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and Jon G. Ayres ESSENTIALS The term ‘occupational and environmental health’ includes any act of emission of any substance, likely to be hazardous in nature, which is either not originally present or is present in a higher concentration than normal in the natural atmosphere. Most air pollutants are gen- erated from human activities (e.g. energy, transportation, industry, agriculture), but natural events in the living (e.g. methane emissions in wetlands) and nonliving environment (e.g. volcanic eruptions) also contribute to atmospheric air pollution, although their relative im- portance has declined since the Industrial Revolution and the advent of modern fossil fuel-based economies. Pollutants may be classified as (1) primary (emitted directly into the atmosphere) or secondary (formed in the air through chemical reactions with other pollutants and gases); (2) indoor or outdoor; (3) gaseous or particulate. The main problem in determining health effects of individual air pollutants is that in real life they are never experienced in isolation, but many large studies have shown a link between air pollution and ad- verse cardiovascular and respiratory outcomes. Long-term exposure to air pollution, especially fine particulate, could also adversely af- fect other outcomes such as diabetes, impaired cognitive function, preterm birth, and lower birthweight. Air pollution—even at low levels—possesses significant health risk, particularly among sensitive individuals. The latest Global Burden of Disease Study 2013 estimated 2.9 million premature deaths could be attributed to ambient air pol- lution, of which 88% occurred in low and middle-income countries. Although by definition all air pollutants are hazardous, only the major ones (e.g. particulate matter, sulphur dioxide, nitrogen dioxide and ozone) in the atmosphere are monitored

and regulated by legis- lation in most countries. Ambient air pollution in high-income coun- tries has reduced significantly in the last few decades, but further reduction in pollutant levels will inevitably require very high cost. As low- and middle-income countries are experiencing fast economic growth, air pollution is likely to continue to be a significant con- tributor of global morbidity and mortality in the future. Introduction Concerns about air quality and its effects on health have been ex- pressed for many hundreds of years. The lime burners of the 1300s were blamed for polluting the air and John Evelyn in his famous diary railed against the polluted air of the City of London. The do- mestic pollution of those years was later compounded by the ad- vent of the Industrial Revolution in the 18th century, when the sulphurous emissions from factories were regarded as a necessary evil meaning, as it did, jobs and payment for the working popula- tion. This grudging acceptance remained until the London smog of 4–9 December 1952 when an increase in coal being burnt in do- mestic grates in a cold week together with a temperature inversion with a ‘blocking’ zone of high pressure accumulated pollutants at street level. Within a week, 4000 excess deaths occurred during

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1678 that six-day period. In fact, a re-assessment of the data suggested a further 13 500 deaths in the weeks and months that followed. As a direct consequence, the Clean Air Act of 1956 was passed, which re- sulted in a marked reduction in particulate pollution and to a lesser reduction in sulphur dioxide levels, such that in the late 1970s there was a general belief that air pollution and its potential health prob- lems had been conquered. However, in the 1980s a different type of air pollution—nitrogen dioxide, fine particulates, and hydrocar- bons arising from vehicle emissions—was realized. These pollutants are not only harmful in their own right but also act as a substrate for the formation of ozone, the combination of all these pollutants comprising the photochemical pollution so characteristic of Los Angeles, Athens, and Mexico City, and increasingly commonly in East and South Asia. As a result, urban dwellers worldwide are now facing long-term exposures to air pollution. As we spend most of our lives indoors, indoor air quality is clearly equally important. In high-income countries as well as in urban areas of low- and middle-income countries (LMICs) most indoor levels of pollutants are driven by outdoor levels. In such settings tobacco smoke becomes the most important source of indoor pol- lutant, exposure to which has been shown extensively to impart ad- verse health effects. However, in the rural and less developed parts of the LMICs combustion products of solid fuels, derived from bio- mass (e.g. wood, animal dung, agricultural residues) and coal for heating and cooking purposes, dominate the scene. It has been esti- mated that nearly one-third of the world’s population are relying on solid fuels, most of whom use traditional unventilated stoves of low efficiency, producing large quantities of pollutants, particularly par- ticulate matter and carbon monoxide. While there has been ample evidence to suggest the global burden of mortality and morbidity associated with household air pollution is substantial and compar- able to ambient air pollution, it has not received adequate attention until recently. At present the extent of the health effect(s) of these pollutants, alone or in combination, are a continuing matter for research and debate. Although acute effects have often been deemed to be small, as more studies of health effects of long-term exposure are under- taken, the true picture of the burden due to air pollution is begin- ning to unravel. Sources and types of air pollutants Very often the term ‘air pollution’ is understood to be an act of emis- sion of any substance, likely to be hazardous in nature, which is either not originally present or is present in a higher concentration than normal in the natural atmosphere. While it is true that most air pol- lutants are generated from human activities (e.g. energy, transporta- tion, industry, agriculture),

