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**Subject:** Traffic Related Air Pollution

To All :

This communication is to promote awareness of the harmful effects of traffic generated air pollution on human health and well-being. Each of you heads or reports to commissions, boards, agencies or legislative bodies that, individually or collectively, regulate and guide present and future growth and development in Ridgefield and surrounding areas.

The attached information is submitted as general information, but, i urge that it be given great weight in any consideration of present or future activity, process, regulation or application that would be reasonably expected to generate increased vehicular traffic. Local and regional traffic congesation is already threatening our way of life and it is imperative that you take notice and act accordingly. Please distribute this communication with attachments to your respective members.

Thank you,

Mike Autuori

**Sampling of Traffic-Related Air Pollution Health Effects Compiled from Available Public Sources**

Michael J. Autuori, Ph.D. March 19, 2025

**AI Overview**

Traffic congestion exacerbates air pollution, leading to various health problems, including respiratory issues, cardiovascular diseases, and potentially increased risk of certain cancers, especially in those living near busy roads.
Here's a more detailed look at the adverse health effects of traffic congestion:
Respiratory Issues:

- **Asthma:**

Traffic-related air pollution (TRAP) can worsen asthma symptoms and even increase the risk of developing asthma, particularly in children.

- **Reduced Lung Function:**

Exposure to pollutants from traffic can lead to reduced lung function in both adults and children.

- **Respiratory Illnesses:**

Long-term exposure to traffic-related air pollution can increase the risk of developing respiratory illnesses, including chronic obstructive pulmonary disease (COPD).

- **PM2.5:**

Fine particulate matter (PM2.5) from traffic exhaust is a major contributor to respiratory problems, as it can penetrate deep into the lungs and enter the bloodstream.
Cardiovascular Diseases:

- **Heart Disease:**

Exposure to traffic-related air pollution is linked to an increased risk of heart disease, heart attacks, and strokes.

- **High Blood Pressure:**

Traffic congestion and the associated stress can contribute to high blood pressure, a major risk factor for cardiovascular disease.
Other Health Effects:

- **Childhood Leukemia:**

Some studies suggest a link between exposure to traffic-related air pollution and an increased risk of childhood leukemia.

- **Premature Births and Low Birth Weight:**

Exposure to air pollution during pregnancy has been associated with an increased risk of premature births and low birth weight.

- **Cancer:**

Long-term exposure to traffic-related air pollution may increase the risk of certain cancers, including lung cancer.

- **Mental Health:**

Road traffic noise can contribute to increased levels of stress, anxiety, and depression.

- **Increased Risk of Accidents:**

Traffic congestion increases the likelihood of accidents, which can lead to injuries and fatalities.

- **Chronic Stress:**

The daily hassles of traffic can contribute to higher levels of chronic stress.

- **Reduced Lung Function:**

Long-term exposure to air pollution can lead to reduced lung function.

- **Impaired Lung Development:**

In children, exposure to air pollution can impair lung development.

- **Oxidative Stress:**

Traffic-related air pollution can cause oxidative stress, which can damage cells and tissues.

- **Genotoxicity:**

Some pollutants can be genotoxic, meaning they can damage DNA and increase the risk of cancer.

- **Inflammatory Responses:**

Traffic-related air pollution can trigger inflammatory responses in the body.

- **Hypertensive Disorders of Pregnancy:**

Exposure to traffic-related air pollution can cause hypertensive disorders of pregnancy

Source: Office of Transportation and Air Quality EPA-420-F-14-044 August 2014

Pollutants directly emitted from cars, trucks and other motor vehicles are found in higher concentrations near major roads. Examples of directly emitted pollutants include particulate matter (PM), carbon monoxide (CO), oxides of nitrogen (NOx), and benzene, though hundreds of chemicals are emitted by motor vehicles. Motor vehicles also emit compounds that lead to the formation of other pollutants in the atmosphere, such as nitrogen dioxide (NO2), which is found in elevated concentrations near major roads, and ozone (O3), which forms further downwind. Beyond vehicles' tailpipe and evaporative emissions, roadway traffic also emits brake and tire debris and can throw road dust into the air. Individually and in combination, many of the pollutants found near roadways have been associated with adverse health effects.

