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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".

Ambient Air Pollution and COVID-19 Risk

The coronavirus disease 2019 (COVID-19) pandemic is severely threatening and challenging public health worldwide. Therefore, exploring the factors influencing COVID-19 transmission and mortality have significant implications for prevention and intervention strategies for this pandemic.

Air pollution is globally the fourth greatest source of lethality. Several epidemiological studies have linked air pollution to the COVID-19 pandemic. However, the conclusions of individual studies are inconsistent.

Generally, both individual susceptibility and ambient conditions contribute to COVID-19 transmission and mortality. The ambient air quality index (AQI) is determined by airborne carbon monoxide (CO), nitrogen dioxide (NO_2), ozone (O_3), particulate matter with diameter <10 μm (PM_{10}) and <2.5 μm ($PM_{2.5}$), and sulfur dioxide (SO_2).

No meta-analysis has yet quantitatively focused on the influence of air pollution on COVID-19 risk and there are inconsistent conclusions of individual studies, while the pandemic is spreading vigorously.

The first meta-analysis on the association of air pollution and COVID-19 was conducted to explore the quantitative effect of outdoor air pollution on COVID-19 incidence and mortality. The effects of two types of AP exposure (long- and short-term) on the two outcomes of COVID-19 (incidence and mortality) were included, to comprehensively explore the relationship between AP and COVID-19 risk.

The results showed that long-term exposure to NO_2, $PM_{2.5}$, and SO_2 was associated with higher COVID-19 incidence. The COVID-19 mortality was positively associated with an increase in NO_2 and $PM_{2.5}$. The COVID-19 incidence was positively associated with an increase in AQI.

Short-term exposure to NO_2, O_3, PM_{10}, $PM_{2.5}$, and SO_2 was associated with higher COVID-19 incidence. No association was found between short-term exposure to CO and COVID-19 incidence.

Subgroup analysis showed that the association between long-term exposure to $PM_{2.5}$ and COVID-19 incidence remained positive when subgroup analysis evaluated countries' income (high- or low-/middle income). The association between short-term exposure to NO_2 and COVID-19 incidence remained positive when subgroup analysis took country income into account.

The burden of the pandemic in most regions worldwide is increasing, underscoring the importance and urgency of attenuating air pollution in order to address the situation.

In conclusion, outdoor air pollutants are detrimental factors to COVID-19 outcomes. Measurements beneficial to reducing pollutant levels might also reduce the burden of the pandemic.

Due to lethality and high infectiousness, COVID-19 remains a significant threat to public health worldwide. With the continuous spread of COVID-19, air pollution has been linked to the pandemic in several epidemiological studies, with inconsistent conclusions.

Highly polluted and severely COVID-19 burdened, low-/middle-income regions should perform epidemiological research to accurately estimate the detrimental effects.

Source: Environmental Research, Part B, Vol. 204, Article 112065, March 2022.

Prenatal Exposure to Metals and Neurodevelopment in Infants

The Agency for Toxic Substances and Disease Registry (ATSDR) lists arsenic (As), cadmium (Cd), lead (Pb), and mercury (Hg) in the Priority Substance List, based on a combination of frequency, toxicity, and potential for human exposure. Chronic exposure to low doses of these metals may cause human health effects affecting the nervous, hematological, and immune systems.

Fetal brain development during pregnancy is susceptible to the action of neurotoxic substances, as the placental barrier is not completely impermeable to the passage of harmful substances, including heavy metals. Several studies have reported an association between metal exposure during pregnancy and early childhood and impaired cognitive function in children.

Neurobehavioral developmental disorders such as autism spectrum disorder, attention deficit hyperactivity disorder, and subclinical decreases in brain function have increased worldwide.

The etiology of metals in neurodevelopmental disorders can occur through different pathways that include genetic and epigenetic mechanisms, neuroendocrine dysfunction, oxidative stress, immune dysregulation, and changes in neurotransmitters.