natural events in the living (e.g. methane emissions in wetlands), and nonliving environment (e.g. volcanic eruptions, lightning strikes, natural radioactive decay) also con- tribute to atmospheric air pollution, but their relative importance has declined since the Industrial Revolution and the advent of modern fossil fuel-based economy, at least in high-income countries. Given the loose definition, it is not surprising that a long list of substances could be defined as air pollutants. They can be classified by number of ways, and Table 10.3.1.1 presents three commonly used criteria with further subclassifications. In the urban environment, the concentration of airborne pollu- tants is usually higher outdoors compared to indoors because of the high volume of vehicular emissions. However, improved thermal insulation and the use of air conditioning systems in modern dwell- ings may also facilitate the accumulation of indoor pollutants, par- ticularly when people smoke in their homes. On the other hand, in LMICs, especially in the rural areas, indoor pollutant level can be many times higher than the outdoor environment because of the use of solid fuels for cooking and heating, a common phenomenon in unventilated dwellings. Pollutants can also be grouped by their physical properties, often by their state and size. Under such a classification scheme particu- late matter (PM), sometimes known as total suspended particulate, is a mixture of solid particles and aerosols suspended in the air with Table 10.3.1.1 Major classifications of air pollutants A. Primary–secondary pollutants a. Primary: Pollutants emitted directly into the atmosphere (e.g. SO2, some NOx species, CO, PM) b. Secondary: Pollutants that form in the air as a result of chemical reactions with other pollutants and gases (e.g. O3, NOx, and some PM) B. Indoor–outdoor pollutants a. Indoor pollutants i. Sources: Combustion, cooking, heating, smoking, particle resuspension, building materials, air conditioning, consumer products, biological agents ii. Products: Combustion products (e.g. tobacco and solid fuel), CO, CO2, sVOC (e.g. aldehydes, alcohols, alkanes, and ketones), microbial agents and organic dusts, radon, manmade vitreous fibres b. Outdoor pollutants i. Sources: Industrial, commercial, transportation, agricultural, natural, transboundary ii. Products: SO2, O3, NOx, CO, PM, VOC, sVOC C. Gaseous–particulate pollutants a. Gaseous: SO2, NOx, ozone, CO, VOC, sVOC (e.g. PAHs, dioxins, benzene, aldehydes, 1,3-butadiene) b. Particulate: Coarse or thoracic particles (aerodynamic diameter 2.5–10 μm; regulatory standard = PM10), fine PM, or respirable (0.1–2.5 μm; regulatory standard = PM2.5); ultrafine PM (<0.1 μm; not regulated) SO2, sulphur dioxide; NOx, oxides of nitrogen; CO, carbon monoxide; CO2, carbon dioxide; sVOC, semi-volatile organic compounds; PM, particulate matter; PAHs, polycyclic aromatic hydrocarbons. Pollutants such as particulate matter (PM), sulphur dioxide (SO2), and carbon monoxide (CO) are examples of primary pollutants which, as combustion product of fossil fuels, are released into the atmosphere directly from sources. Secondary pollutants are formed by primary pollutants in the atmosphere. One such example is ground (tropospheric) level ozone (as opposed to stratospheric ozone, which is naturally occurring), formed by chemical reactions of oxides of nitrogen and hydrocarbons in the presence of sunlight. Ozone is one of the main constituents of the photochemical smog, which is typical in cities with many motor vehicles on warm sunny days. From Bernstein JA, et al. (2004). Health effects of air pollution. Journal of Allergy and Clinical Immunology, 115(5), 1116–23, with permission from Elsevier.

 variations in size, composition, and origin. The major components of PM are inorganic compounds such as sulphates, nitrates, am- monia, black carbon, sea salt, and mineral dust, and organic sub- stances including pollen and mould. Coarse or ‘inhalable particles’ have an aerodynamic diameter of ≤10 μm (known as PM10), are chiefly derived from attrition of larger particles particularly from abraded soil, road dust (from brake and tyre), construction debris, or also from aggregation of smaller combustion particles which can be

inhaled to the lower respiratory system. Fine or ‘respirable par- ticles’ (aerodynamic diameter of ≤2.5 μm; PM2.5) are a subset of the PM10 fraction, which can penetrate deep into the lungs through the bronchioles to the alveoli where gas-exchange takes place. Ultrafine particles are less than 100 nm in size, and may even penetrate into the systemic circulation. Measurement of a health effect The main problem in determining health effects of individual air pollutants is that in real life they are never experienced in isolation. There are multiple potential interactions, not only with other air pollutants, but with other factors such as weather conditions (as in the 1952 London smog), levels of airborne allergen, the presence of a respiratory tract infection, exercise, diet, socioeconomic status, and actively or passively inhaled cigarette smoke. Some health ef- fects may also be attributed to multiple risk factors at a time and hence making determination of specific, pollutant-induced health effects difficult. There are three main ways whereby a health effect can be assessed: animal studies, challenge studies in humans, and by epidemiological studies (including cross-sectional, time series, case-crossover, and cohort studies). Each has their problems but when determining na- tional ambient air quality standards all available types of data may be taken into consideration. When determining national ambient air quality standards, the aim is to try and define a no observable effect level (NOEL), that is, the level of a specific pollutant where no health effect can be demonstrated. The rough rule of thumb that has been used is to take as the standard a level one-tenth of the NOEL obtained from human chamber/challenge studies or one-hundredth of the NOEL determined by animal studies. Risk assessment is difficult and very largely arbitrary. Although individual risks are relatively easily measurable in the workplace, when considering am- bient airborne pollution individual risk and public health load has not been very well characterized to date. This is partly because the health effects of outdoor air pollution are considerably less than the risks of active cigarette smoking or the risks of other well-recognized aetiological factors (e.g. high blood pressure). Moreover, potential sources of confounding, primarily in terms of socioeconomic status and cigarette smoking might have affected the interpretation of the health effects of air pollution. Ambient (outdoor) air pollution and health effects Large-scale multicity and indeed multinational studies, such as the early Harvard Six Cities, American Cancer Society cohort, and Adventist Health Air Pollution (AHSMOG) studies in the United States, and the more recent NMMAPS (National Morbidity Mortality Air Pollution Study), APHENA (Air pollution and health: a European and North American approach), PAPA (Public health and Air Pollution in Asia), and ESCAPE (European Study of Cohorts for Air Pollution Effects) studies have provided evidence showing the link between air pollution and adverse cardiovascular and respiratory outcomes (including ischaemic heart disease, stroke, heart failure, asthma, and chronic bronchitis), particularly in terms of excess mortality associated with short- and long-term exposures. Recent evidence suggests long-term exposure to air pollution, es- pecially fine particulate, could also adversely affect other outcomes such as diabetes, impaired cognitive function, preterm birth, and lower birthweight. Indeed, the latest Global Burden of Disease Study 2017 estimated 4.2 million premature deaths could be attributed to ambient air pol- lution in 2015, of which more than 80% occurred in LMICs; the ma- jority of them in the World Health Organization (WHO) Western Pacific and Southeast Asia regions. However, as most of the studies investigating health effects to date have been done in high-income countries where the current air pollutant level is 15–20-fold lower than that in LMICs, it raises the question whether concentration-response functions, primarily derived from high-income countries, could be extrapolated to estimate the burden in LMICs. More importantly, it is extremely difficult to ascertain and quantify the level and duration of exposure to airborne pollutant at both population and individual levels with high accuracy and precision. Coexposure at the same time to another pollutant will likely affect any physiological response as

