ANNUAL REVIEWS

Annual Review of Environment and Resources

Indoor Air Pollution and Health: Bridging Perspectives from Developing and Developed Countries

Ajay Pillarisetti,[1] Wenlu Ye,[1] and Sourangsu Chowdhury[2]

[1]Division of Environmental Health Sciences, School of Public Health, University of California, Berkeley, California, USA; email: ajayp@berkeley.edu, wenlu.ye@berkeley.edu

[2]CICERO Center for International Climate Research, Oslo, Norway; email: sourangsu.chowdhury@cicero.oslo.no

Annu. Rev. Environ. Resour. 2022. 47:197–229

First published as a Review in Advance on September 20, 2022

The *Annual Review of Environment and Resources* is online at environ.annualreviews.org

https://doi.org/10.1146/annurev-environ-012220-010602

Keywords

household air pollution, biomass, exposure assessment, particulate matter, indoor air quality

Abstract

Much of the global population spends most of their time indoors; however, air pollution measurement, a proxy of exposure, occurs primarily outdoors. This fundamental disconnect between where the people are and where the measurements are made likely leads to misestimation of the true burden of air pollution on human health, which is already substantial, with exposure leading to approximately 6.7 million deaths yearly. In this review, we describe the two disparate but linked fields commonly referred to as indoor air pollution and household air pollution. Both fields focus on the measurement and characterization of exposures and subsequent health effects that occur primarily in the indoor environment. The former tends to focus on issues in the developed world, whereas the latter focuses on issues in low- and middle-income countries reliant on solid fuels, like wood, dung, coal, and crop residues, for basic household energy needs. Both lead to substantial exposures to air pollutants that are damaging to human health. We describe and contrast both contexts and provide potential topics for conversation between the disciplines.

Contents

INTRODUCTION 198
AIR POLLUTION AND THE GLOBAL BURDEN OF DISEASE 200
Outcomes Associated with Air Pollution Exposure 201
Exposure Estimation in the Global Burden of Disease 201
Counterfactual Exposures 202
Concentration- and/or Exposure-Response Functions 202
Non–Household Air Pollution Indoor Air Pollution in the Global Burden of Disease 203
POLLUTANTS OF CONCERN 203
INDOOR AIR POLLUTION IN THE DEVELOPED WORLD 206
Exposure 206
Assessing Exposures 206
Health Impacts 208
Mitigating Exposures 209
HOUSEHOLD AIR POLLUTION PRIMARILY IN THE DEVELOPING WORLD 211
Introduction 211
Inside Out 212
Reducing the Impact of Household Air Pollution Exposures: Shifting Paradigms 213
INDOOR AND HOUSEHOLD AIR POLLUTION: DISTINCTIONS AND DISCOURSE 216
Cooking with Gas: A Strong Source in High-Income Countries, a Strong Solution for Solid Fuel–Using Households 216
Household Air Pollution in the Burden of Disease 216
"Modern" Indoor Air Pollution Concerns in Countries with High Burdens of Household Air Pollution 217
The Place's the Thing! 217
Utilization of Low(er)-Cost Sensors for Air Pollution Assessment 217
CONCLUSION 217

Exposure: the interface between a pollutant concentration in the environment and an individual; a function of duration of contact between people and pollutants

Global Burden of Disease (GBD): quantifies morbidity and mortality from a range of diseases and risk factors for ill-health

INTRODUCTION

Exposure to air pollution is estimated by the Global Burden of Disease (GBD) effort[1] to result in approximately 6.67 million excess deaths per year (1) from a range of outcomes, including cardiovascular and respiratory diseases, diabetes, and cancers, among others. Much of this burden is attributable to ambient exposure to particulate pollution (4.1 million annual deaths, ~61% of the total burden) and ambient ozone (O_3) exposure (0.37 million, 9% of the total). The levels of exposure to ambient air pollution (AAP) experienced by individuals and populations are often

[1]The Institute for Health Metrics and Evaluation at the University of Washington organizes the GBD effort. More than 7,000 researchers in over 156 countries and territories collect and analyze the data. The data estimate premature deaths and lost disability-adjust life years (DALYs) from 350 diseases and injuries in 195 countries, by age and sex, from 1990 to the present. For more details on the GBD effort, see **https://www.healthdata.org/gbd/about**.

estimated by combining ground monitoring data, satellite-based estimates of pollution levels, and modeled concentrations from chemical transport models (2).

For wealthy countries, however, these estimates rarely take into account the location in which many people spend most of their time—the indoor environment (3). They also tend to focus on a narrow range of pollutants for which there is strong epidemiological evidence of health impacts—a justifiable approach, but one that presents perhaps an incomplete picture of the true burden of air pollution. In largely ignoring indoor environments, these estimates also ignore an arguably fundamental tenant of exposure science: to measure where the people are (4).

Of the 6.67 million annual deaths attributable to air pollution, 2.3 million are estimated to arise from direct exposure to household air pollution (HAP) generated during the combustion of solid fuels, typically for cooking, and largely in low- and middle-income countries (LMICs). Solid fuels are often burned indoors, but escape into the outdoor environment, thus contributing to AAP. In some contexts, it is expected that individuals in LMICs are exposed to both solid fuel–related HAP and other sources of indoor air pollution (IAP)—in addition to their ambient exposures.

On one hand, the location of exposure does not matter; reducing the combined or total exposure from all sources and locations is the overarching concern of policy and control measures. On the other hand, and from a health and mitigation standpoint, it is vital to know what causes emissions and exposures—and, critically, where these exposures occur—so that action can be taken to protect health and the environment.

Estimates of where people spend time—their so-called time-activity budgets—have been made for the developed world over the past three decades (3, 5, 6). In these studies, individuals report spending the majority of their time—between 80 and 90% of a typical day—indoors, whether at home, at work, or in transit. Less data are readily available in LMICs (7–9), although they indicate a similar trend, with the majority of time spent indoors. Regardless, given the substantial time spent inside daily by a large fraction of the global population, investigation and characterization of indoor air pollutants and environment are essential to (1) accurately understand and (2) mitigate exposures.

In a 2016 review of indoor air quality (IAQ) and its impacts on humans, Kwok Wai Tham (10) suggested four explanations for the relative lack of focus on the indoor environment, which we expand upon here. First, large episodes—such as the Donora event in the US (11), the London Smog (12), and high air pollution occurrences in Delhi (13), Los Angeles, and Beijing (14)—received large attention and response due to the intensity of pollution levels and the magnitude of impacts on health. During some of these events, entire populations had elevated exposures. Such events are seemingly not as newsworthy for indoor pollutants, despite, at least for the case of solid fuel use, equally high concentrations being regularly experienced by a large fraction of the global population. Second, data collection frequency and availability vary widely for outdoor and indoor environments; significantly more data are available for the former, especially at the population level. Recent advances in air pollution sensor technology, although useful in indoor environments, have widely been applied to outdoor environments to date. The same data discrepancy applies to health data; population-level associations between IAP and health are less well characterized, in part because detailed exposure data are hard to obtain. Third, and primarily in the developed world, there has been a reduction in magnitude and a change in the species emitted and experienced indoors. Emerging concerns in these contexts are a result of new construction materials and techniques, indoor use of chemicals, and the ways in which buildings are ventilated, used, and maintained. Fourth, understanding IAQ as a substantial driver of total exposure is relatively new (15, 16). Finally, we note that the issue of HAP from solid fuels has, until recently, been under-recognized—a trend that shows signs of reversing, from the standpoint of both research

Disability-adjusted life year (DALY): a combined metric of morbidity and mortality; one DALY represents the loss of one year of full, healthy life

Ambient air pollution (AAP): contaminants in outdoor air arising from a variety of sources—transportation, industry, power generation, etc.

Concentration: the mass of a pollutant per volume air, typically measured in a microenvironment

Household air pollution (HAP): arises from the combustion of solid fuels, like wood, dung, coal, and crop residues, for cooking and other end uses

Solid fuels: wood, dung, grass, coal, crop residues, etc.; also referred to as biomass fuels

Emissions: the rate of release of pollutants into the environment, typically per kilogram fuel or per unit time

and policies to mitigate exposure. This reversal has been slow, however, with the absolute number of individuals exposed to HAP remaining relatively static over time, despite the substantial and disproportionate elevation in exposure and the substantial energy access and equity issues.

Indoor air quality/indoor air pollution (IAQ/IAP): air quality within the built environment, including in homes, businesses, workplaces, vehicle cabins, and other places where people spend time

$PM_{2.5}$: particulate matter with an aerodynamic diameter of 2.5 μm or less

In this review, we focus on describing IAP and its health impacts. We distinguish between HAP in developing countries and IAP in developed countries, a division mirrored in the scientific literature on IAQ, science, pollution, and chemistry. The term household air pollution was coined (17) to draw a distinction from the broader indoor air quality/indoor air pollution (IAQ/IAP) literature and as an acknowledgment that much household combustion of solid fuels happens in and around the home, not just indoors; that much of the byproducts of this combustion ends up outdoors, impacting ambient air quality; and that chimneys alone—which may reduce concentrations of indoor air pollutants—do not necessarily resolve the fundamental problem of unclean, inefficient combustion.

The contrast between these contexts can be stark. The use of solid fuels as a source of household energy impacts approximately 3.8 billion people, primarily in LMICs, and results in high exposures to HAP, typically assessed by measurement of particulate matter with an aerodynamic diameter of 2.5 μm or less ($PM_{2.5}$) and/or carbon monoxide (CO). Exposure to HAP results in between 2 and 4 million deaths per year. HAP is often experienced in the home, but exposure also occurs outside of the home (from emissions that escape the indoor environment or from outdoor cooking) and downwind of original emissions.

Non-HAP IAP arises from a variety of sources and likely impacts nearly everyone on the planet. In many regions of the developed and developing world, indoor measurements of fine particulates reveal high infiltration of ambient pollution (18–20). Within the built environment—an area often perceived as clean—numerous factors contribute to degraded IAQ. Exposure sources in these contexts are manifold and include, for example, cooking (with clean or unclean fuels); cleaning; off-gassing from furniture, carpets, and electronic equipment; chemical product use and storage indoors; and chemical reactions (21–23). The concentration of indoor species and exposure to them is influenced by ventilation, other building characteristics, and occupant behaviors. The study of the impacts of these indoor sources on human health and productivity is an ongoing area of concern, with most work being performed in the United States and Europe.

This review summarizes IAP science, with a focus on pollution of indoor origin and its impacts on health. It begins with an exploration and description of the current burden of disease from air pollution exposure and continues with a discussion of measurement techniques and estimates of emissions, exposures, and health effects. We discuss key measurement and mitigation strategies for each context described above, noting areas of commonality and divergence. Additionally, we attempt to identify areas of common interest to both types of IAP; research in each subfield has diverged in recent years. We note that the difference in these types of pollution is not necessarily easily reconcilable, but that the distinct disciplines likely could benefit from approaches taken by the other and by continued dialogue between experts.

AIR POLLUTION AND THE GLOBAL BURDEN OF DISEASE

In the introduction, we stated the somewhat shockingly large numbers associated with exposure to air pollution from both ambient and household sources. These burden of disease estimates, which are generated yearly, have varied substantially in recent years and warrant description to understand what they do and do not include and how they were derived (see the sidebar titled The Global Burden of Disease and Comparative Risk Assessment). For the purposes of this discussion, we focus primarily on particulate pollution, which dominates the air pollution–related burden of disease.

THE GLOBAL BURDEN OF DISEASE AND COMPARATIVE RISK ASSESSMENT

The GBD exercises, which seek to quantify the extent and impact of disease and injuries and associated risk factors for every country in the world, began in the 1990s. Risk factors, in this context, refer to particular activities, behaviors, or exposures associated with disease and death and range from smoking cigarettes to unsafe sex to high salt diets. Since the inception of GBD activities, the effort has evolved substantially, and now results in estimates of disease burden yearly in terms of morbidity and mortality for 369 diseases and injuries and 87 risk factors, for two sexes, and for 204 countries and territories (28, 29). The activity is housed at the Institute of Health Metrics and Evaluation (IHME) at the University of Washington.

Since 2004, AAP burdens have been estimated for exposure to particulate air pollution and ozone and HAP burdens have been estimated for exposure to particulates. Methods for estimating the burden have evolved substantially in the intervening years, leading to changes in burden estimates that may be attributable to changes in actual exposures, changes in GBD estimation methodology, addition of associated health endpoints, and/or better and more resolved exposure assessment.

There are five fundamental inputs to burden of disease calculations (24, 25): (*a*) the diseases or outcomes for which there is sufficient and strong evidence to support association with a specific risk factor or exposure; (*b*) background rates, typically at the country level, for each of these outcomes; (*c*) time- and location-resolved exposures; (*d*) selection of a counterfactual level, that is, a health-based "ideal" exposure; and (*e*) functions relating exposure and disease, known as exposure-response or concentration-response functions.

Outcomes Associated with Air Pollution Exposure

In GBD, any relationships between exposure and disease that meet the criteria of convincing or probable evidence, as defined by the World Cancer Research Fund grading system (26), are eligible for inclusion. Convincing evidence includes multiple epidemiological studies [prospective studies or randomized controlled trials (RCTs)] showing consistent, biologically plausible associations and little or no contrary findings. Probable evidence is based on epidemiological studies that are consistent and biologically plausible, but may be of insufficient duration, number, or sample size and/or have issues with incomplete follow-up.

In the 2019 GBD, $PM_{2.5}$ pollution exposure was associated with numerous health endpoints, including lower respiratory infection in children and adults and five additional diseases in adults: chronic obstructive pulmonary disease (COPD), ischemic heart disease, lung cancer, stroke, and type 2 diabetes mellitus (T2DM). HAP is also associated with cataracts. All particulate air pollution, regardless of source, is associated with low birthweight and gestational age (BWGA; a substantial risk factor in its own right), which is in turn associated with 11 health endpoints: diarrheal diseases, lower respiratory infections, upper respiratory infections, otitis media, meningitis, encephalitis, neonatal preterm birth, neonatal encephalopathy, neonatal sepsis, hemolytic disease and other neonatal disorders. BWGA and T2DM were added to the burden of disease attributable to particulate air pollution exposure in recent assessments, as evidence for these linkages improved.

Exposure Estimation in the Global Burden of Disease

Exposure estimation for HAP and AAP is a complex process involving fusing multiple scales of data to derive global estimates that result in highly spatially and temporally resolved estimates for nearly all populated points on the planet. For AAP, satellite-derived aerosol estimates, ground-based measurements, chemical transport models, emissions inventories, and population

and land-use data are utilized (2, 27). For HAP, the proportion of individuals using solid cooking fuels is estimated; this is then mapped to actual exposures for men, women, and children based on existing ratios between measurements on respective groups and statistical models relating exposure levels with the type of fuel, socioeconomic indicators, and the like. Details on techniques used in GBD estimation of time- and location-resolved exposures and health impacts are available in the appendices to the recent GBD risk assessment papers (28, 29).

Counterfactual exposure: in the context of GBD, an exposure that allows an ideal health state

Exposure-response functions: functions that relate exposure with disease; also referred to as exposure-response curves

Counterfactual Exposures

Counterfactual exposures to particulate air pollution in the GBD are based on theoretical minimum risk exposure levels (TMRELs). TMRELs are exposures that present a minimal risk to exposed populations; using these values allows estimation of a hypothetical population (nearly) free of disease.

For some exposures, like active smoking, a TMREL of 0 would be reasonable; for others, like air pollution, a minimum level is set that (*a*) acknowledges some background exposure and (*b*) represents current scientific understanding of risk at very low levels. For particulate air pollution, this level is a uniform distribution between the minimum and fifth percentile of observed concentrations (between 2.4 and 5.9 $\mu g/m^3$). These values are the mean of the minimum and fifth percentiles of exposure distributions from outdoor air pollution cohort studies conducted in North America.

