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Mercury Exposure and Cardiovascular Disease

There is evidence that exposure to mercury (Hg) may be a risk factor for cardiovascular disease (CVD).

Hg is a global pollutant with known adverse health outcomes. Hg is present in three forms: elemental Hg (Hg^0), inorganic Hg (Hg salts), and organic Hg (methyl mercury (MeHg)). Among these, MeHg is the most harmful form of Hg due to its high bioavailability and neurotoxicity.

The general population is exposed to a relatively low level of inorganic Hg, primarily through dental amalgam, inhalation from anthropogenic sources such as metal mining and smelting, combustion of fossil fuels, and incineration of municipal waste.

Elevated exposure to inorganic Hg is found among Hg miners, gold miners, persons using skin-lightening cosmetic products, dentists, and patients receiving dental amalgams.

Marine and freshwater fish consumption is the most common route of MeHg exposure for the general population. Many coastal and island dwellers, particularly indigenous populations, frequently consume locally available marine seafood species that have high MeHg concentrations.

Hg exposure is a public health concern primarily because of its well-documented neurodevelopmental toxicity in fetuses and children. However, a growing body of evidence suggests that Hg exposure may also lead to adverse cardiovascular outcomes.

Despite age-standardized CVD mortality declines in high-income and some middle-income countries. Cardiovascular diseases (CVDs) remain a major cause of death and loss of health worldwide. Ischemic heart disease (IHD) and stroke are the two leading causes of CVD globally.

Besides the traditional CVD risk factors, such as hypertension, overweight/obesity, diabetes, and high salt intake, concerns about exposure to air pollution and environmental chemicals, such as heavy metals and persistent organic pollutants, are growing. The contribution of chemical pollution to the global burden of disease is almost certainly underestimated.

Reviews indicate that Hg exposure is associated with an increased risk of hypertension and diabetes. Although the mechanisms by which Hg induces CVD are not fully understood, plausible explanations include oxidative stress and inflammation, which promotes endothelial and renal dysfunction, as well as binding of selenoenzymes. A dose-response relationship has been proposed between Hg and cardiovascular outcomes but has yet to be confirmed.

Recently, a systematic review of published studies with meta-analysis of the results was conducted to examine the associations between chronic Hg exposure and CVD outcomes.

Studies were selected according to a priori-defined inclusion criteria, and their qualities were assessed. Study estimates were extracted, and subgroup analyses were conducted to explore potential sources of heterogeneity: 1) fatal vs. nonfatal events, 2) cohort study vs. non-cohort study, and 3) inorganic Hg vs. methyl mercury (MeHg).

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Mercury Exposure and Cardiovascular Disease

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Dose-response meta-analyses were conducted for MeHg exposure and fatal/nonfatal ischemic heart disease (IHD), stroke, and all CVDs.

This is the first systematic review and meta-analysis to quantify the association of Hg exposure with CVDs and all-cause mortality.

A total of 14 studies reporting results collected from more than 34,000 participants in 17 countries were included in the meta-analysis. Hg exposure was associated with an increase in nonfatal IHD, all-cause mortality, CVD mortality, and mortality due to other heart diseases.

No association was observed between Hg exposure and stroke. A heterogeneous relationship was found between studies reporting fatal and nonfatal outcomes and between cohort and non-cohort studies. However, these differences were mainly due to differences in Hg exposure level.

In conclusion, chronic exposure to Hg is associated with an increased risk of all-cause mortality and fatal/nonfatal IHD. More evidence is available for total Hg exposure than inorganic Hg exposure. The risk of multiple cardiovascular endpoints starts to increase consistently at a Hg concentration of 2 μg/g in hair.

More research is needed for populations with low to moderate exposure, especially for inorganic Hg.

Source: Environmental Research, Vol. 193, Article 110538, February 2021.

Ambient $PM_{2.5}$ Exposure and Anemia Outcomes Among Children Under Five Years of Age in India

India has some of the highest levels of ambient $PM_{2.5}$ in the world, exceeding the World Health Organization (WHO)-recommended annual mean air quality guideline of 10 μg/m^3 by orders of magnitude. Exposure to ambient fine particulate matter ($PM_{2.5}$) is the third leading cause of death in India.