The Rio Birth Cohort Study on Environmental Exposure and Child Development (PIPA Project) is a prospective cohort study conducted in Brazil, aiming at the investigation of the effects of environmental pollutants on maternal and child health.

This study investigates potential associations between metal concentrations in maternal and umbilical cord blood and newborn neurological development among the PIPA pilot study population.

The Denver Developmental Screening Test II (DDST-II) was applied to screen neuromotor development, evaluating children from zero to six years old regarding their ability to perform tasks organized in four neurodevelopmental domains: "Personal-Social", "Fine Adaptive Motor", "Gross Motor", and "Language". For the analyses, the children were divided into "fail group" and "not fail group".

The results showed that maternal blood lead concentrations were higher in premature newborns. One-third of the infants exhibited at least one fail in the neurodevelopment evaluation (fail group).

Maternal blood arsenic concentrations were significantly higher in the "fail group" compared to infants who did not fail (not fail group).

Maternal and umbilical cord blood arsenic concentrations were higher in all Denver Test's domains in the "fail group", although non-statistically significant, showing a tendency for the gross motor domain and maternal blood.

Continuous monitoring of concentrations of neurotoxic substances in women of childbearing age is essential to establish preventive measures to eliminate or minimize the risk of fetal exposure during pregnancy. Long-term development studies may reveal other associations.

Future investigations should aim to demonstrate the possible neuro-developmental effects of prenatal exposure to environmental pollutants on child health and identify potential sources of exposure.

Source: International Journal of Environmental Research and Public Health, Vol. 19, Issue 7, Article 4295, April 2022.

Perinatal Exposure to Pesticides and Autism Spectrum Disorder

Agricultural pesticides have been one of the most extensively used compounds throughout the world. Pesticide exposure during pregnancy is associated with neurological disorders in the offspring, including autism spectrum disorder (ASD), attention deficit hyperactivity disorder (ADHD), and cognitive deficits.

ASD (i.e., autism) is a neurological disorder that appears in early childhood and is characterized by markedly persistent deficits in social interaction, communication skills, and repetitive stereotyped behaviors.

It is widely believed that a combination of genetics and the environment contributes to the development of ASD. Individuals with susceptibility genes are more likely to develop ASD when exposed to environmental hazards. Recent studies have shown that environmental factors account for as high as 40-50% of the contribution ratio to ASD.

The present review discusses the available evidence linking exposure to several highly used and frequently studied pesticides (e.g., glyphosate, chlorpyrifos, pyrethroids, and avermectins) with increased risk of ASD from human epidemiology studies, animal studies, and studies of molecular mechanisms.

The review presented a consolidated understanding of pesticides closely related to the etiology of ASD, providing indirect evidence for the plausibility linking pesticides exposure during pregnancy and lactation (the critical developmental periods) to ASD.

Recent studies have shown that low-level agricultural pesticide exposure during the critical period of neurodevelopment (pregnancy and lactation) is closely related to ASD.

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Role of Environmental Toxicants in Hypertension and Cardiovascular Diseases

Hypertension is a complex disease that causes many morbidities and mortalities worldwide. In 2000, an estimated 972 million adults had hypertension, which is predicted to grow to 1.56 billion by 2025. Hypertension is considered the major risk factor for cardiovascular disease (CVD).

The causes for the increase in populations with hypertension are believed that due to industrial activity and exposure to environmental toxicants such as diesel exhaust particles (DEP), polycyclic aromatic hydrocarbons (PAH), residues of organochlorine insecticides (OCI), polychlorinated biphenyls (PCBs), particulate matters (PM), and heavy metals (lead, mercury, cadmium, and arsenic).

High blood pressure for an extended period can cause cardiac dysfunction, kidney damage, and damage to other vital body organs.

Hypertension often leads to diabetes mellitus that strongly puts the patients at an increased risk of cardiovascular, kidney, and/or atherosclerotic diseases.

