

Indoor air pollution: a global health concern

Junfeng (Jim) Zhang* and **Kirk R Smith†**

**Environmental and Occupational Health Sciences Institute & School of Public Health, University of Medicine and Dentistry of New Jersey, NJ and †Environmental Health Sciences, School of Public Health, University of California at Berkeley, Berkeley, CA, USA*

Indoor air pollution is ubiquitous, and takes many forms, ranging from smoke emitted from solid fuel combustion, especially in households in developing countries, to complex mixtures of volatile and semi-volatile organic compounds present in modern buildings. This paper reviews sources of, and health risks associated with, various indoor chemical pollutants, from a historical and global perspective. Health effects are presented for individual compounds or pollutant mixtures based on real-world exposure situations. Health risks from indoor air pollution are likely to be greatest in cities in developing countries, especially where risks associated with solid fuel combustion coincide with risk associated with modern buildings. Everyday exposure to multiple chemicals, most of which are present indoors, may contribute to increasing prevalence of asthma, autism, childhood cancer, medically unexplained symptoms, and perhaps other illnesses. Given that tobacco consumption and synthetic chemical usage will not be declining at least in the near future, concerns about indoor air pollution may be expected to remain.

Long history

One of the basic human needs is shelter. Throughout human history, people have depended upon rock shelters, caves and rude huts to protect themselves from the vagaries of weather and climate. Today, some of these crude shelters can still be found in parts of the world, mainly in less developed countries. On the other hand, civilization has brought a significant fraction of the world's population to live in modern single and multifamily dwellings, work in modern office buildings, and carry out various activities in public facilities and other built environments that provide amenities and convenience far beyond the basic needs of sheltering.

People living in crude shelters have relied on crude fuels for their cooking and heating needs. The combustion of these crude biomass fuels—such as crop residues, animal dung and wood—generates smoke that adversely affects the health of occupants. Industrialization resulted in extensive

**Correspondence to:
Dr Junfeng (Jim) Zhang,
Environmental and
Occupational Health
Sciences Institute & School
of Public Health,
University of Medicine
and Dentistry of New
Jersey, 170 Frelinghuysen
Road, Piscataway, New
Jersey 08854, USA.
E-mail:
jjzhang@eohsi.rutgers.edu*

use of coal, a significant fraction of which was burnt indoors for space heating during winter months. The coal smoke emitted from household chimneys in London, for example, was the main cause of the Great London Smog in the winter of 1952—an event that led to thousands of excess deaths during a week-long episode. This and other similar events triggered the introduction of legislation banning domestic use of coal in many cities of developed countries. Fifty years later, however, coal combustion is still taking place in households worldwide, and indoor smoke from solid fuels (biomass and coal) is affecting something like half the world's households, today¹.

In modern residences, cooking and space-heating needs are usually met by fossil fuels such as natural gas, liquefied petroleum gas, heating oil (petroleum product) and electricity. Occasional carbon monoxide (CO) poisoning cases are reported, mainly as a consequence of the improper use or inadequate ventilation of appliances. Concerns have also been reported about exposure to nitrogen dioxide (NO₂) emitted from gas stoves². Compared to combustion of solid fuels, however, gaseous fuels in simple devices emit substantially smaller amounts of pollution, including particulate matter (PM), CO, eye irritating volatile organic compounds (*e.g.* aldehydes), and carcinogenic compounds such as benzene and 1,3-butadiene and polycyclic aromatic hydrocarbons^{3–5} (Table 1).

Although indoor air pollution from fuel combustion is therefore generally of lesser concern in modern homes and buildings, than in traditional homes in the developing world, there may nevertheless be important sources of exposure, related to materials used in construction, furnishing,

Table 1 Major health-damaging pollutants generated from indoor sources

Pollutant	Major indoor sources
Fine particles	Fuel/tobacco combustion, cleaning, cooking
Carbon monoxide	Fuel/tobacco combustion
Polycyclic aromatic hydrocarbons	Fuel/tobacco combustion, cooking
Nitrogen oxides	Fuel combustion
Sulphur oxides	Coal combustion
Arsenic and fluorine	Coal combustion
Volatile and semi-volatile organic compounds	Fuel/tobacco combustion, consumer products, furnishings, construction materials, cooking
Aldehydes	Furnishing, construction materials, cooking
Pesticides	Consumer products, dust from outside
Asbestos	Remodelling/demolition of construction materials
Lead ^a	Remodelling/demolition of painted surfaces
Biological pollutants	Moist areas, ventilation systems, furnishings
Radon	Soil under building, construction materials
Free radicals and other short-lived, highly reactive compounds	Indoor chemistry

^aPb-containing dust from deteriorating paint is an important indoor pollutant for occupants in many households, but the most critical exposure pathways are not usually through air.

furniture and consumer products. For example, asbestos had been widely used as insulating materials in buildings built before mid-1970s, when its use was banned in the USA and many other countries. These buildings may also contain lead-based paints. It is therefore important to protect workers and occupants from exposure to asbestos fibres and lead due to dust re-suspension during renovation or demolition of old buildings.