will the presence of cofactors such as age, individual susceptibility (pres- ence of comorbidities, concurrent respiratory tract infection, degree of bronchial responsiveness), smoking, physical activity (which in- creases effective lung dose due to enhanced ventilation) or diet. Such variations within and between individuals have added the complexity in interpreting the findings from previous literature. Although health effects of air pollution could be at times difficult to assess and to ascertain from observational studies, interventions (often associated with pollution abatement legislations) have pro- vided indirect evidence to support the link between air pollution and adverse health outcomes. A ban on ‘smoky’ coal sale and burning in Ireland, first introduced in Dublin in 1990 and subsequently ex- tended to cities and towns nationwide, has resulted in substantial reductions in cardiorespiratory hospital admissions and respiratory mortality. Associations between improved air quality and reduc- tion in mortality have also been reported following the reduction of sulphur content of fuel in Hong Kong in 1990. On the other hand, conflicting findings have been reported in studies investigating the effect of low emission/congestion charging zones on pollutant levels. Nevertheless, a study in Rome estimated that residents living near main roads have gained 3.4 days per person following the intro- duction of low emission zones in 2006. Transient changes in air quality have also been demonstrated to have measurable population health benefits. During the 2008 Olympic and Paralympic Games (July–September) the Chinese government implemented a series of aggressive pollution control measures in Beijing and surrounding areas, including restriction or complete shutdown of industrial and construction operations and an alternative-day driving scheme. As a result, large reductions in gaseous and particulate pollutant levels were achieved during this period (-13% to -60%) compared with the pre-Olympic period. During this time, there was a significant reduc- tion in levels of inflammatory and thrombotic biomarkers in healthy

 young adults living in Beijing, but reverting to pre-Olympic period levels as the control measures were relaxed after the Paralympic Games. A reduction in cardiovascular mortality was also observed in the same period. However, it is important to note that such major athletic events inevitably introduce disturbances to the normal city life, such that changes in health events might not have been entirely explained by changes in air quality. In the following paragraphs health effects of the major air pollu- tants will be described in some detail. Sulphur dioxide (SO_2) SO_2 comes from the combustion of sulphur-containing fossil fuel, usually in power stations and ocean-going vessels. Emissions in high-income countries have been reduced drastically thanks to the availability of low-sulphur fuel and improvement in flue-gas desul- phurization. However, in countries with high reliance on coal, SO_2 continues to be a major pollutant and precursor to acid rain. Controlled human challenge studies in both asthmatic and healthy individuals have reported that around 5 minutes’ exposure to inhaled SO_2 induces rapid onset bronchoconstriction resulting in decrease in FEV1 or increase in airway resistance within 2 min- utes of exposure. SO_2 causes tracheitis and reduces ciliary func- tion in vitro, while in vivo it can cause bronchoconstriction in exercising asthmatics at levels experienced in urban areas today. Nonasthmatic individuals, particularly those who are atopic, will also develop bronchoconstriction with SO_2 on exercise but only at significantly with higher exposures (≥1300 μg/m3). This sen- sitivity to the effects of SO_2 could have a genetic component as a recent study among asthmatic patients spirometrically responsive (>12% decrease in forced expiratory volume in 1 s (FEV1)) to ex- posure to SO_2 reported significant association between the TNFα promotor polymorphism and asthma. It has been estimated that 25% of asthmatics are sensitive to less than 0.5 ppm of SO_2 while exercising. Although among

healthy adults the effect is usually short lived and there is a spontaneous recovery within 30 minutes of initial ex- posure, in asthmatic patients, recovery could take up to 4 hours. On the other hand, a meta-analysis of time series studies (focusing on short-term effect) has found positive association in terms of hospital admission as well as mortality for both cardiovascular and respira- tory health outcomes. Many studies worldwide have shown chronic effects of sulphur dioxide on absenteeism, respiratory symptoms, and on prevalence and mortality of chronic obstructive pulmonary disease (COPD). Particulate matter (PM10 and PM2.5) As described previously, PM comprises a wide range of particles or aerosols of different sizes and composition. Recent studies have shown PM to have specific health effects independent of SO2, which is often emitted simultaneously and the individual effects of which were previously thought to be difficult to disentangle. In high- income countries traffic emission, particularly diesel exhaust, is the main source of PM. It has been demonstrated that particle size is negatively related to the extent of adverse health effect, probably ex- plained by the high surface area to volume ratio in smaller particles, which encourages adsorption of more toxic compounds at a given mass. The small size also enables the particles to enter the systemic circulation and into tissues and organs. Human challenge and animal model studies have suggested that exposure to diesel exhaust has potent inflammatory effects involving lung epithelial cells and alveolar macrophages. Healthy volunteers ex- posed to 300 µg/m3 diesel exhaust or particulates for one hour in a controlled chamber have been shown to have increased neutrophil counts in sputum and bronchial biopsy specimens and increases in IL- 6, IL-8, and growth-related oncogene α-levels, with minimal change in lung function. In patients with mild asthma, exposure to diesel can increase airway hyperresponsiveness to methacholine and airway re- sistance. Longer-term (3 months) of higher dose of diesel exhaust in animals increased airway hyperresponsiveness significantly but the ef- fect disappeared when the animals were relocated to cleaner environ- ment for further 3 months. On the other hand, the mechanism leading to adverse cardiovascular health outcome is relatively unclear. It has been reported that PM exposure is associated with up-regulation of fibrinogen and platelet levels, while sequestration of red blood cells in the lung could increase the risk of cardiac arrhythmia. Epidemiological studies, particularly multicity studies, have provided substantial evidence to suggest short- and long-term ex- posure to PM10 and PM2.5 results in increase in cardiorespiratory emergency room visits, hospital admissions and mortality (mainly ischaemic heart disease and stroke). Pooled analysis from 110 time series studies reported for a 10 µg/m3 increase in PM2.5 concentra- tion there is an 1.5% increase in daily respiratory and 0.8% increase in cardiovascular mortality, respectively. With regard to the effects of long-term exposure, the multicentre ESCAPE used data from 22 European cohorts and found 7% increase in all natural cause mor- tality per 5 µg/m3 increase in PM2.5. There is also support for links between long-term PM2.5 exposure and other endpoints, including adverse birth outcomes (low birth weight, preterm birth, and small for gestational age), childhood respiratory disease, and impaired neurodevelopment and cognitive function. Oxides of nitrogen (NOx) The major oxides of nitrogen, nitric oxide (NO), nitrous oxide (N2O), and nitrogen dioxide (NO2) are regarded together as NOx, but it is NO2 that receives most attention as the other two have no known deleterious health effects. While one-third of ambient NO2 comes from diesel engines, the highest exposures to ambient NO2 for most individuals, however, are seen indoors in gas-fired kitchens, where levels may reach 900 µg/m3 for short periods. A more usual indoor level is 190 µg/m3 (100 ppb) compared to an outdoor urban level of around 60 µg/m3. The effects of ambient levels of NO2 is a matter of much debate as some question whether NO2 is merely an indicator of other pollu- tants (particularly PM). Oxides of nitrogen can react with ammonia, moisture, and other compounds to form small particles and is

