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$PM_{2.5}$ as a Potential SARS-CoV-2 Carrier

The rapid spread of SARS-CoV-2 in the COVID-19 pandemic has raised questions as to the route of transmission of this disease. The initial understanding was that transmission originated from respiratory droplets passing from an infected host to a susceptible host. However, indirect contact transmission of viable virus by fomites and through aerosols has also been suggested.

Finer, virus-laden respiratory droplets and particulate matter (≤5 μm) can remain in the air for an extended period and be carried even beyond 6 meters.

Despite numerous studies that have demonstrated the transmission route of SARS-CoV-2 via respiratory droplets, evidence on aerosols-borne transmission remains limited.

Particulate matter, $PM_{2.5}$, are fine solids with a particle diameter of ≤2.5 μm. They are commonly found suspended in ambient air aerosols. No correlation has been found between the virus concentration and particulate matter's diameter. Nevertheless, positive correlations between $PM_{2.5}$ and other respiratory viruses such as influenza have been reported, emphasizing the possibility that particulate matter carries and transports SARS-CoV-2.

$PM_{2.5}$ in indoor environments is mainly derived from common outdoor sources such as motor vehicles, biomass burning, and industrial emissions. Prolonged exposure to $PM_{2.5}$ is particularly detrimental to human health as this fine particulate matter can be easily inhaled and penetrate deep into the lungs.

$PM_{2.5}$ is known to have a significantly longer lifetime in the air where it can be suspended for an extended period, compared to respiratory liquid droplets. This longer lifetime of particles may pose a significant viral exposure to healthcare personnel, especially in indoor environments.

$PM_{2.5}$ can also be deposited on surfaces in indoor environments such as hospital floors, for example. This fine particulate matter also readily becomes airborne in tiny turbulent eddies stirred up by physical activities such as human movements and walking.

Considering the fact that the viability of SARS-CoV-2 on many types of surfaces has been reported (e.g., on metals for 48 hours, plastic for 72 hours, cardboard for 24 hours, and copper for 4 hours), it is likely that the virus on the surface can be potentially lodged on the $PM_{2.5}$ and redistributed/transported back into the air.

Recent findings based on air particle measurements suggest that SARS-CoV-2 can be carried aloft by $PM_{2.5}$ when healthcare workers remove their personal protective equipment (PPE).

It has also been suggested that fine, suspended dust in the air could join with microorganisms of a diameter smaller than 5 μm during aerosolization. Since the diameter of the SARS-CoV-2 is two orders of magnitude smaller, approximately 70-90 nm, the mechanism/mode of its airborne transport is still unclear and, therefore, worth exploring.

In this study, the researchers hypothesize the possible role of $PM_{2.5}$ as a carrier (or transport agent) keeping SARS-CoV-2 airborne. $PM_{2.5}$ was collected over a period of four weeks during 48-h measurement

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$PM_{2.5}$ as a Potential SARS-CoV-2 Carrier

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intervals from four separate hospital wards containing different infected clusters in a teaching hospital in Kuala Lumpur, Malaysia.

The results indicated the highest SARS-CoV-2 RNA on $PM_{2.5}$ in the ward with a number of occupants.

There is a link between the virus-laden $PM_{2.5}$ and the ward's design. The number of patients and their symptoms influence the number of airborne SARS-CoV-2 RNA with $PM_{2.5}$ in an enclosed environment.

The results clearly indicated that SARS-CoV-2 RNA was present within the sampling of ambient particles. Hence, it is crucial to determine whether these RNAs came from intact virus particles or were merely RNA from non-infectious virus particles. Thus, it was suggested that infectious virus be identified by culturing the virus residing on the $PM_{2.5}$ in an appropriate cell culture.

This study could not show a direct link between the concentration of $PM_{2.5}$ and SARS-CoV-2. However, the results did show that $PM_{2.5}$ generated from human activities in healthcare facilities can influence the presence of SARS-CoV-2 RNA in indoor environments.

Furthermore, the degree of viral shedding from symptomatic patients may influence the presence of SARS-CoV-2 RNA on $PM_{2.5}$. Therefore, all possible precautions against airborne transmission in indoor environments should be taken seriously.

Source: Scientific Reports. Vol. 11, Article 2508, January 2021.

