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Association between Short-term Exposure to Air Pollution and COVID-19 Infection: Evidence from China

A novel coronavirus disease, namely COVID-19, was first detected in the city of Wuhan, China in December 2019. In subsequent months, it spread rapidly to the rest of China and later become a global public health problem.

COVID-19 is caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Generally, most SARS-CoV-2 infected patients have mild symptoms including fever, dry cough, and sore throat. However, some patients have severe and even fatal complications such as Acute Respiratory Distress Syndrome (ARDS).

Several early studies have demonstrated that human-to-human contact increases the risk of COVID-19 infection. Population mobility has played a significant role in the COVID-19 epidemic.

Previous studies have suggested that ambient air pollutants are risk factors for respiratory infection. Pollutants make pathogens more invasive to humans, introducing microorganisms which can affect the body's immune system and make people more susceptible to disease.

Since COVID-19 is a respiratory disease and SARS-CoV-2 can remain viable in aerosols for hours, it is interesting to investigate the effect of air pollution on COVID-19 infection.

The present study was conducted to explore the relationship between concentrations of six ambient air pollutants ($PM_{2.5}$, PM_{10}, SO_2, CO, NO_2 and O_3) and daily confirmed COVID-19 cases in 120 cities in China from January 23, 2020 to February 29, 2020.

The results showed significantly positive associations of $PM_{2.5}$, PM_{10}, CO, NO_2 and O_3 with confirmed COVID-19 cases, while SO_2 was negatively associated with the number of daily COVID-19 confirmed cases.

These findings provided evidence that air pollution is an important factor in COVID-19 infection.

Previous studies show that exposure to SO_2, CO and NO_2 is harmful to our health and increases the risk of respiratory disease. A time-series analysis conducted in Thailand observed that PM_{10}, SO_2, CO, NO_2 and O_3 were significantly related to increased risk of respiratory hospital admissions. Overall, all of the six air pollutants investigated could be risk factors in respiratory infection.

The study has some implications for the control and prevention of COVID-19. Governments and the public should pay more attention to regions with high concentrations of $PM_{2.5}$, PM_{10}, CO, NO_2 and O_3, since these regions may suffer a more serious COVID-19 epidemic.

Reducing air pollutants (not including SO_2) could be a useful way to control COVID-19 infection.

Additionally, it is noteworthy that SO_2 has a negative association with COVID-19 confirmed cases. The virucidal property of SO_2 may be a possible reason. Further studies are needed to determine the biological mechanisms behind this phenomenon.

Source: Science of The Total Environment, Vol. 727, Article 138704, July 2020.

Short-term $PM_{2.5}$ Exposure Induces Sustained Pulmonary Fibrosis Development During Post-exposure Period in Rats

According to the Global Burden of Disease Study 2017, the number of deaths attributable to ambient particulate matter (PM) pollution has risen to 2.94 million, with a percentage increase of more than 20% in the period from 2007 to 2017.

$PM_{2.5}$ is one of the most hazardous pollutants in the atmosphere. Inhaled $PM_{2.5}$ can invade bronchi, deposit in alveoli, and even penetrate into blood circulation, exerting detrimental effects on multiple organ systems.

Evidence is accumulating while demonstrates that $PM_{2.5}$ exposure is associated with morbidity and mortality of various respiratory diseases, especially the chronic ones such as asthma, chronic obstructive pulmonary disease (COPD) and pulmonary fibrosis, all of which have brought a great health burdens to human beings.

Pulmonary fibrosis is a chronic and progressive disease with high mortality and limited therapeutic options. It is characterized by excessive deposition of collagen fibers in the pulmonary mesenchyme, scarring of the lung and destruction of the respiratory system, resulting in the inevitable reduction of respiratory capacity.

Some toxicological studies have already identified the pulmonary fibrosis which is induced by long-term $PM_{2.5}$ exposure. There has, however, been no evidence indicating whether short-term exposure can lead to post-exposure development of pulmonary fibrosis.

Multiple mechanisms are involved in the initiation and development of pulmonary fibrosis. Inflammation and epithelial-mesenchymal transition (EMT) play key roles. Inflammation has a pivotal effect in most interstitial lung diseases. Chronic inflammation leads to fibrosis. A host of immune cells and inflammatory mediators promote fibrotic progression.

