New Research

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COVID-19


Due to intense industrialization and urbanization, air pollution has become a serious global concern as a hazard to human health. Epidemiological studies found that exposure to atmospheric particulate matter (PM) causes severe health problems in human and significant damage to the physiological systems. In recent days, PM exposure could be related as a carrier for SARS-CoV-2 virus transmission and COVID-19 infection. Hence, it is important to understand the adverse effects of PM in human health. This review aims to provide insights on the detrimental effects of PM in various human health problems including respiratory, circulatory, nervous, and immune system along with their possible toxicity mechanisms. Overall, this review highlights the potential relationship of PM with several life-limiting human diseases and their significance for better management strategies.


The COVID-19 pandemic has an adverse effect on the environment. This epidemic's effect on the waste composition and management and the impacts of municipal solid waste management (MSWM) on disease transmission or controlling are considered a compelling experience of living in the COVID-19 pandemic that can effectively control the process. This systematic review research was conducted to determine the effects of COVID-19 on the quantity of waste and MSWM. Searches were conducted in three databases (using keywords covid 19, coronaviruses, and waste), and among the published articles from 2019 to 2021, 56
ones were selected containing information on the quantity and waste management during the COVID-19 pandemic. The results showed that COVID-19 caused the quantity variation and composition change of MSW. COVID-19 also has significant effects on waste recycling, medical waste management, quantity, and littered waste composition. On the other hand, the COVID-19 pandemic has changed waste compounds' management activities and waste generation sources. Recognizing these issues can help plan MSWM more efficiently and reduce virus transmission risk through waste.


   The health impact of coronavirus disease 2019 (COVID-19) appears disproportionate across populations. Biological mechanisms of prognosis of COVID-19 (1) suggest that long-term exposure to ambient air pollution may contribute to health disparities. Mortality impacts including from pneumonia and cardiovascular events due to long-term exposure to air pollution are well-established (2-5). Early studies covering multiple neighborhoods (e.g., U.S. counties) suggest that long-term exposure to fine particles (PM2.5) and ozone (O3) may be linked with higher risk of COVID-19 mortality (6-8).


   BACKGROUND: Ecologic analyses suggest that living in areas with higher levels of ambient fine particulate matter air pollution (PM2.5) is associated with higher risk of adverse COVID-19 outcomes. Studies accounting for individual-level health characteristics are lacking.

   METHODS: We leveraged the breadth and depth of the US Department of Veterans Affairs national healthcare databases and built a national cohort of 169,102 COVID-19 positive United States Veterans, enrolled between March 2, 2020 and January 31, 2021, and followed them through February 15, 2021. Annual average 2018 PM2.5 exposure, at an approximately 1 km2 resolution, was linked with residential street address at the year prior to COVID-19 positive test. COVID-19 hospitalization was defined as first hospital admission between 7 days prior to, and 15 days after, the first COVID-19 positive date. Adjusted Poisson regression assessed the association of PM2.5 with risk of hospitalization.

   RESULTS: There were 25,422 (15.0%) hospitalizations; 5,448 (11.9%), 5,056 (13.0%), 7,159 (16.1%), and 7,759 (19.4%) were in the lowest to highest PM2.5 quartile, respectively. In models adjusted for State, demographic and behavioral factors, contextual characteristics, and characteristics of the pandemic a one interquartile range increase in PM2.5 (1.9 µg/m3) was associated with a 10% (95% CI: 8%-12%) increase in risk of hospitalization. The association of PM2.5 and risk of hospitalization among COVID-19 individuals was present in each wave of the pandemic. Models of non-linear exposure-response suggested increased risk at PM2.5 concentrations below the national standard 12 µg/m3. Formal effect modification analyses suggested higher risk of hospitalization associated with PM2.5 in Black people compared to
White people ($p = 0.045$), and in those living in socioeconomically disadvantaged neighborhoods ($p < 0.001$).

CONCLUSIONS: Exposure to higher levels of PM2.5 was associated with increased risk of hospitalization among COVID-19 infected individuals. The risk was evident at PM2.5 levels below the regulatory standards. The analysis identified those of Black race and those living in disadvantaged neighborhoods as population groups that may be more susceptible to the untoward effect of PM2.5 on risk of hospitalization in the setting of COVID-19.