Since the 1970s, many energy-conserving buildings have been built in North America and Europe. Improved energy conservation was mainly achieved through reducing exchanges between outdoor fresh air and indoor air. Meanwhile, synthetic materials and chemical products have been extensively used in these airtight buildings. The combination of low ventilation rate and the presence of numerous sources of synthetic chemicals has resulted in elevated concentrations of volatile organic compounds (VOCs) (*e.g.* benzene, toluene, formaldehyde), semivolatile organic compounds (SVOCs) (*e.g.* phthalate plasticizers and pesticides) and human bioeffluents. This has been suggested as a major contributing factor to occupant complaints of illness symptoms, or so-called 'sick building syndrome', in the last three decades⁶. Although the aetiology is still not clear, many cases of respiratory diseases, allergies and asthma, medically unexplained symptoms including sick building syndrome, and cancer, are believed to be attributable to poor indoor air quality in both developing and developed countries^{7,8}.

Indoor smoke from burning solid fuels

Combustion of solid fuels in households often takes place in simple, poorly designed and maintained stoves. This kind of combustion contributes directly to low energy efficiency, adding pressure on fuel resources. Low combustion efficiency means that a large fraction of fuel carbon is converted to compounds other than carbon dioxide (CO₂)—*i.e.* products of incomplete combustion. These incomplete combustion products mainly comprise CO and fine (respirable) particles, as well as a large suite of VOCs and SVOCs^{3-5,9}. Daily exposure to products of incomplete combustion poses both acute and chronic health risks^{4,5}. A recent World Health Organization (WHO) report estimated that indoor smoke from solid fuels ranked as one of the top ten risk factors for the global burden of disease, accounting for an estimated 1.6 million premature deaths each year¹⁰ (see also Chapter 1). Among all environmental risk factors, it ranked second only to poor water/sanitation/hygiene (Fig. 1)^{10,11}. In this estimate, the burden of disease is defined as lost healthy life years, which includes those lost to premature death and those lost to illness as weighted by a disability factor (severity)¹². It needs to be recognized,

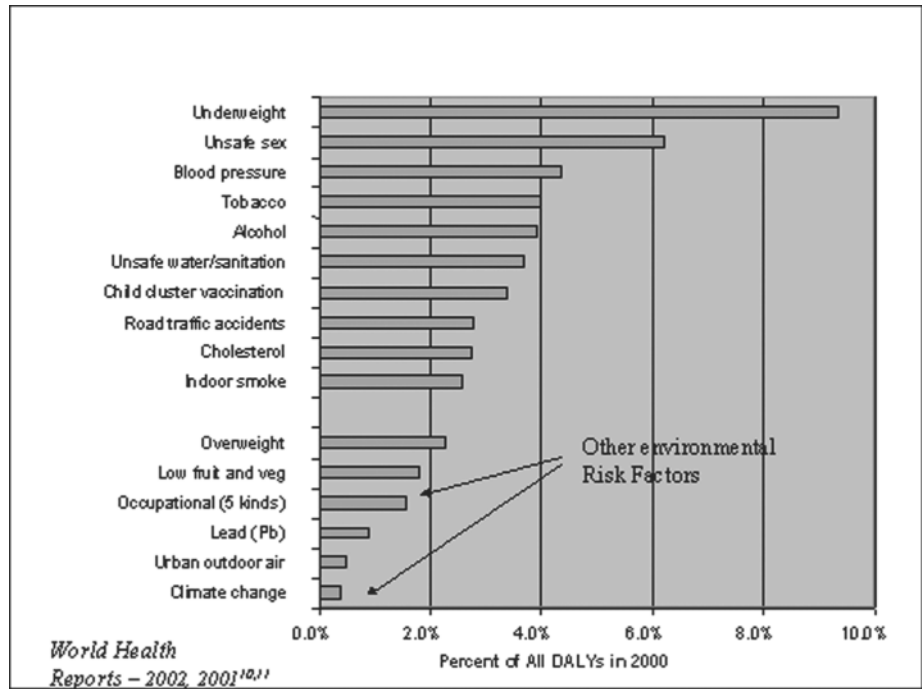


Fig. 1 Global burden of disease from the top 10 risk factors plus selected other risk factors^{10,11}. Note: Indoor smoke category here includes only solid fuel use in households and not smoke from other fuels or tobacco.

however, that such estimates are associated with relatively large uncertainties, because the data available on exposure and on exposure-effects relationships are limited, despite the apparently large risks and populations involved.