EMT is a major source of the fibroblasts and myofibroblasts which are integral in the development of fibrosis in the lung. Oxidative stress, the imbalance between increased levels of oxidants and low activity of antioxidant mechanisms, is also linked to the initiation of pulmonary fibrosis.

Inflammation and oxidative stress have been widely accepted as two fundamental mechanisms underlying PM toxicity. It is clear that EMT can occur in the respiratory system following $PM_{2.5}$ exposure.

However, the researchers hypothesized that short-term $PM_{2.5}$ exposure could initiate long-term pulmonary fibrosis development in post-exposure duration and the fibrosis was mediated by oxidative stress/NF-κB/inflammation/EMT pathway.

To verify these conjectures, researchers in the present study chose Sprague-Dawley rats to establish an animal model successively experiencing short-term $PM_{2.5}$ exposure and long-term post-exposure normal feeding.

The study provides evidence of post-exposure toxicity induced by $PM_{2.5}$ and helps clarify the contribution of $PM_{2.5}$ in the initiation and development of pulmonary fibrosis.

Pulmonary inflammatory gene expression, EMT marker changes, RelA/p65 expression, levels of oxidative-stress indicators including malondialdehyde (MDA), superoxide dismutase (SOD) and glutathione (GSH), and serum inflammatory cytokine levels were explored as possible potential mechanisms responsible for the pulmonary fibrosis development.

Rats were treated (10 times) with $PM_{2.5}$ for 1 month, followed by normal feeding for 18 months. Using micro PET/CT imaging, ^{18}F-FDG intake which is linked with the initiation and development of pulmonary fibrosis in living bodies, was found to gradually increase in the lung, following exposure.

Histolopathological examination revealed continuous deterioration of pulmonary injury post-exposure. Collagen deposition and hydroxyproline content continued to increase throughout the post-exposure duration, indicating pulmonary fibrosis development.

Chronic and persistent induction of pulmonary inflammatory gene expression (Tnf, Il1b, Il6, Ccl2, and Icam1), epithelial mesenchymal transition (EMT, reduction of E-cadherin and elevation of fibronectin) and RelA/p65 upregulation, as well as serum inflammatory cytokine production, were also found in the $PM_{2.5}$-treated rats.

Pulmonary oxidative stress, manifested by increased MDA and a decrease in GSH and SOD, was induced during exposure, but disappeared in later post-exposure duration.

This study, which systematically investigated the post-exposure impact of $PM_{2.5}$ in Sprague-Dawley rats, demonstrated that short-term $PM_{2.5}$ exposure, induced via the oxidative-stress-initiated NF-κB/inflammation/EMT pathway, could lead to continuous pulmonary fibrosis development in later post-exposure duration.

While most studies have focused on the immediate $PM_{2.5}$-induced toxic effects which occur simultaneously with exposure, this study is the first to report sustained and long-term, post-exposure toxicity in the respiratory system triggered by $PM_{2.5}$.

As to the significance of this study for the general population, it becomes more evident that being once exposed to $PM_{2.5}$ may lead to increased risk of pulmonary fibrosis throughout later life, even if the original exposure fades away.

From this perspective, the health hazards of $PM_{2.5}$ appear to be long-term, and could be much greater than we have previously understood. This suggests the need for greater urgency in the abatement of $PM_{2.5}$ pollution. Unflagging efforts will be required in order to minimize the immense health impacts that $PM_{2.5}$ could bring.

Source: Journal of Hazardous Materials, Vol. 385, Article 121566, March 2020.

Overview of Molecular Mechanisms in Cadmium Toxicity

Cadmium (Cd) is widespread in the environment both naturally and as a pollutant emitted or discharged from industrial, agricultural, and other sources. Diet and tobacco smoking are considered the most toxic sources of Cd poisoning from within the general population.

Exposure to Cd is known to be genotoxic and carcinogenic and can produce various toxic effects on kidneys, liver, lungs, and bones, and on the cardiovascular, endocrine and reproductive systems. These effects are the result of Cd's numerous toxic mechanisms, all of which are complexly interrelated, highly complicated, and difficult to understand.

