COVID-19


Since the emergence of Coronavirus disease (COVID-19), the threat of plastic waste pollution has grown exponentially, with a strong attention on the environmental and human health consequences of millions of personal protective equipment (PPE) (e.g., face masks, shields, gloves, and wipes) being used and discarded. In response, a massive research effort has been launched to understand, characterize, and estimate the exposure risks of PPE associated contaminants. While the number of studies examining the impacts of PPE is increasing, this review aimed to provide a quick update on the research conducted to date of this topic, as well as to identify priorities for future research. Specifically, we analyzed recent global peer-reviewed articles on PPE to synthesize methods, control measures, and documented evidence to (1) investigate the discarded PPE in a variety of environments; (2) determine the microplastics discharge in the aquatic environment; (3) examine the intentionally or unintentionally added chemicals in the production of PPE; and (4) assess potential human health hazards and exposure pathways. Despite progress, more research is needed in the future to fully understand the chemical emissions from PPE degradation mechanisms (mechanical, chemical, and biological), as well as the magnitude and density of PPE pollution in the environment.

Environmental problems, such as climate change, pollution, and environmental degradation, are important contributors to the spread of infectious diseases, such as COVID-19 and SARS. For instance, a greater concentration of ambient NO2 was associated with faster transmission of the SARS-CoV-2 virus, which causes COVID-19. However, it remains unclear whether outbreaks of infectious diseases arouse individuals' concern on the need to protect the environment and therefore promote more pro-environmental behaviors. To this end, we examined the relationship between infectious disease vulnerability and pro-environmental behaviors using data from a cross-societal survey (N = 53 societies) and an experiment (N = 214 individuals). At both the societal and the individual levels, infectious disease vulnerability increased pro-environmental behaviors. At the societal level, this relationship was mediated by citizens' level of environmental concern. At the individual level, the relationship was mediated by empathy. The findings show that infectious disease vulnerability is conducive to pro-environmental behaviors.


Currently, there is a massive debate on whether meteorological and air quality parameters play a crucial role in the transmission of COVID-19 across the globe. With this background, this study aims to evaluate the impact of air pollutants (PM2.5, PM10, CO, NO, NO2, and O3) and meteorological parameters (temperature, humidity, wind speed, and rainfall) on the spread and mortality due to the COVID-19 outbreak in Delhi from 14 Mar 2020 to 3 May 2021. The Spearman's rank correlation method employed on secondary data shows a significant correlation between the COVID-19 incidences and the PM2.5, PM10, CO, NO, NO2, and O3 concentrations. Amongst the four meteorological parameters, temperature is strongly correlated with COVID-19 infections and deaths during the three phases, i.e., pre-lockdown (14 March 2020 to 24 March 2020) ($r = 0.79$), lockdown (25 March 2020 to 31 May 2020) ($r = 0.87$), and unlock (1 June 2020 to 3 May 2021) ($r = -0.75$), explaining the variability of about 20-30% in the lockdown period and 18-19% in the unlock period. NO2 explained the maximum variability of 10% and 7% in the total confirmed cases and deaths among the air pollutants, respectively. A generalized linear model could explain 80% and 71% of the variability in confirmed cases and deaths during the lockdown and 82% and 81% variability in the unlock phase, respectively. These findings suggest that these factors may contribute to the transmission of the COVID-19 and its associated deaths. The study results would enhance the ongoing research related to the influence of environmental factors. They would be helpful for policymakers in managing the outbreak of COVID-19 in Delhi, India.

Environmental pollution from microplastics (MPs) in air is a matter of growing concern because of human health implications. Airborne MPs can be directly and continuously inhaled in air environments. Especially high MPs contributions can be found in indoor air due to the erosion and breakage of consumer, domestic and construction products, although there is little information available on their sources and concentrations and the risks they might pose. This is in part due to the fact that sampling and analysis of airborne MPs is a complex and multistep procedure where techniques used are not yet standardized. In this study, we provide an overview on the presence of MPs in indoor air, potential health impacts, the available methods for their sampling and detection and implications from the use of face masks during the COVID-19 pandemic.

Health Impacts of Climate Change


**OBJECTIVE:** Many epidemiological studies have observed the association of air pollutant exposure with the onset, progression, and mortality of stroke. The aim of this study was to investigate the associations of air pollutants, including SO2, NO2, O3, CO, and PM10, with stroke according to exposure duration.

