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INTERNATIONAL CENTRE FOR ENVIRONMENTAL AND INDUSTRIAL TOXICOLOGY (ICEIT)

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"UNEP Centre of Excellence for Environmental and Industrial Toxicology".

International Incidence of Childhood Cancer, 2001-10: A Population-based Registry Study

Cancer is a major cause of death in children worldwide, and the recorded incidence has been increasing. However, during the past two decades, very little internationally comparable data on incidence of childhood cancer has been available.

Since the publication of the International Incidence of Childhood Cancer, volume 1 (IICC-1) in 1988 and IICC-2 in 1998, which covered roughly the 1970s and the 1980s respectively, no internationally comparable data on incidence patterns of childhood cancer have been published.

To address this problem, the International Agency for Research on Cancer (IARC), in collaboration with the International Association of Cancer Registries (IACR), while recognizing the need for a specific approach to the collection and dissemination of childhood cancer data, has coordinated a study to assess the incidence of childhood cancer worldwide, the complete results of which will be published in IICC-3.

A new population-based registry study has collected data on all malignancies and non-malignant neoplasms of the central nervous system (CNS) diagnosed before age 20 years in populations covered by high-quality cancer registries with complete data for 2001–10.

The target age range for IICC-3 is 0–19 years, compared with 0–14 years in IICC-1 and IICC-2. Inclusion of the 15–19 years age group was motivated by the shortage of internationally comparable data for this transition age between childhood and adulthood.

Incidence rates per million person-years for the 0–14 years and 0–19 years age groups were age-adjusted, using the world standard population to provide age-standardised incidence rates (WSRs), with age-specific incidence rates (ASR) for individual age groups (0–4 years, 5–9 years, 10–14 years, and 15–19 years).

All rates were reported for 19 geographical areas or ethnicities by sex, age group, and cancer type. The regional WSRs for children aged 0–14 years were compared with comparable data obtained in the 1980s in IICC-2.

Of 532 invited cancer registries, 153 registries from 62 countries, departments, and territories met quality standards, and contributed data for the entire decade of 2001–10.

Among children aged 0–19 years, 385,509 incident cases were found occurring in 2.64 billion person-years.

The overall WSR was 140.6 per million person-years in children aged 0–14 years (based on 284,649 cases). The most common cancers were leukaemia (WSR 46.4), followed by CNS tumours (WSR 28.2), and lymphomas (WSR 15.2).

In children aged 15–19 years (based on 100,860 cases), the ASR was 185.3 per million person-years. The most common of these were lymphomas (ASR 41.8), and the group of epithelial tumours and melanoma (ASR 39.5).

Incidence varied considerably between and within the described regions, and by cancer type, sex, age, and racial and ethnic group.

(Continued on page 2)

International Incidence of Childhood Cancer, 2001-10: A Population-based Registry Study

(Continued from page 1)

Since the 1980s, the global WSR of registered cancers in children aged 0–14 years has increased from 124.0 to 140.6 per million person-years.

This unique global source of childhood cancer incidence will be used for further aetiological research and to inform public health policy, potentially contributing towards attaining several targets of the Sustainable Development Goals aiming at ensuring healthy lives and promoting wellbeing of all at all ages.

The observed variations of geography, race and ethnicity, age, sex, and time require constant monitoring and research at local, national, and international levels.

Complete documentation and a detailed exposition of the data will be published in both print and electronic formats in the forthcoming IARC Scientific Publication, **International Incidence of Childhood Cancer, Volume III (IICC-3).** More information is available at http://iicc.iarc.fr/about/index.php.

Source: The Lancet Oncology, Vol. 18, Issue 6, Pages 719-731, June 2017.

Residential Exposure to Pesticides as Risk Factor for Childhood and Young Adult Brain Tumors

Descriptive epidemiology of childhood brain tumors (CBTs) has recently been reviewed. Evidence accumulating over the past ten years suggests a positive association between exposure to non-agricultural pesticides and CBTs.