The aim of this review is to present data from the most recent literature pertaining to the most relevant mechanisms and molecular pathways of Cd toxicity.

Cadmium-induced toxicity is manifested through different, reciprocally linked, molecular mechanisms.

The general mechanisms (gene regulation, apoptosis, autophagy, oxidative stress, and interaction with bioelements) and specific molecular pathways of Cd toxicity are intertwined.

The most important mechanisms include changes in gene expression and inhibited repair of damaged DNA, interupted apoptosis and autophagy, oxidative stress, and interaction with bioelements.

The effects of Cd on the autophagy process demonstrate as induction or disruption, most likely depending on gene expression. Autophagy following Cd exposure seems either to suppress or activate apoptosis, but both can be induced by increased accumulation of reactive oxygen species (ROS).

Additionally, Cd-induced intracellular calcium (Ca) elevation results in the induction of ROS, triggering cell apoptosis due to the cross-talk between Ca signaling and ROS in physiology and pathophysiology.

Hence, it is rather difficult if not impossible, to make a general assumption about the particular dominant mechanism and molecular pathway responsible for the expression of a certain Cd toxic effect.

It can be postulated, however, that many factors determine which mechanism(s) will be included and/or have dominance in the development of certain toxic effects.

Keeping in mind the ubiquitous presence of Cd in the environment and its complex toxicity, the molecular mechanisms of Cd should be further investigated with additional attention given to dose, route, and time of exposure, as well as the type of cell and tissue in which the Cd toxic effect is manifest.

Source: Current Opinion in Toxicology, Vol. 19, Pages 56-62, February 2020.

Concentration of Metals in Urine and Dyslexia among Children in China

Most physiologically based learning disabilities are attributable to developmental dyslexia (DD), a common neurodevelopment disorder. Between 5.0-17.5% of school-aged children in English-speaking countries and 3.0-12.6% of such children in China are affected by DD.

The social and economic burdens aggravated by DD are substantial, since dyslexic children who suffer from persistent reading difficulties are typically educational underachievers. Compared with their non-dyslexic peers, they also tend to do less well economically.

While some metals like iron and selenium may help to improve cognitive function, working memory, and early language skills, exposure to heavy metals such as arsenic, lead, and manganese, increases the risk of neurodevelopmental disability, neurological deficits, or decreased cognitive abilities.

Previous studies on the role of metals in neurodevelopment disorders have generally focused on single metals. This does not reflect the fact that humans are often, if not always, exposed to multiple metals concurrently.

In the present case-controlled study embedded in the Tongji Reading Environment and Dyslexia (READ) research program in China, the researchers systematically measured the presence of 21 metals in the urine of dyslexic children. They further estimated the combined and interactive effects of co-exposure to multiple metals in this group.

The one-time collection and measurement of urine metals was considered to reflect what would be a long-term burden, since many of the metals measured have a long half-life.

After adjusting for potential confounders, two metals (selenium and argentum) were found to be significantly associated with dyslexia in single-metal multivariable models.

Argentum was positively associated with the risk of dyslexia, while selenium was negatively associated.

Argentum is a rare element, but it is widely available through various routes, such as silver jewelry. Few studies have so far reported the concentration in children's urine of argentum.

In a joint association analysis of their urine, children with higher levels of argentum and lower levels of selenium had a significantly higher risk of dyslexia than those with low levels of both.

This finding might explain the non-statistically significant associations between different levels of urine argentum and the risk to Chinese children of dyslexia, under higher levels of selenium.

This initial assessment of the associations between exposure to metals and the risk of dyslexia for children in China may provide evidence for the etiology of environmental factors of dyslexia.

Longitudinal studies are needed to further evaluate these relationships and to investigate potential mechanisms in a larger population.

Source: Environment International, Vol. 139, Article 105707, June 2020.

Biomonitoring of Human Exposure to Polycyclic Aromatic Hydrocarbons with Hair Analysis in E-waste Recycling Area

Over the last few decades, a series of reliable methods have been developed to assess polycyclic aromatic hydrocarbon (PAH) levels and hazards for human exposure.

Recently, hair has been introduced as a potential bioindicator for assessing human exposure to PAHs owing to its stability, non-invasive collection and wider detection window.