Hypertension has been identified as a major risk factor for the development of diabetes; patients with hypertension are at two-to-three-fold higher risk of developing diabetes than patients with normal blood pressure.

Causes for the increase in hypertension and diabetes are not well understood. Environmental factors (e.g., exposure to environmental toxicants like heavy metals, organic solvents, pesticides, alcohol, and urban lifestyle) have been postulated as one of the reasons contributing to hypertension and cardiovascular diseases (CVD). The mechanism of action(s) of these toxicants in developing hypertension and CVDs is not well defined.

Studies on monitoring blood pressure in an urban population of developed countries demonstrate a rise in systolic blood pressure and pulse pressure with increasing age. In developed countries, 35-50% of individuals over age 65 are thought to be hypertensive.

Environmental chemicals and drugs are metabolized by CYP P450 based enzymatic system. It has been difficult to interpret the effects of aging on the levels and activities of P450 isozymes. CYPs produce reactive oxygen species (ROS), which are removed by antioxidants. Decreasing levels of antioxidants among the elderly and increased ROS can cause vasoconstriction and exacerbate elderly hypertension.

Research studies have linked hypertension with the chronic consumption of alcohol and exposure to metals like lead, mercury, and arsenic. Workers chronically exposed to styrene have a higher incidence of CVD.

Recent studies have demonstrated that exposure to particulate matter (PM) in diesel exhaust and urban air contributes to increased CVD and mortality.

In the review, the role of environmental toxicants such as heavy metals, organic pollutants, PM, alcohol, and some drugs in hypertension and CVD were discussed along with possible mechanisms and limitations in extrapolating animal data to humans.

At present, humans are exposed to various environmental pollutants, which might influence blood pressure in a meaningful way. Several pollutants such as heavy metals, diesel exhausts, and PM have been linked to increased blood pressure.

A number of studies have been carried out reporting the linkage between the exposure to environmental pollutants and increased blood pressure, while no substantial data provided unequivocal information unraveling the possible mechanisms involved in such phenomena.

However, increased incidence of hypertension worldwide points to the role of environmental pollutants as one of the factors and understanding the mechanisms by which pollutants affect blood pressure is required for meaningful interventions.

Source: Toxicology Reports, Vol. 9, Pages 521-533, March 2022.

Perinatal Exposure to Pesticides and Autism Spectrum Disorder

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The possible associations and mechanisms leading to ASD were also discussed from epidemiological and toxicological evidences.

Abnormalities in gut microbiota, neuronal dendritic morphology, axonal morphology and function, neurotransmission, and immunity (superfluous activation of microglia and astrocyte) may be the underlying pathophysiological processes associating pesticide exposure with the development of ASD.

The results of such studies could provide a scientific basis for further exploring the risk of environmental factors in the etiology of ASD.

Importantly, this review may motivate epidemiological and basic medical research to further focus on the role of pesticides exposure during pregnancy and lactation in the pathogenesis of ASD, which may facilitate the development of prevention or treatment strategies for this disorder.

Source: Environmental Research, Vol. 203, Article 111902, January 2022.

Environmental Air Pollution and Olfactory Decline in Aging

Olfactory deficits are particularly pronounced in older age and associated with a number of health conditions such as depressive symptoms and frailty, as well as shorter survival and diminished quality of life.

It has been speculated that part of the olfactory loss observed in older age may arise from cumulative damage of xenobiotics. An increased exposure to air pollution may lead to olfactory loss, especially among middle-age or older adults for whom xenobiotic exposure has accumulated over a longer time.

The smallest particulates [particulate matter with aerodynamic diameter <2.5 μm ($PM_{2.5}$)] are among the most harmful forms of air pollution for human health.

Due to its small size, $PM_{2.5}$ can penetrate the brain via the olfactory route, where it is likely to cause damage to the olfactory system. Additional types of pervasive air pollutants that can enter the brain via the olfactory system are different forms of nitrogen oxides (NOx) which usually produced during combustion of fuels in air.