In the United States and Canada, tumors of the brain and central nervous system (CNS) are the most frequent solid tumors and are the second leading cause of cancer-related death in children and adolescents 0 to 19 years of age.

In Europe, primary tumors of the CNS are the second most common (after leukemia) and the most lethal in children 0 to 14 years old.

CBTs include several histologic subtypes. Incidence rates differ with age (higher incidence rates observed in children 0 to 4 years of age in USA and Europe), country (overall incidence varies from 1.12 cases to 5.26 per 100,000 persons in Kuwait and in USA), gender (more common in males) and ethnicity.

The higher incidence rate of CBTs before the age of 5 suggests that both prenatal and early postnatal exposures may be especially important.

Improved survival rates after diagnosis of a CNS tumor have been recorded over the past 40 years. This is mostly due to earlier detection and advances in treatment (e.g., surgical techniques, rational use of postoperative radiation and chemotherapy).

However, despite this progress and decades of epidemiological research, the etiology of CBTs remains largely unclear.

A multifactorial process involving genetic and environmental factors is the most likely explanation. Parental exposure to toxicants during pregnancy may be associated with polymorphisms in genes metabolizing these toxicants.

Children are particularly vulnerable to environmental exposure to pesticides due to physiological and behavioral characteristics, including greater food or fluid intake per body weight, “hand-to-mouth” activity, can increase dose and toxicity as compared to adults.

Children can be exposed to pesticides indirectly from parental pesticide exposure via occupational and para-occupational (“take-home”) exposure.

Most epidemiological studies on the relationship between childhood pesticide exposure and brain cancer have considered parental occupational exposures, but fewer studies assess residential exposures.

The purpose of this study is to systematically review and to meta-analyze the available epidemiological data on the relationship between residential pesticide exposure and childhood brain tumors.

The researchers aim to enhance our understanding of the potential involvement of residential exposure in the etiology of CBT by exploring several variables as potential sources of heterogeneity in results, i.e., the quality of the studies, the sources of pesticide exposure and exposure location, critical exposure periods, specific pesticide category, application methods, type of pest treated, specific exposures, type of CBTs, child age at diagnosis and geographic location.

The present systematic review and a meta-analysis of case-control studies reveal a positive association between residential exposure to pesticides and childhood brain tumors, more particularly indoor exposure, involving insecticides and with gliomas as outcome.

Several factors argue in favor of residential pesticide exposure as an etiological factor or at least as a contributing risk factor for childhood brain tumors. These include the consistency of between-study results, the lack of or very low heterogeneity, the absence of evidence of publication bias and biological plausibility.

It is possible that the large number of comparisons could result in some statistically significant findings by chance. However, the consistency of the results

(Continued on page 4)

Epigenetic Mechanisms Underlying Arsenic-associated Toxicity and the Development of Diabetes

Arsenic is a common carcinogenic element of major health concern. Considerable epidemiological evidence is available that links exposure to organic and inorganic arsenic with various human ailments, particularly cancer and diabetes.

The different types of organic, inorganic and biological forms of arsenic have specific toxicity profiles and mechanisms. The biotransformation and toxicity of both inorganic- and organic arsenical compounds differ from each other in substantial ways.

At the same time, the molecular mechanisms underlying arsenic-induced diabetes and carcinogenesis are still unclear, and exploring the exact mechanism is a new subject of study.

In animal models, arsenic increases the level of glucose and insulin when used in relatively high concentrations.

In *in vitro* studies, the uptake of glucose decreases in insulin sensitive cells and inhibits the factors responsible for insulin signaling transduction and insulin sensitivity.

Experimental evidence indicates that epigenetic alterations may play an important role in the development of diseases linked with exposure to environmental toxicants.

Today, epigenetics provide the ideal basis for understanding the mechanisms underlying toxicant-induced diseases. Epigenetic concepts and the application of epigenetic techniques in the area of human ailments have stimulated interest among researchers.

