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$PM_{2.5}$ and Antibiotic Resistance

Antibiotic resistance is increasingly becoming a threat to global health, causing approximately 1.27 million premature deaths worldwide in 2019, substantially exceeding the estimated 0.70 million deaths in 2016.

The One Health approach recognizes that antibiotic resistance is not just a human health issue, but also affects animals and the environment.

Humans are exposed to antibiotic-resistant bacteria and antibiotic-resistance genes via food, the environment, or direct contact with infectious sources, such as animals. Antibiotic-resistant bacteria and antibiotic-resistance genes in hospitals or livestock farming could be transmitted to sewage-treatment facilities or ecosystems, and could even be emitted from these settings into the atmosphere and be exposed to humans through inhalation.

The major air pollutant, in the form of particulate matter $(PM)_{2.5}$, contains diverse antibiotic-resistant bacteria and antibiotic-resistance genes, which are transferred between environments and directly inhaled by humans, causing respiratory-tract injury and infection. $PM_{2.5}$ could also increase cell-membrane permeability to enhance the efficiency of horizontal gene transfer, accelerating the evolution and exchange of antibiotic-resistance elements in bacterial pathogens. However, understanding of the contribution of $PM_{2.5}$ to global antibiotic resistance is poor.

The present study aimed to collect an extensive database of antibiotic resistance and predictors to explore whether $PM_{2.5}$ is a primary factor driving global antibiotic resistance.

Data on multiple potential predictors (ie, air pollution, antibiotic use, sanitation services, economics, health expenditure, population, education, climate, year, and region) were collected in 116 countries from 2000 to 2018 to estimate the effect of $PM_{2.5}$ on antibiotic resistance using univariate and multivariate analysis.

This analysis represents the first global estimates of antibiotic resistance and burden of premature deaths attributable to antibiotic resistance resulting from $PM_{2.5}$ pollution.

The final dataset included more than 11.5 million tested isolates. Raw antibiotic-resistance data included nine pathogens and 43 types of antibiotic agents.

Significant correlations between $PM_{2.5}$ and antibiotic resistance were consistent globally in most antibiotic-resistant bacteria, and correlations have strengthened over time.

The findings show a consistent association between $PM_{2.5}$ and aggregate resistance across regions and pathogens, indicating that $PM_{2.5}$ is one of the primary factors driving global antibiotic resistance.

Furthermore, $PM_{2.5}$ can facilitate the horizontal gene transfer of antibiotic-resistance genes between bacteria. A higher frequency of antibiotic-resistance gene exchanges might lead to a more frequent emergence of antibiotic-resistant bacteria during antibiotic treatment.

Taken together, these findings emphasize the importance of air environments as antibiotic-resistance diffusion vectors and reservoirs.

The results highlight the fact that controlling air pollution to reduce $PM_{2.5}$ concentrations might lead to substantial health and economic benefits by reducing antibiotic resistance.

Source: The Lancet Planetary Health, Vol. 7, Issue 8, Pages e649-e659, August 2023.

Impact of Outdoor Air Pollution in COVID-19 Pneumonia Patients

Air pollution is the world's leading environmental cause of illness and premature death. According to the World Health Organization (WHO, 2018), about seven million deaths a year across the world are attributable to air pollution.

Air pollution is a complex mixture of gaseous and particulate components that vary both temporally and spatially. Outdoor air pollution exposure (OAPE) has been identified as a cause of higher morbidity and mortality in viral and bacterial lower respiratory tract infections and pneumonia.

Epidemiological studies have previously investigated impacts of particulate matter (PM) and gaseous pollutants such as nitrogen oxides (NO_X) and ozone (O_3) on COVID-19 outcomes. Among the pollutants studied, COVID-19 mortality appears to be most closely related to $PM_{2.5}$ and NO_2.

Experimental studies have shown that air pollution can decrease immune response and, in the respiratory tract, facilitate viral entry through angiotensin-converting enzyme 2 by increasing protease activity, which might facilitate SARS-CoV-2 infection. Most severe forms of COVID-19 and deaths associated with the disease have been related to a disproportionate systemic inflammatory response.

Air pollution plus SARS-CoV-2 infection, may have a multiplicative effect on inflammatory response exacerbating the cytokine storm.