Exposure to ambient $PM_{2.5}$ has been associated with adverse cardiovascular, respiratory, and mortality outcomes, and with negative impacts on child health including low birth weight and stunted growth. Even so, there continues to be a general lack of information, especially in terms of exposure-response analyses, on the association between ambient $PM_{2.5}$ exposure and many other health outcomes.

Anemia, measured via low-blood hemoglobin concentration, is characterized by a decreased oxygen-carrying capacity of the blood. Childhood anemia in India is endemic; in 2016, with nearly 60% of the children in India being anemic.

Most of the research examining exposure to ambient particulate matter and anemia outcomes has been conducted in the United States, Europe, and China, where associations has been observed between increased exposure to PM and increased anemia prevalence/ decreased hemoglobin concentration among the elderly.

Long-term ambient $PM_{2.5}$ exposure could lead to chronic systemic inflammation, which could reduce iron absorption, exacerbate the effects of dietary iron deficiency and lead to the onset of anemia.

The present study hypothesized that adjusting for relevant covariates, long-term ambient $PM_{2.5}$ exposure would be associated with decreased hemoglobin levels and increased odds of anemia in children.

Using health data from the National Family and Health Survey 2015-2016, this study examined, through district-level ecological and individual-level analyses, the association between ambient $PM_{2.5}$ exposure and anemia in children under the age of five across India.

The district-level ecological analysis found that there is a 1.90% increase in average anemia prevalence among children with every 10 μg/m^3 increase in ambient $PM_{2.5}$ exposure.

At the individual level, for every 10 μg/m^3 increase in ambient $PM_{2.5}$ exposure, average hemoglobin decreased by 0.14 g/dL. The odds ratio associated with a 10-μg/m^3 increase in ambient $PM_{2.5}$ exposure was 1.09 . There was evidence of effect modification by wealth index, maternal anemia status, and child Body Mass Index (BMI).

The results suggest that ambient $PM_{2.5}$ exposure could be linked to anemia in Indian children, although additional research on the underlying biologic mechanisms is needed.

Future studies on this association should specifically consider interactions with dietary iron deficiency, maternal anemia status, and child BMI.

Across all primary models, the study found that children exposed to higher levels of ambient $PM_{2.5}$ were at higher risk of being anemic after adjusting for potential confounders such as diet, sex, wealth index, maternal anemia status, and accounting for clustering by district.

This study adds to the body of global evidence highlighting the adverse effects of ambient $PM_{2.5}$ exposure on human health and suggests that meeting targets for the National Clean Air Program would improve child health, and that control of air pollution should be a top priority in India.

Source: Environmental Epidemiology, Vol. 5, Issue 1, Article e125, February 2021.

Environmental Substances Associated with Osteoporosis

The World Health Organization (WHO) defines osteoporosis as a systemic skeletal disease characterized by reduced bone mass and micro architectural degrading of bone tissue, which causes an increase in bone frailty and consequent susceptibility to fractures.

Osteoporosis affects approximately 200 million people worldwide and contributes every year to nearly 9 million fractures. Women suffer from osteoporosis at a fourfold higher rate than men. Women start losing bone mass at a younger age.

They begin to suffer fractures 5-10 years before men. By the age of 50 or above, their rate of osteoporosis is twice as high. Although the density and trabecular architecture of bones are equivalent in both genders, the fracture rates in men are lower, mainly because men lose less porous (trabecular) bone compared to women.

A variety of techniques are used to assess the risk of fracture, but there are two main approaches, i.e. clinical assessment of risk factors and physical measurement of skeletal mass. Among the various different ways of assessing bone mineral density (BMD), the most common method is dual-energy X-ray absorptiometry (DXA).

Vulnerability to fractures increases with aging, other contributing factors include, for example, menopause at an early age, a maternal incident of hip fracture, a fracture after the age of 40, low body weight levels, as well as some particular diseases and treatments.

A moderate and nutritious, well-balanced diet with sufficient calcium and vitamin D is basic, along with regular exercise, including weight-bearing activities. These are the cornerstones for building and maintaining healthy bone mass.

In addition to traditional risk factors, certain substances in the environment now appear to increase the risk of osteoporosis.

Substances such as Cd, Pb, phthalates, and PFASs which are found throughout the natural and manmade environment, are suspected of having adverse effects on bone mineral density (BMD) and therefore of increasing the risk of osteoporosis.

This study selects only those substances for which there is the strongest epidemiological evidence vis-a-vis humans, according to current research. Heavy metals such as arsenic (As) and mercury (Hg) are therefore excluded.