Inorganic arsenic seems to be associated with epigenetic modifications such as alterations in DNA methylation, histone modifications, and microRNA (miRNA) abundance.

In this review, the researchers focused on current evidence that points to the epigenetic mechanisms associated with arsenic exposure, especially based on the activity of DNA methyltransferases (DNMTs) and the development of diabetes.

It is clear that exposure to arsenic causes alterations in DNA methylation patterns both at gene-specific and global levels. Exposure also causes modifications of histone and altered expression levels of the miRNAs.

The importance of such arsenic-induced epigenetic effects on the development of diabetes is likely to depend on various factors, including genotype, exposure to other toxicants, and other environmental effects such as nutrition.

In spite of the information regarding the epigenetic effects of arsenic on the status of various cells and tissues, there is still a lack of consensus towards the exact mechanism through which arsenic may cause type 2 diabetes (T2D).

Arsenic initiates hyper- and hypo-methylation at the same time, probably both at the gene-specific and genome levels. Thus, hypermethylation occurs at one gene while hypomethylation occurs at another.

The application of next-generation sequencing and microarray technologies might be helpful in identifying the signature genes that experience both hyper- and hypo-methylation after exposure to arsenic.

In this review, the researchers have focused on the epigenetic alterations of arsenic, especially based on the activity of the DNMTs.

The latest developments in the area of methylation-sensitive sequencing and microarrays techniques provide valuable practices which can elucidate epigenetic alterations both at gene specific and genome-wide levels.

In the near future, the next generation of sequencing techniques will help address shortcomings in the studies of genomic-wide and regional changes in DNA methylation patterns.

From an environmental point of view, it is important to consider how best to model relevant human exposures and what techniques should be assigned to investigate epigenetic alterations induced by various toxicants.

In laboratory experiments, the pertinence of animal models to human exposure remains uncertain. However, the near future may bring consensus to the field of epigenetics.

Presently it is difficult to identify a good model system which will reveal the occurrence of disease associated with chronic human arsenic exposure. If it is recognized that epigenetic alterations are constantly being observed in animal models as well as in specific human tissues, it would be a major breakthrough to identify the link between chronic arsenic exposure and the development of T2D.

In conclusion, the study indicates that exposure to low or moderate concentrations of inorganic arsenic is linked with epigenetic effects.

In addition, it is evident that, arsenic can change the components of the epigenome, inducing diabetes through epigenetic mechanisms, such as alterations in glucose transport and/or metabolism and insulin expression/secretion.

Source: Food and Chemical Toxicology, Vol. 107, Pages 406–417, September 2017.

Effects of Intermediate Frequency Magnetic Fields on Male Fertility Indicators in Mice

Exposure to electromagnetic fields is ubiquitous in the environment because of new technologies and novel applications being actively developed and commercialized. Extremely low frequency (ELF) magnetic fields (MF) associated with the distribution and use of electric power and radio-frequency (RF) electromagnetic fields (EMF) from wireless communication devices have been studied extensively for possible health effects.

However, much less data are available on the possible health effects of intermediate frequency (IF) fields between the ELF and RF ranges from 300 Hz to between 100 kHz and 30 MHz. The upper limit depends on how RF is defined.

Intermediate frequency EMF are increasingly used at work and at home in applications such as electronic article surveillance (EAS) systems, induction heating cookers, magnetic resonance imaging machines, and inductively coupled wireless power transmission.

While several studies have evaluated developmental and reproductive effects of maternal exposure to IF MFs, only two studies have addressed possible effects on male fertility. These studies used 10 kHz MFs of the type used in inductively coupled power transmission, or 20 kHz and 60 kHz fields relevant to induction heating cookers.

