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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 23rd, 2018, 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, for the annual review of progress on collaborative activities previously agreed upon 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 in the technical discussions attended by a team of senior researchers from CRI on February 21st and 22nd, covered the following areas: (1) CRI's capacity building/training programmes in Chemical Safety, Toxicology and Environmental Health, including in-country training that was conducted in Sri Lanka on Risk Assessment and Risk Management of Chemicals in 2017; (2) further development of training modules/courseware for both face-to-face and web-based training in chemical safety, including the use of the developed electronic distance learning tool on risk assessment and risk management of chemicals and a module on the WHO Global Environmental Monitoring System/Food Database; (3) enhancing and widening information dissemination networks, e.g. for raising awareness and disseminating information related to chemical safety and risk assessment, including a database of available training courses in risk assessment and risk management of chemicals; and (4) possible research collaborations in the area of children's environmental health.

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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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CRI organized 4 international training courses in 2017, attended by 57 participants from 16 countries, and 4 have been tentatively planned for 2018. These courses are open to participants from developing countries, primarily from the Asia Pacific region, and taught by international experts from world-renowned academic and research institutions with a wealth of teaching experience in the region.

Courses tentatively scheduled for 2018 include:

	Training Course	Time
1	Detection of Environmental Pollutants and Monitoring of Health Effects	February 5 - 16, 2018
2	Environmental Toxicology	April 19 - 27, 2018
3	Environmental Immunotoxicology and Reproductive Toxicology	October 1 - 12, 2018
4	Environmental and Health Risk Assessment and Management of Toxic Chemicals	December 2018

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.

Cardiovascular Effects of Air Pollution

Air pollution is becoming a major public health issue. It has been clearly implicated in millions of premature deaths worldwide. In particular, 60-80% of air pollution-related deaths give clear evidence of serious cardiovascular diseases.

Air pollution is composed of particulate matter (PM) and gaseous pollutants, such as nitrogen oxides, ozone, sulphur dioxide, volatile organic compounds and carbon monoxide.

PM is classified according to size as coarse particles (PM_{10} with diameter < 10 μm, ≥ 2.5 μm), fine particles ($PM_{2.5}$ with diameter < 2.5 μm, ≥ 0.1 μm) and ultrafine particles (nanoparticles, diameter < 0.1 μm).

In 2015 the European Society of Cardiology launched a campaign to "raise awareness of the detrimental effects that the environment can have on the heart". As part of this effort, an original review of the scientific evidence from epidemiological and experimental studies was performed to examine the cardio-vascular effects of outdoor air pollution.

The results showed that, in addition to differences between countries and continents, strong differences exist between the main sources of pollutants within individual countries, depending on local sources.

Road traffic in large cities is a major contributor to global pollutant emissions, and is also the main source of NO_2 which arises mainly from diesel vehicles.

Pooled epidemiological studies reported that a 10 μg/m^3 increase in long-term exposure to $PM_{2.5}$ was associated with an 11% increase in cardiovascular mortality.

Increased cardiovascular mortality was also related to long-term and short-term exposure to nitrogen dioxide.

Exposure to air pollution and road traffic was associated with an increased risk of arteriosclerosis, as shown by premature aortic and coronary calcification.

Short-term increases in air pollution were associated with an increased risk of myocardial infarction, stroke and acute heart failure.

Although the cardiovascular risk increases with level and duration of exposure, all the studies concluded that there is no safe threshold below which there is no effect.

Reinforcing the evidence from epidemiological studies, numerous experimental studies demonstrated that air pollution promotes a systemic vascular oxidative stress reaction.

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HRH Princess Chulabhorn Mahidol Co-Authors the Lancet Commission on Pollution and Health Report that Outlines Massive Scope of Health and Economic Costs of Air, Water and Soil Pollution

A report published in **the Lancet Commission on Pollution and Health** in October 2017 analyzed and communicated the massive scope of the health and economic costs of air, water and soil pollution.