**METHODS:** Data from the Korean National Health Insurance Service-Health Screening Cohort from 2002 to 2015 were obtained. The 21,240 patients who were admitted for or died due to stroke were 1:4 matched for age, sex, income, and region of residence with 84,960 control participants. The meteorological factors of mean, highest, and lowest temperatures; relative humidity; ambient atmospheric pressure; and air pollutant concentrations (SO2, NO2, O3, CO, and PM10) were analyzed to determine their associations with stroke. The odds ratios for stroke after exposure to each meteorological factor and air pollutant at 7 and 30 days were calculated in the stroke and control groups. Subgroup analyses were conducted according to age, sex, income, and region of residence.

**RESULTS:** The odds ratio associated with seven days of exposure to CO was 1.16 (95% CI = 1.04-1.31) in stroke patients. For 30 days of exposure, the odds ratio associated with CO was 1.16 (95% CI = 1.02-1.32) in stroke patients. Seven and 30 days of NO2 exposure were inversely associated with stroke. The odds ratio associated with seven days of exposure to O3 was 1.16 (95% CI = 1.01-1.32) in ischemic stroke patients. Both ischemic and hemorrhagic stroke had negative associations with 7 and 30 days of NO2 exposure.

**CONCLUSION:** Both short- and long-term exposure to CO were related to stroke.


Chronic kidney disease (CKD), a global disease burden related to high rates of incidence and mortality, manifests as progressive and irretrievable nephron loss and decreased kidney regeneration capacity. Emerging studies have suggested that exposure to air pollution is closely
relevant to increased risk of CKD, CKD progression and end-stage kidney disease (ESKD). Inhaled airborne particles may cause vascular injury, intraglomerular hypertension, or glomerulosclerosis through non-hemodynamic and hemodynamic factors with multiple complex interactions. The mechanisms linking air pollutants exposure to CKD include elevated blood pressure, worsening oxidative stress and inflammatory response, DNA damage and abnormal metabolic changes to aggravate kidney damage. In the present review, we will discuss the epidemiologic observations linking air pollutants exposure to the incidence and progression of CKD. Then, we elaborate the potential roles of several air pollutants including particulate matter and gaseous co-pollutants, environmental tobacco smoke, and gaseous heavy metals in its pathogenesis. Finally, this review outlines the latent effect of air pollution in ESKD patients undergoing dialysis or renal transplant, kidney cancer and other kidney diseases. The information obtained may be beneficial for further elucidating the pathogenesis of CKD and making proper preventive strategies for this disease.


BACKGROUND: Global asthma-related mortality tallies at around 2.5 million annually. Although asthma may be triggered or exacerbated by particulate matter (PM) exposure, studies investigating the relationship of PM and its components with emergency department (ED) visits for pediatric asthma are limited. This study aimed to estimate the impact of short-term exposure to PM constituents on ED visits for pediatric asthma.

METHODS: We retrospectively evaluated non-trauma patients aged younger than 17 years who visited the ED with a primary diagnosis of asthma. Further, measurements of PM with aerodynamic diameter of < 10 μm (PM10), PM with aerodynamic diameter of < 10 μm (PM2.5), and four PM2.5 components (i.e., nitrate (NO3⁻), sulfate (SO4²⁻), organic carbon (OC), and elemental carbon (EC)) were collected between 2007 and 2010 from southern particulate matter supersites. These included one core station and two satellite stations in Kaohsiung City, Taiwan. A time-stratified case-crossover study was conducted to analyze the hazard effect of PM.

RESULTS: Overall, 1597 patients were enrolled in our study. In the single-pollutant model, the estimated risk increase for pediatric asthma incidence on lag 3 were 14.7% [95% confidence interval (CI), 3.2–27.4%], 13.5% (95% CI, 3.3–24.6%), 14.8% (95% CI, 2.5–28.6%), and 19.8% (95% CI, 7.6–33.3%) per interquartile range increments in PM2.5, PM10, nitrate, and OC, respectively. In the two-pollutant models, OC remained significant after adjusting for PM2.5, PM10, and nitrate. During subgroup analysis, children were more vulnerable to PM2.5 and OC during cold days (< 26 °C, interaction p = 0.008 and 0.012, respectively).

CONCLUSIONS: Both PM2.5 concentrations and its chemical constituents OC and nitrate are associated with ED visits for pediatric asthma. Among PM2.5 constituents, OC was most closely related to ED visits for pediatric asthma, and children are more vulnerable to PM2.5 and OC during cold days.

