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Impact of Outdoor Air Pollution on COVID-19

In late 2019, the city of Wuhan, China became the epicenter of a pneumonia epidemic caused by a new coronavirus, the severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2). In February 2020, it was designated by the World Health Organization (WHO) as coronavirus disease 2019 (COVID-19).

COVID-19 infections are not specific. Their forms range from completely asymptomatic to severe, requiring mechanical ventilation or leading to death. Although older age and comorbidities have consistently been reported as risk factors for unfavourable COVID-19 prognosis, younger patients without known risk factors are also being admitted to intensive care, albeit to a lesser extent and with a different range of symptoms.

Reported comorbidities include hypertension, obesity, diabetes, cardio-vascular disease, chronic obstructive pulmonary disease, chronic kidney disease and malignancy. Symptomatology, when present, is quite variable, reflecting systemic involvement, including symptoms of lower respiratory tract infection (e.g. cough, fever and dyspnoea), neurological impairment, cutaneous manifestations, or gastro-intestinal issues, especially in the elderly.

Multiple organ failure and cardiopulmonary complications, such as myopericarditis, pulmonary embolism and acute respiratory distress syndrome, represent some of the major complications of severe COVID-19.

Some of the severe forms and deaths attributed to SARS-CoV-2 infection are suspected to have occurred as a result of a disproportionate inflammatory response leading to a major release of pro-inflammatory cytokines, also referred to as a "cytokine storm".

Emerging epidemiological and experimental data are now suggesting the involvement of air pollution in COVID-19-related outcomes. As suggested by the existing literature on multiple respiratory viruses, air pollution may play a role in COVID-19-related morbidity and mortality, according to specific mechanisms.

Air pollution is a complex mixture of gaseous and particulate constituents that vary both spatially and temporally.

COVID-19-related epidemiological studies have so far investigated impacts based on particulate matter (PM) of two sizes, e.g. inhalable particles (particles with an aerodynamic diameter of 10 μm (PM_{10})) and fine particles (particles with an aerodynamic diameter of 2.5 μm ($PM_{2.5}$)). It is also worthwhile mentioning here that both PM_{10} and $PM_{2.5}$ are proxies for ultrafine particles (diameter <0.1 μm).

Size is important in determining how the PM is moved or carried and where its final destination will be within the respiratory tract or bloodstream. Depending on its source, PM can have different compositions. Carbonaceous PM from coal, fuel or wood combustion is the most harmful and is responsible for many chronic diseases, including cardiopulmonary and metabolic diseases, neurodegenerative conditions, cancer and low birthweight.

The gaseous pollutants investigated so far at the population level in relation to

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Impact of Outdoor Air Pollution on COVID-19

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COVID-19 and other respiratory viruses include nitrogen oxides (NOx) and ozone (O_3). NO_2 is a major air pollutant in urban environments, primarily arising from traffic, particularly from diesel cars NO_2 has been associated with asthma, COPD, bronchiolitis and cardiovascular diseases. Tropospheric O_3 levels are dependent on other emitted pollutants, including NOx and VOCs, as well as on climate parameters, and have been associated with excess cardiorespiratory mortality and morbidity.

Studies have pointed out that air pollution may be a contributing factor to COVID-19 pandemic. However, the specific links between air pollution and SARS-CoV-2 infection remain unclear.

This comprehensive overviews the existing literature and synthesizes key data from *in vitro*, animal and human studies and brings in evidence that sheds light on the question of whether or not air pollution and COVID-19 are related.

Epidemiological investigations have related various air pollutants to COVID-19 morbidity and mortality at the population level, but they suffer from several limitations. Air pollution may be linked to an increase in COVID-19 severity and lethality through its impact on chronic diseases, such as cardiopulmonary diseases and diabetes.

Experimental studies have shown that exposure to air pollution leads to a decreased immune response, thus facilitating viral penetration and replication.

In vitro, animal and human studies have reported that exposure to air pollutants leads to increased mucosal permeability and oxidative stress, decreased antioxidants and surfactant antimicrobial proteins, as well as impaired macrophage phagocytosis.

In addition, SARS-CoV-2 entry in host cells through angiotensin-converting enzyme 2 (ACE2) requires the cleavage of the viral spike protein by proteases, and such protease activity may be increased by air pollution, as is documented in the case of several other respiratory viruses.

