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AIR POLLUTION AND CHILD HEALTH

Prescribing clean air



World Health
Organization

Air pollution and child health: prescribing clean air

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Abbreviations and acronyms

AAP	ambient air pollution
ALRI	acute lower respiratory infection
ASD	autism spectrum disorders
BMI	body mass index
CO	carbon monoxide
DALY	disability-adjusted life-year
FEV1	forced expiratory volume in 1 s
FVC	forced vital capacity
HAP	household air pollution
HIC	high-income country
LMIC	low- or middle-income country
LPG	liquefied petroleum gas
NO _x	nitrogen oxides
O ₃	ozone
PM	particulate matter
PM ₁	particles < 1 μm in diameter
PM _{2.5}	particles < 2.5 μm in diameter
PM ₁₀	particles < 10 μm in diameter
SDG	Sustainable Development Goal
SGA	small for gestational age
SO ₂	sulfur dioxide
TB	tuberculosis

Preface

This report summarizes the latest scientific knowledge on the links between exposure to air pollution and adverse health effects in children. It is intended to inform and motivate individual and collective action by health care professionals to prevent damage to children's health from exposure to air

The evidence is clear: air pollution has a devastating impact on children's health.

pollution. Air pollution is a major environmental health threat. Exposure to fine particles in both the ambient environment and in the household causes about seven million premature deaths each year (1,2). Ambient air pollution (AAP) alone imposes enormous costs on the global economy, amounting to more than

US\$ 5 trillion in total welfare losses in 2013 (3).

This public health crisis is receiving more attention, but one critical aspect is often overlooked: how air pollution affects children in uniquely damaging ways. Recent data released by the World Health Organization (WHO) show that air pollution has a vast and terrible impact on child health and survival. Globally, 93% of all children live in environments with air pollution levels above the WHO guidelines (see Annex 2). More than one in every four deaths of children under 5 years is directly or indirectly related to environmental risks (4). Both AAP and household air pollution (HAP) contribute to respiratory tract infections that resulted in 543 000 deaths in children under 5 years in 2016 (1).

Although air pollution is a global problem, the burden of disease attributable to particulate matter in air is heaviest in low- and middle-income countries (LMICs), particularly in the WHO African, South-East Asia, Eastern Mediterranean and Western Pacific regions (1,5). LMICs in these regions – especially the African Region – have the highest levels of exposure to HAP due to the widespread use of polluting fuels and technologies for basic daily needs, such as cooking, heating and lighting (6). Poverty is correlated with high exposure to environmental health risks. Poverty can also compound the damaging health effects of air pollution, by limiting access to information, treatment and other health care resources.

The enormous toll of disease and death revealed by these new data should result in an urgent call to action for the global community – and especially for those in the health sector. Strong action to reduce exposure to air pollution offers an unparalleled opportunity to protect the health of children everywhere. Health professionals have a central role to play in this effort. Health effects experienced early in life can increase a child's future risk of disease and lead to lifelong consequences. A child who is exposed to unsafe levels of pollution early in life can thus suffer a "life sentence" of illness. Health professionals are well positioned to communicate with families, communities and decision-makers about these and other serious risks of exposure to air pollution.

The Sustainable Development Goals (SDGs) recognize the importance of social and environmental factors as determinants of health. All the SDGs are clearly linked to health-related targets, reflecting the growing awareness that health, environmental and poverty alleviation are interconnected –that ensuring healthy lives for all (SDG 3) and making cities inclusive, safe, resilient and sustainable (SDG 11) require universal access to energy (SDG 7) and hinge upon combating climate change (SDG 13). The launch of the 2030 Agenda for Sustainable Development offers an unparalleled opportunity to increase action to address the environmental hazards that undermine children's health. Implementing evidence-based policies and health practices to protect children from air pollution will, in turn, be essential to realizing the Sustainable Development Agenda: reducing children's exposure can have enormous benefits due to avoided disease, reduced mortality and improved well-being. Reducing air pollution can also improve health and well-being by slowing climate change. It is estimated that, by 2030, climate change will be responsible for 250 000 deaths each year (7). As many of the same pollutants that threaten health, such as black carbon and ozone (O₃), are also important agents of atmospheric warming, interventions that reduce their emissions are likely to result in benefits for both children's health and the climate.