**Traffic Noise and Mental Health: A Systematic Review and Meta-Analysis**

Janice Hegewald [1,2,†], Melanie Schubert [1,†], Alice Freiberg [1], Karla Romero Starke [1,2], Franziska Augustin [1], Steffi G Riedel-Heller [3], Hajo Zeeb [4,5], Andreas Seidler [1,*]

- Author information
- Article notes
- Copyright and License information

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Abstract

Recent evidence suggests that traffic noise may negatively impact mental health. However, existing systematic reviews provide an incomplete overview of the effects of all traffic noise sources on mental health. We conducted a systematic literature search and summarized the evidence for road, railway, or aircraft noise-related risks of depression, anxiety, cognitive

decline, and dementia among adults. We included 31 studies (26 on depression and/or anxiety disorders, 5 on dementia). The meta-analysis of five aircraft noise studies found that depression risk increased significantly by 12% per 10 dB $L_{DEN}$ (Effect Size = 1.12, 95% CI 1.02–1.23). The meta-analyses of road (11 studies) and railway traffic noise (3 studies) indicated 2–3% (not statistically significant) increases in depression risk per 10 dB $L_{DEN}$. Results for road traffic noise related anxiety were similar. We did not find enough studies to meta-analyze anxiety and railway or aircraft noise, and dementia/ cognitive impairment and any traffic noise. In conclusion, aircraft noise exposure increases the risk for depression. Otherwise, we did not detect statistically significant risk increases due to road and railway traffic noise or for anxiety. More research on the association of cognitive disorders and traffic noise is required. Public policies to reduce environmental traffic noise might not only increase wellness (by reducing noise-induced annoyance), but might contribute to the prevention of depression and anxiety disorders.

**How Sitting In Traffic Jam Can Harm Your Health - Dr. Vivek Kumar**

Dr. Vivek KumarMay 01, 2024

Traffic congestion increases vehicle emissions and degrades ambient air quality, studies have shown excess morbidity and mortality for drivers and commuters, stressed and frustrated motorists, encouraging road rage and reduced health of motorists. Commuters who are exposed to air pollution, especially those riding in non-air conditioned vehicles such as autos, e-rickshaws, cycle rickshaws and motorcycles, double their health risk.

Aside from chronic lung diseases, air pollution can cause blood pressure to rise and inflame the arteries, increasing heart attack and stroke risk.Blood pressure goes up with increased traffic pollutants, and electrocardiogram changes show decreased blood flow to the heart.

Pollution that gathers inside cars in traffic jams and at red traffic lights is far higher than that found in cars that are moving. It contributes to lung cancer, asthma, and other respiratory diseases, besides heart disease and stroke. All of these can be fatal.Pollution at intersections is 29 times higher than those found in free-flowing traffic.At intersections, vehicles slow down, stop, rev up to move when lights turn green, and they are closer together. The cars move slowly, so that drivers are exposed for longer. As the output is ongoing, the pollution does not disperse but lingers and accumulates. The cars waiting in traffic jams or at red lights contain up to 40 percent more pollution than those that are moving.

Long commutes eat up exercise time and are associated with higher weight, lower fitness levels, and higher blood pressure—all strong predictors of heart disease, diabetes, and some types of cancer.

Being exposed to the daily hassles of traffic can lead to higher chronic stress.One of the stress triggers while driving during a traffic jam is impatience—having to wait for the traffic to move and dealing with the mistakes of other motorists on the road.Impatience, if not handled at the beginning, tends to turn into resentment, aggressive driving and anger- which all can lead to

road rage.Stress, is a "killer disease" that makes people vulnerable to other diseases and even depression.

