



**CRI/ICEIT
NEWSLETTER**

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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".

CRI's WHO Collaborating Centre on Capacity Building & Research in Environmental Health Science & Toxicology

Official Visit of Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol to the World Health Organization's Regional Office for South-East Asia



On February 13th, 2015, Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol, President of the Chulabhorn Research Institute (CRI), paid an official visit to the World Health Organization's Regional Office for South-East Asia (WHO SEARO) in New Delhi, India, to review progress on collaborative activities between WHO SEARO and CRI, carried out under CRI's International Centre for Environmental Health and Toxicology (ICEHT), a WHO Collaborating Centre for Capacity Building and Research in Environmental Health Science and Toxicology since 2005.

Discussions that took place, including the technical discussions attended by a team

of senior researchers from CRI on February 11th and 12th, which covered the following areas: (1) CRI's capacity building/training programmes in Chemical Safety and Occupational and Environmental Health/Medicine; (2) development of training modules/courseware for both face-to-face and web-based training in chemical safety; (3) promoting the use of the electronic distance learning tool (eDLT) on risk assessment and risk management of chemicals (launched in 2013); (4) raising awareness and disseminating information related to chemical safety and risk assessment; and (5) research.

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Official Visit of Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol to the World Health Organization's Regional Office for South-East Asia

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On the issue of research, Professor Dr. Her Royal Highness Princess Chulabhorn Mahidol gave a special presentation to WHO SEARO technical staff entitled, “**Arsenic Exposure In Utero and During Early Childhood and Its Potential Health Impacts**”. The talk introduced the research CRI had been conducting in the south of Thailand, in Ron Pibul district, in a population of exposed pregnant mothers from five villages, with the objective of understanding how arsenic affects biological systems and the potential outcomes of exposure during pregnancy and early childhood. Arsenic contamination in this particular area of Thailand is attributed to tin mining during the 1960s to the 1980s. Although tin mining in the area ceased in the 1980s, ground water in the area is still contaminated with arsenic.

Her Royal Highness Princess Chulabhorn summarized some of the key results from this research, including the identification of gene expression changes in the cord blood of newborns that were significantly associated with prenatal arsenic exposure, showing a set of 11 key genes involved in the stress and inflammatory response and cell cycle regulation. DNA methylation has been proposed as an epigenetic mechanism for arsenic-induced toxicity. A follow-up study was conducted in the children born from the aforementioned exposed mothers and the data showed that the exposed children had a lower ability to

methylate arsenic, which has been associated with increased biological effects and a greater susceptibility to arsenic-related diseases, such as peripheral vascular disease and bladder cancer. The data also indicated an increase in oxidative DNA damage, and a lower capacity in these same children for repairing the damage in their DNA. Taken as a whole, these results suggested that continued exposure to arsenic in the young children who were prenatally exposed may exacerbate the risk of

cancer development later in life.

Professor Dr. Her Royal Highness Princess Chulabhorn ended the presentation by suggesting that the next step from a research perspective may be to continue to follow-up with these subjects through time, to see if there is indeed evidence of health effects that can be linked with arsenic exposure, keeping in mind the various modifying factors, such as life-style, which may impact on disease manifestation. Meanwhile, what can be done now is to try to implement preventive measures to reduce this risk through reducing exposures and by educating people to become aware of the risks they are faced with in their everyday lives.

In terms of the capacity building/training programmes in Chemical Safety and Occupational and Environmental Health/Medicine, CRI organized 5 international training courses in 2014, including 4 at CRI and one in Vietnam, attended by participants from 20 countries. These courses are open to participants, primarily from the Asia Pacific region, and are taught by international experts from world-renowned academic and research institutions with a wealth of teaching experience in the region.