also a precursor to photochemical smog. An early study showed that 190 µg/m3 NO2 increased airway responsiveness in exercising asth- matics, but subsequent studies, even up to exposures of 700 µg/m3, showed no effect. In a controlled chamber study, healthy smokers exposed to high levels of NO2 had induced inflammatory response in the airway that was characterized by neutrophil influx and re- duced lymphocytes count. NO2 at very high levels are often only present in occupational settings, and can cause acute pulmonary oedema. Cross-sectional and short-term longitudinal epidemio- logical studies have shown NO2 to be associated with airway in- flammation in healthy subjects and increase in respiratory symptoms

10.3.1 Air pollution and health 1681 in asthma patients. Susceptible individuals living close to main roads are likely to have worsened respiratory symptoms leading to in- creased hospital admissions and premature mortality. Similar to PM, there is also evidence that long-term exposure to NO2 is linked to increased cardiovascular emergency admissions and mortality, mainly due to stroke and myocardial infarction. A pooled ana- lysis from 110 time series studies showed per unit µg/m3 increase in NOx would increase the risk of all-cause, cardiovascular and respiratory mortality by 0.17% (0.12–0.23%), 0.11% (–0.12–0.35%), and 0.15% (–0.29–0.59), respectively. Ozone (O3) Ozone is a highly reactive molecule which is formed by the action of ultraviolet light on NOx and hydrocarbon fragments emitted by ve- hicles. It is formed mostly during the summer months and tends to build up over a prolonged warm spell. Levels are often higher in rural areas downwind from cities as urban nitric oxide neutralizes ozone while the formation of ozone takes a little time as polluted air is taken downwind. It is also probable that ozone enhances the formation of aerosol strong acid, thus significantly affecting the ambient pollutant mix. There is a diurnal variation with peak levels being achieved in late afternoon. In winter, ozone levels are almost unmeasurably low but in summer can exceed hourly values of 200 µg/m3. Ozone causes inflammatory changes (mainly neutrophilic) in the bronchial mucosa at levels as low as 160 µg/m3, at which level changes in forced vital capacity and FEV1 can be detected after an exposure of 1 hour and can persist for up to 24 hours. Individual responses to chal- lenge with ozone vary widely between normal and asthmatic individ- uals, but asthma patients appear not to be more sensitive to the effects than normal subjects. It is possible that women are more susceptible. Repetitive exposures seem to produce a lesser response suggesting a latency effect which is difficult to understand in view of the known inflammatory effect of this gas. Summer peaks of ozone have been associated with peaks in hospital admissions for asthma in several areas of North America. Ozone at levels which do not cause an effect on airway function can cause bronchoconstriction when patients are pre-exposed to usual ambient SO2 which suggests a potentiating effect of air pollutants, which has great logical appeal. Ozone at 240 µg/m3 has also been shown to enhance the bronchoconstrictor response to inhaled allergen which again reinforces the very likely possibility that current air pollutants exert their effect in a permissive way. The large multicentre APHENA study has provided data on short-term effects of ozone. It found statistically significant associations for 1-hour ozone and all-cause mortality and cardiovascular mortality. Recent cohort analyses suggest an effect of long-term exposure to ozone on mortality, at least for respiratory or cardiorespiratory mor- tality, especially in people with potential predisposing conditions. Polycyclic aromatic hydrocarbons (PAHs) These form part of the total hydrocarbons, a wide range of com- pounds most of which have no known human health effect. When considering total hydrocarbons methane is usually excluded, the re- maining hydrocarbons largely comprising alkanes. Because of their association with PM, total hydrocarbons are usually expressed as parts per billion of carbon. PAHs are partitioned between the par- ticulate and gas phase, the lower molecular weight molecules being in the gas phase. The carcinogenicity