However, population-based studies investigating the relationship between air pollution and olfactory ability are scarce.

The present study aimed to investigate associations between exposure to common air pollutants ($PM_{2.5}$ or NOx) and longitudinal change in odor identification across 12 years of follow-up in Sweden.

Average pollution emissions were categorized into quartiles with increasing emission concentrations, each comprising 25% of the total sample size. The first quartile was considered the reference category, representing participants exposed to the lowest average pollutant concentration of $PM_{2.5}$ or NOx.

Exposure to higher quartiles of outdoor air pollution was associated with faster decline in olfactory identification ability across follow-up, in comparison with exposure to the lowest quartile. Similar results were observed for the third and fourth quartiles of NOx.

The researchers observed associations between air pollution and olfactory decline at commonly occurring $PM_{2.5}$ and NOx concentrations, the former being on average well below the current World Health Organization (WHO) guideline of 10 $\mu g/m^3$.

The results suggest an association between air pollution ($PM_{2.5}$ and NOx) exposure and subsequent olfactory decline in older adults. Cumulative effects of airborne pollutants on the olfactory system may be one underlying cause of olfactory impairment in aging.

Source: Environmental Health Perspectives, Vol. 130, No. 2, February 2022.

Toxic Footprint and Electronic Components in Printed Circuit Boards

According to the global E-waste monitor 2020, the total amount of waste electric and electronic equipment (WEEE) was 53.6 million tons in 2019, of which China accounted for the highest as 7.5 million tons.

Waste Printed Circuit Boards (WPCBs), account for 4% of WEEE, contain valuable material resources and hazardous substances, thereby posing a challenge for sustainable resource recovery and environmental protection initiatives. Overcoming this challenge will require mapping the toxic footprint of WPCBs to specific materials and substances used in manufacturing electronic components (ECs).

WPCBs typically contain various types of Printed Wire Boards (PWBs) and ECs, and the two categories vary widely in materials composition and concentration.

Best available technology for resource recovery includes removal of mounted ECs from WPCBs. Researchers have focused on strategies such as machinery, hydrometallurgy, pyro-metallurgy and biotechnology to recover valuable metals from PWBs (30-40%), such as copper (16%), aluminum (5%), tin (4%), iron (3%), nickel (2%) and zinc (1%), and also precious metals, such as gold (0.03%), silver (0.05%).

According to the production process, ECs that have not been modified in material composition and structure are recognized as electronic elements (EEs); otherwise, they are called as electronic devices (EDs).

The present study collected 50 EC specimens from WPCBs in five ubiquitous consumer products, such as television, refrigerator, air conditioner, washing machine and computer.

The study extracted and analyzed metal contents and used leachability assessments based on tests adopted by the regulatory policies from China and the United States.

Through this study, ECs both EEs and EDs, including all types of ECs in home appliances that the Chinese WEEE market currently addresses were surveyed.

ECs contain abundant metal resources, especially compared to PWBs, and it is recommended to separately recycle ECs and PWBs from WPCBs. Moreover, it is recommended to sort and recycle ECs according to the most abundant metal contents.

The results found that copper and iron are the most abundant constituents in ECs, whereas abundance of precious metal content is in the order of silver > gold > palladium > platinum.

On the other hand, with increasingly strict environmental regulations and policies, toxic pollutants

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Paraquat Toxicity in Swiss Albino Mice

Paraquat is a fast, effective and non-selective nitrogen herbicide. The toxic effects of paraquat on plants are due to the production of free radicals after oxidation with oxygen molecules that cause photosynthesis damage.

Paraquat is highly toxic to both humans and animals. Although the mechanisms of paraquat are not fully understood, it is hypothesized that toxicity results from the generation of reactive oxygen species (ROS) via the redox cycle process and causes oxidative stress-related damage to cellular organelles, proteins, nucleic acids, and lipids.