This scoping review aims to present an overview of the recent research evidence concerning selected environmental substances and their possible associations with osteoporosis. It takes a disease-oriented approach to risks which environmental substances can pose to human health, especially as regards osteoporosis.

This kind of approach can be informative and useful as it aims to enhance public health and to find ways to reverse the increasing burden of bone health disorders.

The European Human Biomonitoring Initiative (HBM4EU) is a joint program coordinating and advancing human biomonitoring in Europe. HBM4EU investigates the exposure of populations to several environmental substances and the plausible health effects of such contamination with the aim of impacting public policymaking.

In HBM4EU, 18 priority substances or substance groups were selected. For each of these, a scoping document was prepared summarizing existing knowledge and health effects. This scoping review is based on these chemical-specific scoping documents and the complementary literature review.

There is evidence that the other adverse health effects of many substances found in the environment include impacts on human bones.

According to the literature, substances such as bisphenols, As, Hg, and PAHs are suspected of having harmful effects on bone health, but the epidemiological evidence is still incomplete. BMD is widely used as an endpoint to investigate the association of chemical exposure and osteoporosis.

Possible links were identified between osteoporosis and the body burden of heavy metals such as cadmium (Cd) and lead (Pb), and industrial chemicals such as phthalates and per- and poly-fluoroalkyl substances (PFASs).

Epidemiological studies now indicate an inverse association between chemicals and BMD. That is, the higher the chemical levels in the measurement matrix, the lower the measured BMD at different bone sites.

Concerns about chemical exposure are rapidly growing, along with calls for more extensive epidemiological studies on the subject. Multidisciplinary approaches and prompt actions to protect citizens in the EU and around the world will be required.

The study results indicate that osteoporosis may be an underestimated endpoint which has not been sufficiently integrated in epidemiological studies.

People are increasingly being exposed to chemicals in the environment during their daily activities, elevating the body burden and contributing to the possibility of disease. Preventing these excessive exposures is a pressing public health concern.

Source: International Journal of Environmental Research and Public Health, Vol. 18, Issue 2, Article 738, January 2021.

Environmental Chemicals Affect Circadian Rhythms

Circadian rhythms control the life of virtually all organisms. They regulate numerous aspects ranging from cellular processes to reproduction and behavior.

Besides the light-dark cycle, there are additional environmental factors, such as temperature and oxygen, that regulate the circadian rhythms in animals as well as humans.

Research on the basic principles and aspects of circadian rhythms has shown that if they are disturbed, physiological dysfunction will result. The role of contaminants in disturbing circadian interactions in humans and animals is still underexplored.

Natural and anthropogenic chemicals can be circadian disrupters when released into the environment. This can happen, for example, via discharge of industrial, hospital and domestic/municipal waste into surface water. Such pollution plays havoc with the circadian rhythm of aquatic organisms.

This review compiles existing data with a focus on fish, particularly zebrafish (*Danio rerio*), as a representative vertebrate model organism, to summarize current knowledge on contaminant-circadian rhythm interactions.

The review centers on and summarizes 40 natural and anthropogenic environmental circadian disrupters in fish. This disruptors can be divided into six major categories: steroid hormones, metals, pesticides and biocides, polychlorinated biphenyls, neuroactive drugs and other compounds such as cyanobacterial toxins and bisphenol A.

Steroid hormones as well as metals are the most studied. Especially in the case of progestins and glucocorticoids, circadian dysregulation was demonstrated in zebrafish on molecular and physiological levels, displayed mainly as altered behavior.

Most studies demonstrated transcriptional alterations of core circadian rhythm genes due to environmental chemicals. However, the precise links between these alterations and physiological outcomes is not well established.

The present review summarizes the current state of knowledge on circadian disrupters, highlights their risks to fish and identifies knowledge gaps regarding animals and humans. Most studies focus on transcriptional and behavioral alterations. Other effects and consequences remain underexplored.

Forthcoming studies should identify and explore additional environmental circadian disrupters. The underlying molecular mechanisms need clarification, and the consequences for physiological processes need to be better understood.

Source: Environment International, Vol. 149, Article 106159, April 2021.

Occupational Exposure to Aluminium

Aluminium is the most common metal element and, behind oxygen and silicon, the third most common element in nature. Aluminium accounts for 8.3% of the total weight of the earth's crust. Due to its serviceable physical and chemical properties, aluminium is widely used in production and daily life. Consequently, the number of people exposed to aluminium has also been increasing.