Viruses may persist airborne through complex interactions with particles and gases, depending on chemical composition; electric charges of particles; and meteorological conditions such as relative humidity, ultraviolet (UV) radiation and temperature. In addition, by reducing UV radiation, air pollutants may promote viral persistence in air and reduce vitamin D synthesis.

The review emphasizes that both short- and long-term exposures to air pollution may be important aggravating factors for SARS-CoV-2 transmission and COVID-19 severity and lethality through multiple mechanisms

Further epidemiological studies are needed to better estimate the impact of air pollution on COVID-19. *In vitro* and *in vivo* studies are also very much needed, in order to more precisely explore the airborne particle-virus interaction.

Future studies in COVID-19 should also examine the role of indoor air pollution, particularly in biomass and tobacco smoke.

The fact that both biology and atmospheric chemistry are separately implicated suggests that a more holistic approach to disease management and mitigation is necessary, both in addressing the current COVID-19 pandemic and in future viral epidemics.

In the light of these relationships between air pollution and COVID-19, and by virtue of the precautionary principle, the researchers recommend the reduction of air pollution from all sources, especially from road traffic and heat generation, through the reinforcement of public health policy.

Source: European Respiratory Review, Vol. 30, Issue 159, Article 200242, March 2021.

Plasticizers and Cardiovascular Health

Plasticizers, additives that modify the flexibility and rigidity of the product, are ingested as they migrate into food and beverages. Human exposure is continuous and widespread; between 75 and 97% of urine samples contain detectable levels of bisphenols and phthalates, the most common plasticizers.

Numerous studies have revealed that exposure to these synthetic chemicals can lead to reproductive and developmental disorders including infertility and early puberty.

More recently, exposure has been linked to the pathogenesis of cardiometabolic diseases such as obesity, type 2 diabetes and cardiovascular disease (CVD).

CVD lead to an estimated 17.9 million deaths annually, making them one of the leading causes of death worldwide. While obesity is considered an independent risk factor for CVD, it frequently occurs in conjunction with other risk factors, including hypertension, insulin resistance and dyslipidemia, in what is known as the metabolic syndrome (MetS). Presence of the MetS increases the risk of death from CVD by approximately 2-fold.

Over the past decades, the rates of obesity have risen faster in children than in adults. Approximately three-quarters of overweight or obese children will be obese as adults and at risk for cardiovascular complications.

This observation coincided with the emerging theory of endocrine disruption that attributes the homeostasis-disrupting effects of exogenous chemicals to an interference with the synthesis, release, transportation, metabolism, or elimination of endogenous bodily hormones.

Endocrine disrupting chemicals (EDCs) interfere with hormone signaling by mimicking endogenous ligands to

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Childhood PFAS Exposure and Glucose Homeostasis

Exposure to per- and poly-fluoroalkyl substances (PFAS), a prevalent class of persistent pollutants, may increase the risk of type 2 diabetes. Youth-onset type 2 diabetes displays a more aggressive phenotype than adult onset type 2 diabetes and results in the rapid progression of disease complications.

Human and animal studies suggest that exposure to PFAS during sensitive periods of development, including childhood, may predispose individuals to type 2 diabetes later in life because this is when cellular differentiation and development of important metabolic tissues occurs.

During puberty, glucose homeostasis, insulin sensitivity, and β-cell function, which are highly predictive of developing type 2 diabetes, undergo significant changes. This suggests that puberty may be a window of increased susceptibility to the potential diabetogenic effects of PFAS.

The present study aimed to examine the associations of childhood PFAS exposure with longitudinal alterations in glucose metabolism, insulin sensitivity, and β-cell function in two independent cohorts of overweight/obese adolescents and young adults with a history of overweight or obesity during childhood.

Four PFAS including perfluoro-octane sulfonate (PFOS), perfluoro-hexane sulfonic acid (PFHxS), as well as perfluorononanoic acid (PFNA), and perfluorodecanoic acid (PFDA) were measured.

The results showed that PFHxS had a strong relationship with glucose homeostasis, whereas no consistent associations were observed for other PFAS and glucose metabolism.