The existing literature provides strong evidence that smoke from solid fuels is a risk factor for acute respiratory infections (ARI), chronic obstructive pulmonary disease (COPD) and lung cancer (from coal smoke)⁸. Evidence from 13 studies in developing countries indicates that young children living in solid-fuel using households have two to three times more risk of serious ARI than unexposed children after adjustment for potential confounders including socio-economic status¹³. An evaluation of eight studies in developing countries indicates that women cooking over biomass fires for many years have two to four times more risk of COPD than those unexposed after adjustment for potential confounding factors¹⁴. Excess lung cancer mortality rates are reported in Chinese women who had been exposed to the smoke from household use of so-called ‘smoky coal’ which has high sulphur content and emits a large quantity of smoke and PAHs compared to other types of coals¹⁵. The existing epidemiological literature provides moderate evidence that solid

fuel smoke is a risk factor for cataracts, tuberculosis, asthma attacks and adverse pregnancy outcomes. Current literature in developed countries would also suggest that exposure to smoke from solid fuels produces cardiovascular disease, though no studies have been done to date in households in developing countries. More studies involving developing country households are needed to evaluate further the suggestive evidence that has so far been found⁸.

Household coal combustion produces the same kinds of pollutants as biomass fuels, but the amounts vary according to such parameters as the content of volatiles and fixed carbon. Depending on the geological conditions of coal formation, household use of coal can also produce differing levels of sulphur dioxide (SO₂) and certain toxic elements^{3,16}. Chronic fluorine and arsenic poisoning are, for example, particular problems in parts of China relying on local dirty coal deposits used for household fuel¹⁷.

Environmental tobacco smoke (ETS)

Tobacco smoke has been an accepted and well-documented cause of ill health for more than half a century. As shown in Figure 1, tobacco accounted for an estimated 4% of the global burden of disease in 2000, mainly as a result of active smoking exposure. However, there has been growing concern about the health effects of exposure to ETS, also called passive smoking, involuntary smoking, or second-hand smoking. ETS refers to the mixture of primary smoke exhaled by smokers and the secondary smoke produced by the burning tobacco between puffs. ETS exposure is lower than that experienced by active smokers, but the smoke is generally similar and contains the same gases and particles including a wide range of irritating compounds and carcinogens. Available data show that ETS exposure is a significant health risk factor in adults, children and infants (Table 2)^{18–26}. In addition to the health outcomes shown in Table 2, breast cancer²⁷ and pulmonary tuberculosis^{28,29} have been suggested, with limited evidence, to be associated with ETS exposure. In a recent cross-sectional study of 1718 school-age children whose mothers were never smokers, a monotonic exposure–response relationship was observed between paternal smoking and decline of pulmonary function³⁰. Estimates indicate that 3000 lung cancer deaths each year can be attributed to passive smoking in the USA along with hundreds of thousands of childhood respiratory disease cases³¹.

ETS exposure affects large numbers of people living in both developing and developed countries. Globally, ETS is perhaps the largest modern source of indoor air pollution, reflecting the alarmingly high levels of smoking prevalence (29% in adults worldwide)³². In each of the four

Table 2 Diseases and risks associated with ETS exposure

Illness	Population	Exposure assessment	Number of studies in meta-analysis	Relative risk			Reference
				Point estimate	95% CI _{low}	95% CI _{high}	
LRI	Children <3 years of age	Smoking by either parent	24 community and hospital based studies	1.57	1.4	1.77	18
Asthma	Children >1 year of age	Smoking by either parent	14 case-control studies	1.37	1.15	1.64	19
Otitis media (recurrent)	Children <7 years of age	Smoking by either parent	9 case-control, survey, and cohort studies	1.48	1.08	2.04	20
Ischaemic heart disease	Adults	Lifelong non-smokers married to smokers	17 studies	1.25	1.17	1.33	21
Lung cancer	Adults	Lifelong non-smokers with spouses who currently smoke	37 studies	1.24	1.13	1.36	22
Nasal-sinus cancer	Adults	Household exposure to passive smoking	3 studies (no meta-analysis)	2.3 ^a	1.7 ^a	3.0 ^a	23
LBW or SGA	Infants	Prenatal maternal smoking		–	Low 1.5	High 3.5	24
Sudden Infant Death Syndrome	Infants <1 year	Prenatal maternal smoking		–	1.8	2.4	25
LBW or SGA	Infants	ETS exposure of non-smoking mothers		–	1.1	1.3	26
Sudden Infant Death Syndrome	Infants <1 year	Postnatal maternal smoking		–	1.6	2.4	25