Concentration- and/or Exposure-Response Functions

The risk of each disease at a given pollution exposure is estimated using exposure-response functions. In the most recent version of the GBD, IHME transitioned from integrated exposure-response (IER) curves (30) to meta regression-Bayesian, regularized, trimmed (MR-BRT) splines (31). The IERs, which were used between 2010 and 2019, are referred to as integrated because they included data from multiple sources of particulate air pollution, namely AAP, HAP, secondhand smoke, and, anchoring the curves on the high end of the exposure range, active or mainline tobacco smoke.

The MR-BRTs leverage recent measurements around the world, including from Chinese cohort epidemiological studies that provide sufficient range on the high end of exposures to drop active tobacco smoking from the list of sources used to fit the curves. This process is also simplified; it now uses shape-constrained splines with three knots instead of a power function. Finally, the counterfactual used by GBD is not built into the model-fitting process and thus can be altered as needed. Both the IERs and the MR-BRTs assume equitoxicity of particles across source categories.

Figure 1 depicts the MR-BRT curves for five outcomes (32). These curves are supralinear; that is, the marginal benefit of an exposure reduction at higher levels is relatively small, as the curves are flat. Incremental decreases in PM exposures at the low end of the curve provide the most substantial risk reductions.

Relative risks derived from exposure-response functions are used to estimate the population attributable fraction for each disease, which is the fraction of the background disease rate that is attributable to $PM_{2.5}$ pollution (rather than, say, high cholesterol). The difference between the disease attributable to $PM_{2.5}$ at measured versus counterfactual exposure levels is the modeled estimate of the burden of disease.

To avoid double counting of disease burdens for individuals exposed to both AAP and HAP, GBD estimates the burden of exposure to ambient $PM_{2.5}$ and estimates the additional health burden due to cooking with solid fuels. Put another way, the total exposure (ambient plus household) is used to estimate burden; the total burden is then apportioned into ambient and household components based on the fraction of the exposure coming from each source. Contributions of HAP to AAP (discussed below) are attributed to the AAP burden.

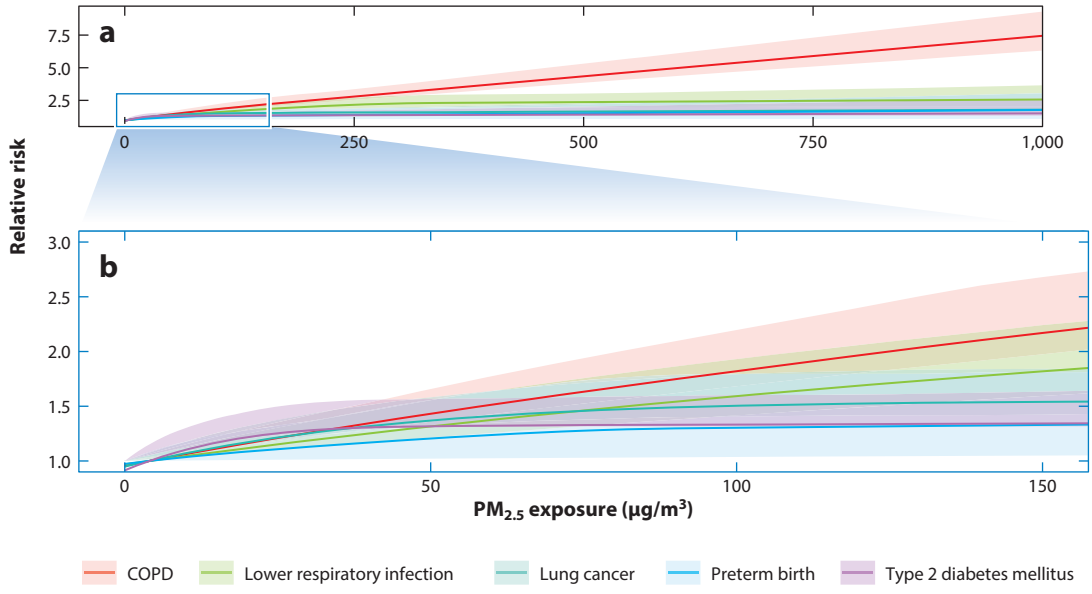

Figure 1

MR-BRT curves for $PM_{2.5}$ exposure. Panel *a* depicts the curves across an average annual exposure range spanning from 0 to 1,000 µg/m^3; panel *b* zooms in on 0 to 150 µg/m^3. The colored lines represent individual diseases associated with $PM_{2.5}$ exposure; the corresponding shading represents 95% uncertainty bounds. Abbreviations: MR-BRT, meta regression-Bayesian, regularized, trimmed; COPD, chronic obstructive pulmonary disease; $PM_{2.5}$, particulate matter with an aerodynamic diameter of 2.5 µm or less.

Non–Household Air Pollution Indoor Air Pollution in the Global Burden of Disease

Given the elements required for inclusion of specific exposure-outcome pairs into the GBD, it is perhaps not surprising that indoor air pollutants beyond $PM_{2.5}$ exposures arising from HAP are not currently included in burden estimates. A recent review of IAQ studies in the developed world has pointed out that, even for these relatively well-studied countries, limited measurements exist for a variety of species, with methodological inconsistencies between studies (33). Associations with health effects are perhaps more robust, but without more comprehensive understanding of emissions and exposures in indoor environments—and their spatial and temporal distribution—estimates in the style of the burden of disease are likely to remain difficult.

This has not stopped some attempts at quantifying the burden of IAQ in the developed world (34–36), however. These methodologies have not yet been applied globally to the best of our knowledge and appear somewhat inconsistent with the methodologies employed by GBD. The implication of this omission, given the paucity of global exposure data, is likely an underestimate of the impact of the indoor environment on health globally above that of HAP. Given global trends of rapid urbanization, changes in building practices, and availability of chemicals that may degrade IAQ, it is possible that households whose exposure is dominated by smoke arising from solid fuel used for cooking also experience a burden from these other, more modern indoor air concerns.

POLLUTANTS OF CONCERN

IAQ is complex and driven by numerous pollutants from a range of sources and pathways (**Figure 2**), in which chemical, biological, and physical contaminants eventually become a portion of the total indoor environment (10, 37). IAQ is influenced by three major factors: (*a*) outdoor

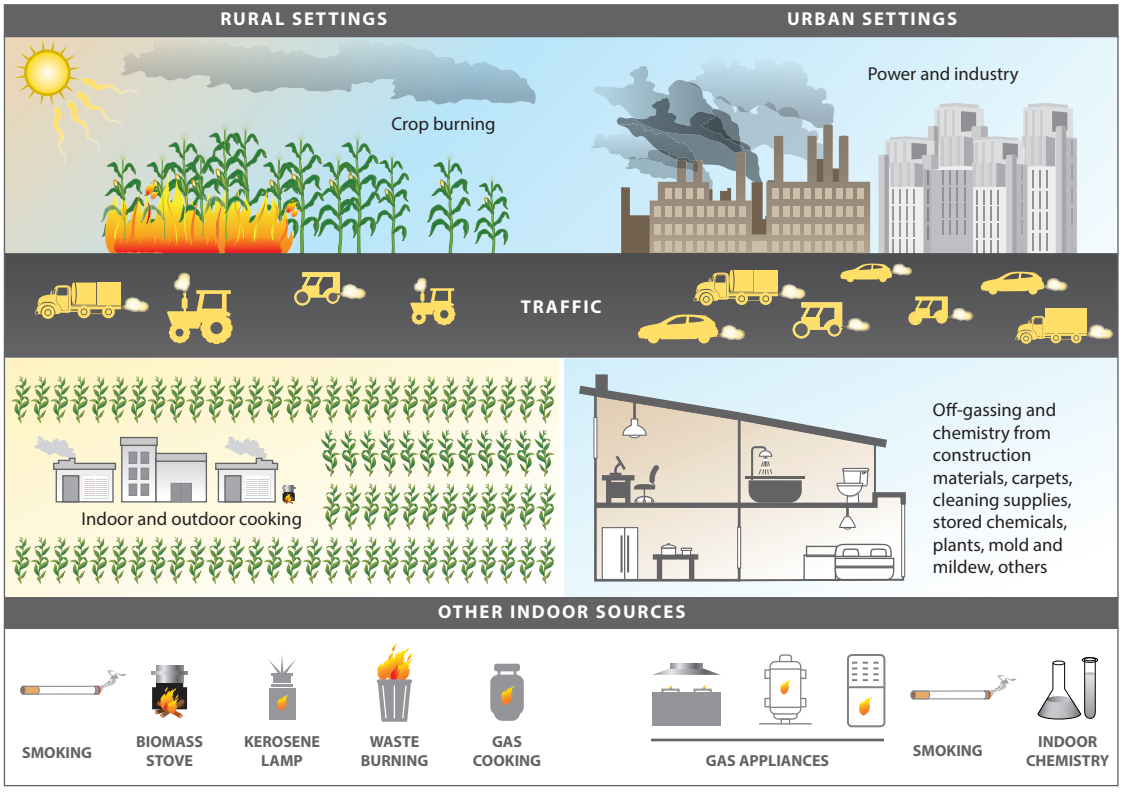

Figure 2

Common sources of indoor air pollution in rural (*left*) and urban (*right*) settings (37, 111). Sources do not vary dramatically between rural and urban contexts, but the magnitudes of emissions are different; for instance, cooking emissions in urban settings tend to be much lower than in rural settings, where biomass combustion dominates. Similarly, the influence of traffic on exposure exists in both settings, but the magnitude of exposure may vary due to individuals' proximity to and frequency of contact with traffic and traffic-related air pollution.

air quality, (*b*) indoor human activities (like cooking, cleaning, or smoking), and (*c*) building and construction materials (38–40).

Important and frequently investigated indoor air pollutants include particulate matter (PM) (including asbestos and fibers), volatile inorganic compounds (CO, CO_2, NO_x, O_3), volatile organic compounds (VOCs; benzene, toluene, ethylbenzene, xylenes, naphthalene, formaldehyde, trichloroethylene, a-pinene, limonene), and biological pollutants (allergens, fungi, bacteria, and viruses). We discuss each briefly below. References 23, 41–43 provide additional detail on the pollutants, their interactions, and their levels.

PM is a complex mixture of solid particles and liquid droplets found in the air. These particles come in many sizes and shapes and can be made up of hundreds of different chemicals (44). Two size classes have been typically measured as part of indoor and outdoor monitoring campaigns: PM_{10} (PM with an aerodynamic diameter of 10 μm or less) and $PM_{2.5}$. Indoor PM may come from outdoor sources (e.g., construction sites, roads, fields, smokestacks, or fires), indoor combustion activities (e.g., the use of stoves, fireplaces, ovens, heaters, and chimneys), and environmental tobacco smoke (ETS). Indoor PM may also form indoors through the reaction between O_3 and some VOCs (e.g., terpenes). PM can also undergo numerous transformations indoors, including

coagulation, vapor deposition onto smaller aerosols, and evaporation of VOC aerosols into the gas phase. Similarly, PM can be removed from the indoor environment by deposition, interception, settling, and exfiltration.

Fibers, such as fiberglass and asbestos, are sometimes included in this group (37). Asbestos is a mineral fiber that occurs naturally in rock and soil and has been widely used in a variety of materials and products (e.g., building material, friction products, heat-resistant fabrics, packaging, insulation, gaskets, and coatings) and as a fire-retardant (45).

CO is an odorless, colorless, toxic gas emitted from incomplete combustion processes. Sources of indoor CO emissions include ETS; unvented cooking and heating devices; leaking chimneys and furnaces; back drafting or incomplete combustion in furnaces, gas water heaters, wood stoves, and fireplaces; generators and other gasoline-powered equipment; vehicle exhaust from attached garages, nearby roads, or parking areas; poorly adjusted or maintained combustion devices; and improperly sized, blocked, disconnected, or leaky flues (22, 40, 46, 47).

Nitrogen oxides (NO_x) are a mixture of gases that are composed of nitrogen and oxygen. Two of the major and most toxicologically significant NO_x are nitrogen monoxide (NO) and nitrogen dioxide (NO_2); both are associated with combustion sources. Indoor NO_x levels can be strongly influenced by infiltration from outdoor air, in particular when indoor spaces are a short distance from roadways or when high-density industrial areas are nearby. Combustion appliances (e.g., open-flame stoves, ovens, heaters), burning of tobacco, and fireplaces can also contribute to NO_x emissions (37, 40, 47).

O_3 is a powerful oxidizing agent mainly produced by photochemical reactions of O_2, NO_x, and VOCs in the atmosphere. The main sources of indoor O_3 come from infiltration of O_3-containing outdoor air and the operation of some electrical devices (e.g., photocopiers), disinfecting devices, air-purifying devices, and other office electronics (40, 48–51).

VOCs are organic chemical compounds with a low boiling point (ranging from 50/100°C to 240/260°C) that can evaporate under normal indoor temperatures and pressures (52, 53). Indoor VOCs are often generated from building material; the number and types of VOCs identified indoors are growing as new materials are being used in construction and interior design (37, 40). VOCs can also be identified from human activities (e.g., cooking, smoking, the use of cleaning and personal care products, and the burning of scented candles and incense), indoor chemical reactions, and the penetration of outdoor air through infiltration and ventilation systems (54–58). Typical VOCs found indoors include benzene, toluene, ethylbenzene, xylenes, naphthalene, formaldehyde, trichloroethylene, limonene, and alpha-pinene (37, 59), among others.

ETS or secondhand smoke is composed of a mixture of both sidestream smoke, the smoke released from burning the end of a cigarette, and exhaled mainstream smoke, the smoke exhaled by the smoker (60). Burning a cigarette results in more than 4,000 measurable chemical compounds; many are toxic and carcinogenic (61, 62). Sidestream smoke is many times more toxic than mainstream smoke (60, 63). It has a similar chemical composition but tends to linger in the environment for 1.5–2 hours. It thus may increase both exposure duration and potential penetration into lung tissue and body cells (60, 63). Thirdhand smoke—smoke that remains in the environment long after the cigarette has been extinguished—has also been identified as a source of potential exposure (64, 65). ETS exposure is further complicated by the smoking habits and frequencies of active smokers sharing the space.

Radon is a naturally occurring gas that originates from uranium ores in the ground. Radon enters indoors primarily through the entry of radon-bearing soil gas through basements and floors and may build up to high concentrations in the air (42). Radon is radioactive and decays in the air, resulting in other radioactive components that can attach to tiny dust particles, be inhaled, and adhere to the lining of lungs, eventually leading to lung cancer (42). Effects of radon

exposure are cumulative, and radon is estimated to cause between 83,000 (29) and 92,000 (66) deaths worldwide yearly.

Biological pollutants are or were living organisms. They promote poor IAQ and can travel through the air and are often invisible (67). Common indoor biological pollutants include animal dander (i.e., minute scales from hair, feathers, or skin), dust mite and cockroach parts, infectious agents (i.e., bacteria or viruses), and pollen.

INDOOR AIR POLLUTION IN THE DEVELOPED WORLD

Exposure

Exposure to IAP has important health implications; in developed countries, adults spend 80%–90% of their time indoors (5, 37, 68, 69); an additional 6% of daily time may be spent inside for transportation (38). Characterizing exposure to IAP is challenging due to the diversity and variability of air pollutants in the indoor environment, as well as the complex physical, chemical, social, and behavioral factors and processes involved in determining IAQ. **Table 1** provides indoor concentrations and/or exposures to common pollutants in developed countries from selected studies, as well as United States Environmental Protection Agency (US EPA) or World Health Organization (WHO) standards.

The average exposure in an indoor space is directly related to room size and ventilation rate (60), among other factors. Season and outdoor temperature impact physiochemical processes, ventilation, and pollution sources (33). For example, during the winter, indoor concentrations of NO_2 can be twice the outdoor levels due to the residential use of gas-fueled heating appliances and reduced natural ventilation (60).