Decreased Serum Bilirubin Levels in Children with Lead Poisoning

Children are especially vulnerable to the toxicity of lead which is now affecting multiple systems..

Lead is a toxic heavy metal that is commonly used in daily necessities, such as paint, gasoline, water pipes, storage batteries, and many other products. Children are vulnerable to lead poisoning because their respiratory and digestive tracts have a higher absorption rate of lead, compared with adults.

Lead can delay growth and development, impair hearing, increase dental caries, and alter children's cognition and behavior after absorption. A total of 75% of absorbed lead is captured by the liver. The second most immediately impacted is the kidney.

Lead damages children in many ways. It impairs neurological development (e.g., behavioral changes, mental impairment, seizures, and coma), creates gastrointestinal issues (e.g., abdominal pain, constipation, nausea, and vomiting) and results in decreased growth in height and delayed sexual maturation.

Oxidative stress is the underlying mechanism of lead-induced organ injury. Lead is a redox-inactive metal. It shows its pro-oxidative activity by generating reactive oxygen species and depleting cellular antioxidant reserves. The pro-oxidative activity of lead results in irreversible neurological damage to children without proper treatment.

Therefore, monitoring the level of oxidative stress in children with lead poisoning is of great importance. Some available biochemical parameters, such as bilirubin, albumin, creatinine, and uric acid, are regarded as endogenous non-enzymatic antioxidants.

The present hospital-based, sex- and age-controlled, paired study aimed to investigate antioxidant levels of biochemical components in children with lead poisoning.

The study analyzed clinical characteristics and measured serum levels of total protein, globulin, albumin, bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), urea, and creatinine.

In the study, 78.9% of patients had neurological symptoms, and some were accompanied by gastrointestinal disorders or developmental retardation.

The results showed that albumin, bilirubin, urea, and creatinine levels were significantly lower and AST, total protein, and globulin levels were higher in children with lead poisoning than in controls.

In conclusion, lead interferes with the non-enzymatic antioxidant system in children, also uniquely showing a decrease in serum bilirubin levels.

Bilirubin is a metabolite of hemoglobin. The interactions between lead and hemoglobin metabolism are complex. Lead may increase bilirubin levels via induction of hemoglobin degradation. However, depletion of the hemoglobin pool may result in less synthesis of bilirubin.

In this study, the decrease in bilirubin levels was not affected by its source (hemoglobin); there was only a weak correlation between them. Therefore, insufficient hemopoiesis did not account for decreased direct bilirubin levels.

Additionally, the role of bilirubin in the body is complex. Bilirubin exerts toxic effects when present in excess, but it acts as a non-enzymatic antioxidant at the proper concentration. The researchers hypothesized that reduced bilirubin levels in children with lead poisoning are most likely due to decompensated depletion against oxidation caused by lead.

The possible association between lead toxicity and decreased serum bilirubin levels in children requires further investigation.

Source: Journal of International Medical Research, Vol. 49, Issue 2, Article 300060521990248, February 2021.

Effects of Air Pollution on Dementia and Cognitive Function in Adult Population: The Epidemiological Evidence

Dementia is arguably the most pressing public health challenge for our age. Its prevalence is strongly age-related: doubling every 5-6 years over the age of 65. The number of people living with dementia worldwide is estimated at 50 million and is expected to reach 152 million by 2050.

Since there is as yet no cure, identifying risk factors that can be controlled has become paramount in attempts to reduce the personal, societal and economic burdens of dementia.

More recently, epidemiological studies have pointed to possible effects on the brain, such as decline of cognitive function and development of dementia, associated with exposure to air pollutants.

There are indirect mechanisms by which pollutants could potentially lead to brain injury, including damage to the vasculature, leading to cerebral ischaemia or extravasation of neurotoxic proteins such as fibrinogen. Brain injury could also be secondary to systemic inflammatory responses to air pollution.

There is great interest in reducing the risk of dementia by identifying preventable risk factors. Epidemiological evidence linking exposure to air pollutants with adverse effects on cognition and the development of dementia has expanded appreciably over the past 15 years.

This review of literature up to December 2019 critically examines the available epidemiological evidence of associations between exposure to ambient air pollutants, cognitive performance, acceleration of cognitive decline, risk of developing dementia and neuroimaging and neurological biomarker studies, following Bradford Hill guidelines for causality.