The organized courses included:

Course	Countries
Detection of Environmental Pollution, Testing and Screening of Toxicity (27 February – 7 March 2014)	6 (Bhutan, Laos, Philippines, Rwanda, Sri Lanka, Vietnam)
Environmental Toxicology (1-9 May 2014)	9 (Bhutan, India, Jordan, Laos, Maldives, Nigeria, Philippines, Thailand, Vietnam)
Environmental Immunotoxicology and Reproductive Toxicology (20-31 October 2014)	13 (Bhutan, Brunei Darussalam, Cambodia, China, India, Indonesia, Laos, Malaysia, Nepal, Philippines, Sri Lanka, Thailand, Vietnam)
Risk Assessment and Risk Management of Chemicals (1-5 December 2014)	Vietnam (in-country training course)
Environmental and Health Risk Assessment and Management of Toxic Chemicals (6-18 December 2014)	11 (Bhutan, Brunei Darussalam, Cambodia, India, Jordan, Laos, Mongolia, Pakistan, Philippines, Sri Lanka, Thailand)

Those who are interested in applying for a fellowship to attend such training courses can check the calendar of events on CRI's website at http://www.cri.or.th/en/ac_actcalendar.php.

Chromium VI and Stomach Cancer

Numerous studies have identified associations between lung cancer and inhaled hexavalent chromium (Cr(VI)) in occupational settings. The International Agency for Research on Cancer has classified Cr(VI) as a group I carcinogen, based primarily on studies of chromate production, chromate pigment production and chromium electroplating involving high exposures.

Several studies also suggest that Cr(VI) may have carcinogenic effects in other internal organs as well, including the gastrointestinal tract. The issue of whether Cr(VI) causes gastrointestinal cancer has implications not only for exposed workers, but also for people who ingest Cr(VI) in drinking water. However, the evidence linking Cr(VI) to gastrointestinal cancer comes primarily from animal studies, and questions have been raised about their relevance to humans.

A meta-analysis of human studies of Cr(VI) and stomach cancer was performed in order to provide a review of the current literature, evaluate causal inference, and assess potential sources of bias and heterogeneity.

The results of this meta-analysis suggest that Cr(VI) exposure is associated with increased risks of stomach cancer. An important feature of this study is that summary relative risks were elevated in a number of different occupational settings and in the subgroup of studies in which lung cancer risks were also elevated.

Another consideration is that drinking water exposures may cause greater toxicity because they can take place over the long term (e.g., lifetime) and are more likely to occur at particularly susceptible life stages (e.g., in fetuses,

children and pregnant women) than exposures occurring at work. Thus, despite the different routes and magnitudes of exposure, these findings could have some relevance to efforts to regulate Cr(VI) in water. They provide evidence that Cr(VI) is a cause of cancer in the human gastrointestinal tract and support the animal and limited human data linking ingested Cr(VI) to stomach cancer.

The results of this study support the efforts of the US EPA and some states to regulate Cr(VI) in drinking water based on its potential carcinogenicity in the gastrointestinal tract. California has recently established the first drinking water standard for Cr(VI) in the USA.

Source: Occupational and Environmental Medicine, Vol. 72, Issue 2, Pages 151-159, February 2015.

Arsenic Exposure, Hyperuricemia, and Gout in US adults

Arsenic, a worldwide environmental pollutant, is an established risk factor for the development of cancer and cardiovascular disease, and possibly for the development of diabetes and chronic kidney disease. Arsenic exposure may result in hyperuricemia secondary to kidney injury, but animal studies have shown mixed results.

Evidence in humans is very limited. Human studies in areas with high arsenic concentrations in drinking water (mean > 50 µg/L) in Mexico and India reported that high exposure to inorganic arsenic is associated with hypouricemia.

In general populations, the major sources of inorganic arsenic exposure are drinking water and food (especially rice and other grains).

In the US, several million people are exposed to arsenic levels in drinking water above 10 µg/L, the US Environmental Protection Agency (EPA)

standard for arsenic in drinking water systems.

A recent cross-sectional study analyzed a representative sample of US adults aged 20 years or older who participated in the 2003-2010 National Health and Nutritional Examination Survey (NHANES) to advance understanding of the association between inorganic arsenic exposure and serum uric acid levels and gout.

In this study, hyperuricemia was defined as serum uric acid higher than 7.0 mg/dL for men and 6.0 mg/dL for women. Gout was defined based on self-reported physician diagnosis and medication use.