Consequently, inferring more severe respiratory epithelium damage and immune dysregulation, pulmonary vascular endothelial cell apoptosis, inflammation and activation of prothombotic state, leading to alveolar edema, acute respiratory distress syndrome (ARDS), multiple organ failure and death.

Additionally, air pollution is associated with the decompensation of pre-existing comorbidities, increasing COVID-19-related morbidity and mortality.

The present study aimed to examine the relationship between exposure to outdoor air pollution and the risk of death in patients with COVID-19 pneumonia, and to investigate the impact of air pollutants on gas exchange and systemic inflammation in this disease.

The results showed that both risk of COVID-19 death and C-reactive protein (CRP) level increased significantly with median exposure to PM_{10}, NO_2, NO and NO_X, independently of the levels of other pollutants analyzed ($PM_{2.5}$, and O_3).

Higher exposure to NO_2, NO and NO_X was associated with lower single peripheral blood oxygen saturation/fraction of inspired oxygen (SpO_2/FiO_2) ratios.

In conclusion, after controlling for socioeconomic, demographic and health-related variables, the results showed evidence of a significant positive relationship between air pollution and mortality in patients hospitalised for COVID-19 pneumonia. Additionally, inflammation (CRP) and gas exchange (SpO_2/FiO_2) in these patients were significantly related to exposure to air pollution.

Therefore, exposure to these pollutants, largely due to vehicle emissions, should be considered an important risk factor for severity and adverse outcomes in COVID-19.

These results highlight the importance of decreasing air pollution levels, and in particular, the need to implement specific public health measures to address this risk factor by reducing people's exposure, such as cutting emissions from road traffic in areas with high levels of NO_2, NO, NO_X and PM_{10}.

Source: Science of The Total Environment, Vol. 894, Article 164877, October 2023.

Exposure to Chemical Pollutants and Sleep Outcomes

Sleep is a dynamic neurophysiological process regulated by a homeostatic sleep drive and a circadian rhythm in wakefulness. Healthy sleep is a multidimensional construct defined by sleep duration, efficiency, timing, alertness, and quality.

Disrupted or impaired sleep and sleep-related disorders such as obstructive sleep apnea (OSA) or chronic insomnia are common. The pervasiveness of these outcomes is alarming because sleep is essential for metabolic, immunologic, developmental, and cognitive functioning.

In addition to structural and sociodemographic factors, health status, and individual behaviors, environmental factors are particularly important for sleep health.

Prior systematic reviews of environmental exposures and sleep have summarized associations with air pollution, second-hand smoke (SHS) exposure, and occupational exposures. However, despite biological plausibility, less is known regarding the effects of chemical pollutants such as pesticides, heavy metals, solvents, and endocrine-disrupting chemicals (EDCs) on sleep health.

Chemical pollutants are common environmental exposures that may disturb sleep by acting upon the biological pathways that regulate sleep-wake behavior, with developmental windows of vulnerability.

Importantly, environmental pollution does not affect all people equally; because of environmental racism, marginalized communities face a disproportionately higher burden of adverse environmental exposures, such as environmental disparities in air pollution, which may contribute to sleep health disparities.

Chemical pollutants may contribute to sleep disruption by influencing the underlying biology of sleep-wake behavior.

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Parental Occupational Exposure to Solvents and Autism Spectrum Disorder

Autism spectrum disorder (ASD) is a neurodevelopmental disorder that includes repetitive behaviors, impairment in reciprocal social interaction, difficulty communicating, and sensory sensitivities.

Environmental and genetic factors have been implicated in the etiology of ASD. Gene-environment interaction research may further elucidate the etiology of ASD and point towards potential preventive opportunities.

Few studies have used single nucleotide polymorphism (SNP) from a broad selection of targeted genes to investigate gene-by-environment contributions to autism risk.

Several reviews cite findings that environmental factors are associated with ASD. In addition, parental occupational exposures have been found to be associated with ASD; in particular, parental occupational exposure to solvents.

Solvents may be absorbed through the skin or lungs and are metabolized into toxic secondary substances including methyl-butyl ketone or n-hexane and are associated with abnormal white matter, smaller corpus callosum volume, and cerebellar atrophy.