The evidence reviewed is consistent in reporting associations between chronic exposure to air pollution and reduced global cognition. The evidence is also consistent in reporting associations between air pollution and reduced performance in visuo-spatial abilities.

The findings are heterogeneous as regards to cognitive domains such as executive function, attention, memory, language and mild cognitive impairment.

Incidence of cognitive decline and dementia has also been consistently associated with exposure to air pollution. The strength of association reported in some studies suggests a potentially important impact on public health.

Neuro-imaging studies reported associations between exposure to air pollution and white matter volume reduction. Other reported effects include reduction in gray matter, larger ventricular volume, and smaller corpus callosum.

Findings related to ischemic (white matter hyperintensities/silent cerebral infarcts) and hemorrhagic (cerebral microbleeds) markers of cerebral small vessel disease have been mixed, as observations on hippocampal volume and air pollution.

The few studies available on neuro-inflammation tend to report associations with exposure to air pollution.

Several effect modifiers have been suggested in the literature. However, very few studies have analysed whether these factors act as effect modifiers, and results are heterogeneous. More replication studies are required to evaluate whether these factors are effect modifiers.

The available evidence, which has been reviewed with reference to Bradford Hill's features of causal associations, suggests that long-term exposure to air pollutants is associated with cognitive decline and with the risk of development of dementia.

Temporal misalignment (of putative causes and effects) could potentially affect the documentation of associations between exposure to air pollution and cognitive and neurological changes.

Most of the studies considered exposures representative in one to ten years prior to cognitive testing, dementia incidence or neuroimaging. However, exposures over one to ten years might not be representative of exposures over a longer term (e.g. 30 years), which may be more relevant to the effect under consideration.

Typical confounding factors have been accounted for the majority of the reviewed studies. Additional factors have also been controlled in some individual studies. However, the adjustment of these factors has been heterogeneously implemented.

A list of possible confounding factors, such as social interactions, physical activity, sleep deprivation and other features of urban living which none of the studies has controlled for, has been compiled.

Despite the efforts to adjust for confounding factors, residual confounding cannot be completely ruled out, especially since the factors affecting cognition and dementia are not yet fully understood.

However, the diversity of study designs, air pollutants, and endpoints examined precludes the attribution of these adverse effects to a single class of pollutant and makes meta-analysis inappropriate.

In conclusion, there is substantial epidemiological evidence to suggest a causal association between exposure to a range of air pollutants and a number of effects on the nervous system, including the acceleration of cognitive decline and the induction of dementia.

Source: Science of The Total Environment, Vol. 757, Article 143734, February 2021.

Serum Nickel and Craniosynostosis Risk: Evidence from Humans and Mice

Heavy metal pollution is increasing in water, soil, and food in many parts of the world, especially in certain developing countries (including China).

Most epidemiological studies on metals to date have investigated single metal and disease outcomes. However, in the real-world environment, people are often exposed to a variety of metals.

Many recent studies have shown that mixed metal exposure affects human health, increasing the risk of atherosclerosis, hypercholesterolemia, type 2 diabetes, osteoporosis, cardiovascular mortality, and breast cancer.

However, there have been few studies on mixed metal exposure and adverse developmental outcomes, and no reported studies have linked metal exposure to craniosynostosis (CS).

Craniosynostosis (CS) is a congenital birth defect in which the bones in an infant's skull join early. Such abnormal and non-physiological suture fusion is considered an adverse developmental outcome because the premature fusion of one or more cranial sutures can lead to skull deformity and facial asymmetry. It is estimated that the prevalence of CS worldwide is 1/2500.

Furthermore, if left untreated, CS can lead to serious complications, including increased intracranial pressure (ICP); delays in reaching language, cognitive, social, or motor skill milestones; facial malformation; sensory deficit; neurological dysfunction affecting the eyes; psychological disorders; fractured ventricular syndrome; and bleeding.

To date, the aetiology of CS remains unclear. Previous studies have found that genetic factors (such as fibroblast growth factor receptor [FGFR] mutation) and environmental exposure, including maternal smoking, drinking, thyroid disease, and antidepressant and antiepileptic drug use, may be predisposing factors for CS.

The purpose of this study is to investigate the association between metal exposure and the risk of CS by conducting epidemiological and experimental studies.