The results showed that low-level arsenic exposure, as measured in urine, is associated with higher serum uric acid levels and increased prevalence of hyperuricemia in men but not with self-reported gout. In women, urine arsenic

was associated with self-reported gout, but not with serum uric acid level and hyperuricemia.

Sex differences in the association between arsenic, uric acid and gout are less conclusive. Female gout has different clinical features relative to men, including more inflammation in the upper limb joints, multiple joints involved, and less recurrence. The risk profile is also different. Female gout is related more to diuretic use and comorbidities such as hypertension and impaired renal function and less to genetic variants.

Further experimental and mechanistic studies at relevant exposure levels and prospective studies in humans are needed to confirm the associations among environmental arsenic exposures, serum uric acid, and gout.

Source: Environmental International, Vol. 76, Pages 32-40, March 2015.

Endocrine Disruptor Activity of Multiple Environmental Food Chain Contaminants

Industrial chemicals, antimicrobials, drugs and personal care products have been identified as global pollutants which enter the food chain. Some have also been classified as endocrine disruptors, based on results of various studies employing a number of *in vitro* and *in vivo* tests.

An endocrine disrupting compound (ED) has been defined as an exogenous substance or mixture that alters function(s) of the endocrine system and consequently causes adverse health effects in an intact organism, or its progeny, or (sub)population. The adverse effects of ED exposure have already been extensively described and include infertility, cancers, disrupted thyroid function, cognitive and behavioural disorders, obesity, metabolic syndrome, type I diabetes and immune dysfunction.

EDs include compounds of natural origin (such as phytoestrogens or mycotoxins) as well as a variety of man-made chemicals (e.g. pesticides, brominated flame retardants (BFRs), ultraviolet (UV) filters and phthalates).

We are exposed to EDs via our environment and through our diet when these compounds may gain entry to the food chain. Many of these compounds have been shown to biomagnify in the food chain and some have been reported

as emerging contaminants in its lower levels. Thus it is important to assess these contaminants for ED activity.

A recent study employed a mammalian reporter gene assay (RGA) to assess the effects of known and emerging food chain contaminants (fifty-nine compounds) on estrogen nuclear receptor transactivation.

The most active estrogen agonists have been reported as two parabens (butyl 4-hydroxybenzoate and propyl 4-hydroxybenzoate), a phthalate (di-*n*-butyl phthalate [DBP]), and three pyrethroid metabolites (3-phenoxybenzoic acid, decamethrinic acid and methyl-3-(2,2-dichlorovinyl)-2,2-dimethyl (cyclopropane)carboxylate [DCCA]).

This study reports five new estrogenic compounds (pyrethroids and their metabolites) which have not been previously described (resmethrin, flumethrin, τ -fluvalinate, DCCA and decamethrinic acid) and highlights for the first time that pyrethroid metabolites are of particular concern, showing much greater estrogenic activity than their parent compounds.

Also, the response enhancement of 17 β -estradiol by three UV filters (Bp-3, UV-9 and UV-328) as well as the anti-estrogenic activity of DBP is reported for the first time.

The high estrogenic activity of parabens is also of interest because these compounds are widely used as food preservatives and in cosmetics as antimicrobial agents. Consequently, they can add up to a pre-existing pool of estrogenic UV filters and phthalates, which are used as solvents in many personal care products.

Finally, this study highlights the challenge in ED assessment and a lack of harmonization of ED testing methods already recognized by the European Food Safety Authority (EFSA, 2010). The range of available literature on the subject is overwhelming and includes a variety of *in vivo* and *in vitro* assays, frequently presenting conflicting findings.

With respect to RGAs, a number of variables may influence the results such as cell line employed, its metabolism, promoter sequence and reporter protein employed, time of exposure, analyte concentration tested and the standards' purity. Thus, before drawing a final conclusion on the ED activity of these compounds, other assays should be employed to verify these findings.

Source: Toxicology in Vitro, Vol. 29, Pages 211–220, February 2015.