Through analyses of existing and emerging data, the report revealed pollution's severe and under-reported contribution to the Global Burden of Disease. It uncovered the economic costs of pollution to low- and middle-income countries and compared the costs of inaction to the costs of available solutions. It highlighted the burden that pollution places on health and economic development, and about cost-effective pollution control solutions and strategies.

Key findings in the report include:

1. Pollution Causes 16% of All Deaths Globally - In 2015, diseases caused by pollution were responsible for an estimated 9 million premature deaths, 16% of all deaths worldwide, 3X more deaths than AIDS, tuberculosis, and malaria combined, and 15X more than all wars and other forms of violence. It killed more people than smoking, hunger and natural disasters and, in some countries, accounted for one in four deaths.

2. Pollution Disproportionately Kills the Poor and the Vulnerable - Nearly 92% of pollution-related deaths occur in low- and middle-income countries, with the toll being greatest in poor and marginalized communities. Children face the highest risks because small exposures to chemicals in utero and early childhood can result in life-long disease, disability, premature death, as well as reduced learning and earning potential.

3. Pollution is Closely Tied to Climate Change and Biodiversity - Fossil fuel combustion in higher-income countries and biomass burning in lower-income countries accounts for 85% of airborne particulate pollution. Major emitters of carbon dioxide are coal-fired power plants, chemical producers, mining operations, and vehicles. Switching to cleaner sources of energy will reduce air pollution and improve human and planetary health.

4. Pollution is Neglected by Funding Agencies Worldwide - Despite significant health impacts, the international development and health agendas have largely overlooked pollution. Funding is sparse when compared to resources for infectious disease and other environmental issues. No large foundations include environmental health and pollution as a focal area. Despite more than 70% of diseases caused by pollution being non-communicable, interventions against pollution are barely mentioned in the Global Action Plan for the Prevention and Control of Non-Communicable Diseases.

5. The Cost of Inaction Is High, While Solutions Can Yield Economic Gains - Pollution-related diseases account for up to 7% of health budgets in middle-income countries. Welfare costs are estimated at $4.6 trillion per year, equivalent to 6.2% of the global GDP. In the US, each dollar invested in air pollution control returned $30 in benefits since 1970. Higher IQs and increased productivity from removing lead from gasoline has returned an estimated $200 billion each year since 1980. Transition toward a circular economy will reduce pollution-related disease and improve health. Decoupling development from the consumption of non-renewable resources will minimize the generation of pollution and other forms of waste by recycling and reuse.

6. The Path Forward Is Clear - Actions for governments include (1) integrating pollution challenges and control strategies into planning processes, and prioritizing programs according to health and economic impact, (2) asking for support from development assistance agencies, and (3) designing and implementing programs that reduce pollution and save lives. Actions for donors, foundations, and individual philanthropists include pollution planning, interventions, and research in their strategies. Actions for people affected by pollution include visiting www.pollution.org to review data related to toxic exposures in their neighborhoods, documenting and uploading their own stories, and connecting with agencies that can help with solutions.

This is the first global analysis of the impacts from all forms of pollution (air, water, soil, occupational) together, as well as exploring the economic costs and social injustice of pollution. The economic costs are enormous.

The report features solutions and recommends how the problem can be solved. It also includes examples and case studies of pollution control success. It details gaps in our data and knowledge, showing a need for research into soil, heavy metals and chemicals related to the burden of disease as these aspects do not have fully-defined risk factors and therefore are significantly under-estimated. Other gaps in knowledge related to exposures, and disease correlations are also indicated, including those associated with developmental neurotoxicants, endocrine disrupters, new classes of pesticides, chemical herbicides, and pharmaceutical wastes.

The report highlights aspects of pollution that have been largely ignored in development circles, despite their significant toll on human health. The report aims to elevate pollution to an issue of global scale, ready for solutions. The authorship is rich and deep in policy and political figures, as well as academics. Input was provided by major global actors, including the World Bank, UNEP, UNDP, European Union, and dozens of bilateral and international organizations, all under the umbrella of the Global Alliance on Health and Pollution. The preparation of the report already has seeded results.