Infants of mothers with solvent exposure show cognitive delays, attention deficit hyperactivity disorder, delayed speech, and motor functioning. Mothers occupationally exposed to solvents were 1.5 times more likely to have a child with ASD compared to a typically developing child further implicating solvents in the risk for ASD.

Growing evidence also points to the increased risk for neurocognitive or behavioral impairments from epigenetic changes, which themselves are modulated by environmental factors. The overlap in regulatory pathways disrupted by both gene mutations and environmental factors highlights convergence between genetic susceptibility and toxic substances.

Given the relationship between parental occupational exposure and ASD, evaluating potential parental occupational exposure to solvents in conjunction with relevant SNPs may contribute to a better understanding of the etiology of ASD, and indicate promising molecular pathways and avenues for prevention.

Whereas research on ASD, until the last decade had primarily focused on clinical aspects and genetics of autism, an emerging body of evidence is uncovering environmental or occupational exposures appearing either as risk or protective factors.

The current study investigates associations between ASD and gene-by-occupational solvent exposure interactions.

SNP in relevant genes (e.g. immune, inflammatory, serotonin) were analyzed. Additive and multiplicative gene-environment interactions between SNPs and parental solvent exposure were evaluated.

The study reports the combinatorial influence of parental solvent exposure and SNP data on the risk of ASD. SNP and solvent interaction is associated with higher rates of ASD compared to the SNP alone or solvent exposure alone.

The results showed statistically significant multiplicative and additive interactions between 31 genes and parental occupational exposure to solvents in their relationships to confirmed ASD diagnoses.

Results of additive interactions can indicate which exposures are associated with the highest risk of disease and therefore, which subgroup is the most appropriate to target for intervention.

Although there were several sub-additive relationships indicating that some gene SNPs in the presence of solvents may be protective of ASD, this also suggests that the wildtype allele may confer higher risk than the minor allele, placing more individuals at risk of ASD, given solvent exposure.

While it is prudent to prevent parental occupational solvent exposure in all workers, results here indicate that some individuals may be more sensitive to the effects of solvent exposure than others. For these individuals, any solvent exposure may put them at the highest risk of ASD.

Further research needs to be done to better understand the gene, solvent relationship, and how best to protect those at greatest risk.

In summary, additive and multiplicative interactions between solvents and gene SNPs in several serotonin, inflammatory, major histocompatibility complex, antioxidant metabolism, and extracellular matrix genes may be associated with ASD.

These interactions may reflect numerous mechanisms affecting brain development, brain wiring, oxidative stress, and inflammation. In contrast, some SNPs potentially protect neurons from inflammation and oxidative stress.

This is one of the first studies to interrogate a relatively large array of SNPs for gene-environment interactions in ASD.

Future research is needed on specific gene SNPs, solvents (or other environmental exposures), and their potential convergent or intersecting pathways.

Source: Environmental Research, Vol. 228, Article 115769, July 2023.

Exposure to Perfluoroalkyl Substances and Pubertal Onset in Girls

Puberty is a window of susceptibility. Environmental exposures during puberty have more of a potential for a long-term health effect.

In girls, estrogen is important for linear growth, breast, and uterine development, menstrual cycles, mood and cognition, and bone density. Timing of puberty, specifically earlier age of peak height velocity and menarche, are associated with risk for breast cancer.

Per- and polyfluoroalkyl substances (PFAS), endocrine disrupting chemicals (EDCs) with worldwide exposure, cause changes in mammary gland development in rodents. A few human studies report delay in pubertal events with increasing perfluorooctanoic acid (PFOA) exposure, but none have examined reproductive hormone levels at initial breast development (thelarche).

The present cohort study was conducted in two areas of the United States with a history of exposure to PFAS to examine the longitudinal relationship between PFAS serum concentrations measured in young girls (6 to 8 years old) to determine whether PFAS exposure is associated with the age at three pubertal milestones: thelarche, pubarche, and menarche.

To identify subclinical outcomes of the PFAS exposures, the relationship between serum reproductive hormone concentrations around the time of thelarche with PFAS exposure was also examined.