Toxicological Effects of Particulate Emissions in Oil and Wood Fuels in Heating Systems

An important source of particulate matter (PM) and gaseous pollution is the combustion of fossil fuels for heating purposes. The gaseous compounds have been blamed for worldwide climate change and global warming. The EU and the U.S. are making efforts to reduce the amount of their overall air pollution and greenhouse gas emissions by discouraging the use of oil for heat production in favor of renewable fuels, including all kinds of wood.

The use of wood instead of oil fuels in heating systems is strongly encouraged in many countries. However, the negative health effects of emissions from such a

large-scale change from oil to wood fuels in heating systems is largely unknown.

Many studies point out that the PM emissions from wood combustion are far from benign. Among the symptoms induced or exacerbated by exposure to wood smoke are asthma and other respiratory diseases.

A new study focused on different toxicological responses evoked by PM emissions from small- and medium-scale oil-fueled heating systems vs. similar-scale wood-fueled heating systems.

PM samples were collected from

these heating systems and their toxic potential analyzed using assays of short-term cytotoxicity, inflammation and genotoxicity (DNA damage) in a murine macrophage cell line.

The results showed that a medium-scale oil-fueled heating system induced a dose-dependent increase of DNA damage, short-term cytotoxic effects, and a cell cycle arrest in the G2/M-phase. The medium-scale wood-fired system also showed significant short-term cytotoxicity but did not induce DNA damage.

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Derivation of Exposure Factors for Infant Lactational Exposure to Persistent Organic Pollutants

Breastfeeding is a major source of exposure to lipophilic persistent organic pollutants (POPs) in newborns and infants. POPs distribute into breast milk lipids and are then ingested by infants. Absorption of these chemicals is estimated to be very high following ingestion.

There is growing concern that high levels of postnatal exposure to POPs through breastfeeding may lead to some negative health impacts, such as altered neurodevelopment and diabetes.

A difficulty linked with risk assessment of POPs in breastfed infants is the exposure assessment of these chemicals. Recently, generic physiologically-based pharmacokinetic (PBPK) modeling methods have been developed and validated for estimating maternal-infant transfer of POPs through *in utero* placental exchange and breastfeeding.

These mathematical models allow accurate estimations of internal POP levels in children from birth up to 45 months of age by considering maternal and child physiology (e.g., infant and maternal body weight), the duration of breastfeeding, the chemical's half-life, and POP levels in maternal blood during pregnancy, cord blood at delivery, or breast milk.

These models have been used to assess infants' lactational exposure to certain POPs in epidemiological studies and to evaluate infants' daily intake in the risk assessment of hexachlorobenzene.

Characterizing infants' daily dose and biological levels is a cornerstone of risk assessment. Although PBPK modeling could be used to estimate lactational exposure and resulting levels in infants, it may be time- and resource-consuming to perform simulations each time risk assessment needs to be carried out.

Risk assessors would benefit from having exposure factors that allow them to estimate infants' dose and biological levels, based on a chemical's half-life and maternal dose or biological levels.

The new study was conducted to understand the behavior of the exposure

ratios between infant and their mothers in the general population and to determine if infant:mother (I:M) internal exposure factors can be derived from model simulations for eventual use in risk assessment to assure infant protection from POP exposures.

Using a validated pharmacokinetic model, Monte-Carlo simulations of infants' exposure during the first 2 years of life for POPs of different half-lives (i.e., 1-20 years) were performed. The I:M ratios for dose and biological levels throughout infancy were derived.

To evaluate model accuracy, simulated I:M biological level ratios were compared to ratios calculated from Inuit mothers' and infants' measured plasma levels of 2,2', 4,4', 5,5'-hexachlorobiphenyl (PCB-153), p,p'-dichlorodiphenyl dichloroethylene (p,p'-DDE) and hexachlorobenzene (HCB).

Practically all measured I:M biological level ratios from the Inuit cohort fell below the 95th percentile of the distributions of simulated PCB-153, p,p'-DDE and HCB levels, which indicates that confidence can be bestowed on the PBPK model to adequately describe the I:M biological level ratios in a population for a variety of POPs.