Ambient air pollution contributes to 7 million premature deaths annually. Concurrently, the ongoing coronavirus disease 2019 (COVID-19) pandemic, complicated with S-protein mutations and other variants, caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has resulted in over 2.5 million deaths globally. Chronic air pollution-mediated cardiopulmonary diseases have been associated with an increased incidence of hospitalization and mechanical ventilation following COVID-19 transmission. While the underlying mechanisms responsible for this association remain elusive, air pollutant-induced vascular oxidative stress and inflammatory responses have been implicated in amplifying COVID-19-mediated cytokine release and vascular thrombosis. In addition, prolonged exposure to certain types of particulate matter (PM2.5, $d < 2.5$ μm) has also been correlated with increased lung epithelial and vascular endothelial expression of the angiotensin-converting enzyme-2 (ACE2) receptors to which the SARS-CoV-2 spike glycoproteins (S) bind for fusion and internalization into host cells. Emerging literature has linked high rates of SARS-CoV-2 infection to regions with elevated levels of PM2.5, suggesting that COVID-19 lockdowns have been implicated in regional reductions in air pollutant-mediated cardiopulmonary effects. Taken together, an increased incidence of SARS-CoV-2-mediated cardiopulmonary diseases seems to overlap with highly polluted regions. To this end, we will review the redox-active components of air pollutants, the pathophysiology of SARS-CoV-2 transmission, and the key oxidative mechanisms and ACE2 overexpression underlying air pollution-exacerbated SARS-CoV-2 transmission.


Converging data would indicate the existence of possible relationships between climate change, environmental pollution and epidemics/pandemics, such as the current one due to SARS-CoV-2 virus. Each of these phenomena has been supposed to provoke detrimental effects on mental health. Therefore, the purpose of this paper was to review the available scientific literature on these variables in order to suggest and comment on their eventual synergistic effects on mental health. The available literature report that climate change, air pollution and COVID-19 pandemic might influence mental health, with disturbances ranging from mild negative
emotional responses to full-blown psychiatric conditions, specifically, anxiety and depression, stress/trauma-related disorders, and substance abuse. The most vulnerable groups include elderly, children, women, people with pre-existing health problems especially mental illnesses, subjects taking some types of medication including psychotropic drugs, individuals with low socio-economic status, and immigrants. It is evident that COVID-19 pandemic uncovers all the fragility and weakness of our ecosystem, and inability to protect ourselves from pollutants. Again, it underlines our faults and neglect towards disasters deriving from climate change or pollution, or the consequences of human activities irrespective of natural habitats and constantly increasing the probability of spillover of viruses from animals to humans. In conclusion, the psychological/psychiatric consequences of COVID-19 pandemic, that currently seem unavoidable, represent a sharp cue of our misconception and indifference towards the links between our behaviour and their influence on the "health" of our planet and of ourselves. It is time to move towards a deeper understanding of these relationships, not only for our survival, but for the maintenance of that balance among man, animals and environment at the basis of life in earth, otherwise there will be no future.

**Health Impacts of Climate Change**


**BACKGROUND:** Fine particulate matter (PM2.5), ozone (O3), and nitrogen dioxide (NO2) are major air pollutants that pose considerable threats to human health. However, what has been mostly missing in air pollution epidemiology is causal dose-response (D-R) relations between those exposures and mortality. Such causal D-R relations can provide profound implications in predicting health impact at a target level of air pollution concentration.

**METHODS:** Using national Medicare cohort during 2000-2016, we simultaneously emulated causal D-R relations between chronic exposures to fine particulate matter (PM2.5), ozone (O3), and nitrogen dioxide (NO2) and all-cause mortality. To relax the contentious assumptions of inverse probability weighting for continuous exposures, including distributional form of the exposure and heteroscedasticity, we proposed a decile binning approach which divided each exposure into ten equal-sized groups by deciles, treated the lowest decile group as reference, and estimated the effects for the other groups. Binning continuous exposures also makes the inverse probability weights robust against outliers.