In females, high PFHxS levels were associated with the development of dysregulated glucose metabolism beginning in late puberty, which may be due to changes in β-cell function. The magnitude of these associations increased postpuberty and persisted through 18 years of age.

In males, no consistent associations between PFHxS and glucose metabolism were observed.

Currently, the research examining mechanisms linking exposure to PFAS with metabolic disorders remain unclear. PFHxS has an estimated biological half-life of between 7 and 25 years, which is the longest half-life of any of the PFAS measured in the present study. Further research is needed to examine the potential mechanisms explaining the differential associations of PFHxS in contrast to other PFAS on glucose homeostasis.

In conclusion, the present study supports the hypothesis that PFAS exposure impairs glucose tolerance in adolescents and young adults.

The associations between PFAS exposure and impaired glucose tolerance were stronger in females compared with males, and the results provide evidence that these associations may be driven by changes in β-cell function. These findings suggest that childhood exposure to PFHxS could predispose females to type 2 diabetes later in life.

Source: Environmental Health Perspectives, Vol. 129, No. 9, September 2021.

Plasticizers and Cardiovascular Health

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nuclear receptors and acting as agonists or antagonists depending on the dose, species, and cell-type.

Plasticizers are among the most pervasive EDCs owing to their high production, slow degradation and leaching into the environment.

There are two main groups of plasticizers: 1) bisphenols, which confer rigidity to hard polycarbonate plastics and 2) phthalates, which provide flexibility to soft plastics and polyvinyl chloride (PVC) products. A large body of evidence indicates that these plastics interfere with adipocyte differentiation and adipose tissue function.

Since adipose tissue is a critical regulator of cardiovascular health, the effects of plasticizers on adipocyte biology may underlie their association with obesity and CVD.

In the 2000s, many governments including Canada, the United States and European countries restricted the use of certain plasticizers in products targeted towards infants and children.

Resultant consumer pressure motivated manufacturers to substitute plasticizers with analogues, which have been marketed as safe. However, data on the effects of these new substitutes are limited and data available to-date suggest that many exhibit similar properties to the chemicals they replaced.

The adverse effects of plasticizers have largely been attributed to their endocrine disrupting properties, which modulate hormone signaling.

The present review discusses the current evidence linking bisphenols and phthalates to obesity and CVD, their relationship with MetS, and their impact on adipose tissue development and function.

Data described include information from recent studies revealing that synthetic analogues marketed as safer alternatives have similar effects on adipogenesis, oxidative stress and adipose tissue function.

These findings emphasize the need for further scientific inquiry into synthetic analogues and their purported safety and continued efforts to limit environmental exposure or develop safer alternatives such as the emerging bio-polymers

Source: Frontiers in Pharmacology, Vol. 211, Article 626448, February 2021.

Environmental Toxicants in the Brain: A Review of Astrocytic Metabolic Dysfunction

Exposure to environmental toxicants is linked to long-term adverse outcomes in the brain and involves the dysfunction of glial and neuronal cells.

Astrocytes, the most numerous cell type, are increasingly implicated in the pathogenesis of many diseases of the central nervous system (CNS), including neurodegenerative diseases.

Astrocytes are critical for proper brain function in part due to their robust antioxidant and unique metabolic capabilities. Through their homeostatic, antioxidant, and metabolic functions, astrocytes are critical for the maintenance of lifelong brain health.

Astrocytes are positioned both at the blood-brain barrier, where they are the primary responders to xenobiotic penetrance of the CNS, and at synapses where they are in close contact with neurons and synaptic machinery.

The etiology of many neuro-degenerative diseases is understood to be multifactorial, involving a deleterious confluence of genetic and environmental influences.

While exposure to several classes of environmental toxicants, including chlorinated and fluorinated compounds, and trace metals, have been implicated in neurodegenerative diseases, their impact on astrocytes represents an important and growing field of research.

The response of astrocytes to environmental toxicants is crucial to understanding the impact on the whole brain, as perturbation of astrocytic function has far-reaching consequences.

While the specific impacts of many toxicants on astrocytes have not been well-studied, the recent scientific view of the CNS is expanding to further appreciate the critical role of glial cells in preserving proper functioning. Hence, there is growth in this area of research.