The new study was conducted to assess possible effects on fertility indicators after long-term exposure to IF MFs. Male C57BL/6J mice were exposed continuously for 5 weeks to 7.5 kHz MF, similar to those emitted by an EAS technology commonly used in supermarkets and other stores to protect merchandise against theft, with the magnetic flux densities used at 12 and 120 µT.

The higher flux density exceeded the International Commission on Non-Ionizing Radiation Protection reference level (100 µT in the frequency range 3 kHz–10 MHz) for occupational exposure, and was higher than the maximum exposure levels (up to 60 µT) found around EAS systems used in supermarkets and libraries.

Sperm cells from cauda epididymis were analysed for motility, total sperm counts, and head abnormalities, as they are considered to be important indicators for detecting the adverse effects of various factors on spermatogenesis. Motile sperm cells were classified as progressive or non-progressive. Testicular spermatid heads were counted as well.

There were no adverse effects on fertility indicators. The body weight development or reproductive tissue weights were not affected by the used IF MFs, and no exposure-related differences were observed in sperm counts or sperm head abnormalities.

Proportion of non-motile cells was significantly decreased in the 120 µT group, and a corresponding increase was seen in the percentage of motile cells (significant in non-progressive motile cells).

In conclusion, no adverse effects on fertility indicators were observed in mice exposed to IF MFs. Increased sperm motility is an interesting finding that needs to be confirmed in further studies.

Source: Environmental Research, Vol. 157, Pages 64–70, August 2017.

Residential Exposure to Pesticides as Risk Factor for Childhood and Young Adult Brain Tumors

(Continued from page 2)

of the present meta-analysis with those observed in this companion meta-analysis on parental occupational exposure to pesticides and childhood brain tumors strongly supports the evidence of a relationship between pesticide exposure and childhood brain cancer.

Even so, the formal causality of pesticide exposure in childhood brain tumors cannot be established mainly due to the rarity of the disease, the absence of positive exposure-response relationships, the scarcity of data on specific histological types of brain tumors, and the lack of precise measurements of specific pesticides.

Future studies can improve exposure assessment through specific pesticide exposure questions using biomarkers and semi-quantitative measures of specific pesticides in environmental or biological specimens to confirm information obtained through questionnaires.

As the critical role of genetic factors in the etiology of several cancers becomes evident, future research focusing on the interaction between genetic and environmental factors and attempts to correlate improved exposure data with genetic predisposition and defined subtypes of CBT would be of great interest.

To this end, well-designed studies including an international consortium of researchers to collect sufficient cases in a reasonable amount of time are required.

While awaiting the results of such studies, prevention may be the most relevant option. Efforts should be made to limit childhood exposure.

Public health policies should be developed for that purpose, including educational measures to increase the awareness of the population, particularly of young couples, women of childbearing age or pregnant women, about the potential impact on children's heath of residential pesticide use.

Source: Environment International, Vol. 106, Pages 69-90, September 2017.

IS ARSENIC AN OBESOGEN?

Over the past 35 years, a four-fold global increase in the prevalence of type 2 diabetes (T2D) has occurred. In 2004, an estimated 422 million adults were suffering from diabetes, reflecting increases in associated risk factors such as overweight and obesity.

Chemicals that promote obesity have been functionally defined as obesogens because they increase the number of adipocytes (hyperplasia) and/or the storage of fat into existing adipocytes (hypertrophia).

Although the obesogen hypothesis is less than 10 years old, approximately 20 environmental chemicals are already known to have obesogenic properties. Diverse compounds have been shown to alter adipocyte differentiation, and several endocrine-disrupting chemicals modulate adipocyte physiology.

In many countries worldwide, arsenic occurs naturally in drinking water from wells or aquifers. From 2001 to 2009, 140 million people are estimated to have been exposed to concentrations above 50 µg/L, which is five-fold higher than the upper limit established by the World Health Organization (WHO).

Arsenic is a recognized risk factor with a specific mechanism for T2D. However, the epidemiological relationship between chronic exposure to high levels of arsenic and obesity development has not been directly established.