LRI = lower respiratory tract infection; LBW = low birth weight; SGA = small for gestational age.

^aRelative risk estimates for nasal-sinus cancer adapted from Ref. 23. See Health outcomes section for more details.

Chinese cities selected for an air pollution epidemiological study, more than 59% of fathers of school-age children were regular smokers, based on a survey³³ of >8000 households during 1993–1996. According to the US Centre for Disease Control and Prevention (CDC), nearly 3000 children under 18 become regular smokers every day in the USA. The CDC also estimates that in the USA active tobacco smoking causes more than 400,000 deaths each year and results in more than \$50 billion in direct medical costs annually. It is true that the direct health impacts of passive smoking are much smaller than those of active smoking. Efforts to control ETS, however, can have a much larger health benefit than just the reduction in ETS exposure itself, because they are also one of the best

avenues by which to change the social acceptance of smoking itself and to encourage smokers to quit.

Indoor inorganic contaminants

Inorganic gases found in contaminated indoor air include CO₂, CO, SO₂, NO₂, ozone (O₃), hydrogen chloride (HCl), nitrous acid (NHO₂), nitric acid vapour (HNO₃) and radon.

In residences where combustion appliances are present, major indoor sources of CO₂ are fuel combustion and occupants' expired air. Exposure to CO₂ itself is normally not a health concern. However, indoor CO₂ levels can be used as an indicator of the presence of other human bioeffluents in occupied facilities (*e.g.* office building, workshops, theatres, commercial buildings) and also are often employed as an indicator of whether the facilities have adequate ventilation. It is generally accepted that CO₂ levels inside an occupied building, when no combustion source is present, should be no more than 650 ppm above outdoor levels.

Household combustion is mainly responsible for elevated indoor levels of CO, SO₂ and NO₂. If properly operated and maintained, appliances that burn gaseous fossil fuels have high combustion efficiencies and thus generate insignificant amounts of CO. However, the high combustion temperature associated with gas combustion favours the formation of NO₂. Epidemiological studies suggest that long-term exposure to NO₂ (through the use of gas stoves) is a modest risk factor for respiratory illnesses compared to the use of electric stoves². The concern about CO, on the other hand, is primarily for its acute poisoning—*i.e.* its ability to bind strongly to haemoglobins (see Chapter 10). Acute exposure to high levels of CO from improperly operated and maintained appliances is the leading cause of poisoning death in USA and claims many lives worldwide³⁴.

Ozone (O₃) is a strong oxidizing agent. For this reason, some so-called air purifiers are intended to produce ozone indoors in order to 'purify' the air, on the largely erroneous belief that O₃ may 'kill' odorous contaminants³⁵. Besides this intentional indoor O₃ source, use of photocopiers and laser printers may generate O₃. However, the amount of O₃ created through typical office activities will normally not significantly elevate indoor O₃ concentrations. Exposure to O₃ may cause breathing problems, reduce lung function, exacerbate asthma, irritate eyes and nose, reduce resistance to colds and other infections, and speed up ageing of lung tissue. Importantly, indoor O₃, whether penetrating from the outdoors or derived indoors, can drive chemical reactions among chemical species present indoors, generating secondary pollutants that may be of greater health concern compared to primary pollutants³⁶. For example,

several terpenes are present indoors at concentrations several orders of magnitude higher than their outdoor concentrations because of the wide use of these terpenes as solvents in consumer products (*e.g.* *d*-limonene contained in lemon scented detergents, α -pinene contained in pine scented paints). Under typical indoor conditions, these terpenes can react with O_3 at a rate faster than, or comparable to, the air exchange rate, to form ultra-fine and fine particles, aldehydes, hydrogen peroxide, carboxylic acids, reactive intermediates, and free radicals including the hydroxyl radical³⁷⁻⁴⁰. The hydroxyl radical can, in turn, further react with almost all the organics present in the air. It is clear that some of the secondary products from O_3 -initiated indoor reactions are strong airway irritants and that exposure to ultra-fine and fine particles have respiratory and cardiovascular effects⁴¹. Therefore, it is not desirable, and can be problematic, to use ozone generators indoors because of the risk associated both with O_3 exposure and with secondary pollutants resulting from indoor O_3 chemistry.

