy complications such as GDM.

The causal mechanisms underlying the associations between air pollution and GDM are still uncertain. However, the results observed in this study are consistent with several potential pathways suggested by previous studies.

Ambient air pollutants such as PM and O_3 have been reported to be associated with increased insulin resistance, dyslipidemia, and systemic metabolic dysfunction, all at which are precursors associated with GDM.

PM contains many toxic chemicals that are regarded as reactive oxygen species (ROS) capable of causing oxidative damage to target tissues. An imbalance between the production of ROS and antioxidant defenses is known to be one of the main causes of insulin signaling–pathways alteration, and a number of studies have linked ROS to insulin resistance.

In this case, the researchers observed increased odds that GDM would occur with per 5-μg/m^3 increase in $PM_{2.5}$ and per 5-ppb increase in O_3 during both the first and second trimester, and in the full pregnancy single-pollutant models.

Compared with the single-pollutant model, the odds ratios (ORs) for O_3 were almost identical in the co-pollutant model. However, the ORs for $PM_{2.5}$ during the first trimester and the full pregnancy were attenuated, and no association was observed for $PM_{2.5}$ during the second trimester in the co-pollutant model.

This population-based study suggests that exposure to air pollution during pregnancy is associated with increased risk of GDM in the USA.

Source: Environmental Health Perspectives, Vol. 123, No. 9, Pages 853–859, September 2015.

Breastfeeding as an Exposure Pathway for Perfluorinated Alkylates

Perfluorinated alkylates (PFASs) are widely used to make industrial products resistant to water, oil, or stains such as waterproof clothing, food packaging, paints and lubricants.

PFASs, which tend to bioaccumulate in food chains and can persist for a long time in the body, are found regularly in the blood of animals and humans worldwide.

PFASs have been linked with reproductive toxicity, endocrine disruption, and immune system dysfunction. Due to the particular vulnerability of the immune system during early development, the sources of PFAS exposure in infants are of special interest.

The elimination of long-chain PFASs in humans is very slow, with half-lives in adults thought to be several years. Exposure of PFASs via human milk could therefore lead to elevated serum concentrations in breastfed infants.

The risks and benefits of breast-feeding are sometimes referred to as "the weanling's dilemma". Although breastfeeding is recommended by WHO as the exclusive food source for infants during their first 6 months and, with supplementary food, up to age 2 years, WHO did not consider the possible impact of environmental chemicals present in human milk.

The present study examines the association between months with exclusive or partial breastfeeding and serum-PFAS concentrations in children up to age 5 to determine the time-dependent impact of this exposure pathway.

The researchers followed children who were born in the Faroe Islands,

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The Chem HelpDesk

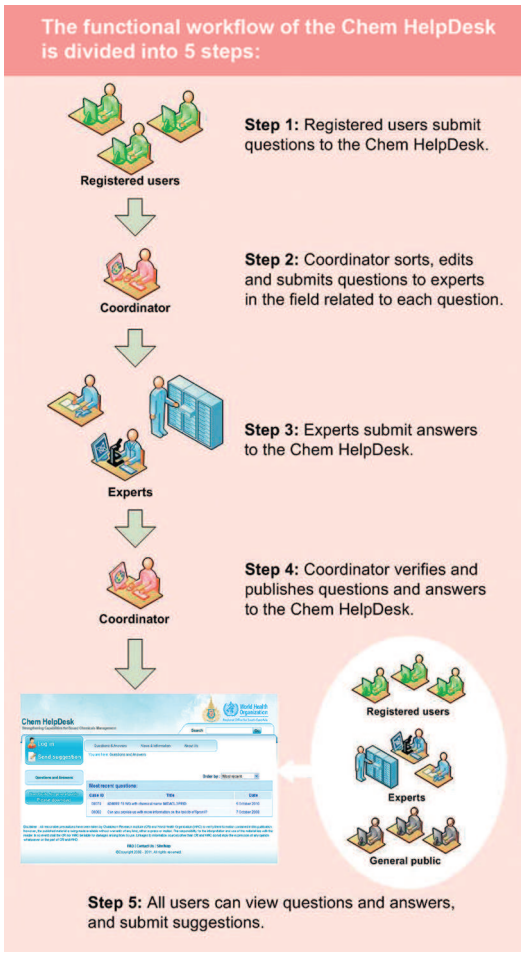
"Strengthening capabilities for sound chemicals management"

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



Breastfeeding as an Exposure Pathway for Perfluorinated Alkylates

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Denmark between 1997–2000, examining the levels of five types of PFASs in their blood at birth and at age 11 months, 18 months, and 5 years. They also checked PFAS levels in the mothers of these children at week 32 of pregnancy.

They found that, in children who were exclusively breastfed, PFAS concentrations in the blood increased by roughly 20% – 30% each month, with lower increases among children who were partially breastfed. The results suggest that breast milk is a major source of PFAS exposure during infancy.

In some cases, by the end of breastfeeding, the children's PFAS serum concentration level exceeded their mothers'.

PFASs have a half-life in people's bodies of more than three years, which is a long time. This makes it difficult for women who might get pregnant to avoid exposure. The results of this study suggest that several months of breastfeeding lowered the mothers' own levels of these compounds, presumably by transferring to their babies.

This study is the first to estimate the transfer of water- and stain-proofing chemicals from mother to baby during breastfeeding and suggests that mother's milk, which provides healthy antibodies, vitamins and nutrients, is also a major source of these harmful compounds for developing children.

Source: Environmental Science & Technology, Vol. 49, No. 17, Pages 10466–10473, August 2015.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute
Schedule for 2016

	Training Course	Date	Duration	Closing Date
1.	Detection of Environmental Pollutants, Testing and Screening of Toxicity	February 15 - 26, 2016	2 weeks	November 25, 2015
2	Environmental Toxicology	May 2016	2 weeks	February 25, 2016

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

Course Description:

1. Detection of Environmental Pollutants, Testing and Screening of Toxicity (February 15 - 26, 2016)

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

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

2. Environmental Toxicology (May 2016)

The course provides students and participants with a background of the major groups of toxic substances encountered by man and animals through food and the environment, and also through exposure at the workplace. These toxicants include mycotoxins, naturally occurring plant and animal toxins, toxic substances in air, water and soil, N-nitroso compounds, solvents, plastics, pesticides and pollutants. The course focuses on the chemistry, fate and distribution in the environment, mechanisms of their action, toxic manifestation in living organisms, as well as toxic syndrome in human beings.

Requirement: Participants should have some basic knowledge of chemistry and the biological/biomedical sciences.

Fellowships:

A limited number of fellowships are available that will cover roundtrip airfare, accommodation (on site) and meals, training materials, and health insurance.

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

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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