Some commuters who are caught in traffic mess are also deprived of sleep, especially those who had to get up early the next day to avoid the morning rush and get to work on time. Sleep deprivation can also affect performance, attention, and long-term memory. Moreover, lack of sleep can cause anxiety, exhaustion, frustration, impulsive behavior, lower immunity, and some mental health problems. It also encourages drug and alcohol.
Traffic jams also don't allow commuters to use rest rooms if required, causing holding of urine for long period of times which can cause urinary infections. Urinary infections can sometimes be so severe as to cause acute urinary retention. If this happens in an elderly individual it can lead to psychotic behavior.
If a commuter held up in a traffic jam feels thirsty and is not carrying a water bottle, it can cause low Blood pressure due to dehydration.

Respir Res
. 2005 Dec 22;6(1):152. doi: 10.1186/1465-9921-6-152

**Long-term air pollution exposure and living close to busy roads are associated with COPD in women**

Tamara Schikowski [1,✉], Dorothea Sugiri [1], Ulrich Ranft [1], Ulrike Gehring [2,3,4], Joachim Heinrich [2], H-Erich Wichmann [2,3], Ursula Krämer [1]

- Author information
- Article notes
- Copyright and License information

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Abstract
Background
Lung function and exacerbations of chronic obstructive pulmonary disease (COPD) have been associated with short-term exposure to air pollution. However, the effect of long-term exposure to particulate matter from industry and traffic on COPD as defined by lung function has not been evaluated so far. Our study was designed to investigate the influence of long-term exposure to air pollution on respiratory symptoms and pulmonary function in 55-year-old women. We especially focused on COPD as defined by GOLD criteria and additionally compared the effects of air pollution on respiratory symptoms by questionnaire data and by lung function measurements.
Methods
In consecutive cross sectional studies conducted between 1985–1994, we investigated 4757 women living in the Rhine-Ruhr Basin of Germany. $NO_2$ and $PM_{10}$ exposure was assessed by measurements done in an 8 km grid, and traffic exposure by distance from the residential address to the nearest major road using Geographic Information System data. Lung function was determined and COPD was defined by using the GOLD criteria. Chronic respiratory symptoms and possible confounders were defined by questionnaire data. Linear and logistic

regressions, including random effects were used to account for confounding and clustering on city level.

Results

The prevalence of COPD (GOLD stages 1–4) was 4.5%. COPD and pulmonary function were strongest affected by $PM_{10}$ and traffic related exposure. A 7 $\mu g/m_3$ increase in five year means of $PM_{10}$ (interquartile range) was associated with a 5.1% (95% CI 2.5%–7.7%) decrease in $FEV_1$, a 3.7% (95% CI 1.8%–5.5%) decrease in FVC and an odds ratio (OR) of 1.33 (95% CI 1.03–1.72) for COPD. Women living less than 100 m from a busy road also had a significantly decreased lung function and COPD was 1.79 times more likely (95% CI 1.06–3.02) than for those living farther away. Chronic symptoms as based on questionnaire information showed effects in the same direction, but less pronounced.

Conclusion

Chronic exposure to $PM_{10}$, $NO_2$ and living near a major road might increase the risk of developing COPD and can have a detrimental effect on lung function.

**Residential Traffic Exposure and Childhood Leukemia**

A Systematic Review and Meta-analysis

Vickie L Boothe [1], Tegan K Boehmer [1], Arthur M Wendel [1], Fuyuen Y Yip [1]

- Author information
- Copyright and License information

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The publisher's version of this article is available at Am J Prev Med

Abstract

Context

Exposure to elevated concentrations of traffic-related air pollutants in the near-road environment is associated with numerous adverse human health effects, including childhood cancer, which has been increasing since 1975. Results of individual epidemiologic studies have been inconsistent. Therefore, a meta-analysis was performed to examine the association between residential traffic exposure and childhood cancer.