We must seize this opportunity to create healthy, sustainable environments for our children. Everyone has a role to play, at every level: individuals, families, paediatricians, family doctors, nurses, obstetricians and gynaecologists, primary health care providers and other community workers, communities, medical students, national governments and international agencies. Their efforts should be guided by the best available evidence on the health effects of air pollution on children and on effective interventions to counter them. This document is designed to support this effort. It reports the latest scientific knowledge on the health effects of air pollution in children. The

breadth and depth of the evidence make clear that air pollution is a formidable disruptor of children's health – one that deserves far greater attention from both policy-makers and health professionals. As children experience the consequences of air pollution in special, specific ways, they deserve to be assessed in a special way. This publication provides practical, reliable information for health professionals, paediatricians and other clinicians in all countries. It will be a useful reference for action: a compendium of the accumulating evidence on the links between air pollution and children's health and a source of guidance for health care providers in their clinical practice and in their collective communication of risks and solutions to the public and to policy-makers.

Children are uniquely vulnerable to the damaging health effects of air pollution.

Children are society's future. But they are also its most vulnerable members. The immense threat posed to their health by air pollution demands that health professionals respond with focused, urgent action. Although more rigorous research into how air pollution affects

children's health will continue to be valuable, there is already ample evidence to justify strong, swift action to prevent the damage it clearly produces. Health professionals must come together to address this threat as a priority, through collective, coordinated efforts. For the millions of children exposed to polluted air every day, there is little time to waste and so much to be gained.

References – preface

1. Ambient air pollution: a global assessment of exposure and burden of disease, second edition. Geneva: World Health Organization; (in press).
2. Burden of disease from the joint effects of household and ambient air pollution for 2016. Version 2 May 2018. Summary of results. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/cities/en/>, accessed August 2018).
3. World Bank, Institute for Health Metrics and Evaluation. The cost of air pollution: strengthening the economic case for action. Washington (DC): World Bank; 2016 (<http://documents.worldbank.org/curated/en/781521473177013155/The-cost-of-air-pollution-strengthening-the-economic-case-for-action>, accessed 20 September 2018). Licence: Creative Commons Attribution CC BY 3.0 IGO.
4. Prüss-Ustün A, Wolf J, Corvalán C, Bos R, Neira M. Preventing disease through healthy environments: a global assessment of the burden of disease from environment risks. Geneva: World Health Organization; 2016 (<http://www.who.int/iris/handle/10665/204585>, accessed August 2018).
5. Exposure to ambient air pollution from particulate matter for 2016. Version 2 April 2018. Summary of results. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/cities/en/>, accessed August 2018). Exposure to household air pollution for 2016. Version 5 April 2018. Summary of results. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/cities/en/>, accessed August 2018).
6. Quantitative risk assessment of the effects of climate change on selected causes of death, 2030s and 2050s. Geneva: World Health Organization; 2014 (<http://www.who.int/iris/handle/10665/134014>, accessed September 2018).

Executive summary

Children's exposure to air pollution

Exposure to air pollution is an overlooked health emergency for children¹ around the world. While such exposure is a persistent problem in some high-income countries (HICs) – especially in low-income communities within those countries – the vast majority of child deaths from exposure to particulate matter air pollution occur in LMICs.

Exposure to air pollution from particulate matter occurs both outdoors and indoors. AAP is primarily derived from fossil fuel combustion, industrial processes, waste incineration, agricultural practices and natural processes such as wildfires, dust storms and volcanic eruptions. The main sources of air pollution may vary from urban to rural areas, but no area is, strictly speaking, safer. AAP was responsible for 4.2 million premature deaths in 2016; of these, almost 300 000 were children under 5 years (1,2).