UN Environment's next global conference will focus exclusively on pollution and the WHO has increased its focus on pollution-related disease. Importantly, the connection between pollution and the Sustainable Development Goals (SDGs) was made, showing pollution playing a part in achieving many aspects of the SDGs.

Source: The Lancet, Vol. 391, No. 10119, Pages 462–512, February 2018.

Particulate Matter and the Risk of Chronic Kidney Disease

Outdoor air pollution has long been linked to major health conditions such as heart disease, stroke, cancer, asthma, and chronic obstructive pulmonary disease.

Elevated levels of fine particulate matter <2.5 μm in aerodynamic diameter ($PM_{2.5}$) are associated with increased risk of cardiovascular complications and death, but their association with chronic kidney disease (CKD) and end stage renal disease (ESRD) is unknown. CKD is diagnosed in five stages, the last of which is ESRD.

Experimental laboratory evidence in murine models suggests that exposure to deep exhaust particles leads to disturbances in renal hemodynamics, and promotes oxidative stress, inflammation, and DNA damage in renal tissue. Such exposure also appears to exacerbate acute kidney injury, and further promulgate chronic renal injury in mice.

Data is lacking on the relationship between air pollution and kidney disease in humans.

These experimental and clinical findings provide biological plausibility and support for the hypothesis that environmental exposure to elevated levels of $PM_{2.5}$ is associated with increased risk of kidney disease.

There have been as yet no major national, longitudinal or epidemiologic studies to determine, whether exposure to elevated levels of $PM_{2.5}$ is associated with increased risk of developing or aggravating CKD.

The present study linked the databases of the Environmental Protection Agency and the Department of Veterans Affairs to build a national longitudinal cohort of more than 2 millions United States veterans in an attempt to characterize the relationship between $PM_{2.5}$, the risk of incident CKD, and the progression to ESRD.

Survival models were used to evaluate the association of $PM_{2.5}$ concentrations and the risk of incident eGFR <60 ml/min per 1.73 m^2, incident CKD, eGFR decline ≥30%, and ESRD over a median follow-up of 8.52 years.

Researchers observed a linear relationship between $PM_{2.5}$ concentrations and the risk of incident CKD and progression to ESRD. The results were consistent where baseline exposure was defined as the annual average $PM_{2.5}$ concentrations in the year 2004, and where exposure was time-varying to reflect movement of cohort participants and changes in $PM_{2.5}$ concentrations over the years.

The researchers also examined a range of kidney outcomes, including development of kidney disease, kidney function decline (eGFR decline ≥30%), and the terminal outcome of ESRD.

The results consistently showed a linear relationship between $PM_{2.5}$ levels and risk of kidney outcomes. The findings were robust in sensitivity analyses including the examination of different distance thresholds from an air monitoring station, and analyses evaluating associations within metropolitan areas.

The results were also consistent in analyses using ambient $PM_{2.5}$ estimates derived from NASA's satellite data. Ambient sodium concentration in the air (used as a negative control) was not associated with increased risk of adverse renal outcomes.

This constellation of research findings suggests that chronic exposure to fine particulate air pollution is a significant risk factor for the development and progression of kidney disease in humans.

They provide a quantitative assessment of the potential reduction in the burden of CKD that could be achieved with improved air quality in the United States, and point to a need for a broader assessment of the global burden of kidney disease attributable to air pollution.

These results demonstrate a significant association between $PM_{2.5}$ concentrations and the risk of development of kidney disease and its progression to ESRD.

It will be important to identify air pollution as a potential contributor to kidney disease. The issue of air quality will have an impact on national and global burdens of disease estimates, will stimulate further policy discussion on the importance to public health of curbing air pollution, and will contribute to the public's better understanding of the vital importance of clean air.