In particular, research on environmental toxicants within the CNS is incomplete without an understanding of how astrocytic metabolic dysfunction, as a result of both acute and chronic exposure to toxicants, contributes to the breakdown of homeostasis and attendant neural degeneration.

Specifically, as the metabolism of astrocytes is so important for brain health, disruption of astrocytic metabolic processes by environmental toxicants is an important, emerging area for understanding neurologic diseases wrought, either in part or in totality, by exposure to these toxicants.

Humans are exposed to multifarious toxicants through our natural and manmade environments, As age is the primary risk factor for neurodegeneration, researchers have long pondered the impact of chronic exposure to toxicants and the ability of these toxicants to cause or contribute to neurodegeneration, as in the context of Alzheimer's disease or Parkinson's disease.

This exposure includes toxicants that are intentionally produced for use against another species. Used in herbicides and insecticides, these compounds have unintended toxic effects. The byproducts of synthetic reactions may have toxic effects and contain biologically relevant metals are toxic at elevated levels.

As glial-centered toxicology research increases, focusing on astrocytic physiological functions, specifically metabolic processes that are understood to be critical for homeostasis, and subsequent dysfunction wrought by exposure to toxicants is critical to understanding the response of the CNS to xenobiotic compounds.

The present review focused on the impact of a range of synthetic compounds on astrocytic function, specifically on perturbed metabolic processes in response to these compounds. The review further considers how perturbation of these pathways impacts disease pathogenesis.

Toxicant exposure results in complex and multi-faceted dysfunction but focusing on astrocytic metabolism, the function of which is critical for CNS homeostasis, enables us to study the engagement of the specific pathways involved in a more global response.

Since the metabolism of astrocytes is indissociably connected to neuronal health and synaptic function, it is critical that understanding of the impact of environmental toxicants on human health include this previously understudied aspect.

From other metabolically linked disorders, such as diabetes, it is clear that environmental toxicant exposure can result in glucose dysfunction, including involvement of the glucose transporter 1 (GLUT1). Within the CNS, this transporter is astrocyte-specific, suggesting an overlap of peripheral and astrocytic mechanisms.

Furthermore, engagement of the cytochrome P450 detoxification system, which is robustly expressed in astrocytes as part of their role as the primary defense against xenobiotic penetrance into the CNS, suggests that astrocytes in particular are crucial for a system-wide response to toxicants.

Importantly, systems that rely on astrocytes, such as glymphatic system clearance and blood-brain barrier function, are disrupted in diabetes, which is known to cause cognitive deficits.

When viewed in conjunction with the perturbation of metabolic processes such as glucose uptake in diabetes as a result of environmental toxicant exposure, these pathological commonalities suggest astrocytic dysfunction as a possible nexus of CNS and peripheral disease.

Overall, evidence is mounting that astrocytic metabolism is important for the response to and neutralization of environmental toxicants within the CNS.

Source: Environmental Toxicology and Pharmacology, Vol. 84, Article 103608, May 2021.

Printing Ink Related Chemicals and Implications for Human Exposure

Although synthetic antioxidants (AOs) and photoinitiators (PIs) are known to be used in printing inks, there are little data on residual concentrations in printing paper products.

AOs, including synthetic phenolic antioxidants (SPAs) and organophosphite antioxidants (OPAs), are widely used in printing inks, plastics, petroleum, personal care products, and even foodstuffs to retard oxidative reactions and prolong shelf lives. SPAs and OPAs are often used simultaneously in products to consume free radicals and peroxides, respectively, creating a synergistic antioxidative effect.

As for PIs, they are a crucial class of additives that generate free radicals under radiation to initiate photopolymerization reactions. Photopolymerization is an emerging technique that is used to produce a large variety of light-curable products such as UV-curable inks, coatings, and resins. On the basis of different structures and functions, the most frequently used PIs can be categorized into four groups: benzophenones (BPs), amine co-initiators (ACIs), thioxanthones (TXs), and phosphine oxides (POs).

In the wake of the wide application of AOs and PIs in commercial products, environmental pollution by these additives has been reported.

Synthetic phenolic antioxidants (SPAs) such as 2,6-di-tert-butyl-4-methylphenol (BHT) and 2,4-di-tert-butyl-phenol (DBP) have been detected in indoor dust, atmospheric particulate matter, water, sludge, and sediment. In many different countries, SPAs have also been detected in human serum, urine, breast milk, and fingernails, suggesting that human exposure to these organic contaminants is ubiquitous.