of PAHs increases with the in- crease of their molecular weight, but reduction in acute toxicity. The main sources of airborne PAHs in the outdoor environment are from evaporation of solvents and fuels and from combustion of car- bonaceous materials at high temperature, predominantly in emissions from motor vehicles. Of the different PAH species, benzene and, to a lesser extent, benzo [a]anthracene, benzo[a]pyrene and dibenz[ah] anthracene are known to be carcinogenic. With introduction of stricter guidelines, current ambient B[a]P concentration has reduced from around 100 ng/m3 in 1945 down to 0.3 ng/m3 in high-income countries. However, the major exposure to PAHs in human is tobacco smoking and burning of fuels, particularly in an indoor environment. Benzene is present in significant quantities in cigarette smoke and, on average, cigarette smokers take in about 2 mg/day compared with less than 0.2 mg/day for most nonsmokers, although passively exposed nonsmokers will be exposed to approximately 60% more benzene than nonpassive nonsmokers. Short-term exposure to PAHs causes impaired lung function in asthmatics and thrombotic effects in people with major coronary heart diseases. Individuals in occupational set- tings exposed to air pollutants with a mixture of PAHs for long dur- ations have reported increases in incidence of skin, lung, bladder, and gastrointestinal cancers, as well as a raised risk of cell damage via gene mutation and cardiopulmonary mortality. Carbon monoxide (CO) The major source of CO for cigarette smokers is, and will remain, cig- arette smoke. For nonsmokers, including children, vehicle emissions passively inhaled cigarette smoke, and indoor cooking are the main contributors. Exposures are far greater in tunnels, car parks, garages, and in dense, slow moving traffic. Kerbside levels in towns are of the order of 20 μg/m3 which, with chronic exposure, would produce a carboxyhaemoglobin level of around 3%. WHO guidelines aim to keep blood levels of carboxyhaemoglobin to less than 2% in non- smokers. In nonsmokers levels of carboxyhaemoglobin rarely exceed 3%. CO exerts its toxic effect not just by the formation of carboxy- haemoglobin but also because it shifts the oxygen dissociation curve to the left. As a result, blood levels of around 3.6% will reduce the time to onset of angina on exercise. The fetuses of smoking mothers might have carboxyhaemoglobin levels up to 2.5 times that of the mother. How the fetus is able to concentrate CO and what the health effects might be to the fetus are unknown. Recent studies have reported that exposure to CO is likely to reduce the maximal exercise capacity in healthy young individuals and hence reduces the time to angina and, in some cases, the time to ST-segment depression in people with car- diovascular disease, albeit at a concentration that is lower than that needed to reduce exercise capacity in healthy individuals. Indoor air pollution and health effects While common air pollutants are often released to the atmosphere from traffic, power stations, and factories, this is not to say air pollu- tion exists only outdoors. It could easily be forgotten that even in in- door setting, where people could spend up to 90% of their time, there are outdoor–indoor transfers, as well as indoor emission sources, and because they are confined environments concentration of pollutants could be much higher indoors than outdoors. Indoor air quality can be considered with respect to the domestic and occupational set- tings. In high-income countries, domestic exposures are predomin- antly PM, CO, NO2, PAHs, and volatile organic compounds due to

 combustion (tobacco smoke and fuel for cooking/heating), domestic activities (cleaning and cooking), and building and furnishing ma- terials (adhesives and radon) and biological pollutants (allergens and moulds). From the occupational point of view, specific expos- ures which lead to occupational asthma are dealt with elsewhere but nonspecific problems, such as the ‘sick building syndrome’ will be covered next in brief. In LMICs, however, poor indoor air quality is related to the burning of

solid fuels using inefficient stoves, which produce very high concentrations of PM, CO, and other combustion products, often many folds higher than what is measured in the am- bient atmosphere. This household air pollution affects one-third of the world's population and is therefore a major cause of morbidity and mortality globally. It is estimated that in 2013, 2.9 million pre- mature deaths and 81.1 million disability adjusted life years (DALYs) can be attributed to household air pollution. India and China alone accounted for 60% of the total premature deaths. Control of indoor pollution is contentious. Most attempts are aimed at increasing room ventilation rates, which have shown sig- nificant improvements in odour levels and occupant satisfaction while air filtration, although attractive, has yet to be shown to be as effective. In LMICs where household air pollution dominates, a wide range of interventions, including use of cleaner alternative fuels (e.g. biogas), replacement of traditional cook stoves with more efficient ones, improving ventilation (e.g. retrofitting chimneys), and change in use behaviour (e.g. keeping children away from smoke), have been implemented with varying levels of success. Ascertainment and quantification of exposure indoors is more difficult than outdoors because there exist very large variations in pollutant levels across different microenvironments (e.g. rooms within the same dwelling) and exposure between and within indi- viduals, determined by several factors including proximity to emis- sion source, permeability and ventilation of the microenvironment, and time-activity pattern of individuals. Consequently, personal ex- posures estimated from detailed diary of indoor and outdoor time-activity patterns will give a better idea of time exposure to specific pollutant(s) when trying to estimate a health risk. Selected indoor exposures in high-income countries Tobacco smoke The most important indoor pollutant is tobacco smoke from the smouldering tobacco and exhaled by smokers. Known as environ- mental tobacco smoke or second-hand smoke, it is a mixture of toxic and carcinogenic chemicals and particulates. A smoker of 20 cigar- ettes a day contributes about 20 µg/m3 to 24-h indoor particulate concentrations, and in a house containing several heavy smokers the 24-h ambient air quality standard of 50 µg/m3 for PM10 can easily be exceeded. Those smokers are exposed not only to mainstream smoke (inhaled through the mouthpiece) but also to the environmental tobacco smoke they generate (also known as sidestream smoke), which is more toxic on a weight for weight basis compared to main- stream smoke. As a consequence, the adverse health effects of ex- posure to environmental tobacco smoke are similar to those caused or exacerbated by direct smoking. In adults, cancers (particularly lung cancer), cardiovascular diseases (ischaemic heart disease and stroke), and respiratory diseases (COPD, onset and exacerbations of asthma, and respiratory tract infections) are the main causes of mortality and morbidity. Associations with other conditions such as cognitive impairment, degenerative eye disease, and mental ill health have been reported. Its irritant effect should not be ignored as this is likely to be the reason most people object to passive smoking. The effect of the irritation per se has no known long-term physical effects, but the effect on quality of life at home (or work) can be con- siderable. In neonates and children, environmental tobacco smoke could have developmental effects (low birth weight, preterm birth, and sudden infant death syndrome) and respiratory effects (retarded lung function growth, lower respiratory tract infection, respiratory symptoms, and asthma onset and exacerbations). Recently a new concept of 'third-hand smoke' has been postulated, which refers to tobacco smoke residue that settles onto surfaces and dust and re-emitted and re-suspended into the air. While currently there is no direct evidence of the harmful effect of this third-hand smoke, tobacco-specific lung carcinogen 4-(methylnitroasmino)-1- (3-pyridyl)-1-butanone (NNK) has been found to be present on sur- faces in most homes occupied by smokers, providing rationale for further research in this field. An updated Cochrane review published in 2016 examined the ef- fects of implementing smoking