The aim of the present study is to investigate the toxic effects of paraquat on Swiss albino mice using physiological, biochemical, oxidative stress, and genetic parameters.

Four groups were randomly formed from male Swiss albino mice (one control and three treatment groups). The control group mice were provided tap water and the mice in the treatment groups were treated orally with three different doses of paraquat (50, 100 and 200 mg/kg b.w) in the drinking water for 28 days.

While feed consumption, body and organ weight were examined as physiological parameters, serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) enzyme activities, blood urea nitrogen (BUN), and liver/kidney malon-dialdehyde (MDA) and glutathione (GSH) levels were examined as biochemical parameters.

Micronucleus (MN) formation in buccal mucosal epithelium, erythrocyte and leukocyte cells and chromosomal aberrations (CAs) in bone marrow cells were examined as genetic parameters.

Comet assay was applied to support the genotoxic effects of paraquat, while the interaction of paraquat with DNA and DNA-related proteins has been elucidated by molecular docking studies.

The results showed that paraquat administration decreased physiological parameters (body, liver and kidney organ weights), and increased biochemical parameters (AST, ALT, BUN, creatinine and MDA).

Paraquat exposure caused a significant dose-dependent increase in MDA levels and a decrease in GSH levels in liver and kidney tissues. The decrease in GSH and increase in MDA is indicative of oxidative stress elicited at all paraquat treatment levels. Kidney and liver damage were confirmed by the trypan blue test.

Paraquat caused a significant dose-dependent increase in MN formation in buccal mucosal epithelium, erythrocyte and leukocyte cells; along with various CAs in bone marrow cells.

Paraquat promoted CAs such as break, fragment, acentric, dicentric, gap and ring in bone marrow cells. Break damage was the most common among these damages.

The percentage of head DNA was significantly decreased in a paraquat dose-dependent manner; while, the percentage of tail DNA, tail moment, and olive tail moment increased significantly with increasing dose. These results, in combination with results of the MN and CA evaluations indicate that paraquat disrupts DNA integrity.

In addition, these observed genotoxic effects occured as a result of the interaction of DNA and DNA-related proteins with paraquat. Molecular docking studies showed that paraquat binds to histone H4 protein with high affinity and has a high intercalation potential.

The high binding of Paraquat to the H4 protein can cause a deterioration in genome stability. Particularly, the DNA integrity disruption observed in the comet test may be associated with the Paraquat-histone H4 interaction.

Intercalation occurs by stacking chemicals between adjacent base pairs in DNA without forming any covalent bonds between the chemical and DNA. Intercalation also causes the supercoiled DNA to unravel, which can ultimately prevent DNA from being recognized by DNA-binding proteins and other regulatory factors.

Intercalators agents such as paraquat have diverse and multiple biological effects on DNA. Inhibition of RNA and DNA synthesis, frameshift mutations, and protein-associated DNA breaks are some of these effects.

Especially DNA breaks lead to high CAs and MN formations. The high frequency of MN and CAs detected in the paraquat group in this study shows that paraquat causes protein-associated DNA breaks.

In conclusion, paraquat herbicide caused a significant toxicity by changing physiological, biochemical, oxidative stress and genetic parameters of Swiss albino mice depending on the application dose.

This is the first study to investigate MN formation caused by paraquat genotoxicity in buccal mucosal epithelium and leukocyte cells.

The genotoxicity mechanism of paraquat was investigated by molecular docking studies and it was found that paraquat was an intercalator, causing deterioration in DNA integrity and protein-associated DNA breaks.

In addition, paraquat caused damage to liver and kidney cell membranes, resulting in an increase in MDA levels, and a decrease in GSH level by promoting the formation of free radicals and induced oxidative stress.

Source: Scientific Reports, Vol. 12, Article 4818, March 2022.