Peak I:M dose ratios were observed at the beginning of lactation with 95th percentile values of 13, 53, 84, 102 and 113 for half-lives of 1, 5, 10, 15 and 20 years, respectively.

In contrast, peak I:M biological level ratios occurred after approximately 1 year of breastfeeding and plateaued at approximately 10.5 (95th percentile) for chemicals of half-lives >5 years.

This is the first systematic study evaluating the distribution of the I:M ratio of exposure (external and internal) and demonstrating how this ratio evolves during the period of breast-feeding. The generic simulations made for POPs of different half lives and the data for three different POPs suggest that a factor derived from the 95th percentile could be used to protect infants from their significantly higher exposures.

Such a factor could be used in risk assessment to consider infant exposure based on knowledge of maternal/women's doses or levels in the population, or to adjust reference dose (RfD) values for this subpopulation.

Source: Regulatory Toxicology and Pharmacology, Vol. 71, Issue 2, Pages 135-140, March 2015.

Toxicological Effects of Particulate Emissions in Oil and Wood Fuels in Heating Systems

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In a small-scale heating systems, both oil and wood combustion emission samples induced DNA damage. However, the short-term cytotoxic effects were greater for the PM emissions than from the oil-fired heating system.

PM mass emissions differed significantly between the tested heating systems. The lowest emissions, 0.1 mg/MJ, were produced by the small-scale oil-fired heating system; the highest emissions, 20.3 mg/MJ, by the medium-scale oil-fired heating system. The wood-fired heating systems' PM mass emissions were in between these concentrations, complicating the direct

comparison of the emissions' health related toxic effects.

The results did indicate however that the emissions from both the small- and the medium-scale wood-fueled heating systems caused less cytotoxicity and DNA damage in a cell model than the emissions from the corresponding oil-fueled heating systems. Hence, controlled wood-fueled heating systems may provide a valid alternative to oil-fueled district heating systems.

Source: Atmospheric Environment, Vol. 103, Pages 321-330, February 2015.

Neurobehavioral Impact from Occupational Exposure to Pesticides

Organophosphates (OPs) became the principal means of agricultural-pest control since the ban of some organochlorine pesticides in the 1970s. While clinical symptoms due to high exposures are primarily experienced by occupationally exposed individuals, sequelae of low-level exposures are of importance in the context of occupational and environmental exposures.

There is agreement about the serious neurological consequences of high exposures to pesticides. Many studies investigated the impact of chronic pesticide exposures by means of neuropsychological performance tests that are capable of reflecting the altered nervous system functioning due to toxic effects. However, the impact of chronic exposures in the absence of acute poisonings is controversial. A systematic analysis of dose-response relationships is still missing.

The importance of dose-response relationships for the proof of causal relationships has been highlighted, and estimate of such relationships is an obstacle to a summary of epidemiological studies on the neurobehavioral impact of pesticides.

The recent study was conducted to (1) quantify the neurotoxic impact of pesticides by an analysis of functional alterations in workers measured by neuropsychological performance tests, (2) estimate the dose-response relationships on the basis of exposure duration, and (3) explore susceptible subgroups.

The meta-analysis employed a random effects model to obtain overall effects for individual performance tests. Twenty-two studies, covered the period between 1965-2010, with a total of 1758 exposed and 1260 reference individuals were included .

Memory and attention were the key domains in which chronically exposed workers showed lower performances than unexposed. Indications for exposure-effect relationships suggested that the lower performance scores were related to exposure.

Relationships exist between the impact of pesticides on performances and exposure duration. A change in test paradigms would help to decipher the impact more specifically. The investigation of biomarkers of effect, for example

oxidative stress or inflammation, appeared as a fruitful approach according to the results on the importance of duration of exposure.

Studies on adolescents had to be analyzed separately due to numerous outliers. The large variation among outcomes hampered the analysis of the susceptibility in this group, while data on female workers was too scant for the analysis.

Further investigations are needed to specify the risk of adolescents and women.