Previous studies have found that aluminium and its compounds are neurotoxic, can inhibit hippocampal nerves, and can cause neurodegenerative diseases similar to Alzheimer's disease (AD).

The decline of cognitive function is closely related to age. At the same time, studies have found that occupational aluminium exposure may also cause age-related cognitive impairment and induce a reduction in attention and memory.

Studies have shown that hippocampal volume mediates age-related decline in spatial memory: young and mature adult rats exhibit different behavior and cognitive learning strategies.

Age-related loss in the allocation and diversion of attention in the middle-aged population has been reported, but poorer performance and lower cognitive test scores have been more common in elderly individuals than among the middle-aged.

Although age-related differences in the relationship between aluminium exposure and cognitive function may be partly due to age cohort effects, epidemiological studies directly evaluating age-related differences in cognitive impairment have been inconsistent.

Cognitive function is composed of multiple cognitive domains, including learning, memory, language, executive function, orientation, attention, and visuospatial reasoning, among other domains. At the same time, there are differences in cognitive impairment among different age groups.

This study aimed to evaluate the different characteristics of cognitive impairment caused by occupational aluminium exposure at different ages in workers in an aluminium plant in China. It also assessed their cognitive function and plasma aluminium concentration.

The results showed that when the plasma aluminium concentration reached 34.52 μmol/L, workers could suffer from auditory, language, memory,

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Maternal Urinary Manganese Concentrations and Newborn Telomere Length

Manganese (Mn), an essential trace element for human growth and development, is related to the function of numerous organ systems such as reproduction, bone growth, neuro-development, enzyme activity, and the prevention of cellular damage due to free radical species.

In the general population, Mn is mainly obtained through diet and nutrition, particularly in plant-based food products, such as grains, brown rice, beans, vegetables, nuts, and tea. Previous studies have found that during pregnancy both Mn deficiency and Mn overload can result in adverse effects.

A series of studies have found that early-life excess Mn exposure can impair neurodevelopment and restrict fetal growth. However, because the body maintains tight control over Mn balance, it is uncommon for the general population to experience excess blood Mn concentrations, except for persons with occupational hazards or individuals living near metal processing plants.

Studies have reported that Mn can be actively passed through the placenta. Fetal demand increases the need for Mn during pregnancy, which suggests the necessity of Mn for fetal growth and development.

Telomeres, nucleotide sequences repeated at the ends of the chromosomes, can maintain the stability of the genome and protect chromosomes from degradation. Telomere length (TL), shortening with each cellular division, is regarded as a biomarker for biological aging.

Previous studies have associated TL shortening with several age-related diseases, such as diabetes, atherosclerosis, and cardiovascular disease, and increased mortality as well.

The intrauterine period is considered to be a critical window for the early programming of diseases in later life. Fetal TL is not only determined by heritability. It is also affected by different environmental factors during pregnancy, such as exposure to heavy metals

Newborn TL is sensitive to maternal metals concentrations, but studies about the association between maternal manganese (Mn) concentrations and newborn TL were not found.

The present study aimed to evaluate the association between maternal Mn concentration during pregnancy and newborn cord blood TL. Data were collected from a birth cohort study of 762 mother-newborn pairs conducted from November 2013 to March 2015 in Wuhan, China.

Unraveling the potential effects of the level of maternal Mn on TL at birth may contribute new insights into the etiology of diseases linked with telomere biological characteristics.

This is the first epidemiology study to report the effect of Mn exposure during pregnancy on newborn TL.

A positive association between maternal Mn exposure during the second trimester and newborn TL was found in the present study.

A doubling of maternal urinary Mn concentration during the second trimester was associated with 2.10% longer newborn cord blood TL. This association was more evident in male infants.

No relationship was found between maternal urinary Mn concentrations and cord blood TL during the first and third trimesters in this study.

The findings suggested that maternal Mn concentration during the second trimester was positively associated with newborn TL.

As the TL at birth has lasting effects throughout the life course, the results might provide epidemiological evidence on the effect of prenatal Mn exposure as well as clues for early prevention of telomere shortening related diseases.

Source: Ecotoxicology and Environmental Safety, Vol. 213, Article 112037, April 2021.