**RESULTS:** Assuming the causal framework was valid, we found that higher levels of PM2.5, O3, and NO2 were causally associated with greater risk of mortality and that PM2.5 posed the greatest risk. For PM2.5, the relative risk (RR) of mortality monotonically increased from the 2nd (RR, 1.022; 95% confidence interval [CI], 1.018-1.025) to the 10th decile group (RR, 1.207; 95% CI, 1.203-1.210); for O3, the RR increased from the 2nd (RR, 1.050; 95% CI, 1.047-1.053) to the 9th decile group (RR, 1.107; 95% CI, 1.104-1.110); for NO2, the DR curve wiggled at low levels and started rising from the 6th (RR, 1.005; 95% CI, 1.002-1.018) till the highest decile group (RR, 1.024; 95% CI, 1.021-1.027).
CONCLUSIONS: This study provided more robust evidence of the causal relations between air pollution exposures and mortality. The emulated causal D-R relations provided significant implications for reviewing the national air quality standards, as they inferred the number of potential early deaths prevented if air pollutants were reduced to specific levels; for example, lowering each air pollutant concentration from the 70th to 60th percentiles would prevent 65,935 early deaths per year.


Air pollution is a major threat to global health, which is associated with several adverse health outcomes and increased mortality. Few studies have investigated the association between air pollution and osteoporosis, and their findings were inconclusive. Our objective is to determine whether exposure to outdoor air pollution is causally associated with risk of osteoporosis. A systematic literature search of PubMed, Web of Science, Embase, and Cochrane Library for publications up to December 2020 was conducted for studies reporting the association between air pollution and osteoporosis. Meta-analysis was performed to estimate the pooled effect size of air pollution on osteoporosis using the relative risk (RR) and 95% confidence intervals (95% CI). Quality assessment was conducted, and all statistical analyses were performed by RevMan 5.3 software. Our search identified 9 eligible studies involving 9,371,212 patients. Meta-analysis revealed that there was an increased risk of osteoporosis (total body BMD and hip fracture) as a result of exposure to air pollution including PM2.5 and NO2. However, no significant excess risk of osteoporosis was found regardless of PM10, NO, and O3. In spite of a few number of epidemiological studies selected in the present literature review, this study indicated that the increased exposure to air pollutants was positively associated with high risk of osteoporosis. Further cohort studies with large sample sizes are needed to investigate different constituents and the duration of exposure of air pollutants.


The Intergovernmental Panel on Climate Change (IPCC) 5th Assessment Report (2014) assessed the state of climate change and health knowledge, globally through the Human Health: Impacts, Adaptation, and Co-Benefits Chapter and regionally through chapters, such as the North America Chapter. With IPCC's 6th Assessment Report scheduled to be released in 2021, we asked: how has climate change and health research in North America advanced since the IPCC's 5th Assessment Report in 2014? Specifically, we systematically identified and examined trends in the extent, range, and nature of climate-health research conducted in North America. We used a scoping review methodology to systematically identify literature and map publication trends. A search string was used to search five academic databases. Two independent reviewers first screened titles and abstracts, and then the full texts of articles for relevance. Research articles and reviews using systematic methods published since 2013 were eligible for inclusion, and no language restrictions were applied. To be included, articles had to
measure and link climatic variables or hazards to health outcomes in North America. Relevant articles were analysed using descriptive statistics to explore publication trends. The number of climate-health articles has significantly increased since the last IPCC Assessment Report. Published research about climate change impacts, heat-related mortality and morbidity, and respiratory illness taking place in urban centres and in the USA continue to dominate the North American climate-health literature, reflected by the high proportion of articles published. Important research gaps on previously neglected climate-sensitive health outcomes, however, are beginning to be filled, including climate change impacts on mental health, nutrition, and foodborne disease. We also observed progress in research that included future projections of climate-health risks; however, projection research is still relatively nascent and under-studied for many climate-sensitive health outcomes in North America, and would benefit from considering social and demographic variables in models. Important research disparities in geographical coverage were noted, including research gaps in Canada and Mexico, and in rural and remote regions. Overall, these publication trends suggest an improved understanding of exposure-response relationships and future projections of climate-health risks for many climate-sensitive health outcomes in North America, which is promising and provides an evidence-base to inform the IPCC 6th Assessment Report. Despite these advancements and considering the urgent policy and practice implications, more research is needed to deepen our understanding of climate-sensitive health outcomes, as well as examine new arising issues that have limited evidence-bases. In particular, transdisciplinary and cross-sector research, that includes the social sciences, examining current and future climate-health adaptation, mitigation, and the adaptation-mitigation nexus should become a top priority for research, given the urgent need for this evidence to inform climate change policies, actions, and interventions.