The risks associated with breathing HAP can be just as great. Breathing clean air at home is essential for children's healthy development, but widespread dependence on solid fuels and kerosene for cooking, heating and lighting results in far too many children living in terribly polluted home environments. About three billion people worldwide still depend on polluting fuels and devices for cooking and heating (3). Women and children spend most of their time around the hearth, exposed to smoke from cooking fires, resulting in indoor concentrations of some pollutants that are five or six times the levels in ambient air. The widespread lack of access to clean household energy has tragic consequences on a vast scale: HAP was responsible for 3.8 million premature deaths in 2016, including over 400 000 deaths of children under 5 years (4).

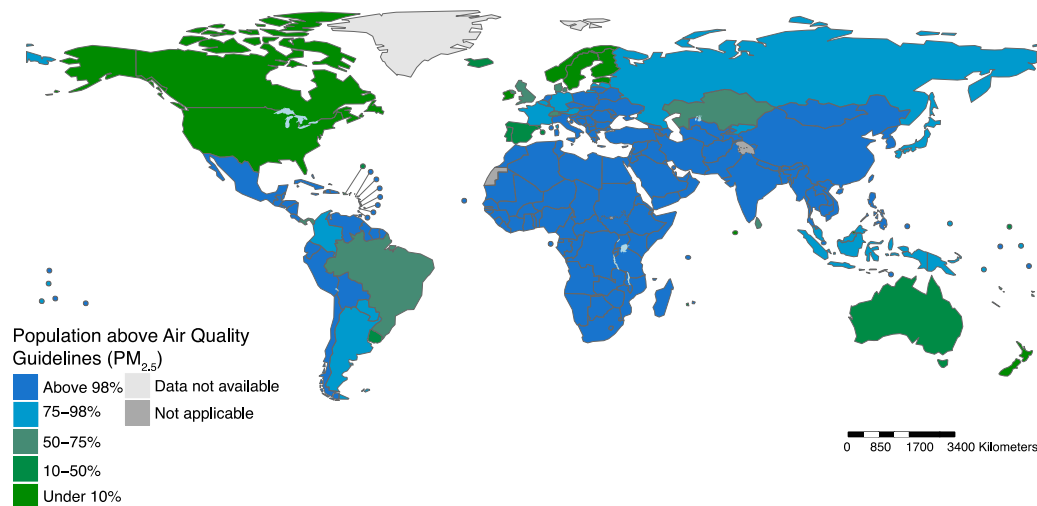
Exposure to ambient air pollution

The proportions of children exposed to levels of fine particulate matter (PM_{2.5}) higher than the WHO air quality guideline levels (Fig. 1) are as follows:

- 93% of all children and about 630 million children under 5 years in the world;
- in LMICs, 98% of all children under 5 years;
- in HICs, 52% of children under 5 years;
- in the WHO African and Eastern Mediterranean regions, 100% of all children under 5 years;
- in LMICs in the South-East Asia Region, 99% of all children under 5 years;
- in LMICs in the Western Pacific Region, 98% of all children under 5 years; and
- in LMICs in the Region of the Americas, 87% of all children under 5 years.

Fig. 1. Proportions of children under 5 years living in areas in which the WHO Air Quality Guidelines (PM_{2.5}) are exceeded, by country, 2016

¹ WHO defines a “child” as a person under 19 years of age, an “adolescent” as a person aged 10–19 years, an “infant” as a person aged 0–11 months and a “newborn” as a person aged 0–28 days. References to “child mortality” usually pertain to children aged 0–59 months.



Source: (5)

Exposure to household air pollution

In 2016, 41% of the world’s population was exposed to HAP from cooking with polluting fuels and technologies. The use of polluting fuels and technologies for cooking is almost exclusively a problem in LMICs, as 83% of the population in the African Region, 59% in the South-East Asia Region and 42% in the Western Pacific Region rely primarily on polluting cooking fuels. The Eastern Mediterranean Region follows, with 31% of its population relying primarily on polluting fuels and devices, while the proportions in the Region of the Americas and the European Region are 13% and 6%, respectively.