Occupational Exposure to Aluminium

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and working memory impairment. Especially in terms of working memory, young workers (<40 years) are more likely to suffer from impairment than middle-aged workers (≥40 years).

This study provides clues suggesting that long-term exposure to aluminium can cause significant cognitive dysfunction, and working memory is likely to become impaired among younger workers.

Relevant departments would therefore find it advisable to monitor the changes in working memory of younger workers and to adopt different monitoring strategies according to the characteristics of cognitive changes at different ages, stages of aging.

Studies have found that selenium and selenium compounds can significantly reduce levels of amyloid plaque and phosphorylated tau in the brain, and can improve cognitive deficits in the AD brain.

Therefore, it is recommended that aluminium workers eat more foods rich in selenium, such as peanuts and soybeans, etc. These can resist the damaging effects of aluminium.

Source: Environmental Toxicology and Pharmacology, Vol. 83, Article 103581, April 2021.

Cadmium Exposure and Type 2 Diabetes Mellitus

Cadmium (Cd) is a recognized human carcinogen, which is raising global concern regarding its ubiquitous presence in the environment and the consequent threat to public health. Since Cd and its compounds degrade only with difficulty, reported exposures via the contaminated environment mean that the toxic effects accumulate in the body and can lead to increasing damage in respiratory, circulatory, endocrine, kidney and skeletal systems.

Cadmium has been reported to disrupt the regulatory system of the hypothalamic-pituitary axis, resulting in alterations of hormone secretion and disorders of the endocrine system.

Due in part to the increasing prevalence of type 2 diabetes mellitus (T2DM), there has been more attention to the diabetogenic effects of exposure to environments contaminated with Cd.

As evidence accumulates, Cd exposure is increasingly being recognized as the driving force of T2DM. However, the longitudinal associations of chronic cadmium exposure with fasting blood glucose changes and T2DM have not been fully elucidated.

The present longitudinal prospective study focused on a community-based population in China. Urinary Cd concentrations were measured repeatedly over three years to test the reproducibility of urinary Cd concentrations.

Internal concentrations of Cd exposure may vary among individuals because of diverse exposure levels, routes, and duration. Blood Cd is reported to reflect only recent exposures, while urinary Cd is proportional to the total body burden.

Associations between urinary Cd, fasting blood glucose (FBG) changes, and T2DM in the general adult population were examined during a three year follow-up.

In the present study, positive dose-response relationships between urinary Cd and FBG and T2DM risk were observed among the general population in China.

The results showed that increased urinary Cd levels significantly related to elevated FBG levels. Upward trends of type 2 diabetes mellitus incidence were also significantly related to increasing urinary Cd levels.

Individuals with the highest urinary cadmium exposure had significant increases in fasting blood glucose change at follow-up. The risk of incident T2DM gradually increased as levels of urinary Cd elevated.

These findings suggest that relatively high chronic cadmium exposure across the adult population generally might contribute to elevated changes of FBG, resulting in the development of T2DM.

Source: Environmental Research, Vol. 192, Article 110259, January 2021.

Long-term Exposure to Ambient $PM_{2.5}$ and Stroke Mortality

Stroke is one of the great threats to global health, contributing to approximately 2.1 million deaths in China and 6.2 million deaths worldwide. Modifiable risk factors such as hypertension, obesity and physical inactivity, which are recognized as closely related to risk of stroke, have not been well controlled during the past decades.

Increasing observational and experimental studies are also finding that pollution in the form of fine particulate matter (particulate matter with aerodynamic diameter ≤ 2.5 μm, $PM_{2.5}$) may be a new risk factor for stroke and other cardiovascular diseases.

Most population-based studies associate short-term ambient $PM_{2.5}$ with the exposure documented risk of hospital admission or mortality due to stroke. However, evidence regarding the role of long-term $PM_{2.5}$ exposure in cerebro-vascular disease among residents in areas of high pollutionis still limited.

In China, ambient air pollution has emerged as one of the top risk factors contributing to cardiovascular disease burdens in 2017. To illustrate the $PM_{2.5}$-stroke relationship accurately, research evidence still requires independent validation from longitudinal cohort data with a wide range of $PM_{2.5}$ exposure.

There are a few studies which have examined how annual temperature variation may interact with air pollution, potentially impacting the association of long-term $PM_{2.5}$ exposure with stroke mortality.