Hot ambient conditions and associated heat stress can increase mortality and morbidity, as well as increase adverse pregnancy outcomes and negatively affect mental health. High heat stress can also reduce physical work capacity and motor-cognitive performances, with consequences for productivity, and increase the risk of occupational health problems. Almost half of the global population and more than 1 billion workers are exposed to high heat episodes and about a third of all exposed workers have negative health effects. However, excess deaths and many heat-related health risks are preventable, with appropriate heat action plans involving behavioural strategies and biophysical solutions. Extreme heat events are becoming permanent features of summer seasons worldwide, causing many excess deaths. Heat-related morbidity and mortality are projected to increase further as climate change progresses, with greater risk associated with higher degrees of global warming. Particularly in tropical regions, increased warming might mean that physiological limits related to heat tolerance (survival) will be reached regularly and more often in coming decades. Climate change is interacting with other trends, such as population growth and ageing, urbanisation, and socioeconomic development, that can either exacerbate or ameliorate heat-related hazards. Urban temperatures are further enhanced by anthropogenic heat from vehicular transport and heat waste from buildings. Although there is some evidence of adaptation to increasing temperatures in high-income countries, projections of a hotter future suggest that without investment in research and risk management actions, heat-related morbidity and mortality are likely to increase.


This study evaluates numerous epidemiological, environmental, and economic factors affecting morbidity and mortality from PM2.5 exposure in the 27 member states of the European Union. This form of air pollution inflicts considerable social and economic damage in addition to loss of life and well-being. This study creates and deploys a comprehensive data pipeline. The first step consists of conventional linear models and supervised machine learning alternatives. Those regression methods do more than predict health outcomes in the EU-27 and relate those predictions to independent variables. Linear regression and its machine learning equivalents also inform unsupervised machine learning methods such as clustering and manifold learning. Lower-dimension manifolds of this dataset's feature space reveal the relationship among EU-27 countries and their success (or failure) in managing PM2.5 morbidity and mortality. Principal component analysis informs further interpretation of variables along economic and health-based lines. A nonlinear environmental Kuznets curve may describe the fuller relationship between economic activity and premature death from PM2.5 exposure. The European Union should bridge the historical, cultural, and economic gaps that impair these countries' collective response to PM2.5 pollution.
Environmental factors play an important role in the development and exacerbation of allergic rhinitis (AR) in childhood. Indoor air pollution, such as house dust mites and secondhand smoke, can significantly increase the onset of AR, while pet dander may affect the exacerbation of AR symptoms in children. Furthermore, traffic related air pollution and pollen are outdoor air pollutants that can affect immune competency and airway responsiveness, increasing the risk of AR in children. Climate change has increased AR in children, as growth patterns of allergenic species have changed, resulting in longer pollen seasons. More extreme and frequent weather events also contribute to the deterioration of indoor air quality due to climate change. Additionally, viruses provoke respiratory tract infections, worsening the symptoms of AR, while viral infections alter the immune system. Although viruses and pollution influence development and exacerbation of AR, a variety of treatment and prevention options are available for AR patients. The protective influence of vegetation (greenness) is heavily associated with air pollution mitigation, relieving AR exacerbations, while the use of air filters can reduce allergic triggers. Oral antihistamines and intranasal corticosteroids are common pharmacotherapy for AR symptoms. In this review, we discuss the environmental risk factors for AR and summarize treatment strategies for preventing and managing AR in children.

In recent decades, the incidence of thyroid cancer has increased more than most other cancers, paralleling the generalized worldwide increase in metal pollution. This review provides an overview of the evidence supporting a possible causative link between the increase in heavy metals in the environment and thyroid cancer. The major novelty is that human thyroid stem/progenitor cells (thyrospheres) chronically exposed to different metals at slightly increased environmentally relevant concentrations show a biphasic increase in proliferation typical of hormesis. The molecular mechanisms include, for all metals investigated, the activation of the extracellular signal-regulated kinase (ERK1/2) pathway. A metal mixture, at the same concentration of individual metals, was more effective. Under the same conditions, mature thyrocytes were unaffected. Preliminary data with tungsten indicate that, after chronic exposure, additional abnormalities may occur and persist in thyrocytes derived from exposed thyrospheres, leading to a progeny population of transformation-prone thyroid cells. In a rat model predisposed to develop thyroid cancer, long-term exposure to low levels of metals accelerated and worsened histological signs of malignancy in the thyroid. These studies provide new insight on metal toxicity and carcinogenicity occurring in thyroid cells at a low stage of differentiation when chronically exposed to metal concentrations that are slightly increased, albeit still in the "normal" range.
Rheumatoid arthritis (RA) flare is related to increased joint damage, disability, and healthcare use. The impact of short-term air pollution exposure on RA disease activity is still a matter of debate. In this cross-sectional study, we investigated whether short-term exposure to particulate matter (PM10, PM2.5), nitrogen dioxide (NO2), and ozone (O3) affected RA disease activity (DAS28 and SDAI) in 422 consecutive RA residents in Lombardy, North of Italy. Air pollutant concentrations, estimated by Regional Environmental Protection Agency (Lombardy-Italy) at the municipality level, were used to assign short-term exposure from the day of enrolment, back to seven days. Some significant negative associations emerged between RA disease activity, PM10, and NO2, whereas some positive associations were observed for O3. Patients were also stratified according to their ongoing Disease-Modifying Anti-Rheumatic Drugs (DMARDs) treatment: no DMARDs (n = 25), conventional synthetic DMARDs (n = 108), and biological or targeted synthetic DMARDs (n = 289). Therapy interaction seemed partially able to influence the relationship between short-term air pollution exposure and RA disease activity (PM2.5 levels and DAS28 at the day of the visit-O3 levels and disease activity scores for the seven days before the evaluation). According to our results, the impact of short-term air pollution exposure (seven days) minimally impacts disease activity. Moreover, our study suggests therapy could alter the response to environmental factors. Further evidence is needed to elucidate determinants of RA flare and its management.