Airborne microorganisms in hospitals have been associated with several hospital-acquired infections (HAIs), and various measures of indoor air quality (IAQ) parameters such as temperature, relative humidity, carbon dioxide (CO2), particle mass concentration, and particle size have been linked to pathogen survival or mitigation of pathogen spread. To investigate whether there are quantitative relationships between the concentration of airborne microorganisms and the IAQ in the hospital environment. Web of Science, Scopus and PubMed databases were searched for studies reporting airborne microbial levels and any IAQ parameter(s) in hospital environments, from database inception to October 2020. Pooled effect estimates were determined via random-effects models. Seventeen of 654 studies were eligible for the meta-analysis. The concentration of airborne microbial measured as aerobic colony count (ACC) was significantly correlated with temperature ($r = 0.25$ [95% CI = 0.06-0.42], $p = 0.01$), CO2 concentration ($r = 0.53$ [95% CI = 0.40-0.64], $p < 0.001$), particle mass concentration ($\leq 5$ µg/m3 ; $r = 0.40$ [95% CI = 0.04-0.66], $p = 0.03$), and particle size ($\leq 5$ and $> 5$ µm), ($r = 0.51$ [95% CI = 0.12-0.77], $p = 0.01$ and $r = 0.55$ [95% CI = 0.20-0.78], $p = 0.003$), respectively, while
not being significantly correlated with relative humidity or particulate matter of size >5 µm. Conversely, airborne total fungi (TF) were not significantly correlated with temperature, relative humidity, or CO2 level. However, there was a significant weak correlation between ACC and TF ($r = 0.31$ [95% CI = 0.07-0.52], p = 0.013). Although significant correlations exist between ACC and IAQ parameters, the relationship is not definitive; the IAQ parameters may affect the microorganisms but are not responsible for the presence of airborne microorganisms. Environmental parameters could be related to the generating source, survival, dispersion, and deposition rate of microorganisms. Future studies should record IAQ parameters and factors such as healthcare worker presence and the activities carried out such as cleaning, sanitizing, and disinfection protocols. Foot traffic would influence both the generation of microorganisms and their deposition rate onto surfaces in the hospital environment. These data would inform models to improve the understanding of the likely concentration of airborne microorganisms and provide an alternative approach for real-time monitoring of the healthcare environment.

11. **Social and environmental risks as contributors to the clinical course of heart failure.** Narita K, Amiya E. Heart Fail Rev. 2021 May 4. doi: 10.1007/s10741-021-10116-7. Online ahead of print. Heart failure is a major contributor to healthcare expenditures. Many clinical risk factors for the development and exacerbation of heart failure had been reported, including diabetes, renal dysfunction, and respiratory disease. In addition to these clinical parameters, the effects of social factors, such as occupation or lifestyle, and environmental factors may have a great impact on disease development and progression of heart failure. However, the current understanding of social and environmental factors as contributors to the clinical course of heart failure is insufficient. To present the knowledge of these factors to date, this comprehensive review of the literature sought to identify the major contributors to heart failure within this context. Social factors for the risk of heart failure included occupation and lifestyle, specifically in terms of the effects of specific occupations, occupational exposure to toxicities, work style, and sleep deprivation. Socioeconomic factors focused on income and education level, social status, the neighborhood environment, and marital status. Environmental factors included traffic and noise, air pollution, and other climate factors. In addition, psychological stress and behavior traits were investigated. The development of heart failure may be closely related to these factors; therefore, these data should be summarized for the context to improve their effects on patients with heart failure. The present study reviews the literature to summarize these influences.