Evidence acquisition

Studies published between January 1980 and July 2011 were retrieved from a systematic search of 18 bibliographic databases. Nine studies meeting the inclusion criteria were identified. Weighted summary ORs were calculated using a random effects model for outcomes with four or more studies. Subgroup and sensitivity analyses were performed.

Evidence synthesis

Childhood leukemia was positively associated (summary OR=1.53, 95% CI=1.12, 2.10) with residential traffic exposure among seven studies using a postnatal exposure window (e.g., childhood period or diagnosis address) and there was no association (summary OR=0.92, 95% CI=0.78, 1.09) among four studies using a prenatal exposure window (e.g., pregnancy period or birth address). There were too few studies to analyze other childhood cancer outcomes.

Conclusions

Current evidence suggests that childhood leukemia is associated with residential traffic exposure during the postnatal period, but not during the prenatal period. Additional well-

designed epidemiologic studies that use complete residential history to estimate traffic exposure, examine leukemia subtypes, and control for potential confounding factors are needed to confirm these findings. As many people reside near busy roads, especially in urban areas, precautionary public health messages and interventions designed to reduce population exposure to traffic might be warranted.

**Outdoor artificial light at night, air pollution, and risk of childhood acute lymphoblastic leukemia in the California Linkage Study of Early-Onset Cancers**


- Charlie Zhong,
- Rong Wang,
- Libby M. Morimoto,
- Travis Longcore,
- Meredith Franklin,
- Tormod Rogne,
- Catherine Metayer,
- Joseph L. Wiemels &
- Xiaomei Ma



*Scientific Reports* **volume 13**, Article number: 583 (2023) Cite this article





**Abstract**

Acute lymphoblastic leukemia (ALL) is the most common type of cancer in children (age 0–14 years); however, the etiology remains incompletely understood. Several environmental exposures have been linked to risk of childhood ALL, including air pollution. Closely related to air pollution and human development is artificial light at night (ALAN), which is believed to disrupt circadian rhythm and impact health. We sought to evaluate outdoor ALAN and air pollution on risk of childhood ALL. The California Linkage Study of Early-Onset Cancers is a large population-based case–control in California that identifies and links cancer diagnoses from the California Cancer Registry to birth records. For each case, 50 controls with the same year of birth were obtained from birth records. A total of 2,782 ALL cases and 139,100 controls were identified during 2000–2015. ALAN was assessed with the New World Atlas of Artificial Night Sky Brightness and air pollution with an ensemble-based air pollution model of particulate matter smaller than 2.5 microns ($PM_{2.5}$). After adjusting for known and suspected risk factors, the highest tertile of ALAN was associated with an increased risk of ALL in Hispanic children (odds ratio [OR] = 1.15, 95% confidence interval [CI] 1.01–1.32). There also appeared to be a borderline association between $PM_{2.5}$ level and risk of ALL among non-Hispanic White children (OR per 10 μg/$m^3$ = 1.24, 95% CI 0.98–1.56). We observed elevated risk of ALL in Hispanic children residing in areas of greater ALAN. Further work is needed to understand the role of ALAN and air pollution in the etiology of childhood ALL in different racial/ethnic groups.

Studies suggest a link between traffic-related air pollution and an increased risk of childhood leukemia, particularly acute myeloid leukemia (AML), with higher risks observed for children under 6 years old and during postnatal exposure.

Some studies indicate that exposure to traffic-related air pollutants, especially benzene and nitrogen dioxide (NO2), is associated with an elevated risk of childhood leukemia.

- **Specific Leukemia Types:**

The link is particularly strong for AML, with some research suggesting a higher relative risk for this subtype of leukemia compared to other types.

- **Exposure Timing:**

The risk appears to be higher when exposure occurs during the postnatal period, rather than during the prenatal period.