Acidic gases such as HCl and HNO_3 are highly corrosive to materials and biological tissues. The main sources of HCl include outdoor-to-indoor transport and thermal decomposition of polyvinyl chloride (PVC). During a fire event, high exposure to HCl from PVC burning can be a real health concern. Major sources of indoor HNO_3 include penetration of outdoor HNO_3 formed in photochemical smog episodes and HNO_3 formed indoors *via* reactions involving O_3 , NO_2 and water vapour⁴². Normally, indoor HCl and HNO_3 concentrations are lower than their outdoor levels due to their high reactivity (loss to walls). Compared to HCl and HNO_3 , HNO_2 is less corrosive. However, indoor levels of HNO_2 can be substantially higher than outdoor levels and indoor concentrations of the other two acidic gases, due to the presence of a strong indoor source of HNO_2 resulting from heterogeneous reactions involving NO_2 and water films on indoor surfaces⁴⁰. Hence, combustion appliances are sources for both NO_2 exposure and HNO_2 exposure. Epidemiological studies of NO_2 health effects should consequently consider the potential confounding effects of HNO_2 and *vice versa*.

Radon-222, an odourless, colourless, and tasteless noble gas, is an isotope produced as a result of the decay of radium-226, which is found in the Earth's crust as a decay product of uranium. Radon has a half-life of 3.8 days and its decay produces a series of short-lived solid-phase daughter products over a few days until lead-210 is produced. These products tend to become associated with airborne particles. The effect of radon decay indoors, therefore, is to make fine particles slightly radioactive and thus expose lung tissue when they deposit during breathing. During the decay process, three types of radiation (α , β , γ) are emitted, all capable of ionizing atoms in living cells, leading to cell damage (see Chapter 13). However, most of the dose is to the respiratory tract and most of this

dose comes from the α radiation. Primary sources of radon in buildings are the soil beneath and adjacent to buildings, domestic water supplies (*e.g.* well water) and building materials. In soil, radon moves through air spaces between soil particles. The fraction of radon that enters soil pores depends on the soil type, pore volume and water content. This is why buildings on sandy or gravelly soils typically have higher radon levels than those on clay soils. Movement of soil radon into buildings is primarily through convection—*i.e.* driven by pressures due to indoor–outdoor temperature differences and pressures associated with winds. Hence, substantial seasonal and diurnal variations are typical in indoor radon concentrations. (Concentrations based on short-term measurements should thus be reported carefully because the measurements may not be representative of long-term cumulative exposure.) Highest radon concentrations in water have been found in drilled wells, particularly in areas with granitic bedrock containing uranium. Radon in groundwater is released when temperature is increased, pressure is increased, and/or water is aerated. (Showering provides optimum conditions for radon release from water.)

The primary concern about radon exposure is its potential to cause lung cancer, which has been shown in uranium miners and others. It has been reported that lung cancer risk is dependent on cumulative dose in a linear dose–response fashion. Although some ecologic studies have suggested links between radon exposure and other types of cancers, these have not been confirmed by data obtained from underground miners^{43,44}. The US Environmental Protection Agency (EPA) has set a guideline value for indoor radon level of 4 pCi/l measured as an annual average. It has been estimated that this guideline value is exceeded in approximately 6% of US residences and in approximately 30% of residences in Midwestern states. Estimated to cause 7–30 thousand deaths annually in the USA, radon exposure is the second leading cause of lung cancer, following smoking. Much of its damage actually occurs in smokers, however, because of a synergistic relationship between the two risk factors. Because radon contamination is naturally derived and imperceptible to human senses, its risk typically causes less alarm than other cancer-causing substances that may pose a significantly smaller risk.