12. **Long-Term Exposure to Particulate Air Pollution Is Associated With 30-Day Readmissions and Hospital Visits Among Patients With Heart Failure.** Ward-Caviness CK, Danesh Yazdi M, Moyer J, Weaver AM, Cascio WE, Di Q, Schwartz JD, Diaz-Sanchez D. J Am Heart Assoc. 2021 May 4:e019430. doi: 10.1161/JAHA.120.019430. Online ahead of print. https://www.ahajournals.org/doi/full/10.1161/JAHA.120.019430 Background Long-term air pollution exposure is a significant risk factor for inpatient hospital admissions in the general population. However, we lack information on whether long-term air pollution exposure is a risk factor for hospital readmissions, particularly in individuals with elevated readmission rates. Methods and Results We determined the number of readmissions and total hospital visits (outpatient visits+emergency room visits+inpatient admissions) for 20
920 individuals with heart failure. We used quasi-Poisson regression models to associate annual average fine particulate matter at the date of heart failure diagnosis with the number of hospital visits and 30-day readmissions. We used inverse probability weights to balance the distribution of confounders and adjust for the competing risk of death. Models were adjusted for age, race, sex, smoking status, urbanicity, year of diagnosis, short-term fine particulate matter exposure, comorbid disease, and socioeconomic status. A 1-µg/m³ increase in fine particulate matter was associated with a 9.31% increase (95% CI, 7.85%-10.8%) in total hospital visits, a 4.35% increase (95% CI, 1.12%-7.68%) in inpatient admissions, and a 14.2% increase (95% CI, 8.41%-20.2%) in 30-day readmissions. Associations were robust to different modeling approaches. Conclusions These results highlight the potential for air pollution to play a role in hospital use, particularly hospital visits and readmissions. Given the elevated frequency of hospitalizations and readmissions among patients with heart failure, these results also represent an important insight into modifiable environmental risk factors that may improve outcomes and reduce hospital use among patients with heart failure.


Background Previous studies have investigated the association of ambient air pollution with blood pressure (BP) in children and adolescents, however, the results are not consistent. We conducted a systematic review and meta-analysis to assess the relationship between short-term and long-term ambient air pollutant exposure with BP values among children and adolescents. Methods and Results We searched PubMed, Web of Science, and Embase before September 6, 2020. Two reviewers independently searched and selected studies, extracted data, and assessed study quality. The studies were divided into groups by composition of air pollutants (NO2, particulate matter (PM) with diameter ≤10 µm or ≤2.5 µm) and length of exposure. The beta regression coefficients (β) and their 95% CIs were calculated to evaluate the strength of the effect with each 10 µg/m³ increase in air pollutants. Out of 36 650 articles, 14 articles were included in this meta-analysis. The meta-analysis showed short-term exposure to PM with diameter ≤10 µm (β=0.267; 95% CI, 0.033–0.501) was significantly associated with elevated systolic BP values. In addition, long-term exposure to PM with diameter ≤2.5 µm (β=1.809; 95% CI, 0.962–2.655), PM with diameter ≤10 µm (β=0.526; 95% CI, 0.095–0.958), and NO2 (β=0.754; 95% CI, 0.541–0.968) were associated with systolic BP values and long-term exposure to PM with diameter ≤2.5 µm (β=0.931; 95% CI, 0.157–1.705), and PM with diameter ≤10 µm (β=0.378; 95% CI, 0.022–0.735) was associated with diastolic BP. Conclusions Our study indicates that both short-term and long-term exposure to some ambient air pollutants may increase BP values among children and adolescents.

Background Previous studies have found associations between fine particulate matter <2.5 µm in diameter (PM2.5) and increased risk of cardiovascular disease (CVD) among populations with no CVD history. Less is understood about susceptibility of adults with a history of CVD and subsequent PM2.5-related CVD events and whether current regulation levels for PM2.5 are protective for this population. Methods and Results This retrospective cohort study included 96 582 Kaiser Permanente Northern California adults with a history of stroke or acute myocardial infarction. Outcome, covariate, and address data obtained from electronic health records were linked to time-varying 1-year mean PM2.5 exposure estimates based on residential locations. Cox proportional hazard models estimated risks of stroke, acute myocardial infarction, and cardiovascular mortality associated with PM2.5 exposure, adjusting for multiple covariates. Secondary analyses estimated risks below federal and state regulation levels (12 µg/m3 for 1-year mean PM2.5). A 10-µg/m3 increase in 1-year mean PM2.5 exposure was associated with an increase in risk of cardiovascular mortality (hazard ratio [HR], 1.20; 95% CI, 1.11-1.30), but no increase in risk of stroke or acute myocardial infarction. Analyses of <12 µg/m3 showed increased risk for CVD mortality (HR, 2.31; 95% CI, 1.96-2.71), stroke (HR, 1.41; 95% CI, 1.09-1.83), and acute myocardial infarction (HR, 1.51; 95% CI, 1.21-1.89) per 10-µg/m3 increase in 1-year mean PM2.5. Conclusions Adults with a history of CVD are susceptible to the effects of PM2.5 exposure, particularly on CVD mortality. Increased risks observed at exposure levels <12 µg/m3 highlight that current PM2.5 regulation levels may not be protective for this susceptible population.