As for organophosphite antioxidants (OPAs), tris(2,4-di-tert-butylphenyl) phosphite (AO168) is the most frequently used. It has been detected in plastics and foams at high concentrations. Although AO168 is not commonly detected in the environment, its oxidation product, tris(2,4-di-tert-butylphenyl) phosphate (AO168O), has been detected in indoor dust and outdoor atmospheric particulate matter.

PIs have also been detected in indoor dust and sewage sludge. Some PIs were detected as well in human serum and breast milk from the United States and from Chinese donors. Although these chemicals have been detected in humans, the potential human exposure pathways are not well-understood.

A large variety of printing paper products, including paperboard food packaging materials and magazines, are ubiquitous in daily life. In order to maintain good performance of paper products, various additional components are used during their production such as sizing agents, surface coating, lacquers, dyes, pigments, etc.

The use of AOs and PIs in printing inks has been reported. Up to 6% of the total production volume of BHT was used in printing inks in 2000. However, there are only very limited publicly available data on residual levels of PIs and the major AO congener, BHT, in food packaging materials, while no data are available on the occurrence of those printing ink related chemicals in magazines.

In the present study, twenty-five PIs, ten AOs, and six transformation products were analyzed in two types of printing paper products, magazines and paperboard food packaging materials, both of which are unavoidable in everyday products in our life. Human exposure to AOs and PIs via contact with these printing products was also evaluated.

This is the first simultaneous detection of a wide range of AOs and PIs in paper printing products.

Food packaging materials had much higher concentrations than magazines; meanwhile, magazine front covers had significantly higher concentrations than magazine inside pages.

AO168O, BHT, bisphenol A (BPA), and BP were among the predominant printing ink related chemicals in those printing paper products.

Preliminary calculations suggest that dermal exposure to AOs and PIs via contact with those printing paper products is a minor exposure pathway compared to food intake and dust ingestion, which is unlikely to cause adverse health effects.

However, as some target chemicals are semi-volatile, human exposure via inhalation while reading magazines should also be evaluated.

When food packaging materials and magazines are brought indoors, they may act as sources of AOs and PIs in the indoor environment. Littering of these paper products may also provide a source of AOs and PIs in the environment.

Further studies should evaluate the migration behaviors of AOs and PIs from these printing paper products.

Source: Environment International, Vol. 149, Article 106412, April 2021.

WHO: Toolkit for Establishing Laws to Eliminate Lead in Paint 2nd Edition

The World Health organization (WHO) has launched the 2nd Edition of **Toolkit for Establishing Laws to Eliminate Lead in Paint** in October 2021.

Lead poisoning from lead in paint is preventable and there are cost-effective, technically feasible alternatives to lead in paint. This toolkit is a collection of materials presented in slide format for advocacy and technical support for the elimination of lead paint.

It includes information on why lead is a public health and environmental concern, explains current testing methods and describes the existing market. It also gives insight into what other countries have done regarding restrictions on lead use in paint.

The toolkit materials can be used as source material by government officials when initiating work on paint regulation. However, all interested stakeholders wishing to understand the issue and galvanize action are welcome to use the information as well.

For more information on the Toolkit, visit the UNEP website - https://www.unep.org/toolkit-establishing-laws-eliminate-lead-paint

This toolkit was developed by a group of partners of the Global Alliance to Eliminate Lead Paint (Lead Paint Alliance), including industry, intergovernmental and nongovernmental organizations and government representatives.

Source: WHO Publications. 7 October 2021. (https://www.who.int/publications/i/item/9789240034549)

Long-term Exposure to Ozone and Sulfur Dioxide Increases the Incidence of Type 2 Diabetes Mellitus

Diabetes mellitus and its complications have long been identified as a major global health concern. The International Diabetes Federation estimates that 463 million adults had diabetes mellitus globally in 2019. That figure is projected to increase to 700 million by 2045.

Asia is presently the major area for the rapid emergence of a type 2 diabetes millitus (T2DM) epidemic. Moreover, the onset of diabetes among Asian populations has been at a much younger age compared to Western populations. Although T2DM was traditionally considered as a disease of the elderly, the age of diagnosis in Asia has dropped in recent years.