bans and found consistent evidence of improving cardiovascular health outcomes and reducing mortality for associated smoking-related illnesses. Several countries have ex- tended the smoking ban to private vehicles in the presence of chil- dren. The impact of such legislation is still too early to be assessed. Electronic cigarettes (e-cigarettes) have gained popularity in re- cent years, either as a substitute of cigarettes or as an aid for smoking cessation. While these battery-powered nicotine vaporizers do not emit combustion products of tobacco, the aerosols produced by e-cigarettes not only significantly increase indoor PM2.5 concen- trations (up to 300 times), but can also contain toxicants including glycols, aldehydes, metals, volatile organic compounds, and PAHs, some of which do not appear in cigarette smoke. Data on the health effects of exposure to e-cigarette aerosols have been sparse. In vitro studies have shown increases in oxidative stress and decreases in epi- thelial cell viability 24 h after e-cigarette aerosol exposure compared to clean air controls. Results from small-scale preclinical studies have been conflicting. Cooking emissions Natural gas cooking appliances are a source of indoor NO2 and CO and contribute to deteriorated air quality when not adequately venti- lated. The health effects of ambient NO2 and CO have been described earlier, and the use of gas for cooking have also been shown to be associated with respiratory symptoms among females. But cooking itself, which is the treatment of food with heat, would promote de- composition and volatilization of lipids and amino acids in food, leading to emissions. The types and levels of pollutants in cooking emissions are highly heterogeneous and depend on food ingredi- ents and methods of cooking (e.g. frying, grilling, and baking), but generally include PM, volatile organic compounds, PAHs, and het- erocyclic amines. Some of these compounds are known mutagens and carcinogens. A link between exposure to cooking emissions and lung cancer has been proposed, although causality has not been to- tally confirmed. It should be noted that all of the currently available data were derived exclusively from the Chinese population.

10.3.1 Air pollution and health 1683 Radon Radon-222 is a noble gas, a natural decay product of radium-226, with a half-life of 3.8 days. It decays through four short-lived ‘radon daughters’ to lead-210. Two of the radon daughters, polonium-218 and polonium-214, are α-emitters. These cause bronchial mucosal damage when they decay within the lung and inhalation can thus lead to lung cancer. It has been estimated that 3–14% of all lung cancer cases could be attributable to radon. In the 1970s it became clear that radon is invariably present in in- door air. Indoor levels vary considerably and in some dwellings levels are unacceptably high. The worldwide average indoor radon level has been estimated at 39 Bq/m3, compared to 5–15 Bq/m3 outdoors. The main sources of indoor radon are the rock or soil on which the house is built, building materials used in the construction of the dwelling, natural gas, and water usage. The pooling of studies from Europe, North America, and China has suggested an increase in risk of lung cancer of 8–13% per 100 Bq/m3 increase in indoor radon level. Biological pollutants Allergens and moulds are the most important biological pollutants found in indoor air. Excrements of pests (house dust mites, cock-roaches, rodents) and dander of pets (particularly cats) are powerful allergens that may lead to the development of asthma and other al- lergic diseases. Several dust mite control measures (e.g. washing of bed linens) have been shown to be highly effective in reducing mite allergen, but the efficacy of these measures in preventing asthma is still under debate. Removal or relocation of pets has been recom- mended as the best measure but the reluctance of owners to give up their pets makes this difficult to implement. Air filtration as an alter- native has been proposed but the results have not been encouraging. Dampness is common in dwellings around the world, especially in overcrowded and poorly ventilated environments. Mould is only but one group of microbes that

thrives in damp conditions. Its spores and fragments are potent allergens that are largely responsible for the health effects. There is sufficient evidence to support a causal link between dampness (and mould) and asthma development and exacerbations. However, a systematic review of trials and controlled before–after studies of the effects of repairing mould-damaged buildings did not find convincing beneficial effect (reduction) in asthma-related symptoms and respiratory infections. Sick building syndrome In the early 1970s, illness or symptom complexes were recognized as being related to occupancy of certain buildings. Sick building syn- drome is characterized by an increased prevalence in a particular building of a range of nonspecific symptoms typical of mucosal ir- ritation (e.g. sore/dry eyes, sore nose, dry mouth, sore throat) often with lethargy and headaches. Symptoms usually disappear as soon as an affected individual leaves the building, but a 12-year follow- up on 239 diagnosed Swedish patients found that symptoms could be long-lasting with significant impact on their social life. Indoor environmental parameters such as ventilation, temperature, relative humidity, and indoor chemical (e.g. formaldehyde, volatile organic compounds) levels have been proposed as risk factors but there does not appear to be a consistent relationship of presence of severity of symptoms. Symptoms are usually reported more often by women than men. It was initially believed that the problem of sick building syndrome was minor, but it is now recognized that the financial costs of the condition are substantial. The acceptance of sick building syn- drome as an entity has led to the condition playing an important role in new building design. In buildings already affected, modification of the ventilation very often produces significant improvements in symptom severity and frequency. Household air pollution in LMICs In LMICs, particularly sub-Saharan Africa and South Asia, indoor air quality is dominated by emissions from solid fuel (coal and bio- mass) for cooking and heating. Although the proportion of the household relying on solid fuel has decreased substantially from 62% in 1980, 53% in 1990, 46% in 2005 to 41% in 2010, the total number of people using solid fuel has remained approximately at 2.8 billion in the last three decades. The types of fuel used depend upon local availability and seasonality, for example, coal is predom- inantly used in China, whereas biomass such as wood, rice husk, twigs, charcoal, and dried animal dungs is used in India. Solid fuels are often burnt in traditional cookstoves of low energy conversion efficiency without chimneys such that combustion is usually incomplete and releases large amounts of carbon (as par- ticulates) and CO. Women, due to their traditional role in domestic purposes in LMICs and young children, who often stay with their mothers, are exposed to very high concentrations of air pollutants. The 24-hour concentrations of PM2.5 measured in kitchen environ- ment from different settings ranges from 100 to 5000 µg/m3 which is many folds higher compared to the WHO recommended guide- lines of 25 µg/m3. Similarly, concentration of CO in enclosed kit- chen with little ventilation can be many folds higher than the WHO recommended level of 7 mg/m3 for 24-hour measurement. In rural communities where solid fuels are predominantly used for domestic purposes, the air pollution from kitchen exfiltrates to outdoor creating higher ambient air pollutant concentrations. In a cross- sectional study of 250 Nepalese rural homes the mean 24-hour in- door PM2.5 concentration in the kitchen where cooking is carried out was 455 µg/m3, the concentration in veranda where the other family members spend most of their time was 129 µg/m3 and nearly 100 m away from house was 7.4 µg/m3 (Fig. 10.3.1.1). This suggests indi- viduals using cleaner fuels but living in a community where solid fuels are used are also likely to be exposed higher air pollution. Like cigarette smoke, biomass smoke is a concentrated cocktail of chemicals, the toxicity of which is dependent on the type of fuel. Animal studies suggest short-term wood smoke exposure could af- fect the physiology of the larynx, airway, and alveoli. Biomass smoke also contains high levels of reactive oxygen species, causing inflam- matory response and DNA damage in vitro.