IAP exposures vary across different socioeconomic groups. Women are at a higher risk of VOC exposure than men because they generally perform more household cleaning tasks and have more exposure to cleaning products (60). Building characteristics (i.e., building quality, airtightness, volume, and ventilation) and occupant behavior (i.e., cooking and cleaning) are important determinants of IAQ, and also differ across socioeconomic groups, leading to variations in exposure (33). A scoping review of exposure to IAP across socioeconomic groups in high-income countries found significantly higher concentrations of PM, NO_2, and VOCs in households of lower socioeconomic status (SES) (33). Conversely, elevated radon levels were observed in households with higher SES (70, 71). Possible reasons include (*a*) higher SES households lived in large dwellings with larger floor areas (72); (*b*) higher internal temperature increased the pressure gradient and led to elevated rates of radon-bearing soil-gas entry into the homes (73); and (*c*) the energy efficiency features (which lower the rates of background air exchange with the outdoors) in higher SES homes may cause radon to accumulate (74) rather than be ventilated to the outdoors, either intentionally or through leakage.

Assessing Exposures

Measurement within homes is the most common method to estimate indoor PM, NO_2, radon, and VOC exposure. Although monitoring devices offer more reliable and objective results than self-reported data, it can be challenging to determine actual exposure levels given the way that occupants interact with monitoring instruments (75) and the single exposure route (i.e., inhalation) being measured. Furthermore, stationary monitors in homes may not reflect the time-activity patterns of residents and may thus misestimate exposure. In addition, sufficient and representative sample sizes are hard to achieve when monitoring IAQ, especially for residential buildings, due to the great variability in building characteristics and occupant behaviors, outdoor pollution levels, the high compliance requirement of residents, and associated costs (33, 72, 76, 77).

Table 1 Indoor concentrations of common pollutants in developed countries

Pollutant	Indoor concentration	Standards
Particulate matter with an aerodynamic diameter of 2.5 μm or less ($PM_{2.5}$)	Kitchen: 25–1,526 $\mu g/m^3$ Bedroom: 13–27 $\mu g/m^3$ Office: 10–44 $\mu g/m^3$ School: 3–23 $\mu g/m^3$ Shopping center: 74–164 $\mu g/m^3$ See References 106, 171–174	Indoor particulate matter (PM) levels have the potential to exceed outdoor PM levels and the United States Environmental Protection Agency (USEPA) National Ambient Air Quality Standards (NAAQS). However, less is known about the specific impacts of indoor PM on health.[a]
Carbon monoxide (CO)	Average indoor level without gas stove: 0.5–5 ppm Levels near properly adjusted gas stoves: 5–15 ppm Levels near poorly adjusted gas stoves: >30 ppm[a]	No standards for CO have been agreed upon for indoor air. The NAAQS for outdoor air are 9 ppm (40,000 mg/m^3) for 8 h and 35 ppm for 1 h.[a] World Health Organization (WHO) guidelines relevant to typical indoor exposure are recommended as follows: 100 mg/m^3 for 15 min, 35 mg/m^3 for 1 h, 10 mg/m^3 for 8 h, and 7 mg/m^3 for 24 h.[b]
Nitrogen oxides (NO_x)	Home: 5–7 $\mu g/m^3$ Office: 16–18 $\mu g/m^3$ Shopping center: <30 $\mu g/m^3$ See References 106, 173, and 175	No standards have been agreed upon for NO_x in indoor air. The American Society of Heating, Refrigerating and Air-Conditioning Engineers and the NAAQS list 0.053 ppm as the average 24-h limit for NO_2 in outdoor air.[a] A 1-h indoor NO_2 guideline of 200 $\mu g/m^3$ and an annual average indoor NO_2 guideline of 40 $\mu g/m^3$ are recommended by WHO.[b]
Ozone	Home: 0.9–21.4 ppb School: 0.9–31 ppb Office: 2.1–8.0 ppb See Reference 176	Health Canada recommends a residential maximum exposure limit of 40 $\mu g/m^3$ (20 ppb) ozone, based on an averaging time of 8 h.[c]
Benzene	Home: 0.7–4.4 $\mu g/m^3$ Office: 1.4–5.5 $\mu g/m^3$ Shopping center: 2.5–48 $\mu g/m^3$ See References 106, 175, 177–181	No safe level of exposure is recommended.
Toluene	Home: 3–20 $\mu g/m^3$ Office: 6–32 $\mu g/m^3$ Shopping center: 15–164 $\mu g/m^3$ School: 1.8 $\mu g/m^3$ See References 106, 173, 175, 177–179, and 181	Toluene is not a confirmed carcinogenic substance.
Ethylbenzene	Home: 3–20 $\mu g/m^3$ Office: 6–32 $\mu g/m^3$ Shopping center: 15–164 $\mu g/m^3$ School: 1.8 $\mu g/m^3$ See References 106, 177–179, and 181	The National Institute of Occupational Safety and Health recommended exposure limit is 435 mg/m^3 (for an 8- to 10-h time-weighted-average exposure).
Xylenes	Home: 3.1 $\mu g/m^3$ Office: 2.2–16 $\mu g/m^3$ Shopping center: 1.3–74 $\mu g/m^3$ School: 0.3 $\mu g/m^3$ See References 106, 173, 175, 177–179	The National Institute of Occupational Safety and Health recommended exposure limit is 435 mg/m^3 (for an 8- to 10-h time-weighted-average exposure). Health Canada recommends an indoor environments short-term (1 h) exposure limit of 7,000 $\mu g/m^3$ (1,700 ppb) and long-term (24 h) exposure limit of 150 $\mu g/m^3$ (36 ppb).[c]

(*Continued*)

Table 1 (*Continued*)

Pollutant	Indoor concentration	Standards
Formaldehyde	Home: 7.7–30 $\mu g/m^3$ Office: 8–17 $\mu g/m^3$ School: 9–17 $\mu g/m^3$ See References 106, 171, 173, 175, 177, and 182	No safe level of exposure is recommended by USEPA.[a] WHO recommends a short-term (30 min) guideline of 0.1 mg/m^3.[b]
Naphthalene	Home: 3–26 $\mu g/m^3$ See References 178, 182, and 183	WHO recommends an annual average guideline of 0.01 mg/m^3.[b]
Trichloroethylene	Home: 0.3–0.6 $\mu g/m^3$	No safe level of exposure is recommended.
Limonene	Home: 32 $\mu g/m^3$ Office: 19 $\mu g/m^3$ School: 11 $\mu g/m^3$ See References 173, 175, 177–179, 181–183	No limits have been set.
Alpha-pinene	Homes: 11–32 $\mu g/m^3$ Libraries: 10–30 $\mu g/m^3$ Office: 6.3 $\mu g/m^3$ School: 1.5 $\mu g/m^3$ See References 173, 175, 177, 179, 181–183	No limits have been set.
Radon	Home: 15–259 Bq/m^3 School: 56–889 Bq/m^3 Office: 54.9 Bq/m^3 See References 184–187	WHO proposes a reference level of 100 Bq/m^3 for 1 year to minimize health hazards due to indoor radon exposure.[b]

[a]See the United States Environmental Protection Agency Indoor Air Quality homepage: **https://www.epa.gov/indoor-air-quality-iaq**.
[b]See *WHO Guidelines for Indoor Air Quality: Selected Pollutants* (188).
[c]See Health Canada's indoor air quality guidelines (**https://www.canada.ca/en/services/health/publications/healthy-living.html**).

Many studies estimate IAQ using models. Shrubsole et al. (78) and Liao et al. (18) used building simulation software to predict IAP exposures from indoor and outdoor sources across different building archetypes and scenarios. Rosofsky et al. (79) estimated infiltration using an air exchange model and spatial data on building properties and metrological conditions. Despite the uncertainties and assumptions associated with the inputs and outputs of the models, modeling techniques allow for extensive estimates of exposure and predictions under future scenarios (33, 78, 80). Such models could be used, in theory, to estimate indoor air burdens for a county, region, country, or the globe.

Health Impacts

Cumulative health impacts from inhalation in US residences of IAP are estimated at 400–1,100 DALYs[2] lost annually per 100,000 persons (34). In EU-26 countries, an annual loss of 2.1 million DALYs is associated with indoor and outdoor originating pollutants, with more than half (1.28 million DALYs) caused by indoor exposure to outdoor air pollution and the remaining

[2]A DALY is equivalent to one year of lost healthy life. DALYs thus occur when individuals die prematurely or when they live with a disability or disease that impacts their life. DALYs are computed as the sum of years of life lost, which estimate premature mortality, and years lost to disability, which quantify lost healthy life due to suboptimal health. For a brief description of DALYs, see Salomon (81).

0.74 million DALYs caused by indoor source pollutants (82). The pathogenesis of IAP exposure results from a combination of effects at different levels (83). Gaseous air pollutants' damage to human tissue depends on solubility in water, concentration, ability to oxidize tissues, and susceptibility of the exposed person (84). PM is usually classified by its sizes. Large particles (e.g., PM_{10}) may affect mucous membranes and the upper airways, causing cough and lacrimation. Fine particles (e.g., $PM_{2.5}$ and $PM_{0.1}$) have greater systemic toxicity, as they easily enter the alveoli and pass through the alveolar-capillary membrane and are carried through the bloodstream (83, 84). In addition to particle sizes, their structure and composition [e.g., metals and polycyclic aromatic hydrocarbons (PAHs)] may also be responsible for the tissue damage on contact (84).

In addition to causing direct damage, exposure to IAP can lead to inflammation with systemic effects, trigger oxidative stress pathways, and activate oxidative stress response genes (83–85). Moreover, epigenetics mediate genetic and physiologic responses to air pollution and are, therefore, an important cause of susceptibility to pollution-related health effects (84, 86, 87).

Common indoor air pollutants and their effects on human health are summarized in **Table 2**. Illnesses caused by indoor environmental factors are commonly divided into two categories: sick building syndrome (SBS) and building-related illness (BRI) (40). SBS refers to a group of symptoms that are linked to the physical environment of specific buildings (88). SBS symptoms caused by IAP can be divided into four categories: (*a*) mucous-membrane irritation (e.g., eye, throat, and nose irritations); (*b*) neurotoxic effects such as headaches, irritability, and fatigue; (*c*) asthma and asthma-like symptoms including chest tightness and wheezing; and (*d*) issues such as skin and gastrointestinal problems (3, 40). Common BRI illnesses include Legionnaire's disease, hypersensitivity pneumonitis, and humidifier fever (89). Indoor environmental pollutants can cause BRI symptoms via four major mechanisms: (*a*) immunologic, (*b*) infectious, (*c*) toxic, and (*d*) irritant (90).

The respiratory system is the primary target of IAP exposure. Although findings are less robust than those from research on the association between outdoor pollution and respiratory health, IAP increases the risk of childhood acute lower respiratory infections (91). There is consistent evidence suggesting that elevated indoor PM concentrations are associated with higher rates of asthma attacks or asthma morbidity among populations with asthma. Indoor exposure to elevated NO_2 is also associated with greater asthma morbidity (92–95). The link between IAP exposure (i.e., $PM_{2.5}$, NO_2, and allergens) and COPD morbidity is also clear in populations living in developed countries, even at relatively low pollution concentrations (96, 97).

Exposure to IAP may affect the natural defense of the body against airborne viruses, making people more likely to contract viral diseases such as COVID-19 (98–100). It has been recognized that short-range inhalation predominates the transmission of SARS-CoV-2 and the transmission occurs mostly indoors in poorly ventilated spaces (101). This transmission pattern may be due to the local climatic conditions with low temperature, mild daytime temperature range, and low humidity that would favor the transmission of viruses, as a study demonstrated in China (102).

Exposure to IAP also increases the risk of specific cardiovascular diseases including ischemic stroke, myocardial infarction, cardiac arrhythmia, heart failure, and atrial fibrillation due to inducing oxidative stress, systemic inflammation, increased blood coagulability, and autonomic and vascular imbalance (103, 104). Moreover, CO in the indoor air environment is likely to decrease tissue oxygenation through carboxyhemoglobin production, which results in a high impact on cardiovascular function (105).

Mitigating Exposures

Overall, improving ventilation is the easiest method to prevent accumulation of IAP. Poorer quality housing and/or occupant behavior may decrease dispersion via inefficient ventilation (33).

Table 2 Health effects of selected indoor air pollutants

Pollutant	Health effects	Studies
Particulate matter with an aerodynamic diameter of 2.5 μm or less	Respiratory symptoms and diseases including eye, nose, throat, and bronchial irritation, asthma, fibrosis, anthracosis, and lung cancer; cardiovascular diseases	189, 190
Carbon monoxide	Mortality; emergency department visits; cardiovascular diseases	190, 191
Nitrogen dioxide	Exacerbate symptoms of respiratory illness; fatal pulmonary edema and pneumonia at high concentration; bronchitis, bronchiolitis, and pneumonia at lower concentrations	60, 92–95, 192
Ozone	DNA damage, lung damage, asthma, decreased respiratory functions	49, 193
Environmental tobacco smoke	Contains many carcinogens; lung cancer, breast cancer, leukemia, lymphoma, and brain tumors in children; nasal and sinus diseases; ischemic heart disease	194–201
Radon	Lung cancer	202, 203
Volatile organic compounds		
Benzene	Carcinogenic and genotoxic under chronic exposure, blood illness, neurological and reproductive problems	189, 190
Toluene	Long-term effect unclear; increased risk of developing asthma and other respiratory conditions; impact on central nervous system	37
Ethylbenzene	Ethylbenzene is a carcinogen. Acute effects include eye and throat irritation and dizziness; long-term effects are unclear.	37
Xylenes	Xylenes are suspected carcinogens. Acute effects include eye and throat irritation, headache, and nausea; long-term effects include issues in respiratory, gastrointestinal, and central nervous systems, lungs, kidneys, heart, and the reproductive system, and increased risks of leukemia, non-Hodgkin's lymphoma, and colon/rectum cancer.	204
Formaldehyde	Formaldehyde is a carcinogen. Respiratory symptoms include cough, sputum production, asthma, colds, chronic bronchitis, and respiratory cancer.	204–208
Naphthalene	Acute intoxication can induce hemolytic anemia and cataracts; long-term effects are unclear.	204
Trichloroethylene	Trichloroethylene is a carcinogen. Acute effects include impacts on the nervous system at concentrations of ~270 mg/m^3. Long-term exposure may cause liver, kidney, and bile duct cancer and non-Hodgkin's lymphoma.	188, 204
Limonene	Eye or skin irritation; long term effects are unclear; there is no evidence of carcinogenicity or genotoxicity.	190, 204
Alpha-pinene	Acute exposure to high concentrations can produce irritation and inflammation; long-term effects are unclear.	204
Biological pollutants		
Molds	Allergic bronchopulmonary aspergillosis, hypersensitivity pneumonitis, and worsening of preexisting asthma; lower respiratory track disease	60, 209–212
Animal dander	Allergen, asthma	213–216
Dust mites	Allergic diseases	217

Furthermore, new designs seeking to reduce energy consumption lead to airtight construction, which can substantially reduce natural ventilation and may thus increase concentrations indoors (37, 106, 107). Thus, the incorporation of adequate ventilation following energy-efficient building modifications is necessary to reduce or prevent high IAP exposures, especially among lower SES households (33).

Ventilation alone cannot prevent all IAP exposure and associated health effects in a cost-effective or technically feasible manner. Current solutions for air purification include combinations of air filtration, ionization, activated carbon absorption, ozonation, and photocatalysis (53, 108). These processes can be integrated into central ventilation systems (in ducts) or used in portable air purifiers designed for limited spaces (108). Emerging air treatment methods include membrane separation, enzymatic oxidation, botanical purification, and biofilters (37, 53, 108). In developed countries, portable air cleaner devices may be effective for reducing IAP exposures (18, 109, 110). Community-level strategies [e.g., educational campaigns (111) and stove exchange programs (112, 113)], bolstered by policy [e.g., smoke-free legislation (114, 115)], have also been used to reduce sources of pollution in developed countries.