In addition to gases and airborne particles, airborne fibres present indoors may pose health risks. Due to the known health effects of asbestos fibres, the use of asbestos in US buildings was banned nearly three decades ago. Avoiding asbestos fibres in old buildings is a top priority in indoor air quality management for those buildings. Although newer buildings do not contain asbestos, synthetic vitreous fibres (also referred to as man-made mineral fibres, glass fibres) can be found in spray-applied fireproofing, ceiling tiles, thermal insulation, sound insulation, fabrics, filtration components, plasters and acoustic surface treatments. Health

concerns relating to synthetic vitreous fibres arise when erosion of fibres occurs from the parent material into the air stream of buildings. Vitreous fibres have been suspected as possible causes of certain SBS symptoms and may cause irritation to the eye, skin, mucous membranes and respiratory tract⁴⁵. It is believed that cancer risks associated with typical building levels of vitreous fibres are low. Marked as a 'healthier' and greener alternative of vitreous fibres, the cellulose fibre is a recycled product made from newsprint. It contains boric acid for fire retardation. However, little information is available on the health effects of cellulose fibre exposure, although concerns have been raised about their potential to cause irritation to the mucous membranes and the upper respiratory tract. Concerns also arise when comparing cellulose fibres with sawdust that is composed mainly of cellulose, polyoses and lignin, as sawdust is classified as a known human carcinogen by IARC⁴⁵.

Indoor organic contaminants

Indoor organic contaminants are conventionally classified by volatility. VOCs have boiling points from <0°C to 240–260°C and are present in the gas phase at typical indoor concentrations. SVOCs have boiling points from 240–260 to 380–400°C, partitioning between the gas phase and the particulate phase under typical indoor conditions. Particulate organic matter comprises components of airborne/suspended dust, with boiling points >380°C.

Indoor organic compounds are released from a variety of building materials such as vinyl tile and coving: compounds include phthalate esters, 2-ethyl-1-hexanol), carpets (4-PCH, 4-VCH, styrene), linoleum (C₅–C₁₁ aldehydes and acids), particleboard (formaldehyde, other aldehydes, ketones) and power cables (acetophenone, dimethylbenzyl alcohol). A large variety of consumer products can contribute to indoor levels of VOCs and SVOCs, including paints (texanols, ethylene glycol, pinene, butoxyethoxyethanol), paint thinners (C₇–C₁₂ alkanes), paint strippers (methylene chloride), adhesives (benzene, alkyl benzenes), caulks (ketones, esters, glycols) and cleaners (2-butoxyethanol, limonene, 2-butanone). Other indoor sources of VOCs and SVOCs include frying foods (1,3-butadiene, acrolein, PAHs), smoking (nicotine, aldehydes, benzene, PAHs), dry cleaned clothing (tetrachloroethylene), deodorizers (*p*-dichlorobenzene), showering (chloroform), moulds (sesquiterpenes) and pesticides (chlorpyrifos, diazinon, dichlorvos). Due to the presence of these numerous indoor sources, many organic compounds are present indoors at concentrations substantially higher than outdoors. High indoor concentrations, coupled with the fact that people spend a larger fraction of time indoors, often make

the outdoor contribution to total personal VOC exposure insignificant or negligible^{46,47}.

Some VOCs and SVOCs are mutagenic and/or carcinogenic—for example, benzene, styrene, tetrachloroethylene, 1,1,1-trichloroethane, trichloroethylene, dichlorobenzene, methylene chloride and chloroform. Long-term exposure to these compounds is thus a concern in terms of cancer risks. Many VOCs and SVOCs found indoors have the potential to cause sensory irritation (*e.g.* aldehydes) and central nervous system symptoms (*e.g.* pesticides). Available studies also suggest that paternal exposure to VOCs (*e.g.* in chlorinated solvents, spray paints, dyes/pigments, cutting oils) during work, and maternal VOC exposures during pregnancy, are responsible for increased risk of childhood leukaemia⁴⁸. There was a conventional misconception that residential and office buildings have VOC concentrations typically two or more orders of magnitude lower than occupational standards or guidelines, in which case exposure to VOCs in residential and office settings would not be likely to be responsible for acute symptoms. However, there are several significant differences between workplace (industrial) exposure and residential/office exposure:

- 1 Personal protection (*e.g.* respirators, safety gargles) is normally used in workplace settings, but not in residences or offices. People usually spend longer time in residences (and offices) than in workplaces where VOC levels are high for a certain period of time. Therefore people may receive higher cumulative VOC doses in residences and offices.
- 2 Residential/office exposure affects a much larger population, including those individuals more susceptible to chemical exposure (*e.g.* asthmatics, children and the elderly).
- 3 Workplace exposure involves one or more known chemicals. However, residential/office exposure usually involves a complex mixture.

For measurement convenience, indoor VOC mixture is often characterized as total volatile organic compounds (TVOC). Indoor TVOC has been used as an indicator of building healthiness because the prevalence rate of SBS symptoms or complaints was suggested to correlate with TVOC concentration⁴⁸. The effectiveness of using TVOC as an indicator of indoor air quality, however, has been increasingly questioned recently, given that large differences in health effects exist among different individual VOCs and that different indoor environments may comprise distinct VOC mixtures.