PAHs are commonly generated by anthropogenic activities, such as incomplete burning of coal, wood biomass, or waste.

The industrial recycling and dismantling of electronic waste (EW) has been active in China since the 1990s. A great variety of toxic chemicals, including PAHs, escape into the environment during the dismantling of EW.

Within such waste dismantling areas, high concentrations of PAHs are present in the air, soil, and sediments; in biological samples, such as fish muscle, and in human samples, including placenta, breast milk, umbilical cord blood and urine.

Human hair has been used to assess the exposure of polyhalogenated aromatic hydrocarbons in EW areas. However, little data is available on the simultaneous assessment of PAHs and its metabolites in hair samples from these areas. Also unclear is the difference between external and internal exposure of PAHs and OH-PAHs detected in hair.

In this study, 96 pairs of hair and urine samples were collected from the e-waste (EW) dismantling workers of an industrial park, and from residents living in surrounding areas.

The present study analyzed the concentrations and exposure patterns of both PAHs and their hydroxylated metabolites (OH-PAHs) in hair external (hair-Ex), hair internal (hair-In), and urine.

Irrespective of the sample (hair-Ex, hair-In or urine) the concentrations of both Σ_{15}PAHs and Σ_{12}OH-PAHs of EW workers were comparable to those of non-EW workers and adult residents. There was no significant difference in exposure levels between EW dismantling workers and residents in surrounding areas.

For the parent PAHs, the concentrations of Σ_{15}PAHs of hair-In were comparable to those of hair-Ex for all populations except for the child residents.

On the contrary, for the OH-PAHs, the concentrations of Σ_{12}OH-PAHs of hair-In were 9-37 times higher than those of hair-Ex for populations.

Moreover, the congener profiles of OH-PAHs of hair-In were different from those of hair-Ex, but similar to that of urine. In particular, 3-OH-Bap, which is a carcinogenic metabolite, was only detected in the hair-In.

These results indicate that OH-PAHs in hair-In, just as in urine, are mainly derived from endogenous metabolism and could be considered as reliable biomarkers for PAHs exposure. However, there were almost no significant correlations between hair-In and urine for OH-PAHs.

These results suggest that more attention should be paid to metabolic compounds when conducting hair analysis. The toxicokinetic characteristics of OH-PAHs in hair-In need to be further confirmed and clarified in order to help establish a more comprehensive PAHs exposure risk assessment system.

Source: Environment International, Vol. 136, Article 105432, March 2020.

Urine Glyphosate Level as a Quantitative Biomarker of Oral Exposure

Since the classification, in 2015, by the International Agency for Research on Cancer (IARC) of glyphosate as a Group 2A substance "probably carcinogenic to humans", health concerns have been raised regarding exposures to operators, bystanders, and consumer exposure to glyphosate residues in food crops.

The literature on glyphosate's toxicokinetics is surprisingly scant, especially in view of the public interest of the possible risk associated with oral consumption of pesticide residues due to consumption of food treated with glyphosate-based pesticides. Specifically, there are no publications discussing urinary glyphosate levels in relation to defined oral exposure.

Although only about 20% of an orally administered dose of glyphosate is absorbed in laboratory animals (EFSA, 2015), urine levels of glyphosate are considered to be good exposure markers because glyphosate does not bio-accumulate. It metabolizes poorly and exclusively to aminomethylphosphonic acid (AMPA).

Based on rat absorption rates, a recent preliminary dietary risk assessment of glyphosate and AMPA levels was conducted, but no unacceptable health risk was identified.

The validity of this dietary risk assessment was limited because rat oral absorption rates were used as a proxy for human rates. Animal absorption rates are not reliable in predicting human oral absorption and may compromise risk assessments based on oral exposure.

Using animal oral absorption rates is especially problematic if they are low, as in the case of glyphosate. If the unknown human absorption rate is significantly higher, the risk to consumers is underestimated. If urinary excretion represents the systemically available dose, this uncertainty can be reduced by comparing human urinary levels to a known intake level.

This study aimed to estimate dietary glyphosate intake by determining the fraction of glyphosate and AMPA excretion in urine after consuming ordinary food with glyphosate residue.