A total of 6 metals (chromium [Cr], nickel [Ni], tin [Sn], arsenic [As], thallium [Tl], and lead [Pb]) in serum were analyzed in a case control study to first evaluate whether an association exists between exposure to multiple metals and CS occurrence.

The metal with the strongest association in the population with regard to inducing CS-like phenotypes was investigated in a mouse model. *In vitro* cell experiments were performed to observe the possible mechanism of metal-induced CS.

This study will provide new conceptual guidance for the study of metal toxicity and will offer new ideas for the prevention of CS in humans in the future.

Bayesian kernel machine regression (BKMR) models were used to account for joint metal effects. Multiple logistic regression analysis was used to explore the association between metal concentration and CS occurrence, with adjustment for potential confounders.

In this study, the results showed that increased levels of mixed metals were associated with an increased risk of CS. The results of single metal analyses also suggested that elevated serum Ni concentrations increased the risk of CS.

Furthermore, the width of the sagittal suture decreased in mice exposed to Ni, similar to the phenomenon of CS, and mineralized nodules in cranial sections increased in a dose-dependent manner. After Ni exposure, osteoblasts showed dose-dependent increases in the gene and protein expression levels of osteocalcin.

Findings from the study highlight the association of Ni with CS and emphasize the importance of assessing the effects of chemical mixtures on health outcomes using multiple statistical methods and experiments in mice and cell lines.

The purpose of this study was to assess the relationship between exposure to a mixture of metals and CS risk. BKMR analysis showed the nonlinear exposure response effect of each chemical, the interaction between mixtures and the overall effect.

The risk of CS increased as exposure to mixed metals increased. According to the BKMR model, Ni, Sn and Cr appear to be the greatest contributors.

Currently, no study has analysed the association between mixed metal exposure and CS risk. Additional research is needed to verify the results.

The researchers carried out experiments in mice and observed early closure of mouse cranial sutures caused by Ni treatment. The results showed that Ni can cause the occurrence of CS in mice and that the classic genetic marker of osteogenic differentiation (OCN) increased in a dose-dependent manner in cell lines.

These results may provide a basis for determining internal exposure limits associated with adverse effects of Ni. Previous studies have found that Ni is toxic and that occupational exposure to Ni has impacts on human health.

Additionally, the Environmental Protection Agency (EPA) recommends that drinking water levels for Ni should not be more than 0.1 mg/L. However, there are recently no regulations or recommendations to regulate the concentration of Ni in the human body.

Metal imported from the external environment to the blood undergoes gastrointestinal digestion and liver metabolism. Hence, the concentration of metals in the blood can better reflect the level of internal exposure in the human body.

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Co-exposure to PCB126 and PFOS Increases Biomarkers Associated with Cardiovascular Disease Risk and Liver Injury in Mice

Polychlorinated biphenyl (PCB) 126 and perfluorooctane sulfonic acid (PFOS) are halogenated organic pollutants of high concern. Exposure to these chemicals is ubiquitous, and can lead to potentially synergistic adverse effects in individuals exposed to both classes of chemicals.

Although the production of PCB126 and PFOS has been banned in many countries and worldwide, and human exposure levels have continued to decline, these chemicals are still detectable in many individuals.

Ingestion of contaminated food and drinking water is the main route of exposure to both PCB126 and PFOS in the general population. Both chemicals are readily absorbed and poorly eliminated from humans and rodents.

The half-life of PCB126 is approximately 17 days in rodents and 4.5 years in humans. The half-lives of PFOS range from days in mice (30-42 days) to years in humans (4.0-5.8 years).

However, little is known about how these two classes of environmental pollutants might interact to impact human health.

The present study was designed to identify possible interactions between PCB126 and PFOS that might promote acute changes in inflammatory pathways associated with cardiovascular disease and liver injury.

Male C57BL/6 mice were exposed to vehicle, PCB126, PFOS, or a mixture of both pollutants. Plasma and liver samples were collected at 48 h after exposure. Changes in the expression of hepatic genes involved in oxidative stress, inflammation, and atherosclerosis were investigated.

The study demonstrated the additive synergistic effects of PCB126 and PFOS on hepatic expression of redox, inflammation, and atherogenic genes, including Nqo1, Icam1 and PAI1.