HOUSEHOLD AIR POLLUTION PRIMARILY IN THE DEVELOPING WORLD

Introduction

More than 20 years into the twenty-first century, one of the oldest sources of air pollution remains the most dominant: Approximately 3.8 billion people (approximately the total world population in 1970) lack access to clean energy for cooking, heating, and other basic needs. Despite national policies and advocacy by global bodies, solid fuels remain by far the dominant energy source for cooking in LMICs and for heating in much of East and Central Europe and Northern China (116–118).

The use of wood as a fuel for fires is, in a way, as old as humanity: It has been described as the distinguishing feature between the prehuman and human state (119, 120). Along with wood, other biomass fuels—crop residues, which began to be used for heating and cooking during the agricultural revolution; coal; and dung—form the major types of solid fuel still used today by just shy of half of the global population (1).

The range of pollutants released from the combustion of these fuels, often in simple, unvented stoves, is wide and varies with fuel and stove type and combustion conditions (**Figure 3** provides an example). Species emitted include PAHs, VOCs, and hundreds of other health-damaging compounds (121–123). In 2018, the WHO and others updated their HAP database, which collects information on all available measurements made in solid fuel–using contexts. Although the database focuses on $PM_{2.5}$ and CO, two of the most commonly measured pollutants, it also includes numerous other pollutants, including PAHs and VOCs. The database contains more than 1,000 measurements from 196 studies performed in 53 countries. Kitchen levels of $PM_{2.5}$ in households using solid fuels varied widely between regions, countries, and fuel types, ranging between approximately 150 and 1,200 $\mu g/m^3$ for wood and dung (124, 125). The burden of disease associated with cooking-related HAP is large—the ninth overall risk factor for mortality globally. HAP exposure results in an estimated 2.3 million deaths yearly and 91 million DALYs—4.1% and 3.6% of all deaths and DALYs, respectively. The distribution of this burden, not surprisingly, follows the distribution of solid fuel use globally, and is most dominant in sub-Saharan Africa and South and East Asia. Age-standardized mortality rates[3] attributable to HAP in sub-Saharan Africa are 6–7 times higher than the global average (200/100,000 versus 30/100,000), and 200 times higher than high-income countries (<1/100,000). This estimate of the burden of disease does not take into account HAP's contribution to AAP—a topic to which we turn our attention next.

[3]Age-standardized rates adjust for population size and the age distribution of each country's population. By standardizing, mortality or morbidity rates can be compared between countries as though the countries had similarly structured populations.

Figure 3

Cooking with wood fuel in a mud stove in Maharashtra, India. Note the kerosene lamp in the window and the proximity of the cook to the fire and smoke. Picture by Ajay Pillarisetti, with permission.

Inside Out

HAP not only has an enormous health impact directly on those exposed to its emissions, but it also escapes outdoors and likely accounts for a large fraction of AAP globally (126). The full impact of HAP is thus composed of the exposures to HAP inside a given house and from its contribution to AAP.

Until recently, the contribution of HAP to AAP was poorly characterized and quantified. This contribution is generally quantified using either top-down or bottom-up source-apportionment methodologies (127). These distinct approaches help in reconstructing the atmospheric concentration of pollutants associated with the different emission sources. Whereas the former is based on inferential methods, the latter is predictive of source contributions and, when operated in conjunction, they may serve as an efficient tool for identifying major sources of $PM_{2.5}$ and thus support air quality management decisions. These source-apportionment studies indicate that HAP contributes to 12–31% of AAP globally (126, 128), with a larger footprint in LMICs. HAP is often a larger contributor than sources that are common indices of development, including road transport, the industrial sector, coal-fired power plants, brick kilns, and construction dust. For instance, in South Asia, studies estimate that more than half of AAP may come from use of solid fuels in households (128), whereas in high-income countries of West Europe and North America, this fraction is approximately 5–10%. Thus, the problem of HAP is confined not only to rural populations but also to all among the shared airshed.

Regardless of the fairly large spread and uncertainty in the exact amount that HAP contributes to AAP, it plays a nontrivial role in total air pollution exposure. These uncertainties arise from differences in input variables, emission inventories, and the ways that different, complex air pollution models account for chemistry and physics in the atmosphere. Furthermore, the definition of residential emissions also differs between studies; emission inventories include varying combinations of cooking, heating, and lighting emissions. Some also group commercial emissions with residential emissions. These discrepancies can be improved by utilizing updated energy service data—for instance, better data on primary and secondary fuel use for specific tasks (like boiling water, cooking food, or space heating) acquired through nationally representative surveys. Detailed characterization of emissions and comparisons of different emissions inventories enable regional comparisons. By applying different emissions inventories in different geographies, the robustness of findings can be evaluated. Such exercises are expected to increase the confidence of modeling studies. Besides, consistent and coordinated efforts in assimilating top-down and bottom-up approaches may be a significant step toward reducing the uncertainties in quantifying the contribution of HAP to AAP. However, attributing the fraction of premature mortality burden to contribution of HAP to AAP depends on additional factors including the shape of the exposure response functions used to estimate the premature mortality burden, baseline mortality rates, and the fraction of exposed population. Regardless of model or method, global source apportionment studies indicate between 0.5 and 1 million deaths from AAP may be attributed to HAP annually, in addition to the 2.3 million deaths from exposure to HAP indoors (29).

Reducing the Impact of Household Air Pollution Exposures: Shifting Paradigms

The late Professor Kirk R. Smith (129)—arguably the father of the field of HAP, the first to measure exposures of women cooking in rural villages, and the principal investigator in the first RCT of a HAP intervention—contextualized in recent years several paradigms for thinking about household energy interventions, explicitly from a health perspective. We summarize and expand upon those paradigms below.

With development comes wealth—and clean cooking. In some countries around the world—including India and China—development trajectories and urbanization are leading to gradual improvements in clean fuel access and use. As households become wealthier, so the hypothesis goes, they switch to clean fuels. Much of the information about solid fuel use—and trends in its prevalence over decadal or longer timespans—comes from national surveys administered at regular intervals, including national censuses, Demographic and Health Surveys, and other energy-related surveys. This type of transition may occur, but often occurs slowly; there is a need, given the large burden of disease, to accelerate movement toward exclusive use of clean fuels. In some regions, despite urbanization and increasing numbers of clean fuel users, rapid population growth has led to a smaller overall fraction using clean fuels. Waiting for development to do its work thus may be insufficient, especially when we have methods to reduce exposures now, rather than waiting on development or waiting for a "silver bullet" solution that may not arrive or may arrive too late to protect populations today.

Make the available clean. Decades of global programs have focused on replacing traditional biomass stoves with more efficient and cleaner burning ones—hence making the available fuel (more) clean. Among the most prominent of these many hundreds of programs were the Chinese National Improved Stove Program and the Indian National Program on Improved *Chulhas*, which installed approximately 130 million and 35 million stoves, respectively. Many of these early programs had the advantage of using the available fuel, being relatively inexpensive and made

from locally available materials. The proliferation of these stoves led to many evaluations of their effectiveness, with mixed results. Although many reduce air pollution concentrations and thus exposures, they do not do so to a level thought to result in relatively safe pollution exposures (130).

In recent decades, the focus shifted from simple stoves to highly engineered devices, so-called advanced biomass combustion stoves, that attempt to burn processed biomass more completely through improved design and fan assistance. Although many of these devices show promise in the lab (131, 132), their real-world performance has been less than ideal. A recent meta-analysis evaluated the impact of various types of interventions, including cleaner cooking interventions, on pollutant concentrations and exposures (130). None of the evaluated improved or advanced biomass interventions reached the WHO Interim Target 1 annual average $PM_{2.5}$ exposure guideline value of 35 $\mu g/m^3$. Reasons for this discrepancy between field and lab tests are manifold and include, for example, differences in fuel preparation and fuel characteristics; the presence of other traditional stoves used concomitantly with intervention; poor performance and maintainability of stoves over time; and design trade-offs that optimize for emissions reductions, sometimes at the expense of usability for village cooks. Finally, these improved or advanced stoves must burn biomass so cleanly as to not reduce pollution by half compared to the traditional stove, but instead reduce it far more—to safer levels, such as the WHO Interim Target 1 Air Quality Guideline value of 35 $\mu g/m^3$ and as evident in the exposure-response curves depicted in **Figure 1**. To date, very few of these stoves have been able to consistently perform at this level—unlike gas, electricity, and other clean fuels, which regularly achieve these exposure reductions relative to traditional stoves.

RCTs of biomass cookstove interventions—in the mode of making the available clean—have occurred in Guatemala (133), Malawi (134), Peru (135), Nepal (136), Rwanda (137), and Ghana (138). These trials have mainly had null results or shown only small health benefits. One exception is the first trial of an improved cookstove, the RESPIRE project in the Western highlands of Guatemala. Although the measured exposure reductions were significant, they did not result in a significant effect on physician-diagnosed pneumonia, but did significantly reduce severe pneumonia. Numerous explanations exist for these findings, including insufficient or unmeasured reductions in exposure, mixed use of traditional and intervention stoves, and other sources of exposure.

Make the clean available. This paradigm acknowledges the challenges of burning biomass efficiently in devices that are durable and reliable, and that cooks want to use. It also recognizes that, for many cuisines and many cultures around the world, gas and electric cooking technologies meet almost all regular needs. Under this mode of thinking, rather than trying to burn biomass cleanly—a challenging task even under ideal circumstances—the focus is on taking clean options, like liquefied petroleum gas (LPG), electricity, and piped natural gas, and making them more accessible, affordable, and available. This sort of transformation suggested by this paradigm is not unprecedented. In the 1970s, Ecuador began heavily subsidizing LPG for household use, resulting in widespread adoption, although some mixed use persists (139–141). Ecuador has further pursued a transition from LPG to induction cooking, in part due to sufficient indigenous hydropower capacity and a desire to reduce the fiscal burden of LPG subsidies. A similar success story is noted for Brazil, where LPG was promoted through a combination of market manipulations, subsidies, and widespread urbanization (142).

Perhaps the most striking ongoing household energy transition—and one of the largest to date—is occurring in India. The transition has been aided by several policy initiatives, beginning in 2015 with Pahal, which sought to stop or slow leakage of subsidized LPG from the household to commercial market. Pahal forced all consumers to purchase LPG at the market price; those eligible for a subsidy received it in their bank account. The second policy, known as "Give

it up," encouraged middle- and upper-class households to voluntarily forego their LPG subsidy in perpetuity. The recovered costs were used by the government to help offset connecting[4] new, poor households to the Indian LPG system. Approximately 10 million households gave up their connection (142). The third policy—Pradhan Mantri Ujjwala Yojana (PMUY)—sought to subsidize the cost of becoming connected to the LPG system by covering nearly all costs. PMUY-eligible consumers were required to purchase a lower-cost two-burner stove, but this purchase could be made via a no-interest loan with the LPG distributor. As of early 2022, the program claims to have connected 90 million households to the LPG system. PMUY has been followed by PMUY-2, which eased some eligibility criteria in order to ensure that all poor households were eligible for the program.

Access and connection to the LPG system is a required step in ensuring a reduction in HAP emissions and exposures. It is, however, insufficient. At least two other parameters must be met: near exclusive use of LPG or clean fuels (143) and cessation of biomass use. Audits and analysis of the PMUY program from India have shown that PMUY beneficiaries consume less fuel on average than do non-PMUY beneficiaries, and likely not enough to cover all of their household energy needs, indicating mixed use of clean and traditional fuels (144, 145). Among the many reasons hypothesized for this incomplete transition, two that occur frequently in the literature are difficulty in accessing refills and their unaffordability (145, 146). Nonetheless, the Indian program has transformed the household energy landscape in India in less than a decade and appears to be a prime example of making the clean available.

Randomized evaluations of LPG and other clean fuel interventions are fairly limited. The largest to date—the multi-country Household Air Pollution Intervention Network (HAPIN) trial (147)—is ongoing. HAPIN recruited 3,200 pregnant women in four LMICs (Guatemala, India, Peru, and Rwanda) and randomized them to an LPG stove and fuel intervention arm or to a control arm (continued traditional cooking). Preliminary results from HAPIN indicate significant reductions between control and intervention groups and between baseline and postintervention measurements in the intervention arm, with approximately 70% of samples falling below the WHO Interim Target 1 annual average $PM_{2.5}$ guideline value of 35 $\mu g/m^3$ (148). Health effects have not yet been reported for the HAPIN trial. Three other trials of an LPG intervention—in Ghana (138), Nepal (136), and Peru (149)—have null findings of the impact of the intervention on health outcomes. The Ghana trial, however, has shown impacts in exposure-response analyses on various health endpoints, including blood pressure (150), child growth (151), and birthweight (152). An ethanol RCT in Nigeria showed no effect on birthweight in intention-to-treat analyses; after controlling for covariates, there was a significant increase in birthweights in the intervention group (153). The same trial reported a significant decrease in diastolic blood pressure (154) among those with the intervention.

Cleaning up after clean cooking. In some contexts, as HAP from cooking decreases, identifying the next most important sources of exposure—potentially from indoor emissions—becomes an essential task. Additional uses of biomass stoves (often indoors, but also outdoors) include space heating, water heating, animal fodder preparation, alcohol or other drink preparation, and commercial cooking (155, 156). This focus—on end uses of energy, rather than on specific appliances and/or fuels—is an emerging area of research, and one for which additional information is

[4]In India, a connection is required to access and become a formal part of the LPG distribution system. Becoming connected typically entails a deposit to a local distributor to cover the cost of the cylinder, regulator, hoses, and paperwork. Connection costs can be a substantial barrier to poor households.

forthcoming, with the adoption of new, standardized household energy questions that specifically focus on stoves, fuels, and end uses.

INDOOR AND HOUSEHOLD AIR POLLUTION: DISTINCTIONS AND DISCOURSE

The distinctions between IAP in the developed world and HAP in the developing world may appear vast. While conducting research for and writing this review, we identified areas where one discipline can learn from the other, and some potential areas of overlapping future research that would benefit both fields.

Cooking with Gas: A Strong Source in High-Income Countries, a Strong Solution for Solid Fuel–Using Households

In many developed countries, as part of a continued push toward electrification, cooking with gas—whether natural gas or LPG—is frowned upon for health, environmental, and climate-related reasons (157, 158). This is in stark contrast with the role of LPG in LMICs, where it is seen as a relatively affordable, transportable, and broadly usable fuel for households currently using solid fuels (142, 159, 160). Even in the context of solid fuel–using homes, there is some concern that levels of NO_2 after intervention with an LPG stove may pose residual health threats (161). Of note, these levels are lower than for cooking with biomass but above health-based guidelines from WHO.

If we view this issue through an exposure apportionment lens, after identifying context-specific strong sources, we could arrive at a transitory solution to the seeming contradiction of LPG promotion. In the developed world, indoor combustion can be reasonably eliminated by replacing gas with clean, low-cost, controllable, efficient electric devices—like induction cooking surfaces. Indeed, such an approach likely makes some sense not only from a health perspective but also from an energy savings and thus climate perspective. In LMICs—where electricity infrastructure is less robust, less reliable, and unable to deliver sufficient energy for cooking—gas is an interim solution, as it has been in the developed world for more than 100 years. There is strong motivation—from health, environmental justice, and equity perspectives, among others—to provide clean fuels to households using solid fuels now. The issue of gas cooking is one where scientists focused on modern IAP and those focused on HAP would likely find fertile ground for discussion around science and measurement, risk assessment and evaluation approaches, and overall mitigation and intervention strategies.