Background Diesel exhaust (DE) emissions are a major contributor to ambient air pollution and are strongly associated with cardiovascular morbidity and mortality. Exposure to traffic-related particulate matter is linked with acute adverse cardiovascular events; however, the mechanisms are not fully understood. We examined the role of the autonomic nervous system during exposure to DE that has previously only been indirectly investigated. Methods and Results Using microneurography, we measured muscle sympathetic nerve activity (MSNA) directly in the peroneal nerve of 16 healthy individuals. MSNA, heart rate, and respiration were recorded while subjects rested breathing filtered air, filtered air with an exposure mask, and standardized diluted DE (300 µg/m3) through the exposure mask. Heart rate variability was assessed from an ECG. DE inhalation rapidly causes an increase in number of MSNA bursts as well as the size of bursts within 10 minutes, peaking by 30 minutes (P<0.001), compared with baseline filtered air with an exposure mask. No significant changes occurred in heart rate variability indices during DE exposure; however, MSNA frequency correlated negatively with total power (r²=0.294, P=0.03) and low frequency (r²=0.258, P=0.045). Heart rate correlated positively with MSNA frequency (r²=0.268, P=0.04) and the change in percentage of larger bursts (burst amplitude, height >50% of the maximum burst) from filtered air with an exposure mask (r²=0.368, P=0.013). Conclusions Our study provides direct evidence for the rapid modulation of the autonomic nervous system after exposure to DE, with an increase in MSNA. The quick increase in sympathetic outflow may explain the strong epidemiological data
associating traffic-related particulate matter to acute adverse cardiovascular events such as myocardial infarction. Registration URL: https://www.clinicaltrials.gov; Unique identifier: NCT02892279.


BACKGROUND: The effect of exposure to particulate matter (PM) on human health is a global public health concern. To develop an effective strategy to reduce PM exposure, we performed detailed questionnaire surveys regarding the type of lifestyle required to avoid PM exposure in patients with chronic obstructive pulmonary disease (COPD). We correlated the data with real-time PM concentration during the winter season.

METHODS: We enrolled 104 patients with COPD aged 40 years or older. Detailed questionnaire surveys were conducted among participants, and internet of things-based sensors were installed at their homes to measure the indoor PM2.5 concentration, which was continuously monitored between December 2019 and February 2020. The associations among PM2.5 concentration, patients' lifestyles, and the impact of both concentration and lifestyle on COPD exacerbation were analyzed.

RESULTS: Mean outdoor PM2.5 concentration was higher than mean indoor PM2.5 concentration during the study period (21.28±5.09 μg/m3 vs. 12.75±7.64 μg/m3), with a mean difference of 8.53±7.99 μg/m3. Among the various social factors and practices that aim to avoid exposure to PM, six practices and economic statuses were confirmed to reduce indoor PM2.5 concentration compared to outdoor concentration; Contrarily, these practices created a significant difference between the outdoor and indoor PM2.5 concentrations. The six practice items that showed a significant difference were 1) checking air quality forecast (the difference: -13.31±1.35 μg/m3, p=0.013), 2) indoor air filter operated (-15.43±1.32 μg/m3, p<0.001), 3) ventilating home by opening the window (-13.14±1.28 μg/m3, p=0.013), 4) checking filters of the air filter (-13.95±1.50 μg/m3, p=0.002), 5) refraining from going out when outside PM is high (-12.52±1.37 μg/m3, p=0.039), 6) wearing a mask when going out (-13.38±1.32 μg/m3, p=0.017). The higher the household income and economic level, the more significant the difference in the PM2.5 concentration. Severe exacerbation was more prevalent among patients with acute exacerbation as the exposure time of PM2.5≥35 μg/m3 or PM2.5≥75 μg/m3.