(Continued on page 5)

Association Between Proximity to Industrial Chemical Installations and Cancer Mortality in Spain

Pollution from chemical facilities will affect the health of any exposed population; however, the majority of scientific evidence available has focused on occupational exposure rather than environmental exposure.

In the interest of public health policy, therefore, the effects of industrial pollution need to be evaluated. Publicly held Pollutant Release and Transfers Registers (PRTRs) provide useful information about a broad variety of industrial pollutant releases into the environment.

With specific regard to Spain, several studies have used PRTR data to evaluate potential associations between different industrial sectors and mortality from various forms of cancer.

Using proximity as a proxy for exposure, these studies suggest that regions of Spain more closely exposed to pollution from certain types of industrial facilities, have around 17% more cancer mortality compared with populations living outside these areas.

There is higher mortality due to digestive and respiratory tract cancers; leukemias; and prostate, breast, and ovarian cancers in places where the impact of industrial pollution is more immediate.

A number of different industrial activities, including chemical industries, have been assessed by these studies. While the potential effects of the chemical sector have not been studied in depth, the complexity and heterogeneity of this sector make such a study both necessary and useful.

Given this paucity of analysis of environmental pollution in the vicinity of chemical installations, the aims of this study are to assess the possibly greater risk of mortality due to different types of cancer among populations in the vicinity of chemical installations in Spain, to identify the potential risks associated with different types of chemical facilities, and to validate the results, using a variety of methodological approaches.

Consequently, this study assessed whether there could have been higher cancer-related mortality associated with environmental exposure to pollution from chemical installations for populations residing in municipalities in the vicinity of chemical industries.

The ecological study was designed to assess municipal mortality due to 32 types of cancer in the period from 1999 to 2008. The exposure to pollution was estimated using distance from the facilities to the centroid of the municipality as a proxy for exposure.

The results suggest that there is greater risk of cancer mortality for some specific cancers for people living in municipalities near to certain types of chemical installations in Spain.

The majority of these installations are located in the north, east, and south of the country. Two-thirds of the total carcinogenic emissions released into the air by the sector come from organic and inorganic chemical facilities.

The results showed higher mortality risk for both sexes for colorectal, gallbladder, and ovarian cancers associated with organic chemical installations.

Notably, pleural cancer in both sexes was related to fertilizer facilities.

Associations were found, specifically for ovarian and breast cancers, for women living in the proximity of explosives/pyrotechnic installations. Increased breast cancer mortality risk was associated with proximity to inorganic chemical installations.

In order to confirm these results, it would be of great interest to analyze cancer incidence, which was not included in the study, or to carry out local studies where individual information was collected (diet, occupational behaviors, etc.) or to use other methodologies for exposure evaluation that include meteorological and orographic data.

Source: Environmental Pollution, Vol. 260, Article 113869, May 2020.

Urine Glyphosate Level as a Quantitative Biomarker of Oral Exposure

(Continued from page 4)

Levels of glyphosate and AMPA in urine served as reliable quantitative biomarkers of oral exposure, thereby providing a more refined human exposure assessment.

The twelve human participants consumed a test meal with a known amount of glyphosate residue and a small amount of AMPA. Urinary excretion was then examined for the next 48 h.

Only 1% of the glyphosate dose was excreted in urine, much lower than previously assumed, based on animal data. The urinary data indicated an elimination half-life of 9 h.

Consequently, the glyphosate intake could be approximately 20 times higher than previously assumed when using urinary data. Since systemic availability in humans is most likely also 20 times lower than in rats, which gives the risk assessment a certain margin of conservatism.

For the AMPA dose, 23% was excreted in urine, assuming that no metabolism of glyphosate to AMPA occurred. If all of the excreted AMPA was a glyphosate metabolite, this corresponds, on a molar basis to 0.3% of the glyphosate dose.

The results indicated that human urinary levels are a reliable indicator of oral dietary exposure to glyphosate residues. This is the first study to investigate glyphosate's urinary excretion characteristics in humans after oral consumption of a defined dose.

Further human toxicokinetic studies should be performed individually. In such studies, blood concentrations need to be determined in order to improve the human bioavailability data.