Metabolic Syndrome and Endocrine Disrupting Chemicals

The burden of non-communicable diseases (NCDs) is increasing and expanding rapidly worldwide. Many NCDs share common risk factors such as sedentary lifestyles and unhealthy diets, increasing the risk of obesity, hypertension, and distorted lipid and glucose metabolism, which together are also known as metabolic syndrome (MetS), a strong predictor of cardiovascular disease morbidity and mortality.

The latest Joint Interim Statement (JIS) definition of MetS requires that at least three of the following five clinical findings: elevated waist circumference, elevated triglycerides, reduced HDL cholesterol, elevated blood pressure, and/or elevated fasting glucose.

Increasing prevalence of MetS is causing a significant health burden among the European population. Several European studies have estimated the prevalence of MetS to be between 13–35% among large European cohorts, mostly around 25%. In addition to the considerable public health burden and increased risk of cardiovascular diseases, type 2 diabetes and non-alcoholic fatty liver disease, the economic impact of MetS on Europe's health care costs is tremendous.

Current knowledge supports the notion that endocrine-disrupting chemicals (EDCs) interfere with human metabolism and hormonal balance, contributing to the conventionally recognized lifestyle-related MetS risk factors. Early life exposure, pregnancy and childhood have been identified as high vulnerability stages for EDC exposure, increasing the risk of disease later in life and in subsequent generations.

The present review aims to identify epidemiological studies focusing on the association between MetS or its individual components (e.g., obesity, insulin resistance, diabetes, dyslipidemia and hypertension), and the most suggestive EDCs prioritized in the European Human Biomonitoring Initiative (HBM4EU).

The eight priority substances in HBM4EU include bisphenol A (BPA), per- and polyfluoroalkyl substances (PFASs), phthalates, polycyclic aromatic hydrocarbons (PAHs), pesticides and heavy metals (cadmium, arsenic and mercury).

Human biomonitoring studies have presented evidence supporting the role of EDC exposures on the development of individual MetS components. The strength of the association varies between the components and EDCs.

The overall toxicological, observational, and human intervention mainstreams of evidence support that BPA exposure during development, but also at adulthood, constitutes a risk factor for obesity, insulin resistance, and other MetS components.

For PFASs, epidemiological evidence shows an association between obesity, dyslipidemia, and diabetes among adults. In utero exposure has been linked with childhood obesity, and exposure among adolescents with increased risk of obesity, dyslipidemia, and hypertension.

For phthalates, reported studies suggest associations with obesity, glucose disturbance, and hypertension among adults. Prenatal exposure is shown to increase risk of childhood obesity, and exposure during the childhood may result increased risk of obesity, lipid, and glucose disturbances. Sex differences in the associations have also been observed.

Exposure to PAHs has been associated with increased risk of obesity, diabetes, and hypertension. Among children, exposure to PAHs has been linked to increased risk of obesity.

For pesticides, existing evidence is still scarce but suggests associations with diabetes and non-persistent pesticides.

For three heavy metals (arsenic, mercury, and cadmium), limited evidence exists but there are some indications that exposure would increase the risk of obesity, diabetes, and hypertension. Additionally, for arsenic and mercury, association with the risk of dyslipidemia has been observed.

Current evidence on metabolic disturbances and EDCs is still limited and heterogeneous, and mainly represent studies from North America and Asia, highlighting the need for well-conducted and harmonized HBM programmes among the European population.

In this review, MetS was examined as an outcome only in few studies, and most available data focused on individual MetS components. Although some MetS components have been widely researched (e.g., obesity and glucose metabolism), others have gained less focus (dyslipidemia and hypertension).

Nevertheless, an increase in the risk of isolated MetS components will lead, over time, to a higher risk of MetS and cardiovascular disease, morbidity, and mortality. Therefore, the role of environmental chemicals on MetS should not be overlooked.