- **Pollutants of Concern:**
    - **Benzene:** Exposure to benzene, a common component of gasoline, has been linked to an increased risk of childhood leukemia, especially AML.
    - **Nitrogen Dioxide (NO2):** Exposure to NO2, a byproduct of vehicle exhaust, has also been associated with an increased risk of leukemia.
    - **Particulate Matter (PM2.5):** Exposure to fine particulate matter, a common component of air pollution, has been linked to increased overall morbidity and mortality.
- **Further Research Needed:**

While these studies suggest a link, more research is needed to confirm these findings and to understand the underlying mechanisms.

- **Precautionary Measures:**

Given the potential risks, public health messages and interventions aimed at reducing exposure to traffic-related air pollution may be warranted, especially in urban areas where a large portion of the population lives near major roads.

**Impacts on Natural Ecosystems: ( AI Overview )**

- **Air Quality Degradation:** TRAP reduces air quality, making it difficult for plants and animals to thrive.
- **Water Quality Degradation:** Pollutants can be deposited on land and enter waterways, harming aquatic life and ecosystems.
- **Damage to Sensitive Plants and Trees:** Air pollutants can be toxic to sensitive plant species, leading to damage or death.
- **Reduced Biodiversity:** Ecosystems with poor air and water quality tend to have lower biodiversity, as certain species cannot survive in polluted environments.
- **Acid Rain:** NOx and sulfur oxides (SOx) emitted by vehicles can contribute to acid rain, which damages forests, lakes, and soil.
- **Ecosystem Health:** Air pollution can disrupt the delicate balance of ecosystems, leading to negative consequences for plants, animals, and the overall environment.

- **Examples of Ecosystem Damage:**
    - **Forests:** Acid rain and ozone pollution can damage trees, making them more susceptible to disease and pests.
    - **Lakes and Rivers:** Pollutants can contaminate water bodies, harming aquatic life and making them unsuitable for human consumption.
    - **Soil:** Pollutants can accumulate in the soil, making it less fertile and harming plant growth.

**Particulate Matter Exposure and Attention-Deficit/Hyperactivity Disorder in Children: A Systematic Review of Epidemiological Studies**


- Gabriele Donzelli [1,*], Agustin Llopis-Gonzalez [1,2], Agustin Llopis-Morales [1], Lorenzo Cioni [3], María Morales-Suárez-Varela [1,2]
- Author information

- Article notes
- Copyright and License information

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- Abstract
- Attention-deficit/hyperactivity disorder (ADHD) is the most common cognitive and behavioural disorder affecting children, with a worldwide-pooled prevalence of around 5%. Exposure to particulate matter (PM) air pollution is suspected to be associated with autism spectrum disorders and recent studies have investigated the relationship between PM exposure and ADHD. In the absence of any synthesis of the relevant literature on this topic, this systematic review of epidemiological studies aimed to investigate the relationship between the exposure of children to PM and ADHD and identify gaps in our current knowledge. In December 2018, we searched the PubMed and EMBASE databases. We only included epidemiological studies carried out on children without any age limit, measuring PM exposure and health outcomes related to ADHD. We assessed the quality of the articles and the risk of bias for each included article using the Newcastle–Ottawa Scale and the Office of Health Assessment and Translation (OHAT) approach, respectively. The keyword search yielded 774 results. Twelve studies with a total number of 181,144 children met our inclusion criteria, of which 10 were prospective cohort studies and 2 were cross-sectional studies. We subsequently classified the selected articles as high or good quality studies. A total of 9 out of the 12 studies reported a positive association between PM exposure to outdoor air pollution and behavioral problems related to attention. Despite these results, we found a significant degree of heterogeneity among the study designs. Furthermore, 11 studies were judged to be at a probably high risk of bias in the exposure assessment. In conclusion, we opine that further high quality studies are still needed in order to clarify the association between PM exposure and ADHD diagnosis.

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**Traffic Related Air Pollution, Particulate Matter, and Autism**

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- Author information
- Copyright and License information

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Abstract

Context

Autism is a heterogeneous disorder with genetic and environmental factors likely contributing to its origins. Examination of hazardous pollutants has suggested the importance of air toxics in autism etiology, yet little research has examined local level air pollution associations using residence-specific exposure assignments.