Although obesity and T2D are not completely equitable, obesity is one of the major risk factors for T2D development.

The mechanisms by which arsenic increases the risk of T2D involve the impairment of glucose-stimulated insulin secretion in pancreatic beta-cells, the induction of pancreatic oxidative damage and insulin resistance in skeletal muscle, increment of gluconeogenesis in the liver, and modulation of other hepatic insulin signaling.

There is, however an integrated computational systems biological approach that examines possible pathogenetic linkages between environmental chemicals to genes and proteins involved in T2D through genome-wide associations, disease similarities, and published epidemiologic and experimental evidence. Studies using this approach put arsenic among the top 10 environmental chemicals potentially linked to T2D. Moreover arsenic also has literature-based associations with obesity.

Thus, arsenic has been considered an important risk factor for T2D, though its obesogenic effects are uncertain, because the effects of arsenic on white adipose tissue (WAT) have been explored non-specifically and with diverse objectives.

Because adipose tissue is a central metabolic organ in the regulation of whole-body energy homeostasis, adipose dysfunction is a critical step in obesity. In particular, WAT functions as a key energy reservoir for other organs. It secretes various hormones, cytokines, and metabolites (termed as adipokines), as well as free fatty acids that control systemic energy balance by regulating appetite signals in the central nervous system and metabolic activity in peripheral tissues.

The aim of this review is to compile the available information about the direct and transgenerational effects of arsenic on adipocytes in order to better understand how arsenic exposure can contribute as a risk factor to obesity and the onset of T2D by specifically focusing on roles that do not lead to pancreas, skeletal muscle or liver impairment.

The impairment of WAT metabolism is crucial in the onset of obesity, and there are some studies which have evaluated the effects of arsenic on it. Only some of these studies, however, were obesity-related.

The known effects of arsenic on WAT/adipocytes have been integrated, based on the diverse metabolic and physiological processes that occur in WAT and which are altered in obesity, specifically: adipocyte growth, adipokine secretion, lipid metabolism, and glucose metabolism.

Weight gain impairs the proper function of many organs, particularly adipose tissue itself. In this sense, the majority of the effects of arsenic evaluated both *in vitro* and *in vivo* on adipose tissue that are known until now suggest that arsenic can negatively affect adipocytes/WAT metabolism.

While arsenic diminishes adipogenesis in pre-adipocytes, it can concomitantly increase adipocyte size or WAT weight.

Arsenic increases basal lipolysis *in vitro* as well as lipogenesis at fasting, reduces basal glucose uptake and ISGU, and down-regulates adiponectin mRNA expression.

The transgenerational effects of arsenic include alterations in birth weight, higher postnatal weight gain with elevated body fat content, glucose intolerance, insulin resistance, and increased serum triglyceride. Trans-generational effects of arsenic also include elevation of leptin in cord blood, in the placenta, and in postnatal serum levels.

Although there is evidence that arsenic alters various aspects of adipose tissue metabolism, it is still not known, as for many endocrine-disrupting chemicals, if its mechanism of action is to promote weight gain by adipocyte hypertrophy directly or primary.

There is evidence that arsenic interacts with other environmental factors, such as high-fat diets, or folate-related nutrients, to modulate positively or negatively its obesogenic effects or arsenic-related diseases. The metabolism of arsenic also plays an important role in its effects.

The metabolism of arsenic is influenced both by polymorphisms in enzymes involved in arsenic metabolism, particularly in arsenic (+3 oxidation state) methyltransferase (known as AS3MT), and by body mass index (BMI). However there is still controversy related to this last point.

To conclude, the currently available information suggests that arsenic can negatively affect WAT metabolism, resulting in arsenic being a potential obesogen. More studies are needed to address this issue accurately.

Source: Molecular and Cellular Endocrinology, Vol. 452, Pages 25-32, September 2017.