The results showed that median PFOA serum concentrations in the study areas were higher than in the U.S. population. Increased PFOA had a statistically significant direct effect of delay on all three milestones, as did body mass index (BMI).

Perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDeA), and 2-(N-methyl-perfluorooctane sulfonamido) acetic acid (Me-PFOSA-AcOH) were also associated with later thelarche, and Me-PFOSA-AcOH also with later pubarche.

PFOA was inversely associated with dihydroepiandrosterone sulfate (DHEAS), estrone (E1), and testosterone concentrations at 6 months prior to puberty.

No relationship was found between PFOA and estradiol (E2), and no association between PFOA and age at thelarche.

Therefore, in contrast to the original hypothesis, the study found that the PFAS class of EDCs were associated with later rather than earlier breast development.

The difference in reported findings across studies suggest possible variation in genetic susceptibility. Incorporating genetic variants into future metabolomic and transcriptomic analyses would enhance the understanding of the effect of environmental PFAS exposure and lead to better clinical care through environmental precision health.

In conclusion, PFAS may delay pubertal onset through the intervening effects on BMI and reproductive hormones. The decreases in DHEAS and E1 associated with PFOA represent biological biomarkers of effect consistent with the delay in onset of puberty.

Future multivariable exposure mixture analyses of the effect on reproductive hormones will determine whether the reproductive hormones with significant findings in the PFAS alone analysis remain significant in the mixture analysis.

Future long-term follow-up of these girls, including their reproductive hormone levels as adults in both the follicular and luteal phases of the menstrual cycle and their pregnancy outcomes, coupled with pubertal findings could provide additional insight into the effects of PFAS, as a disrupted endocrine environment early in life can affect reproductive health in adulthood.

Source: Environmental Health Perspectives, Vol. 131, No. 9, Article 97009, September 2023.

Exposure to Chemical Pollutants and Sleep Outcomes

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To address these gaps in knowledge and to synthesize the current epidemiological evidence, we conducted a systematic review of the relationships between chemical pollutants and sleep health and disorders.

A systematic review was conducted to identify, evaluate, summarize, and synthesize the existing evidence between chemical pollutants (air pollution, exposures related to the Gulf War, EDCs, metals, pesticides, solvents) and dimensions of sleep health (architecture, duration, quality, timing) and disorders (sleeping pill use, insomnia, sleep-disordered breathing (SDB)).

Overall, associations between particulate matter, exposures related to the Gulf War, dioxin and dioxin-like compounds, and pesticide exposure with worse sleep quality.

Air pollution: Particulate matter and nitrogen dioxide were most linked with poor sleep quality, and tobacco smoke exposure in pediatric populations associated with SDB.

Exposures related to the Gulf War: Gulf War Illness were associated with poor sleep quality, insomnia, SDB, and altered sleep architecture.

EDCs: Dioxins, polychlorinated biphenyls (PCBs), and polybrominated diphenyl ethers (PBDEs) were tied to sleep disruption and insomnia.

Metals: Exposure to lead and mercury were associated with insomnia.

Pesticides: Organophosphate, pyrethroid, and/or carbamate pesticides were associated with insomnia. However, pesticide exposure (small sample sizes), particularly in pediatric populations, was not well explored.

Solvents: Toluene were linked to disrupted sleep timing and SDB.

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Cadmium Toxicity and Health Effects

Cadmium (Cd) is one of the most toxic elements to which humans may be exposed at work or in the natural environment.

Cadmium (Cd) is naturally found in soil, minerals, and water. Cd does not decompose in the environment, nor is it easily removed from the soil, where it is often introduced through phosphate fertilizers (e.g., superphosphates).

It easily reacts with other substances that are most commonly used in cells and batteries including nickel-cadmium batteries, alloys, pigments, plastic stabilizers, dyes, and paints, as well as in glass manufacturing and the galvanic industry.

The greatest exposure to Cd occurs in the metallurgical industry. The continued use of Cd in industry drastically affects the environment, resulting in high exposure of humans to the element.

Cd belongs to the group of toxic, carcinogenic, and stimulating elements. The biological half-life of Cd in the human body ranges from 16 to even 30 years. Some chronic lung diseases (such as emphysema, asthma, and bronchitis) and high blood pressure are related to slow poisoning by Cd in small doses. Moreover, long-term exposure to Cd can lead to various diseases, such as cancer, leukemia, and genetic toxicity.