Additionally, exposure to mixtures significantly induced circulating levels of PAI1 protein, a biomarker for cardiovascular disease risk. Furthermore, mice exposed to these mixtures developed liver injuries.

Lipidomic analysis revealed that co-exposure to the mixture enhanced hepatic lipid accumulation and elevated levels of oxidized phospholipids. If translatable to humans, these effects might result in liver injury and increased risk of cardiovascular disease.

Taken together, the results suggest that co-exposure to PCB126 and PFOS could promote redox stress and further enhance the production of oxidized phospholipids (OxPLs), which can potentially lead to the observed upregulation of antioxidant and atherogenic gene/protein expression profile.

We hypothesize that the higher levels of OxPLs induced by the PCB126 and PFOS mixture could also contribute to increased inflammation and the development of cardiometabolic disease.

While this is an acute study, using a high dose of PFOS and looking at short term effects of interactions between two different pollutant classes, it is also important to consider doses relevant to human exposure and to address additional factors such as diet and longer duration of exposures, all of which can modify toxicity outcomes.

In summary, this study shows that acute co-exposure to PCB126 and PFOS in mice results in liver injury and increased cardiovascular disease risk.

Future studies involving high fat diet, chronic co-exposure, and a dose related with human exposure will be useful to better understand the mechanistic outcomes of PCB126 and PFOS mixture effects on the liver-cardiovascular axis and subsequent metabolic alterations.

The study provides new evidence that pollutant mixtures can induce a toxicity profile that is distinct from individual pollutant exposure, thereby underlining the importance of investigating effects of mixtures of chemicals or pollutants in risk assessment and biomonitoring experimental settings.

Source: Toxicology and Applied Pharmacology, Vol. 409, Article 115301, December 2020.

Serum Nickel and Craniosynostosis Risk: Evidence from Humans and Mice

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The Centers for Disease Control (CDC) of the United States has recommended that levels of Pb and other metals, when greater than 5 μg/dL in children's blood should be considered to have damaging effects on health.

The results are similar to previous reports which have suggested that non occupational exposure to Ni may be toxic to child development. Together, these findings may provide a basis for formulation of government regulations or policies.

However, additional studies are needed to demonstrate the threshold values or cut off points for developmental toxicity and other harmful effects of Ni concentrations in the blood.

This study is the first to provide evidence associating increased serum Ni with an increased risk of CS. Early life exposure to Ni promotes osteogenesis during skull growth, which may contribute to the development of CS.

Source: Environment International, Vol. 146, Article 106289, January 2021.

U.S. EPA: The New Lead and Copper Rule

On December 22, 2020, the United States Environmental Protection Agency (U.S. EPA) announced the first major update to the agency's Lead and Copper Rule (LCR) in nearly 30 years. This historic action strengthened every aspect of the LCR and accelerated actions that reduce lead in drinking water to better protect children from lead exposure.

This new Lead and Copper Rule will better protect children and families in the U.S. from exposure to lead in drinking water. For the first time in nearly thirty years, this action incorporates best practices and strengthens every aspect of the rule, including closing loopholes, accelerating the real world pace of lead service line replacement, and ensuring that lead pipes will be replaced in their entirety.

In older homes and buildings, lead can leach from service lines, solder, and fixtures into tap water and become a significant source of lead exposure. In children, lead exposure can cause irreversible and life-long health effects, including decreasing IQ, focus, and academic achievement.

The U.S. EPA has made tremendous progress in lowering children's blood lead levels by phasing lead out of gasoline, banning lead paint, and implementing the old LCR. The old rule included deficiencies that have been corrected by EPA's new Lead and Copper Rule.

For example, the old rule created so many loopholes that since 1991, over nearly 30 years, only 1 percent of utilities actually replaced lead pipes as a result of an action level exceedance.

The old LCR also allowed up to 48 months to pass in small U.S. towns before corrosion control was in place after a water system exceed was found to the action level (15 ppb). It also failed to require all systems to test for lead in drinking water in their elementary schools or child care facilities.

The EPA's new Lead and Copper Rule better protects children and communities from the risks of lead exposure by testing drinking water at elementary schools and child care facilities, removing lead from U.S. citizens' drinking water, and empowering communities through information. Improvements under the new rule include:

- Using science-based testing to better locate elevated levels of lead in drinking water.
- Establishing a trigger level (10 ppb) to jumpstart mitigation earlier and in more communities.
- Driving more complete lead service line replacements.
- For the first time, requiring testing in elementary schools and child care facilities.
- Requiring water systems to identify and make public the locations of lead service lines.