CONCLUSION: Lifestyle and economic levels can affect the indoor PM2.5 concentration, which may impact COPD exacerbation.

WE ACT

PURPOSE OF REVIEW: Following their use for medicinal purposes, volatile inhalational anaesthetic agents are expelled into the atmosphere where they contribute to anthropogenic climate change. We describe recent evidence examining the benefits and harms associated with their use.

RECENT FINDINGS: The environmental harms associated with desflurane and nitrous oxide likely outweigh any purported clinical benefits. Life cycle analyses are beginning to address the many gaps in our understanding, and informing choices made on all aspects of anaesthetic care. There is, however, an urgent need to move beyond the debate about anaesthetic technique A vs. B and focus also on areas such as sustainable procurement, waste management, pharmacological stewardship and joined-up solutions.

SUMMARY: There is now compelling evidence that anaesthetists, departments and hospitals should avoid desflurane completely, and limit nitrous oxide use to settings where there is no viable alternative, as their environmental harms outweigh any perceived clinical benefit. Life cycle analyses seem supportive of total intravenous and/or regional anaesthesia. There are many other areas where choices can be made by individual anaesthetists that contribute towards reducing the environmental burden of healthcare, such as prioritising the reduction of inappropriate resource use and over-treatment. However, this all requires joined up solutions where all parts of an organisation engage.

18. **What mental health professionals and organisations should do to address climate change.**
https://www.cambridge.org/core/journals/bjpsych-bulletin/article/what-mental-health-professionals-and-organisations-should-do-to-address-climate-change/EDDE8DC5454A14AA93EB41C597D82453

AIMS AND METHOD: The climate change emergency is also a mental healthcare emergency. We seek to provide a framework for what mental health professionals and organisations should do to make their practice more sustainable.

RESULTS: There are ethical, legal and organisational imperatives to make mental healthcare more sustainable. Mental healthcare must be refocused with an emphasis on prevention, building social capital and community resilience. Patients must be empowered to manage their own mental health. Efficiencies should be found within the system. Low-carbon ways to deliver care must be found, measured and improved upon. Greater adaptability needs to be built into the system to mitigate the impact of climate change. Sustainability should be integrated into training programmes, and good examples of practice shared and celebrated.

CLINICAL IMPLICATIONS: Mental health organisations and individuals must act now to prevent and adapt for the climate and ecological emergency. Sustainable practice is also good practice.

19. **The Moderating Role of Ethical Leadership on Nurses' Green Behavior Intentions and Real Green Behavior.**
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8068523/

AIM: This study is aimed at exploring the relationship between green behavior intentions and green behavior and analyzing the moderating role of ethical leadership in this relationship.
BACKGROUND: Nurses' green behavior can directly reduce costs and protect the natural environment and organizational sustainability by saving resources and energy. It is not clear how green behavior intention affects green behavior or how the positive influence of green behavior intention on green behavior can be enhanced. Design and Methods. This is a cross-sectional study, and the surveys are collected from 3 hospitals in China. Of the initial cohort of 489 nurses, 89.6% were female. There were 327 subjects (66.9%) aged 35 or less, 267 subjects (54.6%) with 10 years or less of work experience, and 220 unmarried subjects (44.9%). Data were collected from January to July 2018, using three surveys: green behavior intentions, green behavior, and ethical leadership.

RESULTS: Green behavior intentions impacted employee green behavior (b = 0.32, t = 5.37, p < 0.01). The interaction term for green behavior intentions and ethical leadership was significant (b = 0.28, t = 2.53, p ≤ 0.01); the conditional direct effect of green behavior intentions was only significant at a high level of ethical leadership (conditional effect = 0.53, SE = 0.16, t = 3.38, p < 0.01, 95% confidence interval of 0.22-0.84).

CONCLUSION: The intention to engage in green behavior influences nurses' green behavior positively, and the relationship is stronger when ethical leadership is high in the organization than when ethical leadership is low. The results of this study can help both academics and practitioners to understand the micromechanism of environmentally sustainable development in more detail and to identify the mechanisms and boundary conditions of green behavioral intentions, green behavior, and ethical leadership.

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