Objective

To examine the relationship between traffic-related air pollution (TRP), air quality, and autism.

Design, Setting and Population

This study includes data on 279 autism cases and 245 typically developing controls enrolled in the Childhood Autism Risks from Genetics and the Environment (CHARGE) Study in California. The mother's address from the birth certificate and addresses reported from a residential history questionnaire were used to estimate exposure for each trimester of pregnancy and first year of life. TRP was assigned to each location using a line-source air-quality dispersion model. Regional air pollutant measures were based on the Environmental Protection Agency's Air Quality System data. Logistic regression models compared estimated and measured pollutant levels for autism cases and typically developing controls.

Main Outcome Measures

Crude and multivariable-adjusted odds ratios (OR) for autism.

Results

Cases were more likely to live at residences in the highest quartile TRP exposure during pregnancy (OR=1.98, 95%CI 1.20–3.31) and the first year of life (OR=3.10, 1.76–5.57) compared to controls. Regional exposure measures of nitrogen dioxide ($NO_2$) and particulate matter less than 2.5 and 10 microns in diameter ($PM_{2.5}$ and $PM_{10}$) were also associated with autism during gestation ($NO_2$ OR=1.81/2SD, 95%CI 1.37–3.09; $PM_{2.5}$ OR=2.08/2SD, 95%CI 1.93–2.25; $PM_{10}$ OR=2.17/2SD, 95%CI 1.49–3.16) and the first year of life ($NO_2$ OR=2.06, 95%CI 1.37–3.09; $PM_{2.5}$ OR=2.12, 95%CI 1.45–3.10; $PM_{10}$ OR=2.14, 95%CI 1.46–3.12).

Conclusions

Exposure to TRP, $NO_2$, $PM_{2.5}$, and $PM_{10}$ during pregnancy and the first year of life was associated with autism. Further epidemiological and toxicological examination of likely biological pathways will help determine whether these associations are causal.

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**lifetime exposure to traffic-related air pollution and symptoms of depression and anxiety at age 12 years**

Kimberly Yolton [a,*,1], Jane C Khoury [b], Jeffrey Burkle [c], Grace LeMasters [d], Kim Cecil [e], Patrick Ryan [b]

- Author information
- Article notes
- Copyright and License information

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The publisher's version of this article is available at Environ Res
**This article has been corrected.** See Environ Res. 2019 Aug 7;176:108519.

Abstract

Background:

While air pollution has been associated with depression and anxiety in adults, its impact on childhood mental health is understudied.

Objective:

We examined lifetime exposure to traffic-related air pollution (TRAP) and symptoms of depression and anxiety at age 12 years in the Cincinnati Childhood Allergy and Air Pollution Study cohort.

Methods:

We estimated exposure to elemental carbon attributable to traffic (ECAT), a surrogate of diesel exhaust, at birth, age 12 years, and average exposure throughout childhood, using a validated land use regression model. We assessed depression and anxiety at age 12 years by parent report with the Behavior Assessment System for Children-2, and by child report with the Child Depression Inventory-2 (CDI-2) and the Spence Children's Anxiety Scale (SCAS). Associations between TRAP at birth, age 12 years, and childhood average and mental health outcomes were estimated using linear regression models adjusting for covariates including parent depression, secondhand smoke exposure, race, household income, and others.

Results:

Exposure to ECAT was not significantly associated with parent-reported depression or anxiety. However, exposure to ECAT at birth was associated with increased child-reported depression and anxiety. Each 0.25 μg/m$^3$ increase in ECAT was associated with a 3.5 point increase (95% CI 1.6–5.5) in CDI-2 scores and 2.3 point increase (95% CI 0.8–3.9) in SCAS total anxiety scores. We observed similar associations between average childhood ECAT exposures but not for concurrent exposures at age 12.