Household Air Pollution in the Burden of Disease

The identification of HAP as a global risk factor for ill-health—and its inclusion in burden of disease estimates—was a long, multi-year process. Part of this process is outlined by Smith et al. (17) and in the annexes to the *WHO Guidelines for Indoor Air Quality* (162). Regardless, the process of identifying the strongest evidence for exposure-outcome pairs and of identifying relatively precise ways of quantifying specific exposures globally should be instructive for future attempts to quantify the regional, national, or GBD associated with non-HAP indoor air pollutant exposure. We note that some studies have already done this, but, to the best of our knowledge, no global effort has been made. As with IAP, or with tobacco smoke, there are hundreds or thousands of species that could be measured in biomass combustion smoke. Studies of HAP focused on measuring CO and PM, pollutants for which there is (*a*) evidence of health effects; (*b*) relatively easy, inexpensive, and validated measurement techniques; and (*c*) precedent for risk assessment. A similar, narrow selection of pollutants for "modern" IAP concerns may be appropriate.

"Modern" Indoor Air Pollution Concerns in Countries with High Burdens of Household Air Pollution

Given the large, global movement toward cities—and the identification of biomass use both within and on the fringes of large mega-cities—there is clear risk overlap between the traditional HAP risks and more modern IAP risks. Indeed, we speculate that there is likely a substantial population experiencing both risks regularly. Furthermore, given the proliferation of commoditized goods, like cleaning supplies and other household chemicals and construction materials, it is likely that the burden of these "modern" exposures is experienced even by the rural poor. As above, identification of the most important species—from an exposure, toxicity, or risk perspective—may help enable global measurement databases using comparable techniques.

The Place's the Thing![5]

Several papers reviewed in the course of writing this article cited Klepeis et al.'s (3) seminal National Human Activity Pattern Survey (NHAPS), from which the finding of time spent indoors is derived. Although there have been follow-up studies—in California, Canada, and beyond—the original NHAPS data are more than 25 years old. Personal devices that track location—including smartwatches, cellular phones, and others—have widely proliferated and may provide a way forward to more precisely—and with more spatiotemporal granularity—identify where people spend time. Understanding these activity patterns can help better understand where the potential for exposure is largest and, concurrently, which sources in which microenvironments may be controllable. This type of technology likely has broad application for both HAP and IAP contexts and could additionally contribute to better total air pollution exposure assessment.

Utilization of Low(er)-Cost Sensors for Air Pollution Assessment

HAP has a rich history of developing and using lower-cost air pollution sensors to measure exposure, appliance usage, and time-activity patterns (163–167). Over the past decade, there has been an explosion of low-cost particle and gas monitoring, leading to wide deployment of lower-cost sensor networks for measurement of more finely spatiotemporally resolved air pollution, both indoors and outdoors (168–170). These sensors are often not purpose built for the environmental conditions experienced in kitchens using solid fuels and may not be suitable for indoor or outdoor measurements in contexts where continuous power and network access are not feasible (like, for example, rural India or Kenya). Discussion of technologies, calibration techniques, placement, and other issues between sectors would likely benefit both.

CONCLUSION

Likely every individual on the planet is exposed to some form of IAP, whether from electrical devices, like printers; chemicals, like cleaning agents; or cooking-related aerosols, from either the cooking itself or the combustion of fuels like gas, wood, or dung to provide energy for food preparation. In LMICs, a dominant form of indoor emissions is HAP arising from the inefficient combustion of solid fuels for cooking, heating, lighting, and other energy end uses. These activities contribute to a substantial burden of disease globally. Although much progress has been made on reducing this burden, it remains persistent in many parts of the globe—and many of the same individuals exposed to HAP may also be exposed to other forms of IAP. In developed countries,

[5] With apologies to Shakespeare.

cooking is a common and substantial source of exposure to indoor air pollutants; because of the types of combustion occurring indoors, these emissions and exposures are much smaller than those of households using solid fuels.

The global impact of "modern" IAP concerns—those not associated with household solid fuel combustion—are not fully globally quantified to date in a way that enables easy comparison with other risk factors. Given what is known about exposures based on where people spend time, however, it is likely that there is a substantial burden of disease associated with IAP. This likely means that the strikingly large number of deaths and DALYs associated with air pollution exposure are in fact underestimated.

SUMMARY POINTS

1. Air pollution imposes a substantial burden on human health, but current estimates do not account for exposures indoors, other than for household air pollution (HAP) from solid fuel use.
2. Exposure to air pollution occurs across a range of environments, including indoors at home, indoors and outdoors at work, in transit, and outdoors; a total exposure approach considers all environments.
3. Individuals that rely on solid fuels, like wood, dung, crop residues, and coal for household energy, experience high exposures to harmful household air pollutants and substantial ill-health, as a result of these exposures.
4. Hundreds, if not thousands, of harmful airborne pollutants have been measured in the indoor environment; the magnitude of exposure to these pollutants depends on behavior, the strength of the source, the duration of proximity to the source, and the underlying health status of the exposed individual.
5. Particulate matter, carbon monoxide, and volatile organic compounds are among the most commonly measured pollutants in both HAP and developed world indoor air pollution (IAP) contexts.
6. It is likely that there is substantial risk overlap in developing countries with high solid fuel use between HAP and "modern" IAP concerns; quantifying this overlap would help more precisely estimate the total burden of disease.
7. In HAP contexts, liquefied petroleum gas interventions have, in some contexts, been shown to reduce air pollution exposures substantially, to WHO Interim Target Guideline values; improved and advanced biomass stoves have not been shown to do so, despite promising laboratory findings.
8. The fields of indoor air quality/indoor air pollution and HAP benefit from continued dialogue between experts; areas of synergy may arise around low-cost sensors, around prioritization of pollutants to measure and mitigate, and around better time-activity estimation.

FUTURE ISSUES

1. The Global Burden of Disease currently estimates ill-health attributable to household air pollution (HAP), ambient particulate air pollution, and ambient ozone. An additional

risk factor—for indoor air pollution (IAP) in middle- and upper-income countries—may be appropriate, although there would need to be consistent measurement of a subset of measurable pollutants and related health effects.

2. The issue of gaseous fuels poses an interesting conundrum for indoor air scientists: They are out of favor in the developed world, as they are fossil fuels that contribute to climate change, ill-health, and environmental degradation, whereas for many developing economies, they represent a transitional fuel on the pathway to truly clean cooking with renewably generated electricity. They offer a solution now to a pressing global health threat, whereas other solutions—like cooking with electricity—may be years or decades away.
3. Better time-activity estimates would benefit all: exposure scientists interested in improving models of air pollution exposure; HAP scientists seeking more explanations for residual exposure, after cooking is cleaned up; and burden of disease modelers, who seek the best possible exposure estimate.
4. Low(er)-cost sensors, although not a panacea, can help fill gaps in knowledge about exposure in both developed and developing world contexts, and should be applied more broadly in indoor microenvironments, whether at home, in transit, or in occupational settings, to name a few.
5. For HAP, beginning to consider what to clean up after cleaning up cooking is vital. In many contexts, there is significant exposure even when using clean fuels; identifying the next target for characterization and mitigation can help reduce exposures and health effects.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

ACKNOWLEDGMENTS

This work is dedicated to the memory of Professor Kirk R. Smith, who helped plan early outlines of this review and discussed its evolution with A.P. shortly before his unexpected death. His insights, humor, dedication, and humanity are deeply missed; we acknowledge and honor his lifelong commitment to decreasing exposure to all forms of air pollution.

LITERATURE CITED

1. Health Effects Institute. 2020. *State of Global Air 2020. Special Report*. Health Eff. Inst., Boston, MA
2. Shaddick G, Thomas ML, Amini H, Broday D, Cohen A, et al. 2018. Data integration for the assessment of population exposure to ambient air pollution for global burden of disease assessment. *Environ. Sci Technol.* 52(16):9069–78
3. Klepeis NE, Nelson WC, Ott WR, Robinson JP, Tsang AM, et al. 2001. The National Human Activity Pattern Survey (NHAPS): a resource for assessing exposure to environmental pollutants. *J. Expo. Sci. Environ. Epidemiol.* 11(3):231–52
4. Wallace LA. 2001. Human exposure to volatile organic pollutants: implications for indoor air studies. *Annu. Rev. Energy Environ.* 26:269–301

5. Matz C, Stieb D, Davis K, Egyed M, Rose A, et al. 2014. Effects of age, season, gender and urban-rural status on time-activity: Canadian Human Activity Pattern Survey 2 (CHAPS 2). *Int. J. Environ. Res. Public Health* 11(2):2108–24
6. Lee S, Lee K. 2017. Seasonal differences in determinants of time location patterns in an urban population: a large population-based study in Korea. *Int. J. Environ. Res. Public Health.* 14(7):672
7. Gao Q, Wang F, Hu L, Yu J, Liu R, et al. 2017. Changes in time spent outdoors during the daytime in rural populations in four geographically distinct regions in China: a retrospective study. *Photochem. Photobiol.* 93(2):619–25
8. Sanchez M, Ambros A, Salmon M, Bhogadi S, Wilson RT, et al. 2017. Predictors of daily mobility of adults in peri-urban South India. *Int. J. Environ. Res. Public Health*. 14(7):783
9. Saksena S, Prasad R, Joshi V. 1995. Time allocation and fuel usage in three villages of the Garhwal Himalaya, India. *Mt. Res. Dev*. 15(1):57–67
10. Tham KW. 2016. Indoor air quality and its effects on humans—a review of challenges and developments in the last 30 years. *Energy Build*. 130:637–50
11. Jacobs ET, Burgess JL, Abbott MB. 2018. The Donora smog revisited: 70 years after the event that inspired the Clean Air Act. *Am. J. Public Health* 108(S2):S85–88
12. Fowler D, Brimblecombe P, Burrows J, Heal MR, Grennfelt P, et al. 2020. A chronology of global air quality. *Philos. Trans. Math. Phys. Eng. Sci.* 378(2183):20190314
13. Basu M. 2019. The great smog of Delhi. *Lung India* 36(3):239–40
14. Zhang JJ, Samet JM. 2015. Chinese haze versus Western smog: lessons learned. *J. Thorac. Dis.* 7(1):3–13
15. Sundell J. 2004. On the history of indoor air quality and health. *Indoor Air* 14(Suppl. 7):51–58
16. Sundell J. 2017. Reflections on the history of indoor air science, focusing on the last 50 years. *Indoor Air* 27(4):708–24
17. Smith KR, Bruce N, Balakrishnan K, Adair-Rohani H, Balmes J, et al. 2014. Millions dead: How do we know and what does it mean? Methods used in the comparative risk assessment of household air pollution. *Annu. Rev. Public Health* 35:185–206
18. Liao J, Ye W, Pillarisetti A, Clasen TF. 2019. Modeling the impact of an indoor air filter on air pollution exposure reduction and associated mortality in urban Delhi household. *Int. J. Environ. Res. Public Health* 16(8):1391
19. Datta A, Suresh R, Gupta A, Singh D, Kulshreshtha P. 2017. Indoor air quality of non-residential urban buildings in Delhi, India. *Int. J. Sustain. Built Environ.* 6(2):412–20
20. Zuo J, Ji W, Ben Y, Hassan MA, Fan W, et al. 2018. Using big data from air quality monitors to evaluate indoor PM2.5 exposure in buildings: case study in Beijing. *Environ. Pollut.* 240:839–47
21. Kelly FJ, Fussell JC. 2019. Improving indoor air quality, health and performance within environments where people live, travel, learn and work. *Atmos. Environ.* 200:90–109
22. Jones AP. 1999. Indoor air quality and health. *Atmos. Environ.* 33(28):4535–64
23. Farmer DK, Vance ME. 2019. Indoor air: sources, chemistry and health effects. *Environ. Sci.-Process. Impacts* 21(8):1227–28
24. Ostro B, Spadaro JV, Gumy S, Mudu P, Awe Y, et al. 2018. Assessing the recent estimates of the global burden of disease for ambient air pollution: methodological changes and implications for low- and middle-income countries. *Environ. Res.* 166:713–25
25. Pillarisetti A, Mehta S, Smith KR. 2016. HAPIT, the Household Air Pollution Intervention Tool, to evaluate the health benefits and cost-effectiveness of clean cooking interventions. In *Broken Pumps and Promises: Incentivizing Impact in Environmental Health*, ed. EA Thomas, pp. 147–69. Cham, Switz.: Springer Int. Publ.
26. World Cancer Res. Fund, Am. Inst. Cancer Res. 2018. *Judging the evidence.* Third Exp. Rep., Contin. Update Proj., World Cancer Res. Fund Int., Lond.
27. Shaddick G, Thomas ML, Green A, Brauer M, van Donkelaar A, et al. 2018. Data integration model for air quality: a hierarchical approach to the global estimation of exposures to ambient air pollution. *J. R. Stat. Soc. Ser. C Appl. Stat.* 67(1):231–53
28. Vos T, Lim SS, Abfafati C, Abbas KM, Abbasi M, et al. 2020. Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet* 396(10258):1204–22

29. Murray CJL, Aravkin AY, Zheng P, Abbafati C, Abbas KM, et al. 2020. Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet* 396(10258):1223–49
30. Burnett RT, Pope CA 3rd, Ezzati M, Olives C, Lim SS, et al. 2014. An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure. *Environ. Health Perspect.* 122(4):397–403
31. Ghosh R, Causey K, Burkart K, Wozniak S, Cohen A, Brauer M. 2021. Ambient and household $PM_{2.5}$ pollution and adverse perinatal outcomes: a meta-regression and analysis of attributable global burden for 204 countries and territories. *PLOS Med.* 18(9):e1003718
32. Murray CJL, Aravkin AY, Zheng P, Abbafati C, Abbas KM, et al. 2020. Supplementary appendix 1 to: Global burden of 87 risk factors in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. *Lancet* 396:78–122
33. Ferguson L, Taylor J, Davies M, Shrubsole C, Symonds P, Dimitroulopoulou S. 2020. Exposure to indoor air pollution across socio-economic groups in high-income countries: a scoping review of the literature and a modelling methodology. *Environ. Int.* 143:105748
34. Logue JM, Price PN, Sherman MH, Singer BC. 2012. A method to estimate the chronic health impact of air pollutants in U.S. residences. *Environ. Health Perspect.* 120(2):216–22
35. Logue JM, McKone TE, Sherman MH, Singer BC. 2011. Hazard assessment of chemical air contaminants measured in residences. *Indoor Air* 21(2):92–109
36. Schram-Bijkerk D, van Kempen EEMM, Knol AB. 2013. The burden of disease related to indoor air in the Netherlands: Do different methods lead to different results? *Occup. Environ. Med.* 70(2):126–32
37. González-Martín J, Kraakman NJR, Pérez C, Lebrero R, Muñoz R. 2021. A state-of-the-art review on indoor air pollution and strategies for indoor air pollution control. *Chemosphere* 262:128376
38. Marć M, Śmiełowska M, Namieśnik J, Zabiegała B. 2018. Indoor air quality of everyday use spaces dedicated to specific purposes—a review. *Environ. Sci. Pollut. Res. Int.* 25(3):2065–82
39. Peng Z, Deng W, Tenorio R. 2017. Investigation of indoor air quality and the identification of influential factors at primary schools in the north of China. *Sustainability* 9(7):1180
40. Tran VV, Park D, Lee Y-C. 2020. Indoor air pollution, related human diseases, and recent trends in the control and improvement of indoor air quality. *Int. J. Environ. Res. Public Health* 17(8):2927
41. Abbatt JPD, Wang C. 2020. The atmospheric chemistry of indoor environments. *Environ. Sci.-Process. Impacts* 22(1):25–48
42. Paleologos KE, Selim MYE, Mohamed A-MO. 2021. Indoor air quality: pollutants, health effects, and regulations. In *Pollution Assessment for Sustainable Practices in Applied Sciences and Engineering*, ed. A-MO Mohamed, EK Paleologos, FM Howari, pp. 405–89. Oxford, UK: Butterworth-Heinemann
43. Miller SL. 2018. Indoor air pollution. In *Handbook of Environmental Engineering*, ed. M Kutz, pp. 519–63. Hoboken, NJ: Wiley
44. U.S. Environ. Prot. Agency. 2022. Particulate matter (PM) basics. *United States Environmental Protection Agency*. **https://www.epa.gov/pm-pollution/particulate-matter-pm-basics**
45. U.S. Environ. Prot. Agency. 2021. Learn about asbestos. *United States Environmental Protection Agency*. **https://www.epa.gov/asbestos/learn-about-asbestos**
46. Spengler JD, Sexton K. 1983. Indoor air pollution: a public health perspective. *Science* 221(4605):9–17
47. World Health Organization. 2018. *WHO Housing and Health Guidelines*. Geneva: World Health Organ.
48. Guo C, Gao Z, Shen J. 2019. Emission rates of indoor ozone emission devices: a literature review. *Build. Environ.* 158:302–18
49. Huang Y, Yang Z, Gao Z. 2019. Contributions of indoor and outdoor sources to ozone in residential buildings in Nanjing. *Int. J. Environ. Res. Public Health* 16(14):2587
50. Lee SC, Lam S, Kin Fai H. 2001. Characterization of VOCs, ozone, and PM_{10} emissions from office equipment in an environmental chamber. *Build. Environ.* 36(7):837–42
51. Zhang Q, Jenkins PL. 2017. Evaluation of ozone emissions and exposures from consumer products and home appliances. *Indoor Air* 27(2):386–97
52. U.S. Environ. Prot. Agency. 2021. Volatile organic compounds' impact on indoor air quality. *United States Environmental Protection Agency*. **https://www.epa.gov/indoor-air-quality-iaq/volatile-organic-compounds-impact-indoor-air-quality**