BACKGROUND: Pollution has been suggested as a precipitating factor for cardiovascular diseases. However, data about the link between air pollution and the risk of out-of-hospital cardiac arrest (OHCA) are limited and controversial.
METHODS: By collecting data both in the OHCA registry and in the database of the regional agency for environmental protection (ARPA) of the Lombardy region, all medical OHCA and the mean daily concentration of pollutants including fine particulate matter (PM10, PM2.5), benzene (C6H6), carbon monoxide (CO), nitrogen dioxide (NO2), sulphur dioxide (SO2), and ozone (O3) were considered from January 1st to December 31st, 2019 in the southern part of the Lombardy region (provinces of Pavia, Lodi, Cremona and Mantua; 7863 km2; about 1550000 inhabitants). Days were divided into high or low incidence of OHCA according to the median value. A Probit dose-response analysis and both uni- and multivariable logistic regression models were provided for each pollutant.
RESULTS: The concentrations of all the pollutants were significantly higher in days with high incidence of OHCA except for O3, which showed a significant countertrend. After correcting for temperature, a significant dose-response relationship was demonstrated for all the pollutants
examined. All the pollutants were also strongly associated with high incidence of OHCA in multivariable analysis with correction for temperature, humidity, and day-to-day concentration changes.

CONCLUSIONS: Our results clarify the link between pollutants and the acute risk of cardiac arrest suggesting the need of both improving the air quality and integrating pollution data in future models for the organization of emergency medical services.

WE ACT

Comment on
Acknowledging that children are estimated to bear ~90% of the global burden of disease secondary to climate change,6 the urgency of addressing the climate crisis falls even more squarely on us as pediatric subspecialists. Lee and colleagues prove to our specialty that we have the capability to provide quality care in a less energy intensive manner if we are willing to re-imagine the methods by which we provide it. As pediatric dermatologists, we must recognize those innovations in response to COVID-19 that have benefited society, and institute them as part of our daily practice. In turn, we are challenged to continually conceive of ways in which we deliver care that prioritizes sustainability and equity, to the benefit of children today and in the future.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8349167/
BACKGROUND: Climate change, including global warming, will cause poorer global health and rising numbers of environmental refugees. As neurological disorders account for a major share of morbidity and mortality worldwide, global warming is also destined to alter neurological practice; however, to what extent and by which mechanisms is unknown. We aimed to collect information about the effects of ambient temperatures and human migration on the epidemiology and clinical manifestations of neurological disorders.
METHODS: We searched PubMed and Scopus from 01/2000 to 12/2020 for human studies addressing the influence of ambient temperatures and human migration on Alzheimer's and non-Alzheimer's dementia, epilepsy, headache/migraine, multiple sclerosis, Parkinson's disease, stroke, and tick-borne encephalitis (a model disease for neuroinfections). The protocol was pre-registered with PROSPERO (2020 CRD42020147543).
RESULTS: Ninety-three studies met inclusion criteria, 84 of which reported on ambient temperatures and nine on migration. Overall, most temperature studies suggested a relationship between increasing temperatures and higher mortality and/or morbidity, whereas results were more ambiguous for migration studies. However, we were unable to identify a single adequately designed study addressing how global warming and human migration will
change neurological practice. Still, extracted data indicated multiple ways by which these aspects might alter neurological morbidity and mortality soon.

CONCLUSION: Significant heterogeneity exists across studies with respect to methodology, outcome measures, confounders and study design, including lack of data from low-income countries, but the evidence so far suggests that climate change will affect the practice of all major neurological disorders in the near future. Adequately designed studies to address this issue are urgently needed, requiring concerted efforts from the entire neurological community.