Conclusions:

TRAP exposure during early life and across childhood was significantly associated with self-reported depression and anxiety symptoms in children. The negative impact of air

pollution on mental health previously reported among adults may also be present during childhood.

EDITORIAL

# Is the air we breathe safe?

Is the air we breathe safe? This seemingly straightforward question is often unanswerable because regulations, monitoring approaches, and reporting structures cannot keep up with the rapid human-induced shifts to the planet and atmosphere. The air is becoming more burdened by pollutants from new sources such as intense fires at the wildland-urban interface, an ever-expanding area where human development meets natural habitats. Air pollution is the second leading cause of human death worldwide, a statistic that excludes numerous indoor and acute outdoor exposures. To protect public health everywhere, air quality standards must be modernized, advanced instrumentation mobilized, and collaborative networks established to move air quality assessment into the 21st century.

A major challenge is updating the Air Quality Index (AQI). The AQI was developed by the United States Environmental Protection Agency and is a key global metric for communicating air quality. However, it only accounts for a handful of pollutants targeted in the 1970s, leaving out thousands of more recently identified volatile organic compounds and other airborne toxins that originate from contemporary building materials, industrial processes and accidents, and urban fires. This includes the burning of homes, cars, plastics, batteries, and other matter at burgeoning urban-wildland boundaries. Consider the 2025 fires in Los Angeles, California, where AQI readings reported "good" air quality once visible smoke blew offshore. This misleadingly suggested that the air was safe even though invisible toxins were still being released. In its current form, the AQI cannot accurately convey the risks of toxins released from such urban fires.

**"Air pollution is the second leading cause of human death worldwide..."**

Airborne bioparticles, such as bacteria, fungi, and viruses, also affect human health, yet remain understudied. Although respiratory viruses such as influenza and severe acute respiratory syndrome coronavirus 2 have drawn attention, other microbes—particularly those released outdoors by fires—are often overlooked. Inconsistent sampling and analysis protocols make it difficult to compare their prevalence over time and space and to assess overall health impacts. A coordinated global mapping system that draws on contributions from government, academic, private, and community scientists could merge bioparticle and chemical pollutant data from a given location with a time-linked event map. This would provide a comprehensive view of atmospheric composition and air quality and substantially improve predictive models.

Existing air quality metrics, monitoring networks, and pollutant databases are insufficient to quantify the extent to which urban fires increase harmful air pollutants. Furthermore, standards for human exposures based on the effects of a growing multitude of air pollutants on health are needed. The gap between research and regulations is reminiscent of the aftermath of 9/11, when first responders were wrongly assured of safe air, only to face serious health issues later. Sadly, responses to episodic and evolving air pollution threats remain inadequate.

Although indoor air pollutants pose health risks—particularly in urban settings where people spend about 90% of their time indoors—the problem is more manageable with specialized filters for fine particles and activated carbon filters for harmful gases. Such interventions are inexpensive and can reduce daily exposure, but are unfortunately limited to major pollution events such as wildfires. Increasing accessibility to these filtration methods for indoor air management would substantially improve global public health.

Meanwhile, although technology exists to characterize air pollutants, epidemiological studies are hindered by fragmented data collection methods, inconsistent analysis protocols, and siloed data storage. Transformative progress requires coordinated measurement efforts, standardized protocols, and open-access global frameworks that link pollutant levels with location-specific variables. By integrating meteorological data, health data, computational systems biology, and artificial intelligence into a global mapping system, baselines can be established, anomalies identified, and the pollutant mixtures posing great risks to health identified.

People everywhere face air quality challenges that demand equitable solutions. In the United States, concern over air pollutants is surging at a moment when federal support for environmental protections is under threat. Yet, with adequate funding from diverse sources, scientists can tackle this solvable problem. By harnessing existing technologies, developing a global mapping system, forming strong inclusive partnerships, and establishing health-based metrics for emerging airborne pollutants, the ubiquitous and challenging question of whether the air is safe to breathe could well be answered.

**–Kimberly Prather and Kelley Barsanti**

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