Emissions arising from coal burning have been classified as definite carcinogens and those from biomass fuels as probable carcinogens. There have been very few controlled biomass smoke-exposure studies in humans, but acti- vation of circulating platelets, neutrophils, and monocytes has been reported with high levels of leukocyte-platelet aggregates in chron- ically biomass smoke-exposed individuals. Long-term exposure to coal smoke has been associated with over 130% increased risk of lung cancer and over threefold increase in nasopharyngeal cancer compared to unexposed individuals. PAHs, particularly benzo[a]pyrene, appear to be directly involved in car- cinogenesis, as they are readily absorbed through the respiratory tract, gastrointestinal tract, and skin and circulated systemically.

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1684 There is good evidence to suggest that exposure to wood smoke is associated with an increased risk of COPD even among never smokers. A meta-analysis reported 80% of excess risk of COPD and 132% of excess risk for chronic bronchitis among those ex- posed to household air pollution compared to those who used clean fuel for cooking. It has been suggested that the mechanism of how biomass smoke develop COPD might be similar to that of cigarette smoking. Both are generated from the combustion of plant materials, which generates complex carbon-based par- ticles coated with PAHs and irritant gases such as formaldehyde and acrolein and this could increase the expression of some of the matrix metalloproteinases as in the case of tobacco smoke. In children, exposure to biomass smoke (average PM10 level up to 1000–2000 μg/m3) increases acute lower respiratory infections. The underlying mechanism by which biomass smoke predisposes individuals to respiratory infections is unknown, although evi- dence from controlled chamber study suggested biomass com- bustion may enhance susceptibility to bacterial pneumonia. Early life exposure to household air pollution might be associated with reduced lung function and increased respiratory symptoms in childhood and ultimately in adulthood, but this needs to be con- firmed in long-term prospective cohorts. Although the association between cardiovascular diseases and ambient air pollution has been well established, there is no direct evidence to conclude the same with household air pollution. Few short-term observational studies have reported increased sys- tolic and diastolic blood pressure, elevated oxidized low-density lipoprotein, significant increase in platelet aggregation, platelet P-selectin expression, raised anticardiolipin IgG and IgM anti- bodies, increased mean carotid intima-media thickness and in- creased prevalence of atherosclerotic plaques among those exposed to biomass smoke. A randomized controlled trial reported reduced ST-segment depression among those who received improved cook stoves, hinting exposure to wood smoke could affect ventricular repolarization. Other endpoints such as adverse pregnancy outcomes and peri- natal risks (birth defects, low birthweight, and sepsis), and impaired physical and cognitive development in children have been reported. Biomass users, especially in rural communities, often spend con- siderable amount of time collecting firewood and are likely to have injuries from falls and muscloskeletal problems. A significant in- creased risk of cataract and teary eyes among biomass smoke-ex- posed individuals have also been reported. Switching to cleaner fuels, such as electricity, is the most effective intervention but this option is not always feasible. Adoption of im- proved cookstoves that are more efficient is a viable alternative. At present, 27% of the total population using solid fuels have access to improved cookstoves, most being in China (70%). Major pro- jects have been launched to produce and disseminate improved cookstoves on a global scale. A recent systematic review examined the effect of stove interventions and concluded while most studies reported a reduction of household air pollution particularly PM and CO levels, they were not enough to meet WHO air quality recom- mendations. Nevertheless, reductions in

self-reported respiratory and nonrespiratory symptoms among those who received im- proved cookstoves have been found. On the other hand, there was no conclusive evidence of improvement in objective measures of lung function and birth weight. The results from these intervention studies highlighted importance of user behaviour and compliance (e.g. exclusive use of improved cookstoves above traditional op- tions) and the long-term proper maintenance of the stoves. Air quality guidelines and standards Although by definition all air pollutants are hazardous, only the major ones (e.g. PM, SO2, NO2, and ozone) in the atmosphere are monitored and regulated by legislation in most countries. While no threshold concentration has been identified below which air pollu- tion has no effect on population health, different limits and target values have been proposed by various international organizations and national governments, driven by a basket of factors including scientific evidence of adverse health effect, cost, feasibility, and pol- itical will. In most cases, the national guidelines in LMICs tend to be more relaxed compared to those in the high-income coun- tries. Table 10.3.1.2 shows three most widely used contemporary guidelines and standards from the WHO, the US Environmental Protection Agency (US EPA), and the European Union (EU). (a) (b) Fig. 10.3.1.1 Household air pollution in kitchen. (a) A man cooking food on traditional stove without chimney using wood; (b) a cot hanging from the kitchen ceiling where an infant is usually kept when the mother is performing the cooking activities. Photos taken by Om P Kurmi.