53. Luengas A, Barona A, Hort C, Gallastegui G, Platel V, Elias A. 2015. A review of indoor air treatment technologies. *Rev. Environ. Sci. Biotechnol.* 14(3):499–522
54. Dunagan SC, Dodson RE, Rudel RA, Brody JG. 2011. Toxics use reduction in the home: lessons learned from household exposure studies. *J. Clean. Prod.* 19(5):438–44
55. Huang Y, Ho SSH, Ho KF, Lee SC, Yu JZ, Louie PKK. 2011. Characteristics and health impacts of VOCs and carbonyls associated with residential cooking activities in Hong Kong. *J. Hazard Mater.* 186(1):344–51
56. Lee K, Choi J-H, Lee S, Park H-J, Oh Y-J, et al. 2018. Indoor levels of volatile organic compounds and formaldehyde from emission sources at elderly care centers in Korea. *PLOS ONE* 13(6):e0197495
57. Liu S, Li R, Wild RJ, Warneke C, de Gouw JA, et al. 2016. Contribution of human-related sources to indoor volatile organic compounds in a university classroom. *Indoor Air* 26(6):925–38
58. Tang X, Misztal PK, Nazaroff WW, Goldstein AH. 2015. Siloxanes are the most abundant volatile organic compound emitted from engineering students in a classroom. *Environ. Sci. Technol. Lett.* 2(11):303–7
59. Vardoulakis S, Giagloglou E, Steinle S, Davis A, Sleeuwenhoek A, et al. 2020. Indoor exposure to selected air pollutants in the home environment: a systematic review. *Int. J. Environ. Res. Public Health* 17(23):8972
60. Seguel JM, Merrill R, Seguel D, Campagna AC. 2017. Indoor air quality. *Am. J. Lifestyle Med.* 11(4):284–95
61. Hecht SS. 1999. Tobacco smoke carcinogens and lung cancer. *J. Natl. Cancer Inst.* 91(14):1194–1210
62. NRC (Natl. Res. Counc.) Comm. Inst. Means Assess. Risks Public Health. 1983. *Risk Assessment in the Federal Government: Managing the Process*. Washington, DC: NRC
63. Schick S, Glantz SA. 2006. Sidestream cigarette smoke toxicity increases with aging and exposure duration. *Tob. Control* 15(6):424–29
64. Schick SF, Farraro KF, Perrino C, Sleiman M, van de Vossenberg G, et al. 2014. Thirdhand cigarette smoke in an experimental chamber: evidence of surface deposition of nicotine, nitrosamines and polycyclic aromatic hydrocarbons and de novo formation of NNK. *Tob. Control* 23(2):152–59
65. Whitlatch A, Schick S. 2019. Thirdhand smoke at Philip Morris. *Nicotine Tob. Res.* 21(12):1680–88
66. Gaskin J, Coyle D, Whyte J, Krewski D. 2018. Global estimate of lung cancer mortality attributable to residential radon. *Environ. Health Perspect.* 126(5):057009
67. U.S. Environ. Prot. Agency. 2021. Biological pollutants' impact on indoor air quality. *United States Environmental Protection Agency*. **https://www.epa.gov/indoor-air-quality-iaq/biological-pollutants-impact-indoor-air-quality**
68. Diffey BL. 2011. An overview analysis of the time people spend outdoors: time spent outdoors. *Br. J. Dermatol.* 164(4):848–54
69. EC (Eur. Comm.). 2003. *Indoor air pollution: new EU research reveals higher risks than previously thought*. Press Release IP/03/1278, Joint Res. Cent., EC, Sept. 22. **https://ec.europa.eu/commission/presscorner/detail/en/IP_03_1278**
70. Casey JA, Ogburn EL, Rasmussen SG, Irving JK, Pollak J, et al. 2015. Predictors of indoor radon concentrations in Pennsylvania, 1989–2013. *Environ. Health Perspect.* 123(11):1130–37
71. Kendall GM, Miles JCH, Rees D, Wakeford R, Bunch KJ, et al. 2016. Variation with socioeconomic status of indoor radon levels in Great Britain: the less affluent have less radon. *J. Environ. Radioact.* 164:84–90
72. Taylor J, Shrubsole C, Davies M, Biddulph P, Das P, et al. 2014. The modifying effect of the building envelope on population exposure to $PM_{2.5}$ from outdoor sources. *Indoor Air* 24(6):639–51
73. Turk BH, Prill RJ, Grimsrud DT, Moed BA, Sextro RG. 1990. Characterizing the occurrence, sources, and variability of radon in Pacific Northwest homes. *J. Air Waste Manag. Assoc.* 40(4):498–506
74. Symonds P, Rees D, Daraktchieva Z, McColl N, Bradley J, et al. 2019. Home energy efficiency and radon: an observational study. *Indoor Air* 29(5):854–64
75. Foulds C, Powell J, Seyfang G. 2013. Investigating the performance of everyday domestic practices using building monitoring. *Build. Res. Inf.* 41(6):622–36
76. Fabian MP, Lee SK, Underhill LJ, Vermeer K, Adamkiewicz G, Levy JI. 2016. Modeling environmental tobacco smoke (ETS) infiltration in low-income multifamily housing before and after building energy retrofits. *Int. J. Environ. Res. Public Health* 13(3):327

77. Milner JT, ApSimon HM, Croxford B. 2006. Spatial variation of CO concentrations within an office building and outdoor influences. *Atmos. Environ.* 40(33):6338–48
78. Shrubsole C, Taylor J, Das P, Hamilton IG, Oikonomou E, Davies M. 2016. Impacts of energy efficiency retrofitting measures on indoor $PM_{2.5}$ concentrations across different income groups in England: a modelling study. *Adv. Build. Energy Res.* 10(1):69–83
79. Rosofsky A, Levy JI, Breen MS, Zanobetti A, Fabian MP. 2019. The impact of air exchange rate on ambient air pollution exposure and inequalities across all residential parcels in Massachusetts. *J. Expo. Sci. Environ. Epidemiol.* 29(4):520–30
80. Taylor J, Davies M, Mavrogianni A, Shrubsole C, Hamilton I, et al. 2016. Mapping indoor overheating and air pollution risk modification across Great Britain: a modelling study. *Build. Environ.* 99:1–12
81. Salomon JA. 2014. Disability-adjusted life years. In *Encyclopedia of Health Economics*, ed. AJ Culyer, pp. 200–3. Amsterdam: Elsevier
82. Asikainen A, Carrer P, Kephalopoulos S, de Oliveira Fernandes E, Wargocki P, Hänninen O. 2016. Reducing burden of disease from residential indoor air exposures in Europe (HEALTHVENT project). *Environ. Health* 15(S1):S35
83. Rosário Filho NA, Urrutia-Pereira M, D'Amato G, Cecchi L, Ansotegui IJ, et al. 2021. Air pollution and indoor settings. *World Allergy Organ. J.* 14(1):100499
84. Schraufnagel DE, Balmes JR, Cowl CT, De Matteis S, Jung S-H, et al. 2019. Air pollution and noncommunicable diseases: a review by the Forum of International Respiratory Societies' Environmental Committee, Part 1: the damaging effects of air pollution. *Chest* 155(2):409–16
85. De Grove KC, Provoost S, Brusselle GG, Joos GF, Maes T. 2018. Insights in particulate matter-induced allergic airway inflammation: focus on the epithelium. *Clin. Exp. Allergy* 48(7):773–86
86. Breton CV, Yao J, Millstein J, Gao L, Siegmund KD, et al. 2016. Prenatal air pollution exposures, DNA methyl transferase genotypes, and associations with newborn LINE1 and Alu methylation and childhood blood pressure and carotid intima-media thickness in the children's health study. *Environ. Health Perspect.* 124(12):1905–12
87. Jardim MJ. 2011. microRNAs: implications for air pollution research. *Mutat. Res.* 717(1–2):38–45
88. Marmot AF, Eley J, Stafford M, Stansfeld SA, Warwick E, Marmot MG. 2006. Building health: an epidemiological study of "sick building syndrome" in the Whitehall II study. *Occup. Environ. Med.* 63(4):283–89
89. U.S. Environ. Prot. Agency. 1991. *Indoor Air Facts No. 4: sick building syndrome*. Doc. 6609J (Air Radiat.)/MD-56 (Res. Dev.), U.S. Environ. Prot. Agency, Washington, DC. **https://www.epa.gov/sites/default/files/2014-08/documents/sick_building_factsheet.pdf**
90. Seltzer JM. 1994. Building-related illnesses. *J. Allergy Clin. Immunol.* 94(2):351–61
91. Grief SN. 2013. Upper respiratory infections. *Prim. Care.* 40(3):757–70
92. Gaffin JM, Hauptman M, Petty CR, Sheehan WJ, Lai PS, et al. 2018. Nitrogen dioxide exposure in school classrooms of inner-city children with asthma. *J. Allergy Clin. Immunol.* 141(6):2249–55.e2
93. Hansel NN, Breysse PN, McCormack MC, Matsui EC, Curtin-Brosnan J, et al. 2008. A longitudinal study of indoor nitrogen dioxide levels and respiratory symptoms in inner-city children with asthma. *Environ. Health Perspect.* 116(10):1428–32
94. Kattan M, Gergen PJ, Eggleston P, Visness CM, Mitchell HE. 2007. Health effects of indoor nitrogen dioxide and passive smoking on urban asthmatic children. *J. Allergy Clin. Immunol.* 120(3):618–24
95. Paulin LM, Williams D'AL, Peng R, Diette GB, McCormack MC, et al. 2017. 24-h Nitrogen dioxide concentration is associated with cooking behaviors and an increase in rescue medication use in children with asthma. *Environ. Res.* 159:118–23
96. Hansel NN, McCormack MC, Belli AJ, Matsui EC, Peng RD, et al. 2013. In-home air pollution is linked to respiratory morbidity in former smokers with chronic obstructive pulmonary disease. *Am. J. Respir. Crit. Care Med.* 187(10):1085–90
97. Jamieson DB, Matsui EC, Belli A, McCormack MC, Peng E, et al. 2013. Effects of allergic phenotype on respiratory symptoms and exacerbations in patients with chronic obstructive pulmonary disease. *Am. J. Respir. Crit. Care Med.* 188(2):187–92
98. Wu X, Nethery RC, Sabath BM, Braun D, Dominici F. 2020. Air pollution and COVID-19 mortality in the United States: Strengths and limitations of an ecological regression analysis. *Sci. Adv.* 6(45):eabd4049

99. Conticini E, Frediani B, Caro D. 2020. Can atmospheric pollution be considered a co-factor in extremely high level of SARS-CoV-2 lethality in Northern Italy? *Environ. Pollut.* 261:114465
100. Setti L, Passarini F, De Gennaro G, Barbieri P, Perrone MG, et al. 2020. SARS-Cov-2RNA found on particulate matter of Bergamo in Northern Italy: first evidence. *Environ. Res.* 188:109754
101. Li Y, Nazaroff WW, Bahnfleth W, Wargocki P, Zhang Y. 2021. The COVID-19 pandemic is a global indoor air crisis that should lead to change: a message commemorating 30 years of indoor air. *Indoor Air* 31(6):1683–86
102. Liu J, Zhou J, Yao J, Zhang X, Li L, et al. 2020. Impact of meteorological factors on the COVID-19 transmission: a multi-city study in China. *Sci. Total Environ.* 726:138513
103. Kampfrath T, Maiseyeu A, Ying Z, Shah Z, Deiuliis JA, et al. 2011. Chronic fine particulate matter exposure induces systemic vascular dysfunction via NADPH oxidase and TLR4 pathways. *Circ. Res.* 108(6):716–26
104. Peters A, Liu E, Verrier RL, Schwartz J, Gold DR, et al. 2000. Air pollution and incidence of cardiac arrhythmia. *Epidemiol. Camb. Mass.* 11(1):11–17
105. Kim K-H, Jahan SA, Kabir E. 2011. A review of diseases associated with household air pollution due to the use of biomass fuels. *J. Hazard Mater.* 192(2):425–31
106. Broderick Á, Byrne M, Armstrong S, Sheahan J, Coggins AM. 2017. A pre and post evaluation of indoor air quality, ventilation, and thermal comfort in retrofitted co-operative social housing. *Build. Environ.* 122:126–33
107. Wei W, Ramalho O, Mandin C. 2015. Indoor air quality requirements in green building certifications. *Build. Environ.* 92:10–19
108. Guieysse B, Hort C, Platel V, Munoz R, Ondarts M, Revah S. 2008. Biological treatment of indoor air for VOC removal: potential and challenges. *Biotechnol. Adv.* 26(5):398–410
109. Butz AM, Matsui EC, Breysse P, Curtin-Brosnan J, Eggleston P, et al. 2011. A Randomized trial of air cleaners and a health coach to improve indoor air quality for inner-city children with asthma and secondhand smoke exposure. *Arch. Pediatr. Adolesc. Med.* 165(8):741–48
110. Lanphear BP, Hornung RW, Khoury J, Yolton K, Lierl M, Kalkbrenner A. 2011. Effects of HEPA air cleaners on unscheduled asthma visits and asthma symptoms for children exposed to secondhand tobacco smoke. *Pediatrics* 127(1):93–101
111. Raju S, Siddharthan T, McCormack MC. 2020. Indoor air pollution and respiratory health. *Clin. Chest Med.* 41(4):825–43
112. Noonan CW, Ward TJ. 2007. Environmental tobacco smoke, woodstove heating and risk of asthma symptoms. *J. Asthma.* 44(9):735–38
113. Ward TJ, Palmer CP, Houck JE, Navidi WC, Geinitz S, Noonan CW. 2009. Community woodstove changeout and impact on ambient concentrations of polycyclic aromatic hydrocarbons and phenolics. *Environ. Sci. Technol.* 43(14):5345–50
114. Faber T, Kumar A, Mackenbach JP, Millett C, Basu S, et al. 2017. Effect of tobacco control policies on perinatal and child health: a systematic review and meta-analysis. *Lancet Public Health*. 2(9):e420–37
115. Been JV, Nurmatov UB, Cox B, Nawrot TS, van Schayck CP, Sheikh A. 2014. Effect of smoke-free legislation on perinatal and child health: a systematic review and meta-analysis. *Lancet* 383(9928):1549–60
116. Balakrishnan K, Ghosh S, Ganguli B, Sambandam S, Bruce N, et al. 2013. State and national household concentrations of $PM_{2.5}$ from solid cookfuel use: results from measurements and modeling in India for estimation of the global burden of disease. *Environ. Health* 12(1):77
117. Smith KR, Pillarisetti A. 2017. Household air pollution from solid cookfuels and health. In *Disease Control Priorities* (3rd ed.), Volume 7: *Injury Prevention and Environmental Health*, ed. CN Mock, R Nugent, O Kobusingye, K Smith, pp. 133–52. Washington, DC: World Bank
118. Chowdhury S, Pozzer A, Haines A, Klingmüller K, Münzel T, et al. 2022. Global health burden of ambient $PM_{2.5}$ and the contribution of anthropogenic black carbon and organic aerosols. *Environ. Int.* 159:107020
119. Wrangham RW. 2009. *Catching Fire: How Cooking Made Us Human*. New York: Basic Books
120. Goudsblom J. 1992. The civilizing process and the domestication of fire. *J. World Hist.* 3(1):1–12