Cd may play a role in the development of diseases related to the central nervous system (CNS), such as Alzheimer's disease (AD), Parkinsonism and Parkinson's disease (PD), Huntington's disease (HD), amyotrophic lateral sclerosis (ALS), and multiple sclerosis (MS), or in the deterioration of cognitive and behavioral functions, as well as chronic diseases, such as osteoporosis and osteomalacia of pelvic bones, femurs, vertebral bodies, and bones of the shoulder blades.

It can cross the placenta and the barrier to the fetus, exerting teratogenic effects, and is associated with Itai-Itai disease, cardiovascular disease, lung function abnormalities, damage caused in the kidneys, etc.

The kidneys are the main target organ and the most sensitive to Cd contamination and a reduced glomerular reabsorption rate.

The symptoms of Cd poisoning may vary depending on the time of exposure, the type of diet, and the age and health status of the exposed people. For non-smokers and non-occupational exposures, the only source of exposure is diet.

The FAO/WHO recommends that the tolerable Cd intake for an adult is approximately 0.4-0.5 mg/week (60-70 µg per day).

Cd is primarily absorbed through the respiratory system (about 13-19% of Cd from the air), but it can also enter through the digestive system (about 10-44%), when dust is mixed and swallowed with saliva.

The amount of accumulated Cd ranges from 0.14 to 3.2 ppm in muscles, 1.8 ppm in bones, and 0.0052 ppm in the blood.

People who are most frequently exposed to heavy metals should be continuously monitored in order to maintain a healthy lifestyle, as well as to implement effective preventive measures and improve public health.

Cd can induce epigenetic changes that play a key role in the development of various cancers, chronic diseases, or other pathogenic disorders. Chronic exposure to Cd in humans may induce carcinogenesis.

The other effects of Cd include oxidative stress and ROS production, which are normally counterbalanced by activating enzymatic (SOD, CAT, and GPx) and non-enzymatic (GSH, vitamin C, and vitamin E) barriers.

It should be mentioned that providing adequate food with micronutrients (Zn, Fe, and Ca) can protect against the absorption and toxicity of cadmium.

The continuous monitoring of individuals who are occupationally exposed to heavy metals such as cadmium is essential to maintain a healthy lifestyle, as well as implement effective preventive measures and improve public health.

Source: Molecules, Vol. 28, No. 18, Article 6620, September 2023.

Exposure to Chemical Pollutants and Sleep Outcomes

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In general, the evidence presented in this review indicates environmental pollutants may be detrimental to sleep health and disorders among adult and pediatric populations.

Possible underlying mechanisms between chemical pollutant exposure and sleep relate to cholinergic signaling, inducing oxidative stress or inflammation, altering neurotransmission, and endocrine disruption.

Future studies should aim to evaluate environmental exposures on sleep across the lifespan, with a particular focus on developmental windows and biological mechanisms, as well as in historically marginalized or excluded populations. There is a large gap in current knowledge about environmental pollutant exposure and pediatric sleep health, especially regarding pesticides.

Overall, future studies should be robustly designed to evaluate environmental exposures and sleep health, with device-based measures of exposures and outcomes.

Longitudinal study design incorporating measures of actigraphy and wearable devices, including representation of diverse backgrounds, as well as clear reporting and data availability will advance our understanding of the environmental contributions to sleep health across the life course.

Source: Sleep Medicine Reviews, Vol. 70, Article 101805, August 2023.

Exposure to Mercury Increases Arrhythmia and Mortality in Post-myocardial Infarction Rats

Mercury (Hg) exposure remains a major problem to the population, which requires action for proper control. Hg compounds can be categorized into three classes: elemental mercury, inorganic mercury compounds (e.g., $HgCl_2$) and organic mercury compounds (e.g., methylmercury). Each mercury class has distinct chemical properties that contribute to different toxicokinetics and toxicodynamics.

Hg interferes with the activity of proteins involved in cardiovascular function. The high affinity of Hg for sulfhydryl groups in amino acids, proteins, enzymes, and sulfur-containing antioxidants (such as N-acetylcysteine, α-lipoic acid, and glutathione) induces oxidative stress and mitochondrial dysfunction, which in turn affects calcium homeostasis in cardiomyocytes and hemodynamic performance.