The study combined satellite-based $PM_{2.5}$ exposure estimates with data from a cohort of urban adults to investigate the long-term effects of $PM_{2.5}$ exposure on stroke mortality. It also explored the associated effects of temperature variation on $PM_{2.5}$-mortality in northern China.

In the cohort data collected from among 38,140 participants in northern China during the period of 2000-2009, a significant association was observed between long-term exposure to $PM_{2.5}$ and stroke mortality among urban residents who had been living in highly polluted environments with annual average levels of $PM_{2.5}$ ranging from 39.0 $\mu g/m^3$ to 100.6 $\mu g/m^3$.

This extended follow-up study provided direct evidence that long-term exposure to high concentrations of $PM_{2.5}$ pollution in northern China has significantly increased stroke mortality.

These findings also support the association between annual temperature range, stroke mortality and air pollution. Despite existing uncertainties, these new findings expand our understanding that increasing rates of mortality are likely linked to long-term $PM_{2.5}$ exposure and temperature variations.

Source: Ecotoxicology and Environmental Safety, Vol. 213, Article 112063, April 2021.

WHO Report Warns that E-waste Affects the Health of Millions of Children

In June 2021, the World Health Organization (WHO) released its first comprehensive report on the dimensions of the global electronic waste (e-waste) problem. The report covers pathways through which children are exposed, the health effects associated with the different pathways of exposure, and actions that the health sector can take alongside other sectors globally, nationally and locally, and calls for more effective and binding action to protect children from the ever-growing health threat.

According to WHO's report, 53.6 million tons of e-waste were generated worldwide in 2019, a 21% increase over the past five years. This trend is expected to continue, with global e-waste generation projected to grow to 74.7 million tons by 2030.

Of the e-waste produced in 2019, only 17.4% reached formal management or recycling facilities, while the rest was illegally dumped, overwhelmingly in low- or middle-income countries, where it is recycled by informal workers.

The e-waste is dismantled, recycled and refurbished in environments where infrastructure, training and environmental and health safeguards may be non-existent or poorly adhered to. This places e-waste workers, their families and communities in greater danger of adverse health effects from e-waste recycling.

As many as 12.9 million women are working in the informal waste sector, which potentially exposes them to toxic e-waste and puts them and their unborn children at risk. Meanwhile, more than 18 million children and adolescents, some as young as 5 years of age, are actively engaged in the informal industrial sector, of which waste processing is a sub-sector. Children are often engaged by parents or caregivers in e-waste recycling because their small hands are more dexterous than those of adults. Other children live, go to school, and play near e-waste recycling centers, where high levels of toxic chemicals, mostly lead and mercury, can damage their intellectual abilities

Children exposed to e-waste are particularly vulnerable to the toxic chemicals they contain due to their smaller size, less developed organs, and rapid rate of growth and development. They absorb more pollutants relative to their size and are less able to metabolize or eradicate toxic substances from their bodies. Workers at these recycling sites, aiming to recover valuable materials such as copper and gold, are at risk of exposure to over 1,000 harmful substances, including lead, mercury, nickel, brominated flame retardants and polycyclic aromatic hydrocarbons (PAHs).

For an expectant mother, exposure to toxic e-waste can affect the health and development of her unborn child for the rest of its life. Potential adverse health effects include negative birth outcomes, such as stillbirths and premature births, as well as low birth weight and length. Exposure to lead from e-waste recycling activities has been associated with significantly reduced neonatal behavioral neurological assessment scores, increased rates of attention deficit/hyperactivity disorder (ADHD), behavioral problems, changes in child temperament, sensory integration difficulties, and reduced cognitive and language scores.

Other adverse child health impacts linked to e-waste include changes in lung function, respiratory and respiratory effects, DNA damage, impaired thyroid function and increased risk of some chronic diseases later in life, such as cancer and cardiovascular disease.

Effective and binding action is urgently required to protect the millions of children, adolescents and expectant mothers worldwide, whose health is jeopardized by the informal processing of discarded electrical or electronic devices according to this new ground-breaking report from the World Health Organization. Regulations and policies need to be put in place such that exporters, importers and governments ensure the environmentally-sound disposal of e-waste and the health and safety of workers, their families and communities; monitor e-waste exposure and health outcomes; facilitate better reuse of materials; and encourage the manufacture of more durable electronic and electrical equipment.