Source: Journal of the American Society of Nephrology, Vol. 29, No. 1, Pages 218-230, January 2018.

Cardiovascular Effects of Air Pollution

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Radical oxygen species induce endothelial dysfunction, monocyte activation and some proatherogenic changes in lipoproteins, which initiate plaque formation.

Furthermore, air pollution favours thrombus formation, because of an increase in coagulation factors and platelet activation.

Combustion-derived particles have strong adverse effects attributable to their small sizes (ultrafine particles) and to the polycyclic aromatic hydrocarbons and metals that they carry on their surface.

While ultrafine particles have a major cardiovascular effect, their concentrations in ambient air are strongly underestimated by actual standards and measurements.

Air pollution is a major contributor to cardiovascular diseases. Promotion of safer air quality appears to be a new challenge in cardiovascular disease prevention.

As recently endorsed by European Society of Cardiology, the available scientific evidence supports the plan that efforts to reduce exposure to air pollution should urgently be intensified and supported by appropriate and effective legislation.

Source: Archives of Cardiovascular Diseases, Vol. 110, Issue 11, Pages 634-642, November 2017.

Metals and Neurodegenerative Diseases

Neurodegenerative processes encompass a large variety of diseases with different pathological patterns and clinical presentation such as Amyotrophic Lateral Sclerosis (ALS), Alzheimer's Disease (AD) and Parkinson's disease (PD).

Although genetic mutations have a known causative role, the large majority of cases have a multifactorial aetiology in interactions between genotype, lifestyle and environmental factors.

Among environmental risk factors, metals are gaining increasing attention because a large percentage of population is exposed to industrial and chemical pollution through food, air and water.

Of particular importance, oxidative stress and neurodegeneration have been reported as consequences of toxic exposures to essential metals, along with dyshomeostasis in essential metal metabolism.

Exposure to metals has been hypothesized to increase oxidative stress in brain cells leading to cell death and neurodegeneration.

The neurotoxicity of metals has been demonstrated by several *in vitro* and *in vivo* experimental studies. It is likely that each metal is toxic through its own specific pathways.

The possible pathogenic role of different metals has become more of an issue in epidemiological evidence coming from occupational and ecological studies.

Through the production of reactive oxygen species (ROS) and interaction with cell signalling pathways, metals may induce DNA damage and lead to apoptosis.

By evaluating the metals concentration in different biological specimens such as blood/serum/plasma, cerebrospinal fluid (CSF), nail and hair, several case-control studies have attempted to assess the possible association between metals and neurodegenerative disorders. However, the results have often been conflicting.

The aim of the present study is to provide an updated summary of evidence in the literature on possible relationship between metals, with ALS, AD and PD representing the main neurodegenerative disorders.

The possible relationship between metals and ALS is supported in some occupational cohort studies. Some spatial clusters have been identified, in particular, the exposure to lead and selenium.

Several case-control studies have evaluated metal concentrations in biological specimens such as blood and CSF in ALS cases and control subjects, but these results have also been inconsistent.

A link between metals and pathological processes has been hypothesized also for AD. Although epidemiological and case-control studies have found evidence only for aluminum, some relevant data also points to the role of mercury as a disease-associated factor.

A few epidemiological and case-control studies suggest a link between the PD and exposure to metals.

However, based on different meta-analyses, evidence remains insufficient to support a significant association between iron serum levels and PD. Bias and the methodological heterogeneity of available studies complicate assessment. Less heterogeneity among studies would facilitate the process of summing-up results.

Data about a possible positive and significant association between manganese and PD seems more consistent, based on the results of long-term exposure and on detected differences in serum levels compared to controls. Even so, evidence from previous meta-analysis shows that manganese exposure is not associated with an increased risk of PD. These results, however, may not preclude the possibility that high manganese exposure can lead to a manganism-related parkinsonism.