Despite efforts made over the past two decades, the aetiology of SBS is still poorly understood. Other terminologies, such as medically unexplained symptoms and non-specific-building-related illness, have appeared in the literature, describing SBS symptoms or similar symptoms and illnesses⁴⁹. Non-specific-building-related illness (NSBRI) is characterized

by the following symptoms: mucous membrane irritation (ocular, nasal), headache, fatigue, shortness of breath, rash and odour complaints⁵⁰. Recent studies suggest that indoor pollutant mixtures, along with psychophysiological factors, may play an important role in causing NSBRI. Epidemiological investigations of building-related health complaints document multiple factors including VOCs, characteristics of the ventilation system, work-related stressors and gender as contributory to symptoms⁵¹. Laboratory animal studies suggest that formation of irritating particles from reactions between ozone and terpenes could contribute to the health effects of NSBRI⁴¹. Controlled human exposure studies have consistently shown that several hours exposure to a VOC mixture representative of typical problem buildings, compared to clean air, increased symptoms and complaints about the odour⁵²⁻⁵⁵. Objective effects of this VOC exposure have been demonstrated for neurobehavioural performance⁵², lung function⁵³ and nasal inflammation⁵⁶, in some but not all studies.

Exposure to SVOCs can occur not only *via* inhalation but, because they may be present in settled dust, also *via* ingestion. Ingestion of house dust is an important exposure route for small children who usually have frequent hand-to-mouth activities. It is suggested that exposure to plasticizer chemicals (*e.g.* diethylhexyl phthalate) may be partly responsible for the significant increase in asthma prevalence in the last two decades because the hydrolysis products of diethylhexyl phthalate cause bronchial hyper-reactivity in rats. It is also known that prostaglandin can mediate inflammatory responses such as those that cause asthmatic attacks, yet diethylhexyl phthalate and other phthalic acid esters have a similar chemical structure to prostaglandin. In addition, Finnish scientists reported an association between plastic interior surfaces and bronchial obstruction in young children⁴⁸. Another group of SVOCs commonly found indoors are pesticides. It has been demonstrated that furniture, stuffed toys and carpeting can serve as reservoirs of pesticides for weeks after application and that ingestion exposure can be the most important exposure route in children⁵⁷. It has been suggested that the extensive use of indoor pesticides may contribute to acute symptoms, cancer, immunological effects and reproductive effects. The magnitude of risk is not well known, however, due to the lack of data.

A global health concern in the future?

Given the wide range of indoor chemical contaminants highlighted above, it is clear that concern about indoor air pollution is nearly ubiquitous, although the pollutants of concern in modern buildings are different from those in solid-fuel-burning households. Nevertheless, indoor

air pollution fits within the risk transition framework⁵⁸, in which the traditional risks of household fuel combustion subside and modern risks from building materials emerge. As shown in the conceptual diagram in Figure 2, the absolute risk declines dramatically as traditional solid fuels are replaced, but the trend of relative risk (percent of total risk) is less clear because overall health risk declines as well. However, total risk is likely to peak in cities in developing countries where there is an overlap between the traditional and modern risks. In Figure 2, a question mark is placed at the arrow end of each curve, presenting questions on the future trends of traditional and modern risks of indoor air pollution. Whether indoor air pollution is a future health concern, therefore, would depend on the future trends of the two curves. Globally the traditional risk curve will approach zero as the use of solid fuels for household energy continues to decline. The modern risk curve, however, is more difficult to predict, but may be a cause for pessimism at least for the near future given that the consumption of tobacco and use of synthetic chemicals and materials do not seem likely to fall. Although society routinely substitutes new materials and chemicals for existing ones, the health effects of most of these are not fully understood before they have been put on the market. Chronic effects of cumulative exposure to low-level multiple chemicals have been recognized recently to be an important area of research. It is vital to understand whether this type of exposure has contributed to the increase in the prevalence of asthma, autism, childhood cancer and medically unexplained symptoms and to the apparent decline in human reproductive function (*e.g.* sperm counts) in some populations.

Unfortunately, health risks associated with indoor air pollution have not, in general, received adequate attention from the regulatory sector, building designers or even health professionals. Although the health and other benefits brought by switching the use of solid fuels to that of liquid or gas fuels, or just introducing improved stoves with chimneys, are tremendous and can well offset their cost⁵⁹, only one large-scale implementation effort has been made—the improved stove programme in China, which has introduced nearly 200 million stoves since the early 1980s⁶⁰. Just as clean water and sanitation at the household level have come to have high priority as primary health measures in all poor parts of the world, however, so should clean household fuels and ventilation.