Indeed, additional studies are needed and should be focused on increasing study quality: harmonization of MetS definitions, harmonization of exposure assessment with certified laboratories undergoing interlaboratory comparisons, representative population samples, longitudinal designs with repeated measurements, harmonization of effect biomarkers implemented and quality control measures, and harmonization of statistical protocols including a selection of the most relevant covariates based on causal graphs.

There is a need, however, to establish harmonized and standardized HBM procedures among the European population, in addition to rigorous and continuous human biomonitoring combined with health monitoring, including novel effect biomarkers which could provide comprehensive information on EDC exposure and association of metabolic disturbances.

Source: International Journal of Environmental Research and Public Health, Vol. 18, Issue 24, Article 13047, December 2021.

ILO - Exposure to Mercury in the World of Work

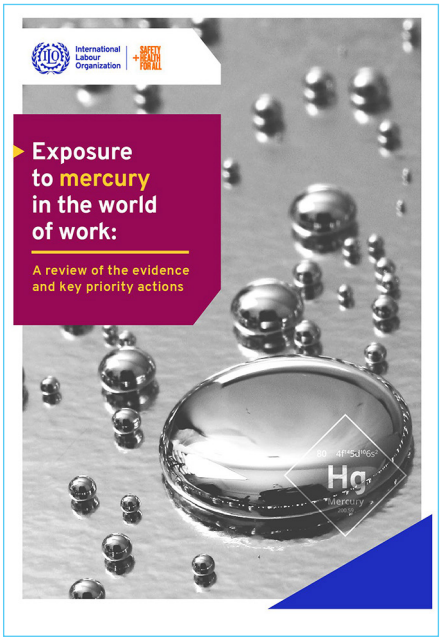
The report **"Exposure to mercury in the world of work: a review of the evidence and key priority actions"** by the International Labour Organization (ILO) provides a critical review of the current evidence on occupational mercury exposures and health impacts, as well as highlighting key priority areas for action.

Occupational mercury exposures present a significant threat to the health of workers in a variety of industries around the world. Workers in artisanal and small-scale gold mining are particularly at risk, however numerous other sectors have been identified as areas of concern, including vinyl chloride monomer production, manufacturing, healthcare, waste-picking and recycling. While exact figures are lacking, it is estimated that millions of workers around the world continue to be exposed and to suffer from the serious health impacts of this toxic heavy metal.

Mercury is toxic to the nervous, digestive and immune systems, as well as specific organs, such as the liver, heart, brain and skin. Serious health effects can result, including chronic metallic mercury vapour intoxication, heart palpitations, kidney abnormalities, cognitive dysfunction, respiratory failure and death. Even low levels of chronic exposure can result in severe disability and debilitating chronic conditions, impacting long-term health and well-being. This significant burden for workers, and society as a whole, is entirely preventable.

The ILO has long recognized the occupational risks posed by mercury exposure, and in fact, mercury poisoning was recognized as one of the very first occupational diseases, listed in the ILO list of occupational diseases already in 1925. Since then, the ILO has developed and promoted numerous International Labour Standards relevant to occupational safety and health, chemical hazards and mining.

In 2013, the Minamata Convention was adopted, with an aim to reduce global mercury emissions and to protect human health and the environment from hazardous mercury exposures.



Source: ILO. Exposure to Mercury in the World of Work: A Review of the Evidence and Key Priority Actions. 18 January 2022.