Since results are not unequivocal, no clear relationship can be drawn from the literature regarding other metals and other neurological diseases.

To repeat, assessments of the concentration of metals in different biological specimens (blood, CSF, nail etc) in previous case-control studies, whether positive, negative or without association, have not shown any general agreement.

The most important consideration concerns the timing of the events. Retrospective case-control studies generally involve prevalent cases, while metal concentrations in biological samples reflect current rather than past exposure.

Thus, since the pathogenesis of neurodegenerative diseases considered in this review is thought to begin several years before the onset of symptoms, their use could be inadequate, and a possible reverse causality cannot be ruled out.

Furthermore, there is typically such a long interval of time from the biological to the clinical onset of neurodegenerative disorders (e.g. PD), the use of incident cases does not help in determining that it is very difficult to determine the exact sequence of the events.

Finally, the heterogeneity of the biological samples, including blood, CSF, nail, hair or urine and the different techniques used to evaluate the concentration of metals also need to be taken into account in interpreting the results.

Clearly, a number of physiological factors can interact with the concentration of different metals in different biological specimens. It is therefore imperative that these variables be taken into account when performing such studies.

Although it is highly possible that metals can play a role in the risk of neurodegenerative disease, there is still insufficient evidence in retrospective case-control studies.

Therefore, based on these selected epidemiological and case-control studies, this research concludes that no causal relationship has been established between most of the evaluated metals exposure and the selected neuro-degenerative diseases. Possible reasons for the lack of association as well as the conflicting results provided by the studies should be considered in terms of specific methodological issues.

Source: Environmental Research, Vol. 159, Pages 82-94, November 2017.

Exposure to Traffic-related Air Pollutants as a Potential Risk Factor for Amyotrophic Lateral Sclerosis (ALS)

Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative disease, affecting approximately 450,000 individuals worldwide. In ALS, motor neuron loss results in paralysis of limbs, speech and swallowing difficulties, and eventual respiratory failure.

ALS has a median survival time of just under three years after the onset of symptoms. The disease results from a complex set of genetic and exogenous factors. However, the vast majority of cases occur in people whose family history includes no such factors.

Long-term exposure to air pollutants has been linked to increased mortality rates, especially in cases of cardiovascular and respiratory diseases. It has also been implicated, though to a lesser extent in neurodegenerative diseases, including Parkinson's and Alzheimer's diseases.

The increasing evidence of a possible link between air pollution and neurodegenerative diseases, together with the observation of an association between smoking and the development of ALS, suggest the possible involvement of fine particulates in the etiology of ALS.

It has been hypothesized that ultrafine airborne particles are able to cross or impair the blood-brain barrier after systemic translocation, leading to chronic brain inflammation, microglia activation, oxidative stress, and white-matter abnormalities, which are potential biological pathways contributing to ALS.

The present study investigated the association between multiple air pollutants and the risk of ALS using historic residential data from a large population-based, case-control study which included more than 900 ALS cases and exposure data from the European Study of Cohorts for Air Pollution Effects (ESCAPE) project.

Using home addresses, the researchers estimated exposures of the participants to six measures of air pollution: the nitrogen oxides NO_2 and NOx; three measures of particulate matter ($PM_{2.5}$, PM_{10}, and PM_{coarse}, which is the fraction of PM calculated as the concentration of PM_{10} minus that of $PM_{2.5}$); and fine particulate matter absorption ($PM_{2.5}$ absorbance, a marker for black soot or carbon).

The report from this large population-based, case-control study presented evidence of the association between long-term exposure to traffic-related air pollution and increased susceptibility to ALS.

Of the various air pollutants studied here, $PM_{2.5}$ absorbance, NO_2 and NOx are primary traffic-related pollutants which have larger spatial concentration differences in urban areas. In contrast, major contributors to inhalable and especially coarse particles (PM_{10} and PM_{coarse}) are secondary road dust, agricultural materials, and construction industries.