Epidemiological studies have shown an increased risk of developing premature microvascular and macrovascular complications during working life among persons with early-onset T2DM, compared with those with later-onset T2DM. From a socio-economics perspective, adult early-onset T2DM indicates an increasing and high burden of the disease on healthcare system.

There is evidence that genetic and environmental factors are important in the development of T2DM. The effects of environmental factors such as ambient air pollution should also be investigated.

Emerging research observes that long-term exposure to surrounding particulate matter with an aerodynamic diameter of <2.5 μm ($PM_{2.5}$) seems to link with a higher risk of incident T2DM in Asian populations, where air pollution is common and inevitable. However, such research has, as yet, focused only on particulate matter. It has not considered the impact of gaseous pollutants such as ozone (O_3) and sulfur dioxide (SO_2) or the mixed effects of various air pollutants on the development of T2DM.

The present study aimed to investigate, while considering other air pollutants, the association between long-term effects of exposure to ambient O_3 and SO_2 on the incidence of T2DM in Taiwanese adults aged 30 to 50 years.

This is the first nationwide population-based cohort study to evaluate O_3 and SO_2 and the subsequent risk of developing T2DM.

The results demonstrated that higher O_3 and SO_2 exposure led to an increased risk of developing T2DM in the 30-50 years old population, compared to adults with lesser exposure.

The underlying mechanisms of exposure to O_3 and SO_2 on T2DM have not been fully investigated. A recent study summarized some pathophysiological mechanisms of how air pollution components including reactive gases and particulates can cause oxidative stress, inflammation, endothelial dysfunction and consequently cardiovascular disease.

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The Chem HelpDesk

"Strengthening capabilities for sound chemicals management"

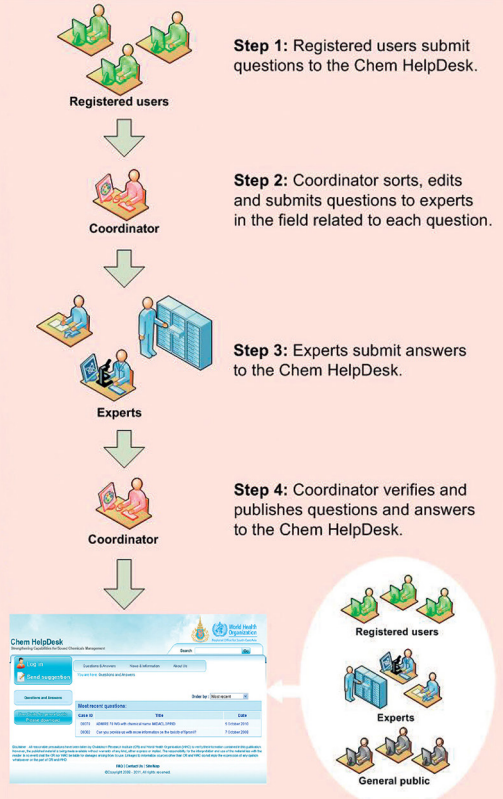
The Regional HelpDesk for Chemical Safety, or **Chem HelpDesk** was established as a joint-initiative between WHO SEARO and CRI, through the WHO Collaborating Center for Capacity Building and Research in Environmental Health Science and Toxicology. The aims of the Chem HelpDesk are to address the issue 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
or e-mail: coordinator@chemhelpdesk.org

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



Long-term Exposure to Ozone and Sulfur Dioxide Increases the Incidence of Type 2 Diabetes Mellitus

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The current study provided a longitudinal population-based evaluation of gaseous air pollutants and the risk of developing T2DM in an Asian country which is also a major area experiencing a rapidly emerging epidemic of T2DM.

The study's large sample size and long follow-up duration provided the necessary power to detect in greater detail the effects of air pollutants on the risk of T2DM development and to identify susceptible subgroups.

Moreover, this study was able to generate consistent results and accurate estimates. Nevertheless, although the present study followed subjects for 11 years, this might be not long enough to observe the development of all T2DM cases, particularly because the study population was relatively young (average age of the cohort was 39.8 years). Thus, the risk of T2DM due to exposure to air pollutants might have been underestimated.