Not well recognized by many observers in this context is the huge difference in what is called the ‘intake fraction’—that is, the fraction of material released that actually passes across the population’s body barriers and is thus swallowed or inhaled. (Previously called exposure factor, exposure efficiency, and exposure effectiveness, among other terms, it is now agreed to use the term ‘intake fraction’ for this concept⁶¹.) The intake fractions for typical air pollution sources vary by several orders

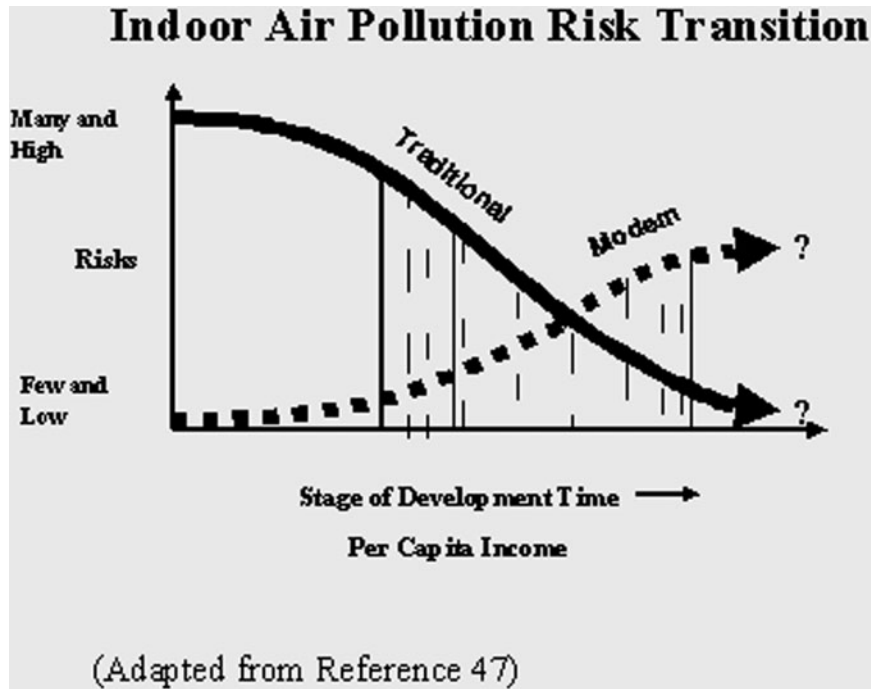


Fig. 2 Conceptualized indoor air pollution risk transition (adapted from Ref. 27).

of magnitude, for example from some 10^{-6} for a large power plant to 5×10^{-2} for a cigarette smoked indoors⁶². In general, one can use the 'rule of one thousand' (*i.e.* a gram of pollution released indoors produces about 1000 times more exposure than one released outdoors), although this obviously varies by situation^{63,64}. This means that the place (and time) of release for a pollutant is just as important in determining health effects as its toxicity, which also can vary by several orders of magnitude for different pollutants. Thus, to extend Paracelsus' famous dictum that the 'dose makes the poison', it is also true to say that 'the place makes the poison'⁶⁵. Consequently, small changes in indoor pollutant sources can have the equivalent health benefit as large changes in outdoor sources for the same pollutant.

Nazarof and Weschler state that '[In the USA], health risk assessment constitutes part of the basis for governmental action on environmental matters... Environmental regulations for carcinogens commonly aim to limit the individual lifetime risk of premature death to $\sim 10^{-6}$ – 10^{-5} for contaminants in drinking water and outdoor air. Yet, average lifetime risks of premature death from exposure to indoor air pollutants are at least $\sim 10^{-4}$ – 10^{-3} , and maximum individual risks exceed 10^{-2} . Does it make sense to spend large sums to mitigate environmental risks at a hazardous waste site to 10^{-6} when indoor air quality risks remain unchecked in the

range of 10^{-4} to 10^{-2} ?' Without the active participation of building designers who appreciate the importance of indoor air quality, it would be impossible to design and construct healthy buildings. Likewise, without the active participation of health scientists and professionals in resolving the puzzles of building-related medically unexplained symptoms and other illnesses, it would be impossible to develop reliable and effective guidelines to prevent excess health risks associated with poor indoor air quality.

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