Source: International Journal of Hygiene and Environmental Health, Vol. 228, Article 113526, July 2020.

Air Pollution Causing Oxidative Stress

Air pollution remains a major factor for adverse health effects and premature death worldwide. Accelerated urban development and rapid population growth contribute significantly to poor air quality. Because of this, developing countries are generally the most affected. However, associations between air pollution and mortality are still evident even in countries where pollution levels are well below target standards.

Animal inhalation studies have demonstrated that the adverse effects related to reactive oxygen species (ROS) vary, depending on the composition and emission sources of the particles. These latter have changed over the last few decades, with anthropogenic, combustion-derived air pollutants being the main concern for public health today.

A pressing research topic has now, therefore, become clarification of the causative relation between air pollutants and adverse effects, and the deciphering of associated molecular mechanisms.

Air pollution is a heterogeneous, complex mixture of gaseous and particulate components which differs according to emission sources, time variables and atmospheric conditions.

Particulate matter with aerodynamic diameter ≤2.5 μm ($PM_{2.5}$) and mainly originating from combustion processes is considered to be the most toxic. It consists of carbonaceous particles with associated adsorbed organic molecules like nitrates, sulfates, and polycyclic aromatic hydrocarbons, as well as reactive metals such as iron, copper, nickel, zinc, and vanadium.

The physicochemical properties of the particles, their size, structure, chemical composition, reactivity, and solubility, determine their health impacts and the mechanisms by which they induce their adverse effects.

Primary particles emitted directly from a source may exhibit a different toxicity from secondary particles formed by gas-to-particle conversion in the atmosphere

Epidemiological and experimental studies have consistently related $PM_{2.5}$ exposure with a wide range of negative effects, especially on the health of cardiovascular and respiratory system.

$PM_{2.5}$ has been linked to the endocrine activity which promotes the development of metabolic diseases such as obesity and diabetes, two well known cardiovascular risk factors.

While the causative relations between air pollutants and exposure-associated health effects have not yet been fully established, recent studies using different methodologies have consistently shown peroxides and ROS to be crucial mediators of particle toxicity.

PM itself contains ROS as well as redox-active components that can lead to ROS generation upon interaction with specimens of biological origin. The capacity of inhaled PM to elicit cellular damage via oxidative reactions is termed oxidative potential (OP). It can be measured using cell-free assays. Such assays have the advantage of being fast, inexpensive, easy to perform and suitable for automation.

The different sensitivities and readouts of OP-assays reduce difficulties regarding comparability and interpretation of results. When used in combination, they allow a finer distinction of harmful particle types and characteristics.

Experiments using respiratory tract lining fluid (RTLF) are bringing us closer to biological models which mimick the inner lung surface, the primary target of inhaled particles upon deposition.

Further, the biochemistry- and molecular biology-assisted untangling of the complex redox mechanisms allows a better understanding of the direct adverse effects of PM, of oxidative stress-associated development, and the progression of disease.

Clarification of the dual activity of redox-active molecules, including ROS, in cell signaling and as master regulator of inflammatory processes has been essential for progress in research on air pollution and its health impacts.

Despite the benefits of cell-free assays, experimental cell-culture and animal studies are still indispensable in the investigation of the adverse effects of air pollution.

This review is an excerpt of results from experimental studies and methodological developments from the past 2 years that have enhanced our understanding of oxidative molecules in particles, their transmission to the target organ, and the molecular pathways generating ROS in physiological and pathological processes.

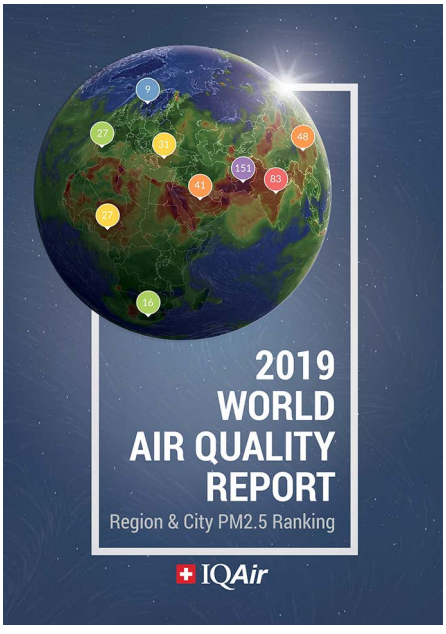
Finally, advances in the development of minimally invasive methods of assessing oxidative stress-related cellular damage in humans have opened the possibility for studies which would otherwise not have been feasible.