WHO - REVIEW OF MINAMATA CONVENTION INITIAL ASSESSMENT REPORTS

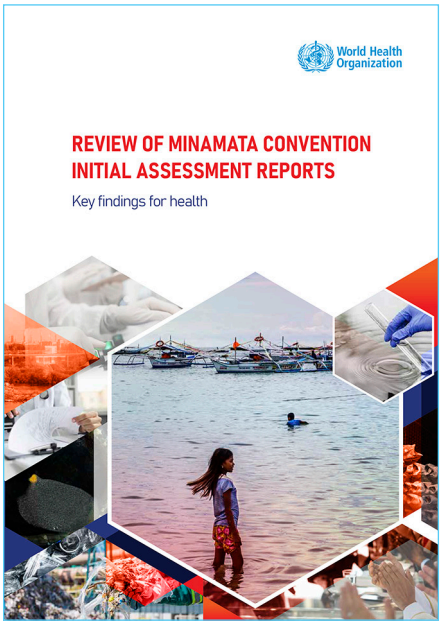
The Minamata Convention on Mercury is a global, legally binding treaty, which was adopted in 2013 and entered into force on 16 August 2017. The core of the Convention is protection of human health, as stated in *Article 1: "to protect human health and the environment from anthropogenic emissions and releases of mercury and mercury compounds".* Implementation of the Convention requires multisectoral action, including the health sector.

In order to raise awareness about health ministries' preparedness and outstanding needs to be able to implement the health-related articles of the Convention, WHO reviewed all the 59 Minamata initial assessment (MIA) reports that had been submitted to the Secretariat of the Convention up to 31 July 2021 as well as two national implementation plans.

The review found that Member States have put in place many measures to implement the health-related articles of the Convention, although it was also found that more work is necessary.

Priorities for action were identified for all of the health-related articles, some of which exceeded the minimum requirements of the Convention. The main priorities, cited in about two thirds of the reports, were phasing out use of mercury-containing medical measuring devices, beyond not allowing their import, export or manufacture, and phasing down the use of dental amalgam. Other priorities for action, identified in more than half the reports, were measures to implement *Article 18, "Public information, awareness and education",* and *Article 11, "Mercury waste".*

Despite the strong presence of WHO Member States among the Parties to the Minamata Convention and despite progress made in implementing relevant measures, this review of MIA reports raises concern about the extent of engagement of ministries of health in the MIA process and therefore in the implementation of the health-related articles of the Convention.



Source: WHO Publication. Review of Minamata Convention Initial Assessment Reports: Key Findings for Health, 20 January 2022.

CALENDAR OF EVENTS

International Training Courses at Chulabhorn Research Institute, Year 2022

	Training Course	Date	Duration	Closing Date
1	Environmental Toxicology and Health	June 15-20, 2022	5 work days	May 5, 2022
2	Environmental and Health Risk Assessment and Management of Toxic Chemicals	December 6-16, 2022	10 work days	October 20, 2022

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

Course Description:

Environmental and Health Risk Assessment and Management of Toxic Chemicals (December 6-16, 2022)

The course is an integration of science and policy, covering the fundamental basis of environmental and health risk assessment and management from exposure assessment and risk characterization; mode of action and human relevance framework; the relationship between risk assessment and risk management; and the need for open, transparent and participatory acceptance procedures and credible communication methods. Emphasis will be placed on human health risk assessment, although the principles of ecological risk assessment will also be covered. Importantly, 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 an electronic distance learning tool on risk assessment and risk management of chemicals and the WHO IPCS Human Health Risk Assessment toolkit will be introduced.

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

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

Toxic Footprint and Electronic Components in Printed Circuit Boards

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such as chromium and mercury were found to not exceed the standard thresholds established by regulatory policies in China and the U.S.

The work found new toxic threats from arsenic and selenium leached from 20 of 50 ECs exceeding regulatory standards. These results will aid manufacturers and recyclers in protecting workers' health and environmental quality from arsenic and selenium pollution, and should initiate discussion about regulating these toxic components as part of a comprehensive program to reduce the toxic footprint of electronic products.

This research contributes results to bridge major knowledge gaps in WPCBs recycling in a way that protects environmental quality and human health.

Source: Waste Management, Vol. 141, Pages 154-162, March 2022.

The ICEIT NEWSLETTER is published quarterly by the International Centre for Environmental and Industrial Toxicology of the Chulabhorn Research Institute. It is intended to be a source of information to create awareness of the problems caused by chemicals. However, the contents and views expressed in this newsletter do not necessarily represent the policies of ICEIT.

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