Moreover, Hg reduces nitric oxide (NO) production, suppresses the inducible NO synthase gene expression, and induces the production of reactive oxygen species (ROS). In addition, Hg inactivates paraoxonase, an enzyme that prevents atherosclerosis, by decreasing the oxidation of LDL and acting in the reverse transport of cholesterol.

Previous studies have demonstrated that chronic exposure to Hg, even at low doses, is associated with increased systolic pressure, dysregulation of the renin-angiotensin system, hypertension, endothelial dysfunction, impaired vascular reactivity, and inflammation. Therefore, Hg exposure could be associated with the development of coronary artery disease, carotid atherosclerosis, and myocardial infarction (MI).

Chronic exposure to methylmercury in rat cardiomyocytes reduces cardiac rhythm, prolongs rate-corrected ventricular repolarization time, and increases ventricular repolarization dispersion, which associated with an increased risk of cardiac arrhythmia.

However, there is no consensus on the dose-response relationship between Hg exposure and cardiotoxicity. Indeed, the susceptibility to Hg toxicity differs among individuals and can be influenced by genetic factors.

Groups with high consumptions of fish, shellfish and marine mammals are likely higher exposure. However, these foods also offer various health benefits, creating a dilemma when it comes to determining their impact on wellbeing through consumption.

Exposures of infants *in utero* to these pollutants through maternal consumption of contaminated seafood can damage developing brains, reduce IQ and increase children's risks for autism, Attention-Deficit/Hyperactivity Disorder. Adult exposures to methylmercury increase risks for cardiovascular disease and dementia.

The evidence linking mercury exposure to cardiovascular disease is extremely limited. Therefore, the acceptable levels of Hg in the blood for individuals with cardiac diseases or those who are susceptible to them should be thoroughly studied and clearly defined.

Given the importance of understanding the relationship between Hg and cardiovascular disease, the present study aimed to investigate whether Hg could worsen the myocardial repercussions following ischemic injury.

Male Wistar rats received intramuscular injections of either saline (NaCl 0.9%) or mercuric chloride ($HgCl_2$, first dose of 4.6 µg/kg, and subsequent doses of 0.07 µg/kg/day) for 4 weeks. The used concentrations were below the threshold value of 15 µg/L in the blood recommended by WHO.

Three weeks post-exposure, transmural infarction was induced in the left ventricle free wall through coronary artery occlusion surgery.

MI surgically-induced model was performed to induce transmural infarction between 40% and 60% of the left ventricular surface without damaging the interventricular septum. This experimental model is the most widely used to study the remodeling process of ventricles after MI injury, which leads to congestive heart failure syndrome.

ECG recordings obtained from MI groups demonstrated alterations in the rhythm of the heartbeat/heart electrical activity, including ventricular extra-systoles and ventricular tachycardia. However, the MI group exposed to Hg (MI-Hg) exhibited augmented ventricular extrasystoles and ventricular tachycardia compared to the MI group.

The results indicate that the significantly increased mortality in MI-Hg groups when compared to MI is correlated with greater occurrence of arrhythmias.

Arrhythmic events in the MI-Hg group were more strongly linked to mortality. While arrhythmias are expected after an MI, those that occurred after exposure to even low concentrations of Hg following the MI were more pronounced. Clearly, even low concentrations of Hg worsen the hemodynamic repercussions after MI.

To assess the presence of ROS, researchers harvested papillary muscles from rats from all groups 1 week after ECG recordings and found higher levels of nitric oxide and superoxide anions after MI. When comparing the animals from MI and MI-Hg groups, it was observed that exposure to the metal caused a notable increase of the production of ROS.

Furthermore, ROS stimulates inflammatory cell activation and endothelial dysfunction. Several studies showed that Hg both stimulates the production of ROS and promote the reduction of antioxidant proteins in various organs and systems of animals and humans, promoting a redox imbalance and stimulating cell death

These findings demonstrate that the Hg toxicity cause worsened outcome in MI. Also, there was an increase in ROS production due to Hg toxicity. Moreover, exposure to Hg in MI resulted in ROS production further elevated.