In conclusion, this large and national-level cohort study found long-term exposure to ambient O_3 and SO_2 to be significantly associated with a higher risk of developing T2DM in a Taiwanese population aged 30 to 50. Exposure to O_3 and SO_2 may play a role in adult early-onset T2DM.

The estimated effects in the incidence analysis of O_3 and SO_2 were robust, even after multiple adjustments for other pollutants. Further studies are necessary to confirm the consistency of the effects of O_3 and SO_2 exposure at different exposure levels.

Source: Environmental Research, Volume 194, Article 110624, March 2021.

Heavy Metals and Pesticides Toxicity in Agricultural Soil and Plants: Ecological Risks and Human Health Implications

Environmental pollutants are toxic substances that enter the environment from both anthropogenic and natural sources. Certain environmental processes, such as synthetic industries, coal conversion, and waste burning, are hazardous to abiotic elements (water, air, and soil) and biotic communities (animals, plants, and humans).

Heavy metals and pesticides are at the top of the list of environmental toxicants endangering nature. Environmental toxicity exceeding standard maximum residue limits (MRL) has received heightened consideration from think tanks worldwide.

This review focuses on the toxic effects of heavy metals [cadmium (Cd), lead (Pb), copper (Cu), and zinc (Zn)] and pesticides (insecticides, herbicides, and fungicides) adversely influencing the agricultural ecosystem (plant and soil) and human health.

Cd, Pb, Cu, and Zn cause an alarming combination of environmental and health problems. Heavy metal pollution arises from many sources, such as industry, mining, and agriculture. In terms of sources in the agricultural sector, these can be categorized into fertilizers, pesticides, livestock manure, and wastewater.

The harmful effects on different plant species ranging from infection to death have been discussed. The novelty of this study is to provide an integrated synthesis of knowledge on the complete pathways of both heavy metals and pesticides, starting from their various sources, their accumulation in soil and plants, and their arrival in the human body.

In addition, the synergistic and antagonistic interactions between heavy metals and pesticides, and their combined toxicity in soil, plants, and humans are reported.

Cd is extremely mobile in the soil. As a result, it affects essential microorganisms and absorbs soil's organic matter. Soil pH and sorption capacity can also be negatively affected by Pb accumulation.

Cu has a harmful effect on soil microbial groups such as Rhizobiales, and Zn can inhibit the activities of beneficial microbes and bacteria. Cd causes inhibition of mineral transport and negatively affects microbial growth in plants. Pb accumulation in plants causes DNA damage, chlorophyll content reduction, and inhibition of seed germination.

Decreasing crop yields and inhibited biosynthesis of chlorophyll are the most negative consequences of Cu toxicity. Zn blocks the translocation of nutrients to leaves and decreases photosynthesis, causing plant death.

The discussion mentioned above highlights the direct and indirect damage to fauna and flora, and to the physicochemical and biological properties of agricultural soil due to pesticide residues. Furthermore, these residues decrease enzymatic activity and inhibit soil microbial communities.

Pesticides can cause chlorosis, necrosis, leaf twisting, and malfunctioning of photosynthesis due to oxidative stress. Many studies have also found that different classes of pesticides lead to nitrogen metabolism suppression, increasing and decreasing some enzyme activity. Moreover, leaf pigmentation can be changed, and fruits and grains may stop growing.

For human health, heavy metals and pesticides have deleterious implications. Different body organs and systems can be affected.

Heavy metal toxicity causes serious problems for children and adults by ingestion, inhalation, and dermal adsorption. The harmful health implications of heavy metals can be summed up as neurodegenerative disorders, musculoskeletal diseases, and reproductive hormonal imbalances.

Pesticide exposure has hazardous effects, such as soft tissue sarcoma, ovarian cancer, lung cancers, asthma, and endocrine disruption. Moreover, pesticides cause genetic damage. They also play an important role contributing to Parkinson's disease and to DNA damage in sperm.

As future perspectives and recommendations, the co-occurrence of toxic mixtures, their interactions, and combined toxicity must be investigated in detail. Further studies should be carried out on new approaches to the phytoremediation and bioremediation of environmental toxicants.

Source: Toxics, Vol. 9, Issue 3, Article 42, February 2021.

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