Ongoing efforts to establish the causal links between particle properties and specific health impacts are needed. This means that cooperation among all disciplines (i.e. multidisciplinary research) will be indispensable.

In-depth knowledge on mechanisms of particle-ROS induced health effects and the relevance of individual emission sources is required in order to better regulate ROS emission and to increase the effectiveness of efforts to reduce the risk of air-pollution and its related health impacts.

Source: Current Opinion in Toxicology, Vol. 20-21, Pages 1-8, April-June 2020.

2019 World Air Quality Report



The 2019 World Air Quality Report is based on data from the world's largest centralized platform for real-time air quality data. It reflects the combined efforts of thousands of initiatives undertaken by citizens, communities, companies, non-profit organizations and governments.

By aggregating, validating and visualizing real-time data from sensors operated by individuals, organizations, and governments. IQ*Air* strives to raise public awareness about air pollution. When alerted to the danger to their own health, people are more likely to attempt to improve the quality of the air they breathe.

The 2019 World Air Quality Report is based on a subset of information provided through its extensive data platform. Only $PM_{2.5}$ (fine particulate matter) data acquired from ground-based air quality monitoring stations with high data availability is included.

An extended presentation of the world's most polluted cities in 2019 is available online. The interactive format allows further exploration of air quality in that year across different regions and subregions.

Air quality information, presented live or forecast for all included locations can also be explored through the IQAir Air Quality Map, which brings together in the presentations a real-time overview of the world's air quality data.

Air pollution constitutes the most pressing environmental health risk facing our global population. It is estimated to be a contributing factor in 7 million premature deaths annually. At present, the air quality of 92% of the world's population is estimated to be toxic (WHO, 2016). In less developed countries, 98% of children under age five breathe toxic air, and air pollution is believed to be the main cause of death for children under the age of 15, killing 600,000 every year (WHO, 2018). In financial terms about $5 trillion in welfare losses are exacted worldwide due to premature deaths associated with air pollution (The World Bank, 2016).

This report highlights the state of particulate pollution around the world by presenting $PM_{2.5}$ data made publicly available during 2019 in order to raise awareness about the public's need to access air quality data. The majority of this data has in fact already been published in or near real-time by governmental sources, and by independently operated and validated non-governmental air quality monitors.

Access to air quality information remains very limited even as 90% of the global population is surviving on air which already exceeds WHO exposure targets.

Regionally, and overall, South Asia, Southeast Asia, and Western Asia carry the highest burden of fine particulate matter ($PM_{2.5}$) pollution. Only 6 of 355 cities in these regions meet WHO annual targets in these areas collectively. Cities within these regions are high in global city ranking. All of the world's top 30 most polluted cities during 2019 were within greater Asia. There were 21 in India and 27 in South Asia.

Using a weighted population average and based on available data, Bangladesh emerges as the most polluted country for $PM_{2.5}$ exposure. Pakistan, Mongolia, Afghanistan and India follow, respectively, deviated among themselves by less than 10%. Bosnia and Herzegovina is the highest ranking country in $PM_{2.5}$ pollution in Europe and the 14th most polluted country globally, with only 4 $\mu g/m^3$ less than China's national $PM_{2.5}$ weighted average.

Vast numbers of people around the world still lack access to real-time air pollution data, especially in Africa and the Middle East. However, increasing numbers of global citizens and non-governmental organizations (NGOs) are deploying their own low-cost air quality sensors to fill in data gaps where they exist. Owing to these efforts, more continuous public air quality data is now available for the first time for Angola, the Bahamas, Cambodia, DR Congo, Egypt, Ghana, Latvia, Nigeria, and Syria.

Awareness of air pollution remains low in areas where pollution levels may be high but real-time monitoring is still limited.

Publishing real-time monitoring data is essential to tackle the urgent issue of air pollution. What is not measured cannot be managed. Sharing live data enables populations to respond quickly and thus better safeguard their health.