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The Chem HelpDesk

"Strengthening capabilities for sound chemicals management"

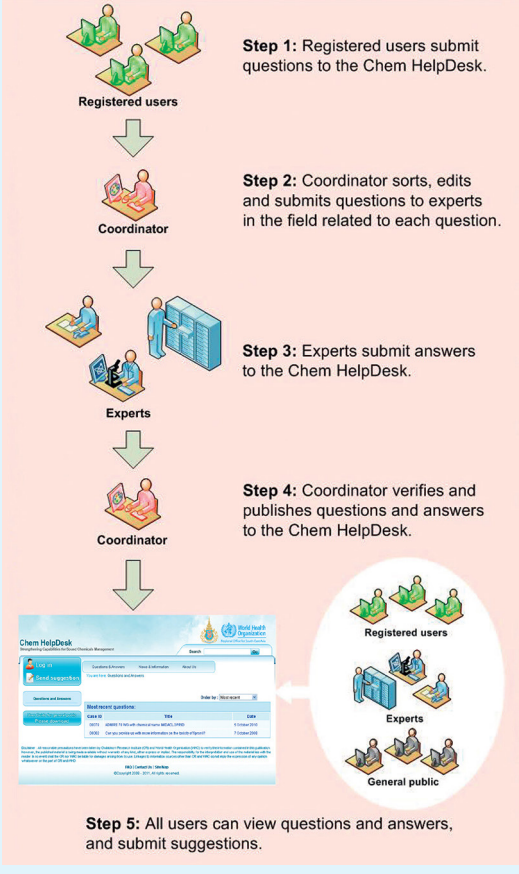
The Regional HelpDesk for Chemical Safety, or **Chem HelpDesk** was established as a joint-initiative between WHO SEARO and CRI, through the WHO Collaborating Center for Capacity Building and Research in Environmental Health Science and Toxicology. The aims of the Chem HelpDesk are to address the issue of the widening gap in the fields of chemical safety and chemicals management between developed and developing countries, and to empower countries in the South-East Asia Region to manage the import, manufacture and processing, storage, distribution, transport, use, recycling and disposal of chemicals, in ways that minimize significant adverse impacts on health and the environment.

The Chem HelpDesk is not-for-profit, and through its website provides cost-free answers to questions submitted by registered users. These answers are provided by experts in the respective fields, who supply technical and scientific advice as part of a Community of Practice (CoP). It is the aim of the Chem HelpDesk to benefit users and to help countries in areas of most need to protect human health and the environment through the safe use and management of chemicals.

In addition to the **"Questions & Answers"** service for registered users, the website also provides information on the safe use of chemicals, as well as a comprehensive list of links to other important websites related to chemicals management in the region. General users have access to the database of questions and answers, as well as all other information on the website.

For more information, please visit: http://www.chemhelpdesk.org

The functional workflow of the Chem HelpDesk is divided into 5 steps:



Exposure to Mercury Increases Arrhythmia and Mortality in Post-myocardial Infarction Rats

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While rats survived after MI, the toxicity of Hg exacerbated the metabolic conditions due to oxidative stress, which in turn could trigger an inflammatory cascade in cardiovascular disease.

The findings suggest that rats in the MI-Hg group, who survived the MI surgery, did not exhibit elevated blood pressure and may have maintained favorable hemodynamic conditions. This could have played a crucial role in their survival following the MI event even Hg exposure.

Hg induces ventricular electrical remodeling that increases the likelihood of cardiac arrhythmias, similar to the effects seen in MI. Both Hg exposure and MI have the potential to promote Ca2+ overload through exacerbated β-adrenergic stimulation, which may contribute to the arrhythmogenesis process.

Researchers propose to conduct a thorough investigation into channels and calcium handling to elucidate the underlying mechanisms by which they are affected when Hg exposure and myocardial infarction co-occur, potentially contributing to increased mortality rates.

In conclusion, this study further supports that exposure to Hg should be recognized as a significant risk factor that exacerbates the impact of cardiac ischemic injury, potentially leading to an increased mortality rate among patients experiencing acute MI.