Evidence from other fields showed that the proof of the impact could be more specific if test paradigms would be altered. This would help to corroborate the relationship between pesticide exposure and effects in addition to a biomarker that appears appropriate for low exposure concentrations and allows exposure-effect estimates beyond crude proxies like exposure duration.

Source: Environmental Research, Vol. 136, Pages 234-245, January 2015.

Risk Assessment for Children Exposed to DDT Residues in Various Milk Types from the Greek Market

Despite the fact that the production and usage of the great majority of organochlorine pesticides (OCPs) has been banned in Europe and substantially reduced worldwide, their residues may still be detected in food products from different regions since they are persistent and highly stable under most environmental conditions. OCPs are also considered as endocrine-disrupting chemicals and carcinogenic compounds.

Dichlorodiphenyl-trichloroethane (DDT), a previously widely used OCPs, is officially classified according to EU regulation as suspected of causing cancer (Carc 2) and responsible for

causing damage to organs through repeated and prolonged exposure, while the Integrated Risk Information System (IRIS) of United States Environmental Protection Agency (US EPA) has classified DDT and some of its major metabolites as probable human carcinogens (Group 2B).

Human exposure to OCPs, including DDT, occurs mainly through the food chain according to their fat solubility. They accumulate in fat-rich food products including dairy products, such as cheese, butter and milk.

Quantitative exposure assessment is now widely used to estimate human

exposure to xenobiotics through the consumption of food and therefore to provide a quantitative estimate of possible risks to human health. Risk assessment outputs are the scientific basis for risk management decisions and option analysis.

The new study was conducted to evaluate children's exposure to DDTs (DDT and its metabolites) via dietary milk consumption in Greece, and to assess the respective potential risks to children's health in terms of cancer and non-cancer effects.

(Continued on page 7)

Long-term Use of Mobile and Cordless Phones and the Risk for Glioma

The recent worldwide increase in the use of wireless communications has resulted in greater exposure to radiofrequency electromagnetic fields (RF-EMF). The brain is the main target of RF-EMF where the highest exposure is on the same side of the brain when the handheld phone is used (ipsilateral).

The International Agency for Research on Cancer (IARC) has classified RF-EMF as Group 2B. This means that RF-EMF exposure is 'possibly' a human carcinogen (2011), based on fairly short latency period (time from first exposure until diagnosis), with results on at most the latency group ≥ 10 years.

The previous study reported the first indication of an increased brain tumour risk associated with use of wireless phones some 15 years ago. The brain tumours associated with the use of wireless phones are the malignant types, mostly glioma, and acoustic neuroma, a benign tumour of the 8th cranial nerve.

To study longer periods of use, the new study encompassed two case-control studies on malignant brain tumours with patients diagnosed during 1997-2003 and 2007-2009. They were aged 20-80 years and 18-75 years,

respectively, at the time of diagnosis. Only cases with histopathological verification of the tumour were included.

Exposures were assessed by questionnaire. The whole reference group was used in the unconditional regression analysis adjusted for gender, age, year of diagnosis, and socio-economic index.

The study clearly shows an increased risk for glioma associated with use of both mobile and cordless phones, a risk that increased significantly with latency and cumulative use.

The highest risk was in the longest latency group (>25 years), giving a statistically significant 3-fold increased risk. Overall a high risk was found for use of the third generation (3G; UMTS) mobile.

Use of cordless phones increased the risk with highest risk in the >15 -20 years latency group. The OR increased in a statistically significant manner both per 100h of cumulative use, and per year of latency for mobile and cordless phone use.

Based on the distribution of RF-EMF on the brain, ipsilateral use of both mobile and cordless phones gave a statistically significant increased risk. There was a higher risk for glioma in the

temporal or overlapping lobes, especially for glioma localized only in the temporal lobe.

Children and adolescents are more exposed to RF-EMF than adults due to thinner skull bone, higher conductivity in the brain tissue, and a smaller head. Also the developing brain is more vulnerable than in adults and continues to develop until about 20 years of age.

Glioma risk was analysed in different age groups for first use of a wireless phone and found the highest risk among subjects with first use before 20 years of age in both mobile and cordless phones use. The risk increased further for ipsilateral use for mobile phone use and cordless phone use.