10.3.1 Air pollution and health 1685 In contrast, indoor air quality is not normally regulated, except for airborne pollutants generated in the workplace, the standards of which are enforced separately by occupational safety and health regulations beyond the scope of this Section. Certain countries have set up emission standards of volatile compounds (e.g. formal- dehyde) from building and interior furnishing materials, primarily aiming to control the associated sick building syndrome. WHO has issued nonlegal binding guidelines based on health risk of damp- ness and mould (WHO 2009) and common chemicals found in indoor environment (WHO 2010), but the adoption of such guide- lines has not been widespread in its member states. WHO has also developed air quality guidelines on household air pollution, par- ticularly to those using solid fuel for cooking. The guideline is sum- marized in Table 10.3.1.3. Table 10.3.1.2 Various major national and international guidelines on major air pollutants Pollutants Mean concentration allowed when averaged over timea 10-min 1-hr 8-hr 24-hr Annual PM10 (µg/m3) WHO – – – 50 20 US EPA – – – 150a EU – – – 50 40 PM2.5 (µg/m3) WHO – – – 25 10 US EPA – – – 35† 12† or 15‡ EU – – – – 25 Nitrogen dioxide (µg/m3) WHO – 200 – – 40 US EPA 190# – – – 99.7 EU – 200 – – 40 Ozone (µg/m3) WHO – – 100 – – US EPA – – 140## – – EU 120 Sulphur dioxide (µg/m3) WHO 500 – – – 20 US EPA – 200 b – – – EU – 350 – 125 – a Not to be exceeded more than once per year over three years; † 98th percentile averaged over 3 years; †Primary pollutant: Annual mean averaged over 3 years; ‡ Secondary pollutant: Annual mean averaged over 3 years; # 98th percentile of 1-hour daily maximum concentration, averaged over 3 years; ## Annual fourth-highest daily maximum 8-hour concentration, averaged over 3 years; b Primary pollutant: 99th percentile of 1-hr daily maximum concentration, averaged over 3 years. The latest guidelines used in tables can be obtained from WHO (http://apps.who.int/iris/bitstream/10665/69477/1/WHO_SDE_PHE_OEH_06.02_eng.pdf); US EPA (http://www3.epa.gov/ttn/naaqs/criteria.html) and EU (http://ec.europa.eu/environment/air/quality/standards.htm) webpages. Table 10.3.1.3 Summary of published WHO air quality guideline values for household air pollution Pollutant (unit for guidelines) Mean concentration over averaging time Unit risk Comments 15 min 30 min 1 hour 8 hours 24 hours 1 year PM2.5 (µg/m3) – – – – 25a 10 – 24–hour guideline not to exceed max.

3 days/year PM10 (µg/m3) – – – – 50a 20 – 24–hour guideline not to exceed max. 3 days/year Benzene (risk of leukaemia per 1 µg/m3) – – – – – – 6.0 × 10–6 No safe level Carbon monoxide (mg/m3) 100 – 35 10 7 – – – Formaldehyde (mg/m3) – 0.1 – – – – – – Naphthalene (mg/m3) – – – – – 0.01 – – Nitrogen dioxide (µg/m3) – – 200 – – 40 – – PAHsb (risk of lung cancer per 1 ng/m3) B[a]P – – – – – – 8.7 × 10–5 No safe level a The 24-hour average values for PM10 and PM2.5 refer to the 99th percentile of the distribution of daily values (i.e. the fourth next highest value of the year). b PAHs, polycyclic aromatic hydrocarbons, B[a]P, benzo[a]pyrene: In view of the difficulties in developing guideline for PAH mixtures, B[a]P was considered to represent the best single indicator compound.

SECTION 10 Environmental medicine, occupational medicine, and poisoning 1686 Table 10.3.1.4 The Daily Air Quality Index, showing exposure concentration and accompanied health messages (accessed from http://uk-air.defra.gov.uk/) Air pollution index bands (value) → Low (1) Low (2) Low (3) Moderate
(4) Moderate
(5) Moderate
(6) High (7) High (8) High (9) Very high (10) Pollutants (µg/m3) Sampling time Concentration of specific pollutants at different air pollution index bands PM10 24-hr mean 0–16 17–33 34–50 51–58 59–66 67–75 76–83 84–91 92–100 ≥101 PM2.5 24-hr mean 0–11 12–23 24–35

> 36–41 42–47 47–53 54–58 59–64 65–70 ≥71 SO2 15-min mean 0–88 89–177 178–266 267–354 355–443 444–532 533–710 711–887 888–1064 ≥1065 NO2 1-hr mean 0–67 68–134 135–200 201–267 268–334 335–400 401–467 468–534 535–600 ≥601 O3 8-hr mean 0–33 34–66 67–100 101–120 121–140 141–160 161–187 188–213 214–240 ≥241 Accompanying health messages for individuals at risk Enjoy your usual outdoor activities. Adults and children with lung problems, and adults with heart problems, who experience symptoms, should consider reducing strenuous physical activity, particularly outdoors. Adults and children with lung problems, and adults with heart problems, should reduce strenuous physical exertion, particularly outdoors, and particularly if they experience symptoms. People with asthma may find they need to use their reliever inhaler more often. Older people should also reduce physical exertion. Adults and children with lung problems, adults with heart problems, and older people, should avoid strenuous physical activity. People with asthma may find they need to use their reliever inhaler more often. Accompanying health messages for the general population Enjoy your usual outdoor activities. Enjoy your usual outdoor activities. Anyone experiencing discomfort such as sore eyes, cough, or sore throat should consider reducing activity, particularly outdoors. Anyone experiencing discomfort such as sore eyes, cough, or sore throat should consider reducing activity, particularly outdoors. PM10, particulate matter less than 10 µm aerodynamic diameter; PM2.5, particulate matter less than 2.5 µm aerodynamic diameter; SO2, sulphur dioxide; NO2, nitrogen dioxide; O3, ozone.

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