Serum Levels of Heavy Metals in Relation to Coronary Artery Disease in the Elderly

There is a continuing attempt to identify novel factors that can predict the risk of cardiovascular disease beyond the established coronary risk factors.

It has been observed that environmental factors and chemical pollutants may also affect the susceptibility to coronary artery disease (CAD).

Among the novel coronary risk factors are heavy metals. They are commonly defined as metalic elements having a specific density of more than 5 g/cm3 such as lead, mercury, aluminum, nickel and cadmium. Of these metals, lead (Pb), cadmium (Cd) and mercury (Hg) are among the most threatening to human health. They are ranked in the top 10 list of hazardous substances according to the Agency for Toxic Substances and Disease Registry (ATSDR).

People mostly come in contact with and are exposed to these metals through occupational exposure, water and soil pollution, or consumption of contaminated food, particularly plants and fruits grown in contaminated soil. Prolonged exposure to low doses of heavy metals is a matter of concern because the body excretes these pollutants slowly, so they accumulate in different organs over the years.

Heavy metal toxicity can counteract biological antioxidant defense and promote oxidative stress via increased free radical generation, DNA damage and lipid peroxidation. Heavy metals can also impair endothelial homeostasis via inactivation of the vasorelaxant factor, nitric oxide. Direct damage to arterial walls may subsequently cause cholesterol accumulation, foam cell formation and atherosclerosis.

The present study aimed to evaluate serum concentrations of lead (s-Pb), mercury (s-Hg) and cadmium (s-Cd) in patients with CAD in comparison with healthy individuals in Isfahan, Iran. The correlation between serum levels of these heavy metals and lipid profile parameters, including serum total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C) and TC:HDL-C ratio, was also investigated.

Isfahan is an industrial province in Iran. Several aluminum, steel and other metal melting industries are located in this province. According to a previous study, sewage and industrial waste from Isfahan metal melting and steel factories contain high levels of Pb and Cd. The sewage, especially, enters the environment and pollutes both soil as well as ground and surface water.

It was observed that the mean concentration of s-Pb and s-Cd and s-Hg was significantly higher in CAD patients compared with control subjects. The same result was also obtained after adjusting for cardiovascular risk factors including age, dyslipidemia, diabetes mellitus and hypertension.

The mean concentration of TC, HDL-C and TC:HDL-C ratio was significantly higher in CAD patients. There was no significant association between serum metal concentrations with TC, HDL-C and TC:HDL-C ratio.

In conclusion, these results indicate elevated serum levels of three important heavy metals (Pb, Cd and Hg) in patients with angiography-defined CAD. Raised serum levels of these elements were also found to be independent of serum TC and HDL-C concentrations.

Long-term exposure to trace levels of Pb, Cd and Hg may play a role in the development of coronary atherosclerotic plaques.

Further studies are warranted to estimate the cardiovascular risk that is conferred by elevated serum levels of Pb, Hg, and Cd, and mechanisms underlying the detrimental effects of these elements on heart and vessels.

Source: Chemosphere, Vol. 180, Pages 540-544, August 2017.

Heart Toxicity Induced by Chronic Arsenic Exposure in Rats

Arsenic is a toxic metalloid widespread in natural environment. Millions of people have been exposed to arsenic through environmental, agricultural and occupational routes. One of the major threats to human health is contaminated drinking water.

Arsenic has been classified as a carcinogen. A significant relationship has been observed between arsenic levels in ground water and mortality from skin, lung, kidney and bladder cancers.

Arsenic is also associated with a wide range of other chronic illnesses, such as diabetes, hypertension, neurological disorders, gastro intestinal disturbance, and dermal, liver, renal and vascular diseases.

The adverse effects of arsenic exposure on the cardiovascular system are well documented. However, the mechanisms responsible for arsenic-induced heart toxicity remain not unclear.