Children’s vulnerability and susceptibility to air pollution

Air pollution is a global public health crisis. Exposure to pollutants in the air threatens the health of people of all ages, in every part of the world, in both urban and rural areas, but it affects the most vulnerable among us – children – in unique ways. Children are at greater risk than adults from the many adverse health effects of air pollution, owing to a combination of behavioural, environmental and physiological factors. Children are uniquely vulnerable and susceptible to air pollution, especially during fetal development and in their earliest years. Their lungs, organs and brains are still maturing. They breathe faster than adults, taking in more air and, with it, more pollutants. Children live closer to the ground, where some pollutants reach peak concentrations. They may spend much time outside, playing and engaging in physical activity in potentially polluted air. New-borns and infants, meanwhile, spend most of their time indoors, where they are more susceptible to HAP. Children spend much time near their mothers while the latter cook with polluting fuels and devices.

Children have a longer life expectancy than adults, so latent disease mechanisms have more time to emerge and affect their health. Their bodies, and especially their lungs, are rapidly developing and therefore more vulnerable to inflammation and other damage caused by pollutants. In the womb, they are vulnerable to their mothers’ exposure to pollutants. Health effects from preconception exposure can also impose latent risks on the fetus. Even after birth, they often remain powerless to change their environment: the very youngest cannot simply get up and walk out of a smoke-filled room. The consequences of their exposure – through inhalation, ingestion or in utero – can lead to illness and other health burdens that last a lifetime. But children depend entirely on us – adults – to protect them from the threat of unsafe air.

Children’s burden of disease related to air pollution

Tables 1 and 2 show the joint burden of disease from AAP and HAP.

- Globally in 2016, one in every eight deaths was attributable to the joint effects of AAP and HAP – a total of 7 million deaths.
- Some 543 000 deaths in children under 5 years and 52 000 deaths in children aged 5–15 years were attributed to the joint effects of AAP and HAP in 2016.
- Together, HAP from cooking and AAP cause more than 50% of acute lower respiratory infections (ALRI) in children under 5 years of age in LMICs.
- Of the total deaths attributable to the joint effects of HAP and AAP worldwide in 2016, 9% were in children.

Table 1. Death rate per 100 000 children attributable to the joint effects of HAP and AAP in 2016, by WHO region and income level

WHO region	Income level	Children < 5 years	Children 5–14 years
African	LMIC	184.1	12.9
	HIC	4.3	1.4
Americas	LMIC	14.2	0.7
	HIC	0.3	0
South-East Asia	LMIC	75	2.5
European	LMIC	8.8	0.6
	HIC	0.3	0
Eastern Mediterranean	LMIC	98.6	3.6
	HIC	5.3	0.4
Western Pacific	LMIC	20.5	1
	HIC	0.3	0
All	LMIC	88.7	4.5
	HIC	0.6	0.1
World		80.5	4.1

LMIC, low- and middle-income country; HIC, high-income country

Table 2. Population attributable fractions of child mortality due to ALRI as a joint effect of HAP and AAP, 2016, by WHO region, the world and income level

WHO region	Income level	Children < 5 years (%)	Children 5–14 years (%)
African	LMIC	66	66
	HIC	25	24
Americas	LMIC	34	34
	HIC	8	7
South-East Asia	LMIC	63	62
European	LMIC	27	27
	HIC	13	14
Eastern Mediterranean	LMI	58	55
	HIC	40	40
Western Pacific	LMIC	53	52
	HIC	12	11
All	LMIC	62	62