For more information on the new Lead and Copper Rule, visit: https://www.epa.gov/ground-water-and-drinking-water/final-revisions-lead-and-copper-rule.

Source: U.S. EPA. The New Lead and Copper Rule, December 22, 2020.

Long-Term Exposure to Outdoor Black Carbon and Carcinogenicity of Air Pollution

Black carbon (BC), a component of fine particulate matter [particles with an aerodynamic diameter ≤2.5 μm ($PM_{2.5}$)], comes from incomplete combustion processes, mainly from anthropogenic sources such as fossil fuel or biomass burning.

Strong evidence over recent decades has led to the classification of outdoor air pollution and $PM_{2.5}$ as carcinogenic (IARC, 2016). Yet the separate effects of each $PM_{2.5}$ component (sulfates, nitrate, ammonium, organics, metals, etc.) have rarely been quantified.

Studies are accumulating which link exposure to BC with increased morbidity and mortality, including lung cancer mortality, lower lung function and slower cognitive development in children, increased bone loss, and decreased cognitive functions in the elderly.

BC may contribute to the carcinogenic effects of air pollution. Although evidence has accumulated as to the toxicity of BC, there has been little evidence about the effects of chronic low-level exposure on cancer risk.

This study aimed to estimate the associations between long-term exposure to BC and risk of cancer in the population-based French Gazel cohort with a 26 year follow-up.

In the Gazel cohort of a predominantly male population of French adults who were employees of the national gas and energy company, long-term BC exposure was positively associated with incident all-site cancer and lung cancer, based on single-pollutant models of cumulative exposure, and on models of BC residuals used to estimate the effect of BC as a $PM_{2.5}$ constituent, while holding the effect of total $PM_{2.5}$ constant.

The Gazel cohort provides a large and well characterized study population with many variables collected throughout the follow-up.

The findings suggest that BC may partly explain the association between $PM_{2.5}$ and lung cancer. Additional studies are needed to further disentangle the effects of BC, total $PM_{2.5}$, and other constituents.

Source: Environmental Health Perspectives, Vol. 129, No. 3, Article EHP8719, March 2021.

The Toxic Truth:
Children's Exposure to Lead Pollution Undermines A Generation of Future Potential

The joint report "The Toxic Truth: Children's exposure to lead pollution undermines a generation of potential" by UNICEF and Pure Earth, notes that lead is a potent neurotoxicant that causes irreparable harm to children's brains.

Around 1 in 3 children, up to 800 million globally, have blood lead levels at or above 5 micrograms per decilitre (μg/dL), a level that the World Health Organization and the United States Centers for Disease Control and Prevention have stated requires global and regional interventions.

Lead is particularly destructive to babies and children under the age of 5 as it damages their brain before they have had the opportunity to fully develop, causing them lifelong neurological, cognitive and physical impairment.

Childhood lead exposure has also been linked to mental health and behavioral problems and an increase in crime and violence. Older children suffer severe consequences, including increased risk of kidney damage and cardiovascular diseases in later life.

Childhood lead exposure is estimated to cost lower- and middle-income countries almost USD $1 trillion due to lost economic potential of these children over their lifetime.

Informal and substandard recycling of lead-acid batteries is a leading contributor to lead poisoning in children living in low and middle-income countries.

Other sources of childhood lead exposure include lead in water from the use of leaded pipes; lead from active industry, such as mining and battery recycling; lead-based paint and pigments; leaded gasoline; lead solder in food cans; and lead in spices, cosmetics, ayurvedic medicines, toys and other consumer products.

Parents whose occupations involve working with lead often bring contaminated dust home on their clothes, hair, hands and shoes, thus inadvertently exposing their children to the toxic element.

The Toxic Truth: Children's Exposure to Lead Pollution Undermines a Generation of Future Potential

While blood lead levels have declined dramatically in most high-income countries since the phase-out of leaded gasoline and most lead-based paints, blood lead levels for children in low- and middle-income countries have remained elevated and, in many cases, dangerously high even a decade after the global phase-out of leaded gasolines.