The results showed an increased risk of ALS associated with long-term exposure to air pollution, specifically $PM_{2.5}$ absorbance and the nitrogen oxides. Only the association with $PM_{2.5}$ absorbance and NO_2 persisted after adjustment for the degree of urbanization.

The study provides new clues for pathogenic pathways in ALS, which will ultimately help to improve understanding of the mechanisms involved and which ultimately may lead to prevention strategies.

As this was the first large population-based study to collect such evidence and to draw such conclusions, it will be important that the findings be replicated in similar studies.

The increased risk of developing ALS due to ambient air pollution was observed at levels well below the existing European annual mean limits of 25 $\mu g/m^3$ for $PM_{2.5}$, and 40 $\mu g/m^3$ for PM_{10} and NO_2 (European Parliament and the Council of the European Union 2008).

Because ambient air pollution levels can be improved, these data support the necessity for increased public health interventions to regulate and limit public exposure to traffic-related air pollutants.

Source: Environmental Health Perspectives, Vol. 126, No. 2, February 2018.

Air Pollution and Type 2 Diabetes: A Large Cohort Study

The links between long-term exposure to air pollution and several health outcomes, including natural and cause-specific (cardiovascular and respiratory) mortality and incidence of various debilitating diseases, including chronic obstructive pulmonary disease, cerebrovascular events, acute coronary events, have been widely studied.

Fine particles ($PM_{2.5}$), nitrogen dioxide (NO_2), and ozone (O_3) have also been recognized as important risk factors for human health.

Among the health outcomes investigated, diabetes has been suggested as positively associated with long-term exposure to air pollution. This relationship is supported by mechanistic hypotheses involving endothelial dysfunction and hyper-activity of the sympathetic nervous system.

The hypothesis that air pollution is associated with diabetes is extremely relevant as the World Health Organization (WHO) has estimated that this metabolic disorder afflicts > 9% of the global population (WHO, 2014) and entails severe health consequences.

Although their results remain controversial, several studies and systematic reviews have investigated the role of air pollution in the occurrence of the most common form of diabetes that involves adult subjects, type 2 diabetes.

The present study aimed to evaluate the potential association

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Type 2 Diabetes Mellitus and Alzheimer's Disease

A number of studies over the past two decades have suggested that Type 2 diabetes mellitus (T2DM) patients have an increased risk of developing Alzheimer's disease (AD). Both disorders are age-related.

These findings have global implications in the coming decades for the world's increasingly aging populations. By 2030, AD is likely to directly affect nearly 65.7 million people worldwide. At the same time, it is estimated that 552 million people will be suffering from T2DM in the one of the world's fastest growing chronic disease epidemics.

Observational studies increasingly link T2DM and AD to exposure to overlapping environmental factors, including lifestyle, smoking, diet and physical activity, as well as environmental/ occupational toxicants, air pollution, pesticides, and heavy metals. In the etiology of both diseases, several common molecular pathways to cellular and metabolic dysfunction have been implicated.

Collectively, environmental and occupational studies provide strong evidence that air pollution and pesticides are associated with greater risk of T2DM, and there is increasing evidence for a link with AD. For example, particulate matter and traffic-related air pollution have been widely associated with T2DM, while multiple studies have associated such exposure with AD or disrupted cognitive function.

Organochlorine (OC) and organophosphate (OP) pesticides have been associated with T2DM in multiple independent populations. Two populations demonstrated increased risks for Alzheimer's associated with OC and OP exposures. Other studies, though limited in assessment, have reported increased risk of AD with any pesticide exposure.

The present study reviews the emerging evidence from observational studies investigating the relationship between T2DM and AD and with shared environmental risk factors, specifically air pollution and pesticides both associated with these chronic disorders.

The researchers hypothesize that these shared environmental risk factors may initiate relevant pathogenic events, with T2DM exacerbating neuronal and metabolic dysfunction, thereby further increasing the risk of developing AD.