The report also calls on the health community to take action to reduce the adverse health effects from e-waste, by building health sector capacity to diagnose, monitor and prevent toxic exposure among children and women, raising awareness of the potential co-benefits of more responsible recycling, working with affected communities and advocating for better data and health research on the health risks faced by informal e-waste workers.

The WHO Initiative on E-waste and Child Health, launched in 2013, aims to increase access to evidence, knowledge and awareness of the health impacts of e-waste; improve health sector capacity to manage and prevent risks, track progress and promote e-waste policies that better protect child health; and improve monitoring of exposure to e-waste and the facilitation of interventions that protect public health.

Source: WHO Report. Children and digital dumpsites: e-waste exposure and child health. 15 June 2021. (https://www.who.int/publications/i/item/9789240023901)

Immunotoxic Role of Organophosphates: An Unseen Risk Escalating SARS-CoV-2 Pathogenicity

Organophosphates (OPs) are extensively used in agriculture, horticulture, forestry, veterinary-medicine, for domestic purposes and for the control of vector-borne diseases. Due to overuse, OP-residues have contaminated drinking water, grains, vegetables, fruits, soft drinks and other food items, provoking global health concerns.

Adequate studies have suggested that the "immunotoxic" potential of OP compounds in human and non-target organisms might increase the risk of infectious disease.

Cytochrome (CYP) P450 gene families are major determinants of biotransformation of OPs and other pesticides. Genetic polymorphism in CYPs is known to alter metabolic pathways, induce false cellular response, and may provoke a false pathological response. Thus, individuals with genetic polymorphism in CYP genes face less elimination of OP pesticides from the body.

It can be assumed that the gene-environment interaction between CYPs and OP exposure might induce oxidative stress (OS) and increase susceptibility to SARS-CoV-2 infection through immune suppression.

Unfortunately in many developing countries, OPs are being indiscriminately used in agriculture. This could lead to more unintentional exposure to these compounds, contributing to the suppression of immune responses to viral infections including SARS-CoV-2.

The current review article will cover the structure, transmission-pattern and respiratory-immunopathologies of SARS-CoV-2 in infected patients.

To curb SARS-CoV-2 infection, a healthy immune system is needed, despite any potent vaccine which can alleviate morbidities in patients. But unintentional exposure to OP compounds from several sources can rupture the antiviral defense against SARS-CoV-2. Moreover respiratory ailments may also be fueled by OP compounds.

Evidence cited in the review confirms that OPs disturb various components of immune machinery. These disruptions can be openings for opportunistic viral infections. It is already known that several respiratory difficulties, including allergic-asthma, pneumonitis, and chronic bronchitis are positively correlated with OP exposure.

Since organophosphate-exposure promotes inflammation and respiratory troubles, during the current pandemic situation any additional exposure to such chemicals could exacerbate inflammatory outcomes and pulmonary maladies in patients. Pre-exposure to OPs might turn out to be a risk factor for compromised immunity.

OP induced oxidative stress is the major driver of apoptosis in lymphoid organs involved in maturation of lymphocytes and the development of antibodies of high affinity against viral antigens. Therefore, OP mediated apoptotic lesions in lymphoid tissues may also dampen to some extent the efficacy of otherwise potent vaccines.

Since direct experimental works dissecting the collaborative impacts of OPs and SARS-CoV-2 are still lacking, this article will urge the scientific community to concentrate on the proposed hypothesis to unveil the synergism between these two new threats to the human-race.

Serious health problems discussed in the current literature will also draw the attention of global environment policy makers and concerned government/ nongovernment organizations towards the perilous impacts of OP-exposure in humans. At the same time, it will insist that they adopt necessary resolutions and amend policies that could limit human contacts with OPs.

In summary, OP compounds are immunotoxic since they promote apoptosis of immunocytes, lower immunoglobulin titer and autoimmune responses in organisms. Similar to OPs, SARS-CoV-2 targets the immune system by modulating interferon response, lymphocyte count, cytokine homeostasis and autoimmune reaction.

Hence, it is not unreasonable to claim that, co-exposure to both of the agents during the existing pandemic milieu may alter the disease outcome of patients with COVID-19.

Finally, it is important to note that, antioxidants may rescue the immune system from detrimental impacts of OP compounds and hence diets rich in antioxidants would be helpful in maintaining the healthy immune system that is essential to overcoming COVID-19.

Source: Food and Chemical Toxicology, Vol. 149, Article 112007, March 2021.

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