The report features five country case studies where lead pollution and other toxic heavy metal waste have affected children. These are Kathgora, Bangladesh; Tbilisi, Georgia; Agbogbloshie, Ghana; Pesarean, Indonesia; and Morelos State, Mexico.

It is clear from evidence compiled that lead poisoning is a much greater threat to the health of children than previously understood. Although much more research needs to be conducted, enough data have recently emerged for decisive action to begin, and it must begin now.

Global and regional action includes creating global standard units of measure to verify the results of pollution intervention on public health, the environment and local economies; building an international registry of anonymous results of blood lead level studies; and creating international standards and norms around recycling and transportation of used lead acid batteries.

For more information, please visit: https://www.unicef.org/reports/toxic-truth-childrens-exposure-to-lead-pollution-2020

Source: UNICEF and Pure Earth. The Toxic Truth. July 2020.

Mercury and Alzheimer's Disease

Mercury is a common environmental toxicant. It is found in the atmosphere mostly as a result of human activities, such as coal burning for heating and cooking, but it is also released naturally by volcanic eruptions, in the form of vapor, or in the weathering of rocks.

The form of mercury most toxic to humans is methylmercury, to which humans are exposed by ingestion of fish. Methylmercury exerts its toxic effects on different parts of the human body, especially the brain.

Mercury toxicity depends on dose, form, and duration of exposure. Therefore, continued scrutiny and assessment of the safety of mercury is paramount in order to preempt unexpected consequences. One of these consequences is postulated to be neurodegeneration, which might lead to Alzheimer's dementia.

Dementia is a devastating, multifactorial disease affecting around 50 million people worldwide. It is most prevalent in the elderly population. The most common form of dementia is Alzheimer's disease (AD), a neuro-degenerative disorder which is a product of interactions among genes, environment, socioeconomic status and lifestyle.

This recent review investigates a specific environmental-disease inter-action between mercury exposure and the hallmarks of Alzheimer's disease. It summarize the avenues by which mercury can affect the pathogenesis of this disorder.

There is no safe concentration for mercury in the atmosphere. Even trace amounts can be harmful to humans in the long term.

Alzheimer's disease (AD) is a neurodegenerative disease which primarily impacts the memory of affected individuals. It is most common in the elderly. There are many cumulative causative agents potentially contributing to the progress of the disease.

Nearly 50 million people worldwide are affected. AD is considered to be one the most devastating diseases, not only for the patient, but also for their families and caregivers.

The effects of mercury on AD, the hallmarks of formation, the extracellular senile plaques and the intracellular neurofibrillary tangles, have been widely studied.

This review demonstrates the involvement of mercury, in its different forms, in the pathway of amyloid beta deposition and tau tangles formation. It aims to understand the link between mercury exposure and Alzheimer's so that prevention strategies can be applied to halt progression of disease.

The progressive nature of AD imposes particularly profound socioeconomic burdens. Therefore, the quest to understand the underlying neuropathology of the disease and to resist its onslaught has been steadily and tenaciously pursued.

The interplay of many converging components gives Alzheimer's disease its pathogenesis, but the role of mercury may be more insidious than we know.

It would definitely be good to lend more focus to studying individuals who are occupationally exposed to high levels of mercury. For future endeavors, the researchers suggest the integration of mercury screening and regular checkups, including collection of serum and cerebro-spinal fluid (CSF) levels of AD, in order to retrieve more data and to better interpret the information.

Differences in responses to mercury might also arise according to duration of exposure, whether lifelong/ chronic (as in pollution) or acute. Chronic effects could be assessed in longitudinal studies in countries where levels of mercury are above average, in India or China, for example.

Some physiological characteristics, like chronic disease, sex, or pregnancy may account for differences in response to mercury toxicity. These variables should also be assessed.

Several avenues of research are evident in the investigation of myriad aspects of mercury neurotoxicity and its potential contribution to a better understanding of neurodegenerative diseases in general.

Recognizing the sporadic and chronic nature of AD development, the long term, including the prenatal, must be considered in addition to the environmental milieu of pollutant mixtures. There are major risk factors and targets of mitigation, but there are also avenues of exploration towards new diagnostic and prognostic modalities.

Source: Metabolic Brain Disease, Vol. 36, Pages 361-374, January 2021.

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