The etiology of T2DM and AD is complex and heterogeneous. Researchers relying on medical records and aging cohorts have previously linked air pollutants and pesticides to both T2DM and cognition, sometimes in the same studies.

In future research, T2DM should also be investigated as both a mediator and a modifier between exposure and cognition. In addition to environmental factors generally, it will be important to consider relevant features of exposures such as the types and timing of exposures and the mixtures of toxicants.

Methods of ambient air pollution exposure assessment have been reviewed and have been steadily improved over the past decades.

For pesticides, one research challenge is to address a multitude of sources (e.g., occupational, home and gardening, diet, and proximity to agriculture), as well as the large number of different chemical compounds and classes that are involved and which change over time. For some compounds, such as the POPs, biomarkers may be the best option.

With chronic diseases such as T2DM and AD, long-term low-level exposures are probably important.

Ambient monitoring for air pollution has become widespread in the USA since the 1990s, creating a basis for future research of long-term exposures based on address histories. Publicly accessible databases of commercial pesticide use do exist, such as the California Pesticide Use Reports, however, few countries record such information.

Ultimately, careful consideration of environmental factors attention to the complexities of exposure assessment, and focus on exposures related to both T2DM and AD could be key to new insights on shared disease mechanisms. Such research could be instrumental in shaping innovative preventative measures and policy decisions.

It will be important to elaborate on the role of shared environmental risk factors which contribute to these disorders. They include but are not limited to air pollution and pesticides. How metabolic dysfunction may modify the impact of these exposures on cognitive decline is a question with global implications for the future.

Source: Current Environmental Health Reports, Vol. 5, Issue 1, pp 44–58, March 2018.

Air Pollution and Type 2 Diabetes: A Large Cohort Study

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between long-term exposure to particulate matter (PM_{10}, $PM_{2.5–10}$, $PM_{2.5}$), soot $PM_{2.5}$ absorbance, nitrogen oxides (NO_2, NOx), and ozone (O_3) and adult diabetes in the residence of a large administrative cohort in Rome (Italy). The study also tested for the possible modifying effects of sociodemographic factors and preexisting health conditions.

The risk of diabetes in this large population-based cohort was associated with long-term exposure to nitrogen oxides and ozone.

The results showed positive associations between exposure to nitrogen oxides and prevalence of diabetes at baseline, and between exposure to NOx and O_3 and incidence of diabetes among 35^+ year olds. No association was found for the other pollutants in the survival analysis.

Higher modifying effects estimate were seen in women for O_3 compared to men. Furthermore, subjects aged < 50 years were found to have a stronger association between NOx and O_3 exposure and risk of diabetes than other age groups.

This is the first European study that evaluates an association between exposure to O_3 and incidence of diabetes.

Source: Environment International, Vol. 112, Pages 68-76, March 2018.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute
Schedule for 2018

	Training Course	Date	Duration	Closing Date
1.	Environmental Immunotoxicology and Reproductive Toxicology	October 1 - 12, 2018	2 weeks	July 31, 2018
2	Environmental and Health Risk Assessment and Management of Toxic Chemicals	December 2018	2 weeks	September 30, 2018

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

Course Description:

Environmental Immunotoxicology and Reproductive Toxicology (October 1-12, 2018)

This course consists of 2 parts. The first part provides an overview of blood cells and the mammalian immune system, including a detailed description of all three arms of the immune response, how toxic chemicals can impact normal immune system homeostasis to result in adverse health outcomes, cellular/functional methodologies for determining the potential immunotoxicity of a given chemical, and how immunotoxicology relates to other scientific fields such as drug development and risk assessment. The second part provides an introduction to hormonally active agents and their mechanisms of action; routes of exposure, bioaccumulation, distribution and metabolism; effects on reproduction and development in humans and animals; and methods for studying changes in gene expression.

Requirement: Participants should have some basic knowledge on the biology of the immune and reproductive systems, as well as methodologies for assessing the functioning of these systems.

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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