2019 saw a significant increase in air quality monitoring coverage. The number of monitoring stations included in this report have increased by more than 200% since the previous year. These gains are due both to new or expanded governmental monitoring networks and to sensor contributions from individuals, NGOs and private industry.

Still, vast populations around the world continue to lack access to air quality information. Often these are areas estimated to endure some of the most severe air pollution, putting at risk the health of huge populations. More monitoring data is needed to bridge the information gap and to better tackle air pollution globally.

Note: IQ*Air* is a Swiss-based air quality technology company that since 1963 has been empowering individuals, organizations and communities through information, collaboration and technology solutions to breathe cleaner air.

Source: IQ*Air* Press Release, 18 March 2020. (https://www.iqair.com/blog/report-over-90-percent-of-global-population-breathes-dangerously-polluted-air)

WHO HEALTH STATISTICS 2020

All over the world, the COVID-19 pandemic is causing significant loss of life, disrupting livelihoods, and threatening the recent advances in health and progress towards global development goals highlighted in the **2020 World Health Statistics published by the World Health Organization (WHO).**

WHO's World Health Statistics, an annual check-up on the world's health, reports progress in terms of a series of key health and health service indicators, revealing some important lessons in terms of progress made towards Sustainable Development Goals (SDGs) and also gaps that need filling.

Life expectancy and healthy life expectancy have increased, but unequally.

The biggest gains were reported in low-income countries, which saw life expectancy rise 21%, or by 11 years, between 2000 and 2016 (compared with an increase of 4%, or 3 years, in higher income countries).

One driver of progress in lower-income countries has been improved access to services preventing and treating HIV, malaria and tuberculosis, as well as a number of neglected tropical diseases such as guinea worm. Another positive sign was better maternal and child healthcare, which reduced child mortality by half between 2000 and 2018.

In a number of other areas, however, progress has been stalling. Immunization coverage has barely increased in recent years, and there are fears that malaria gains may be reversed. Furthermore, within and outside the health system there is an overall shortage of services to prevent and treat noncommunicable diseases (NCDs) such as cancer, diabetes, heart and lung disease, and stroke. In 2016, 71 per cent of all deaths worldwide were attributable to NCDs. The majority (85%) of these 15 million premature deaths occurred in low and middle-income countries.

This uneven progress broadly mirrors inequalities in access to quality health services. Only one third to one half of the world's population was able to obtain essential health services in 2017. Services in low- and middle-income countries remains very limited, compared to health care in wealthier ones. The same disparities prevail in terms of health workforce densities.

In more than 40% of all countries, there are fewer than 10 medical doctors per 10,000 people. Over 55% have fewer than 40 nursing and midwifery personnel per 10,000 people.

The inability to pay for healthcare is another major challenge. Based on current trends, WHO estimates that this year (2020) approximately 1 billion people (almost 13% of the global population) will be spending at least 10% of their household budgets on health care. The majority of these people live in lower middle-income countries.

The COVID-19 pandemic highlights the need to protect people from health emergencies, as well as the wisdom of promoting universal health coverage to sustain healthier populations. Multisectoral interventions like improving basic hygiene and sanitation will reduce the burdens on over taxed health services.

In 2017, more than half (55%) of the global population was estimated to lack access to safely-managed sanitation services. More than one quarter (29%) lacked safely-managed drinking water. In the same year, two in five households globally (40%) lacked basic handwashing facilities with soap and water.

World Health Statistics also highlights the need for stronger data and health information systems. Uneven capacities for systematically collecting and using accurate and timely health statistics, undermine countries' ability to understand population health trends, develop appropriate policies, allocate resources and prioritize interventions.

In the studies of almost a fifth of the countries globally, over half of the key indicators have no recent primary or direct underlying data. It will be a major challenge to enable countries to prepare for, prevent and respond to health emergencies such as the ongoing COVID-19 pandemic. WHO is therefore supporting countries in strengthening their surveillance, data and health information systems so they can measure their status and manage their improvements.

Source: WHO News Release, 13 May 2020. (https://www.who.int/gho/publications/world_health_statistics/2020/en/)

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