121. Naeher LP, Brauer M, Lipsett M, Zelikoff JT, Simpson CD, et al. 2007. Woodsmoke health effects: a review. *Inhal. Toxicol.* 19(1):67–106
122. Weinstein JR, Asteria-Peñaloza R, Diaz-Artiga A, Davila G, Hammond SK, et al. 2017. Exposure to polycyclic aromatic hydrocarbons and volatile organic compounds among recently pregnant rural Guatemalan women cooking and heating with solid fuels. *Int. J. Hyg. Environ. Health* 220(4):726–35
123. Shen H, Luo Z, Xiong R, Liu X, Zhang L, et al. 2021. A critical review of pollutant emission factors from fuel combustion in home stoves. *Environ. Int.* 157:106841
124. Shupler M, Balakrishnan K, Ghosh S, Thangavel G, Stroud-Drinkwater S, et al. 2018. Global household air pollution database: kitchen concentrations and personal exposures of particulate matter and carbon monoxide. *Data Brief* 21:1292–95
125. Shupler M, Godwin W, Frostad J, Gustafson P, Arku RE, Brauer M. 2018. Global estimation of exposure to fine particulate matter ($PM_{2.5}$) from household air pollution. *Environ. Int.* 120:354–63
126. Chafe ZA, Brauer M, Klimont Z, Van Dingenen R, Mehta S, et al. 2014. Household cooking with solid fuels contributes to ambient $PM_{2.5}$ air pollution and the burden of disease. *Environ. Health Perspect.* 122(12):1314–20
127. Johnson TM, Guttikunda S, Wells GJ, Artaxo P, Bond TC, et al. 2011. *Tools for improving air quality management: a review of top-down source apportionment techniques and their application in developing countries.* Rep. 339/11, Energy Sector Manag. Assist. Progr., World Bank, Washington, DC
128. Chowdhury S, Chafe Z, Pillarisetti A, Lelieveld J, Guttikunda S, Dey S. 2019. *The contribution of household fuels to ambient air pollution in India—a comparison of recent estimates*. Policy Brief CCAPC/2019/01, Collab. Clean Air Policy Cent., New Delhi
129. Balakrishnan K, Clasen T, Mehta S, Peel J, Pillarisetti A, et al. 2020. In Memoriam: Kirk R. Smith. *Environ. Health Perspect.* 128(7):71601
130. Pope D, Johnson M, Fleeman N, Jagoe K, Duarte R, et al. 2021. Are cleaner cooking solutions clean enough? A systematic review and meta-analysis of particulate and carbon monoxide concentrations and exposures. *Environ. Res. Lett.* 16(8):083002
131. Bilsback KR, Dahlke J, Fedak KM, Good N, Hecobian A, et al. 2019. A laboratory assessment of 120 air pollutant emissions from biomass and fossil fuel cookstoves. *Environ. Sci. Technol.* 53(12):7114–25
132. Jetter J, Zhao Y, Smith KR, Khan B, Yelverton T, et al. 2012. Pollutant emissions and energy efficiency under controlled conditions for household biomass cookstoves and implications for metrics useful in setting international test standards. *Environ. Sci. Technol.* 46(19):10827–34
133. Smith KR, McCracken JP, Weber MW, Hubbard A, Jenny A, et al. 2011. Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial. *Lancet* 378(9804):1717–26
134. Mortimer K, Ndamala CB, Naunje AW, Malava J, Katundu C, et al. 2016. A cleaner burning biomass-fuelled cookstove intervention to prevent pneumonia in children under 5 years old in rural Malawi (the Cooking and Pneumonia Study): a cluster randomised controlled trial. *Lancet* 389:67–175
135. Hartinger S, Lanata C, Hattendorf J, Verastegui H, Gil A, et al. 2016. Improving household air, drinking water and hygiene in rural Peru: a community-randomized-controlled trial of an integrated environmental home-based intervention package to improve child health. *Int. J. Epidemiol.* 45(6):2089–99
136. Katz J, Tielsch JM, Khatry SK, Shrestha L, Breysse P, et al. 2020. Impact of improved biomass and liquid petroleum gas stoves on birth outcomes in rural Nepal: results of 2 randomized trials. *Glob. Health Sci. Pract.* 8(3):372–82
137. Kirby MA, Nagel CL, Rosa G, Zambrano LD, Musafiri S, et al. 2019. Effects of a large-scale distribution of water filters and natural draft rocket-style cookstoves on diarrhea and acute respiratory infection: a cluster-randomized controlled trial in Western Province, Rwanda. *PLOS Med.* 16(6):e1002812
138. Jack DW, Ae-Ngibise KA, Gould CF, Boamah-Kaali E, Lee AG, et al. 2021. A cluster randomised trial of cookstove interventions to improve infant health in Ghana. *BMJ Glob. Health.* 6(8):e005599
139. Gould CF, Schlesinger S, Toasa AO, Thurber M, Waters WF, et al. 2018. Government policy, clean fuel access, and persistent fuel stacking in Ecuador. *Energy Sustain. Dev. J. Int. Energy Initiat.* 46:111–22
140. Gould CF, Schlesinger SB, Molina E, Lorena Bejarano M, Valarezo A, Jack DW. 2020. Long-standing LPG subsidies, cooking fuel stacking, and personal exposure to air pollution in rural and peri-urban Ecuador. *J. Expo. Sci. Environ. Epidemiol.* 30(4):707–20

141. Troncoso K, Soares da Silva A. 2017. LPG fuel subsidies in Latin America and the use of solid fuels to cook. *Energy Policy*. 107:188–96
142. Goldemberg J, Martinez-Gomez J, Sagar A, Smith KR. 2018. Household air pollution, health, and climate change: cleaning the air. *Environ. Res. Lett.* 13(3):030201
143. Johnson MA, Chiang RA. 2015. Quantitative guidance for stove usage and performance to achieve health and environmental targets. *Environ. Health Perspect.* 123(8):820–26
144. Kar A, Pachauri S, Bailis R, Zerriffi H. 2019. Using sales data to assess cooking gas adoption and the impact of India's Ujjwala programme in rural Karnataka. *Nat. Energy* 4(9):806–14
145. Mani S, Jain A, Tripathi S, Gould CF. 2020. The drivers of sustained use of liquified petroleum gas in India. *Nat. Energy* 5(6):450–57
146. Jain A, Mani S, Patnaik S, Shahidi T, Ganesan K. 2018. *Access to Clean Cooking Energy and Electricity: Survey of States 2018*. Rep., Counc. Energy Environ. Water, New Delhi
147. Clasen T, Checkley W, Peel JL, Balakrishnan K, McCracken JP, et al. 2020. Design and rationale of the HAPIN study: a multicountry randomized controlled trial to assess the effect of liquefied petroleum gas stove and continuous fuel distribution. *Environ. Health Perspect.* 128(4):047008
148. Johnson M, Pillarisetti A, Piedrahita R, Balakrishnan K, Peel JL, et al. 2021. Exposure contrasts of pregnant women during the Household Air Pollution Intervention Network randomized controlled trial. medRxiv 21265938. **https://doi.org/10.1101/2021.11.04.21265938**
149. Checkley W, Williams KN, Kephart JL, Fandiño-Del-Rio M, Steenland NK, et al. 2021. Effects of a household air pollution intervention with liquefied petroleum gas on cardiopulmonary outcomes in Peru. A randomized controlled trial. *Am. J. Respir. Crit. Care Med.* 203(11):1386–97
150. Quinn AK, Ae-Ngibise KA, Jack DW, Boamah EA, Enuameh Y, et al. 2016. Association of Carbon Monoxide exposure with blood pressure among pregnant women in rural Ghana: evidence from GRAPHS. *Int. J. Hyg. Environ. Health* 219(2):176–83
151. Boamah-Kaali E, Jack DW, Ae-Ngibise KA, Quinn A, Kaali S, et al. 2021. Prenatal and postnatal household air pollution exposure and infant growth trajectories: evidence from a rural Ghanaian pregnancy cohort. *Environ. Health Perspect.* 129(11):117009
152. Quinn AK, Adjei IA, Ae-Ngibise KA, Agyei O, Boamah-Kaali EA, et al. 2021. Prenatal household air pollutant exposure is associated with reduced size and gestational age at birth among a cohort of Ghanaian infants. *Environ. Int.* 155:106659
153. Alexander D, Northcross A, Wilson N, Dutta A, Pandya R, et al. 2017. Randomized controlled ethanol cookstove intervention and blood pressure in pregnant Nigerian women. *Am. J. Respir. Crit. Care Med.* 195(12):1629–39
154. Alexander DA, Northcross A, Karrison T, Morhasson-Bello O, Wilson N, et al. 2018. Pregnancy outcomes and ethanol cook stove intervention: a randomized-controlled trial in Ibadan, Nigeria. *Environ. Int.* 111:152–63
155. Patil R, Roy S, Gore M, Ghorpade M, Pillarisetti A, et al. 2021. Barriers to and facilitators of uptake and sustained use of LPG through the PMUY in tribal communities of Pune district. *Energy Sustain. Dev.* 63:1–6
156. Lam NL, Upadhyay B, Maharjan S, Jagoe K, Weyant CL, et al. 2017. Seasonal fuel consumption, stoves, and end-uses in rural households of the far-western development region of Nepal. *Environ. Res. Lett.* 12(12):125011
157. Krasner A, Jones TS, LaRocque R. 2021. Cooking with gas, household air pollution, and asthma: little recognized risk for children. *J. Environ. Health* 83(8):14–18
158. Zhu Y, Connolly R, Lin Y, Mathews T, Wang Z. 2020. *Effects of residential gas appliances on indoor and outdoor air quality and public health in California*. Rep., Dep. Environ. Health Sci., Fielding Sch. Public Health, Univ. Calif., Los Angel.
159. Quinn AK, Bruce N, Puzzolo E, Dickinson K, Sturke R, et al. 2018. An analysis of efforts to scale up clean household energy for cooking around the world. *Energy Sustain. Dev.* 46:1–10
160. Rosenthal J, Quinn A, Grieshop AP, Pillarisetti A, Glass RI. 2018. Clean cooking and the SDGs: integrated analytical approaches to guide energy interventions for health and environment goals. *Energy Sustain. Dev.* 42(Suppl. C):152–59

161. Kephart JL, Fandiño-Del-Rio M, Williams KN, Malpartida G, Lee A, et al. 2021. Nitrogen dioxide exposures from LPG stoves in a cleaner-cooking intervention trial. *Environ. Int.* 146:106196
162. WHO (World. Health Organ.) 2014. *WHO Guidelines for Indoor Air Quality: Household Fuel Combustion*. Geneva: WHO
163. Pillarisetti A, Allen T, Ruiz-Mercado I, Edwards R, Chowdhury Z, et al. 2017. Small, smart, fast, and cheap: microchip-based sensors to estimate air pollution exposures in rural households. *Sensors* 17(8):1879
164. Liao J, McCracken JP, Piedrahita R, Thompson L, Mollinedo E, et al. 2019. The use of bluetooth low energy Beacon systems to estimate indirect personal exposure to household air pollution. *J. Expo. Sci. Environ. Epidemiol.* 30:990–1000
165. Pillarisetti A, Carter E, Rajkumar S, Young BN, Benka-Coker ML, et al. 2019. Measuring personal exposure to fine particulate matter ($PM_{2.5}$) among rural Honduran women: a field evaluation of the Ultrasonic Personal Aerosol Sampler (UPAS). *Environ. Int.* 123:50–53
166. Chartier R. 2015. Pilot testing and evaluation of an Enhanced Children's MicroPEM. *Environ. Health Perspect.* 2015:3043 (Abst.)
167. Ruiz-Mercado I, Canuz E, Smith KR. 2012. Temperature dataloggers as stove use monitors (SUMs): field methods and signal analysis. *Biomass Bioenergy* 47:459–68
168. Holstius DM, Pillarisetti A, Smith KR, Seto E. 2014. Field calibrations of a low-cost aerosol sensor at a regulatory monitoring site in California. *Atmos. Meas. Tech.* 7(4):1121–31
169. Collier-Oxandale A, Feenstra B, Papapostolou V, Zhang H, Kuang M, et al. 2020. Field and laboratory performance evaluations of 28 gas-phase air quality sensors by the AQ-SPEC program. *Atmos. Environ.* 220:117092
170. Polidori A, Papapostolou V, Feenstra B, Zhang H. 2017. *Field evaluation of low-cost air quality sensors: field sensing and testing protocol*. Rep., Air Qual. Sens. Perform. Eval. Cent., S. Coast Air Qual. Manag. Dist., Diamond Bar, CA
171. Canha N, Lage J, Candeias S, Alves C, Almeida SM. 2017. Indoor air quality during sleep under different ventilation patterns. *Atmos. Pollut. Res.* 8(6):1132–42
172. Barmparesos N, Assimakopoulos MN, Assimakopoulos VD, Loumos N, Sotiriou MA, Koukoumtzis A. 2018. Indoor air quality and thermal conditions in a primary school with a green roof system. *Atmosphere* 9(2):75
173. Mandin C, Trantallidi M, Cattaneo A, Canha N, Mihucz VG, et al. 2017. Assessment of indoor air quality in office buildings across Europe—the OFFICAIR study. *Sci. Total Environ.* 579:169–78
174. Vasile V, Petran H, Dima A, Petcu C. 2016. Indoor air quality—a key element of the energy performance of the buildings. *Energy Procedia*. 96:277–84
175. Langer S, Bekö G, Bloom E, Widheden A, Ekberg L. 2015. Indoor air quality in passive and conventional new houses in Sweden. *Build. Environ.* 93:92–100
176. Nazaroff WW, Weschler CJ. 2022. Indoor ozone: concentrations and influencing factors. *Indoor Air* 32(1):e12942
177. Dodson RE, Levy JI, Spengler JD, Shine JP, Bennett DH. 2008. Influence of basements, garages, and common hallways on indoor residential volatile organic compound concentrations. *Atmos. Environ.* 42(7):1569–81
178. Du L, Batterman S, Godwin C, Rowe Z, Chin J-Y. 2015. Air exchange rates and migration of VOCs in basements and residences. *Indoor Air* 25(6):598–609
179. Edwards RD, Jurvelin J, Saarela K, Jantunen M. 2001. VOC concentrations measured in personal samples and residential indoor, outdoor and workplace microenvironments in EXPOLIS-Helsinki, Finland. *Atmos. Environ.* 35(27):4531–43
180. Geiss O, Giannopoulos G, Tirendi S, Barrero-Moreno J, Larsen BR, Kotzias D. 2011. The AIRMEX study—VOC measurements in public buildings and schools/kindergartens in eleven European cities: statistical analysis of the data. *Atmos. Environ.* 45(22):3676–84
181. Rösch C, Kohajda T, Röder S, von Bergen M, Schlink U. 2014. Relationship between sources and patterns of VOCs in indoor air. *Atmos. Pollut. Res.* 5(1):129–37
182. Zhong L, Su F-C, Batterman S. 2017. Volatile organic compounds (VOCs) in conventional and high performance school buildings in the U.S. *Int. J. Environ. Res. Public Health* 14(1):100