	HIC	18	15
World		62	62

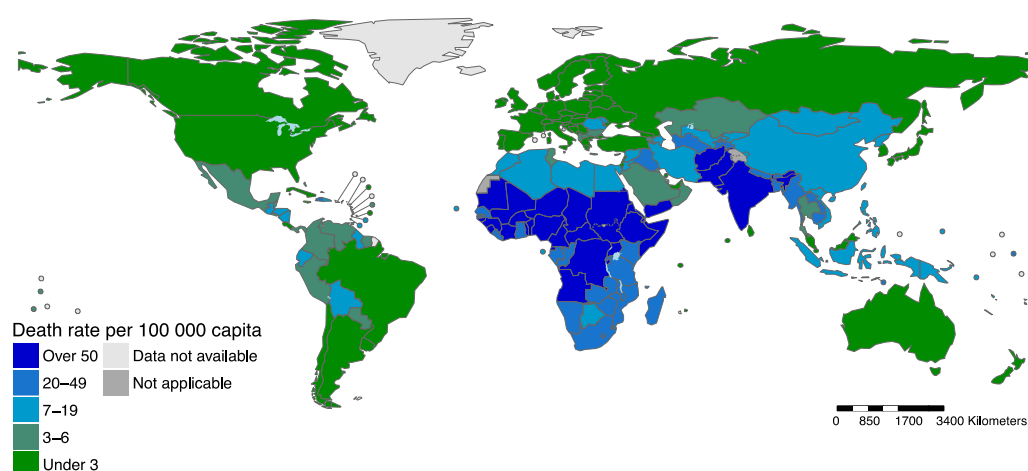
LMIC, low- and middle-income country; HIC, high-income country.

ALRI, acute lower respiratory infection; HAP, household air pollution; AAP, ambient air pollution.

Burden of disease due to AAP: In 2016, AAP was responsible for approximately 261 000 deaths from ALRI and almost 24 million disability-adjusted life years (DALYs) among children under 5 years. The numbers of deaths from ALRI due to AAP in children under 5 years of age are shown in Fig. 2.

The numbers of DALYs due to AAP in children under 5 years and children aged 5–14 years are shown in Annex 2 in Fig. 15 and Fig. 16, respectively.

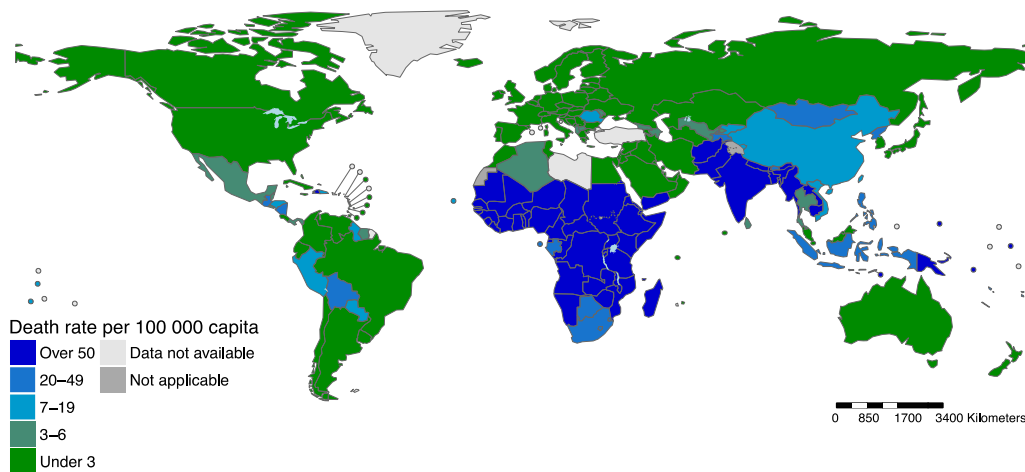
Fig. 2. Death rate per 100 000 per population from ALRI due to AAP in children under 5 years, 2016



Source: see Annex 2

Burden of disease due to HAP: In 2016, HAP was responsible for approximately 403 000 deaths from ALRI and 37 million DALYs among children under 5 years (Fig. 3).