It is crucial to implement stricter measures to combat Hg contamination in the environment and emphasize that Hg contamination is a substantial public health concern, particularly for individuals vulnerable to cardiovascular diseases.

Source: Frontiers in Physiology, Vol. 14, Article 1260509, October 2023.

Relationship of Arsenic Exposure with Hypertension and Wide Pulse Pressure

Arsenic is a natural component of the earth's crust and is widely distributed throughout the environment in the air, water and land. It is highly toxic in its inorganic form.

The standard for water arsenic exposure concentration is recommended as 10 μg/L by the World Health Organization (WHO), while over 200 million people in various countries such as Bangladesh, India, Mexico, Chile, the United States, and China are living in areas with arsenic exposure levels exceeding the standard.

There is mounting evidence that chronic arsenic exposure is related to stroke, cerebrovascular disease, and risk factors for cardiovascular disease.

Hypertension, or high blood pressure, is a progressive cardiovascular syndrome arising from complicated and interrelated etiologies and is another risk factor for both heart disease and stroke. The Global Burden of Disease Study estimated that over 10 million deaths were due to hypertension in 2016.

Evidence from epidemiological studies suggests that chronic arsenic exposure may be associated with a higher incidence of hypertension in the population.

However, the effects of arsenic on adverse health are closely related to the different oxidation states and several different chemical forms in which arsenic can be found.

Inorganic arsenic species [trivalent arsenic (As^{3+}) and pentavalent arsenic (As^{5+})] are regarded as human carcinogenic agents by the International Agency for Research on Cancer (IARC), with As^{3+} being more toxic than As^{5+}.

After absorption from the gastrointestinal tract, inorganic arsenic (As^{3+} and As^{5+}) undergoes several oxidation-reduction reactions and methylation metabolism processes primarily in the liver, which results in the final organic species of arsenic, monomethylated arsenicals (MMA) or dimethyl arsenic (DMA) for elimination in urine.

Studies have shown that, in addition to inorganic arsenic, some organic arsenic metabolites can produce toxic effects on cells and proteases. Therefore, it is necessary to investigate the association of different arsenic species with blood pressure and associated adverse outcomes.

The present study aimed to investigate the relationship between arsenic exposure and blood pressure and the occurrence of hypertension and wide pulse pressure (WPP) in patients with coal-burning arsenicosis.

Hypertension status was defined as systolic blood pressure (SBP) $\geq$ 140 mm Hg or diastolic blood pressure (DBP) $\geq$ 90 mm Hg. Pulse pressure (PP) is defined as SBP-DBP, and WPP is defined as PP $\geq$ 60 mmHg, a key indicator for evaluating cardiovascular function. WPP is a sign of deteriorating cardiovascular health that carries an increased risk for mortality.

Total arsenic (t-As), MMA, DMA, As^{3+}, and As^{5+} in urine were measured to reflect the arsenic exposure of the population.

The results show that arsenic exposure is related to an increased incidence of hypertension and WPP in the arsenicosis population, primarily due to an induced increase in SBP and PP.

The dose-effect relationships between MMA, As^{3+}, hypertension, and WPP were characterized following trend analyses in the coal-burning arsenicosis population.

The data reveal that with increasing arsenicosis severity, a significant increase in hypertension and WPP incidences is observed in the arsenicosis population. These findings are consistent with a recent meta-analysis, which demonstrated that arsenic was mainly associated with an increased SBP but not significantly related to DBP.

The high level of MMA exposure increases the risk of hypertension by 1.99 times and the WPP by 2.42 times. Similarly, the high level of As^{3+} exposure increases the hypertension risk by 3.68 times and the WPP by 3.84 times.

The results revealed that urinary MMA and As^{3+} levels are mainly associated with increased SBP and induce a higher incidence of hypertension and WPP.

In conclusion, the study demonstrated that the levels of SBP and PP were increased in the arsenicosis population. This abnormal change was associated with the accumulation of urinary MMA and As^{3+} and induced a higher incidence of hypertension and WPP in the coal-burning arsenicosis population.

The study provides preliminary population evidence that cardiovascular-related adverse events such as hypertension and WPP ought to be noticed in the coal-burning arsenicosis population.

Source: Toxics, Vol. 11, Issue 5, Article 443, May 2023.

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