The researchers concluded that glioma and also acoustic neuroma are caused by RF-EMF emissions from wireless phones, which are therefore regarded as carcinogenic, under Group 1 according to the IARC classification, indicating that current guidelines for exposure should be urgently revised. This pooled analysis gives further support to that conclusion regarding glioma.

Source: Pathophysiology, Vol. 22, Pages 1-13, March 2015.

Risk Assessment for Children Exposed to DDT Residues in Various Milk Types from the Greek Market

(Continued from page 6)

The occurrence of residues of DDT and its metabolites was monitored in 196 cow milk samples of various pasteurized commercial types collected from the Greek market. Residue levels were determined by Gas Chromatography-Mass Spectrometry (GC-MS) analysis.

In 97.4% of the samples at least one DDT isomer or one of the DDT metabolites was detected, in levels not exceeding the maximum permitted residue level (MRLs = 40 mg/kg for sum

of DDTs) by the EU (2008). Most of DDT residue levels in milk samples of examined types did not differ significantly.

Exposure assessment scenarios were developed for children aged 1, 3, 5, 7 and 12 years old based on estimated body weights and daily milk consumption.

Hazard Index (HI) for both carcinogenic and non-carcinogenic effects was estimated and the results indicate no significant health risk for children.

However, the highest HI values are at ages 1-3 years old, probably due to the higher ratio of milk consumed relatively to body weight at this age.

Finally, the results of the study support that stricter agricultural policy is needed for the reduction of DDT levels in milk and in other agricultural products.

Source: Food and Chemical Toxicology, Vol. 75, Pages 156-165, January 2015.

CALENDAR OF EVENTS

Advanced International Training Courses in Occupational & Environmental Health August 17-22, 2015 at Chulabhorn Research Institute, Bangkok, Thailand

Course Director: Philip J. Landrigan, MD, MSc (Icahn School of Medicine at Mount Sinai, USA)

Course Coordinator: *Khunying* Mathuros Ruchirawat, PhD (Chulabhorn Research Institute, Bangkok, Thailand)

Course Description:

The course is intended to be an advanced course in occupational and environmental health. It is designed for physicians, medical students, nurses, industrial hygienists, environmental health scientists and other health care workers. Previous participation in the Introduction to Occupational & Environmental Medicine course, which was organized in 2011 and 2013 is advisable, but not a requirement.

The theme of the course is occupational and environmental causes of disease, and approaches to prevention. The course will present information on major current occupational and environmental hazards and the diseases they cause, with particular emphasis on hazards, including emerging hazards, in South and South East Asia. It will describe the scientific methodologies used in occupational and environmental health to assess toxic hazards and to establish linkages between hazards and disease. It will discuss the multidisciplinary nature of research and practice in occupational and environmental health. It will provide information on the on-line literature resources available in occupational and environmental health, and on new advances in e-learning and communication technology. It will also discuss approaches for prevention and control of occupational and environmental diseases, with particular focus on strategies for translating scientific findings into public policy.

The course will combine lectures with supervised case studies in which participants will have the opportunity to work in small groups and to discuss topics directly with faculty of renowned institutes from the USA and Thailand.

These include:

1. **Philip J. Landrigan**, M.D., M.Sc. (Icahn School of Medicine at Mount Sinai, New York, U.S.A.)
2. **Roberto Lucchini**, M.D. (Icahn School of Medicine at Mount Sinai, New York, U.S.A. and University of Brescia, Italy)
3. **Melissa A. McDiarmid**, M.D., M.P.H. (University of Maryland School of Medicine, Maryland, U.S.A.)
4. **William A. Suk**, Ph.D., M.P.H. (National Institutes of Health, NC, U.S.A.)
5. **Khunying Mathuros Ruchirawat**, Ph.D. (Chulabhorn Research Institute, Bangkok, Thailand)

Requirement:

Participants should be part of a network of health sciences professionals involving physicians, medical students, nurses, industrial hygienists, environmental health scientists and other health care workers.

Fellowships:

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

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

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