Compared with conventional toxicological end point assays, proteomics technology, which provides global protein information, is a much more powerful tool for investigating the toxic mechanisms triggered by exposure to environmental pollutants

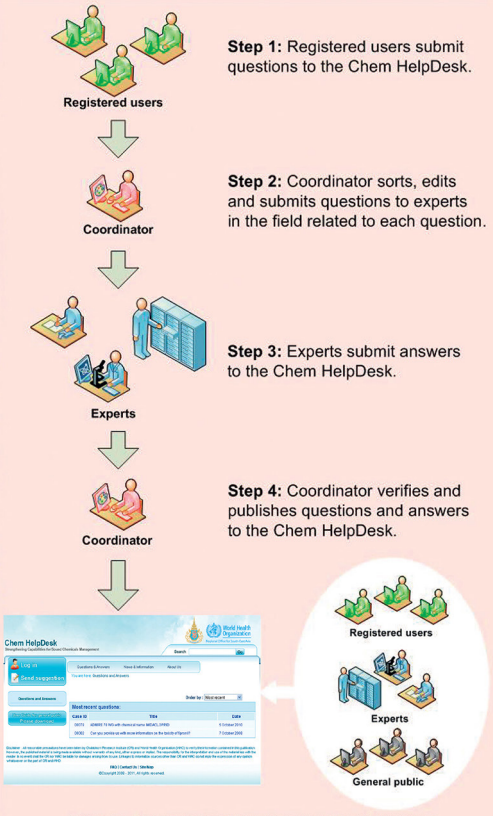
The present study uses a high-throughput label-free quantitative proteomics approach to investigate alterations in proteomic profiles of rat heart tissue after long-term exposure to arsenic. The signaling pathways in relation to arsenic action were also analyzed.

(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**

Heart Toxicity Induced by Chronic Arsenic Exposure in Rats

(Continued from page 6)

The results may offer comprehensive insights into the mechanisms of arsenic-induced cardio toxicity, and could help develop potential biomarkers for human health risk assessment of environmental arsenic exposure.

The investigation found that the abundance of 81 proteins was significantly altered by arsenic treatment (35 up-regulated and 46 down-regulated).

Among these proteins, 33 were specifically associated with cardiovascular system development and function, including heart development, heart morphology, cardiac contraction and dilation, and other cardiovascular functions.

The research results suggest that the aberrant regulation of 14 proteins induced by arsenic mediated via the Akt/p38 MAPK signaling pathway, disturbs cardiac contraction and relaxation, impairs heart morphogenesis and development, and induces thrombosis in rats.

Although the results cannot be applied directly to humans, these data are expected to provide novel insights into the molecular mechanism and biomarker discovery for arsenic-induced cardiovascular disease.

The associations of detected protein dysregulation with arsenic exposure should be further investigated in human populations to validate the proposed mechanisms derived from rat models.

Overall, these findings will augment the knowledge of the involved mechanisms and develop useful biomarkers for cardiotoxicity induced by environmental arsenic exposure.

Source: Environmental Pollution, Vol. 229, Pages 210-218, October 2017.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute

Schedule for 2017 - 2018

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

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

Course Description:

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

The course is an integration of science and policy, covering the fundamental basis of environmental and health risk assessment and management, from identification of hazard, assessment methods, the mode of action and human relevance framework, the inherent uncertainties in each step, the relationship between risk assessment and risk management, and the need for open, transparent and participatory acceptance procedures and credible communication methods. Emphasis is placed on human health risk assessment, although the principles of ecological risk assessment will also be covered. The course teaches the practical application of risk assessment methods to various problems, e.g. hazardous waste site release, through the use of case studies relevant to problems faced in developing countries, and describes the policy context in which decisions to manage environmental health risks are made. Teaching and learning aids such as electronic distance learning tools and IPCS risk assessment toolkit will be introduced.

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

2. Detection of Environmental Pollutants, Testing and Screening of Toxicity (February 2018)

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

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