183. Xu J, Szyszkowicz M, Jovic B, Cakmak S, Austin CC, Zhu J. 2016. Estimation of indoor and outdoor ratios of selected volatile organic compounds in Canada. *Atmos. Environ.* 141:523–31
184. Fahiminia M, Fouladi Fard R, Ardani R, Mohammadbeigi A, Naddafi K, Hassanvand MS. 2016. Indoor radon measurements in residential dwellings in Qom, Iran. *Int. J. Radiat. Res.* 14(4):331–39
185. Madureira J, Paciência I, Rufo J, Moreira A, de Oliveira Fernandes E, Pereira A. 2016. Radon in indoor air of primary schools: determinant factors, their variability and effective dose. *Environ. Geochem. Health.* 38(2):523–33
186. Bochicchio F, Žunić ZS, Carpentieri C, Antignani S, Venoso G, et al. 2014. Radon in indoor air of primary schools: a systematic survey to evaluate factors affecting radon concentration levels and their variability. *Indoor Air* 24(3):315–26
187. Su C, Pan M, Zhang Y, Kan H, Zhao Z, et al. 2022. Indoor exposure levels of radon in dwellings, schools, and offices in China from 2000 to 2020: a systematic review. *Indoor Air* 32(1):e12920
188. World Health Organization. 2010. *WHO Guidelines for Indoor Air Quality: Selected Pollutants*. Geneva: WHO Reg. Off. Eur.
189. Carazo Fernández L, Fernández Alvarez R, González-Barcala FJ, Rodríguez Portal JA. 2013. Indoor air contaminants and their impact on respiratory pathologies. *Arch. Bronconeumol. Engl. Ed.* 49(1):22–27
190. Leung DYC. 2015. Outdoor-indoor air pollution in urban environment: challenges and opportunity. *Front. Environ. Sci.* 2. **https://doi.org/10.3389/fenvs.2014.00069**
191. U.S. Environ. Prot. Agency. 2021. Carbon monoxide's impact on indoor air quality. *United States Environmental Protection Agency*. **https://www.epa.gov/indoor-air-quality-iaq/carbon-monoxides-impact-indoor-air-quality**
192. Kjaergaard SK, Rasmussen TR. 1996. Clinical studies of effects of nitrogen oxides in healthy and asthmatic subjects. *Cent. Eur. J. Public Health.* 4(Suppl.):23–26
193. Salonen H, Salthammer T, Morawska L. 2018. Human exposure to ozone in school and office indoor environments. *Environ. Int.* 119:503–14
194. Benninger MS. 1999. The impact of cigarette smoking and environmental tobacco smoke on nasal and sinus disease: a review of the literature. *Am. J. Rhinol.* 13(6):435–38
195. Hirayama T. 1981. Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. *Br. Med. J. Clin. Res. Ed.* 282(6259):183–85
196. Health Hum. Serv. 2006. *The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General*. Washington, DC: U. S. Dep. Health Hum. Serv., Public Health Serv., Off. Surgeon General
197. Svendsen KH, Kuller LH, Martin MJ, Ockene JK. 1987. Effects of passive smoking in the Multiple Risk Factor Intervention Trial. *Am. J. Epidemiol.* 126(5):783–95
198. Brennan P, Buffler PA, Reynolds P, Wu AH, Wichmann HE, et al. 2004. Secondhand smoke exposure in adulthood and risk of lung cancer among never smokers: a pooled analysis of two large studies. *Int. J. Cancer* 109(1):125–31
199. Hole DJ, Gillis CR, Chopra C, Hawthorne VM. 1989. Passive smoking and cardiorespiratory health in a general population in the west of Scotland. *BMJ* 299(6696):423–27
200. Whincup PH, Gilg JA, Emberson JR, Jarvis MJ, Feyerabend C, et al. 2004. Passive smoking and risk of coronary heart disease and stroke: prospective study with cotinine measurement. *BMJ* 329(7459):200–205
201. Glantz SA, Parmley WW. 1991. Passive smoking and heart disease. Epidemiology, physiology, and biochemistry. *Circulation* 83(1):1–12
202. Samet JM. 2006. Residential radon and lung cancer: end of the story? *J. Toxicol. Environ. Health A.* 69(7):527–31
203. Krewski D, Lubin JH, Zielinski JM, Alavanja M, Catalan VS, et al. 2006. A combined analysis of North American case-control studies of residential radon and lung cancer. *J. Toxicol. Environ. Health A.* 69(7):533–97
204. Kotzias D, Koistinen K, Kephalopoulos S, Schlitt C, Carrer P, et al. 2005. *The INDEX Project: critical appraisal of the setting and implementation of indoor exposure limits in the EU*. Rep. EUR 21590EN, Eur. Comm., Luxemb.

205. Malaka T, Kodama AM. 1990. Respiratory health of plywood workers occupationally exposed to formaldehyde. *Arch. Environ. Health* 45(5):288–94
206. Kastner PE, Casset A, Pons F. 2011. Formaldehyde interferes with airway epithelium integrity and functions in a dose- and time-dependent manner. *Toxicol. Lett.* 200(1–2):109–16
207. Collins JJ, Acquavella JF, Esmen NA. 1997. An updated meta-analysis of formaldehyde exposure and upper respiratory tract cancers. *J. Occup. Environ. Med.* 39(7):639–51
208. Partanen T. 1993. Formaldehyde exposure and respiratory cancer—a meta-analysis of the epidemiologic evidence. *Scand. J. Work. Environ. Health* 19(1):8–15
209. Knutsen AP, Bush RK, Demain JG, Denning DW, Dixit A, et al. 2012. Fungi and allergic lower respiratory tract diseases. *J. Allergy Clin. Immunol.* 129(2):280–91; quiz 292–93
210. Denning DW, O'Driscoll BR, Hogaboam CM, Bowyer P, Niven RM. 2006. The link between fungi and severe asthma: a summary of the evidence. *Eur. Respir. J.* 27(3):615–26
211. Denning DW, O'Driscoll BR, Powell G, Chew F, Atherton GT, et al. 2009. Randomized controlled trial of oral antifungal treatment for severe asthma with fungal sensitization: the Fungal Asthma Sensitization Trial (FAST) study. *Am. J. Respir. Crit. Care Med.* 179(1):11–18
212. Sahakian NM, Park J-H, Cox-Ganser JM. 2008. Dampness and mold in the indoor environment: implications for asthma. *Immunol. Allergy Clin. North Am.* 28(3):485–505, vii
213. Sporik R, Squillace SP, Ingram JM, Rakes G, Honsinger RW, Platts-Mills TA. 1999. Mite, cat, and cockroach exposure, allergen sensitisation, and asthma in children: a case-control study of three schools. *Thorax* 54(8):675–80
214. de Blay F, Heymann PW, Chapman MD, Platts-Mills TA. 1991. Airborne dust mite allergens: comparison of group II allergens with group I mite allergen and cat-allergen *Fel d* I. *J. Allergy Clin. Immunol.* 88(6):919–26
215. Bollinger ME, Eggleston PA, Flanagan E, Wood RA. 1996. Cat antigen in homes with and without cats may induce allergic symptoms. *J. Allergy Clin. Immunol.* 97(4):907–14
216. Gelber LE, Seltzer LH, Bouzoukis JK, Pollart SM, Chapman MD, Platts-Mills TA. 1993. Sensitization and exposure to indoor allergens as risk factors for asthma among patients presenting to hospital. *Am. Rev. Respir. Dis.* 147(3):573–78
217. Arbes SJ, Gergen PJ, Elliott L, Zeldin DC. 2005. Prevalences of positive skin test responses to 10 common allergens in the US population: results from the third National Health and Nutrition Examination Survey. *J. Allergy Clin. Immunol.* 116(2):377–83

Annual Review of Environment and Resources

Volume 47, 2022

Contents

The Great Intergenerational Robbery: A Call for Concerted Action Against Environmental Crises
Ashok Gadgil, Thomas P. Tomich, Arun Agrawal, Jeremy Allouche, Inês M.L. Azevedo, Mohamed I. Bakarr, Gilberto M. Jannuzzi, Diana Liverman, Yadvinder Malhi, Stephen Polasky, Joyashree Roy, Diana Ürge-Vorsatz, and Yanxin Wang 1

I. Integrative Themes and Emerging Concerns

A New Dark Age? Truth, Trust, and Environmental Science
Torbjørn Gundersen, Donya Alinejad, T.Y. Branch, Bobby Duffy, Kirstie Hewlett, Cathrine Holst, Susan Owens, Folco Panizza, Silje Maria Tellmann, José van Dijck, and Maria Baghramian 5

Biodiversity: Concepts, Patterns, Trends, and Perspectives
Sandra Díaz and Yadvinder Malhi 31

COVID-19 and the Environment: Short-Run and Potential Long-Run Impacts
Noah S. Diffenbaugh 65

Shepherding Sub-Saharan Africa's Wildlife Through Peak Anthropogenic Pressure Toward a Green Anthropocene
P.A. Lindsey, S.H. Anderson, A. Dickman, P. Gandiwa, S. Harper, A.B. Morakinyo, N. Nyambe, M. O'Brien-Onyeka, C. Packer, A.H. Parker, A.S. Robson, Alice Ruhweza, E.A. Sogbohossou, K.W. Steiner, and P.N. Tumenta 91

The Role of Nature-Based Solutions in Supporting Social-Ecological Resilience for Climate Change Adaptation
Beth Turner, Tahia Devisscher, Nicole Chabaneix, Stephen Woroniecki, Christian Messier, and Nathalie Seddon 123

Feminist Ecologies
Diana Ojeda, Padini Nirmal, Dianne Rocheleau, and Jody Emel 149

Sustainability in Health Care
Howard Hu, Gary Cohen, Bhavna Sharma, Hao Yin, and Rob McConnell 173

Indoor Air Pollution and Health: Bridging Perspectives from Developing and Developed Countries
Ajay Pillarisetti, Wenlu Ye, and Sourangsu Chowdhury 197

II. Earth's Life Support Systems

State of the World's Birds
Alexander C. Lees, Lucy Haskell, Tris Allinson, Simeon B. Bezeng, Ian J. Burfield, Luis Miguel Renjifo, Kenneth V. Rosenberg, Ashwin Viswanathan, and Stuart H.M. Butchart 231

Grassy Ecosystems in the Anthropocene
Nicola Stevens, William Bond, Angelica Feurdean, and Caroline E.R. Lehmann 261

Anticipating the Future of the World's Ocean
Casey C. O'Hara and Benjamin S. Halpern 291

The Ocean Carbon Cycle
Tim DeVries 317

Permafrost and Climate Change: Carbon Cycle Feedbacks From the Warming Arctic
Edward A.G. Schuur, Benjamin W. Abbott, Roisin Commane, Jessica Ernakovich, Eugenie Euskirchen, Gustaf Hugelius, Guido Grosse, Miriam Jones, Charlie Koven, Victor Leshyk, David Lawrence, Michael M. Loranty, Marguerite Mauritz, David Olefeldt, Susan Natali, Heidi Rodenhizer, Verity Salmon, Christina Schädel, Jens Strauss, Claire Treat, and Merritt Turetsky 343

III. Human Use of the Environment and Resources

Environmental Impacts of Artificial Light at Night
Kevin J. Gaston and Alejandro Sánchez de Miguel 373

Agrochemicals, Environment, and Human Health
P. Indira Devi, M. Manjula, and R.V. Bhavani 399

The Future of Tourism in the Anthropocene
A. Holden, T. Jamal, and F. Burini 423

Sustainable Cooling in a Warming World: Technologies, Cultures, and Circularity
Radhika Khosla, Renaldi Renaldi, Antonella Mazzone, Caitlin McElroy, and Giovani Palafox-Alcantar 449

Digitalization and the Anthropocene
Felix Creutzig, Daron Acemoglu, Xuemei Bai, Paul N. Edwards, Marie Josefine Hintz, Lynn H. Kaack, Siir Kilkis, Stefanie Kunkel, Amy Luers, Nikola Milojevic-Dupont, Dave Rejeski, Jürgen Renn, David Rolnick, Christoph Rosol, Daniela Russ, Thomas Turnbull, Elena Verdolini, Felix Wagner, Charlie Wilson, Aicha Zekar, and Marius Zumwald 479

Food System Resilience: Concepts, Issues, and Challenges
Monika Zurek, John Ingram, Angelina Sanderson Bellamy, Conor Goold, Christopher Lyon, Peter Alexander, Andrew Barnes, Daniel P. Bebber, Tom D. Breeze, Ann Bruce, Lisa M. Collins, Jessica Davies, Bob Doherty, Jonathan Ensor, Sofia C. Franco, Andrea Gatto, Tim Hess, Chrysa Lamprinopoulou, Lingxuan Liu, Magnus Merkle, Lisa Norton, Tom Oliver, Jeff Ollerton, Simon Potts, Mark S. Reed, Chloe Sutcliffe, and Paul J.A. Withers 511

IV. Management and Governance of Resources and Environment

The Concept of Adaptation
Ben Orlove 535

Transnational Social Movements: Environmentalist, Indigenous, and Agrarian Visions for Planetary Futures
Carwil Bjork-James, Melissa Checker, and Marc Edelman 583

Transnational Corporations, Biosphere Stewardship, and Sustainable Futures
H. Österblom, J. Bebbington, R. Blasiak, M. Sobkowiak, and C. Folke 609

Community Monitoring of Natural Resource Systems and the Environment
Finn Danielsen, Hajo Eicken, Mikkel Funder, Noor Johnson, Olivia Lee, Ida Theilade, Dimitrios Argyriou, and Neil D. Burgess 637

Contemporary Populism and the Environment
Andrew Ofstehage, Wendy Wolford, and Saturnino M. Borras Jr. 671

How Stimulating Is a Green Stimulus? The Economic Attributes of Green Fiscal Spending
Brian O'Callaghan, Nigel Yau, and Cameron Hepburn 697

V. Methods and Indicators

Why People Do What They Do: An Interdisciplinary Synthesis of Human Action Theories
Harold N. Eyster, Terre Satterfield, and Kai M.A. Chan 725

Carbon Leakage, Consumption, and Trade
Michael Grubb, Nino David Jordan, Edgar Hertwich, Karsten Neuhoff, Kasturi Das, Kaushik Ranjan Bandyopadhyay, Harro van Asselt, Misato Sato, Ranran Wang, William A. Pizer, and Hyungna Oh 753

Detecting Thresholds of Ecological Change in the Anthropocene
Rebecca Spake, Martha Paola Barajas-Barbosa, Shane A. Blowes, Diana E. Bowler, Corey T. Callaghan, Magda Garbowski, Stephanie D. Jurburg, Roel van Klink, Lotte Korell, Emma Ladouceur, Roberto Rozzi, Duarte S. Viana, Wu-Bing Xu, and Jonathan M. Chase 797

Remote Sensing the Ocean Biosphere
Sam Purkis and Ved Chirayath 823

Net Zero: Science, Origins, and Implications
Myles R. Allen, Pierre Friedlingstein, Cécile A.J. Girardin, Stuart Jenkins, Yadvinder Malhi, Eli Mitchell-Larson, Glen P. Peters, and Lavanya Rajamani 849

Indexes

Cumulative Index of Contributing Authors, Volumes 38–47 889

Cumulative Index of Article Titles, Volumes 38–47 897

Errata

An online log of corrections to *Annual Review of Environment and Resources* articles may be found at http://www.annualreviews.org/errata/environ