Fig. 3. Death rate per 100 000 per population from ALRI due to HAP in children under 5 years, 2016

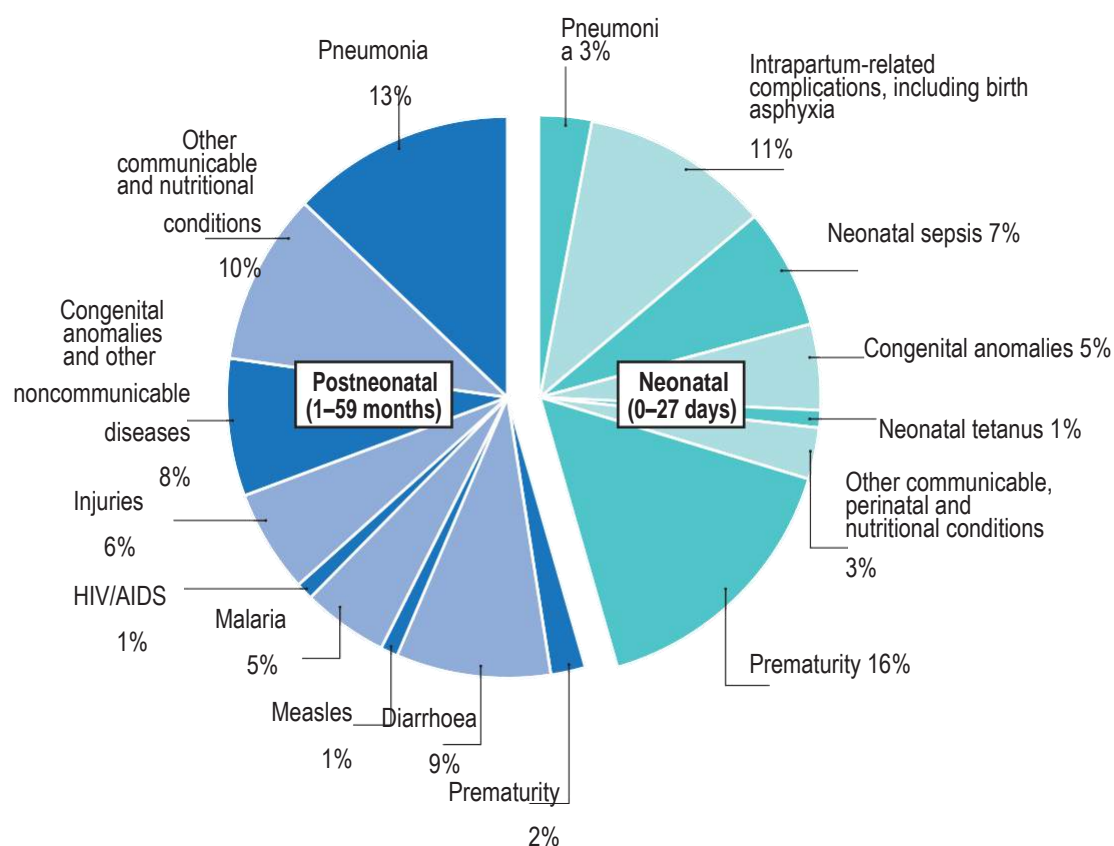


Source: see Annex 2

This tragically high toll is for just one disease, ALRI. The total burden of mortality and morbidity among children due to exposure to AAP and HAP is much greater. Evidence of the many different adverse health effects of exposure to air pollution is discussed below.

Exposure to air pollution contributes to more than half of all deaths from ALRI in children under 5 years in LMICs, making it one of the leading killers of children worldwide. The five leading causes of death in children under 5 years globally are prematurity, acute respiratory infections, intrapartum-related complications (including birth asphyxia), other group 1 conditions and congenital anomalies (6). Premature birth is the only factor that kills more children under 5 years globally than acute respiratory infections (Fig. 4). In the African Region, acute respiratory infection is the leading cause of death of children under 5 years.

Fig. 4. Causes of deaths among children under 5 years, 2016



Source: Reference (6).

Several global strategies and initiatives to improve child health have included targets, policies and interventions related to air pollution. These include the Global Action Plan for the Prevention and Treatment of Pneumonia and Diarrhoea, the Every Newborn Action Plan and the Nurturing Care Framework for Early Childhood Development (see Annex 1).

Sources of air pollution

Ambient air pollution: health toll, sources and solutions

AAP caused about 4.2 million premature deaths in 2016 (2). It is estimated that, in 2016, 286 000 children under 15 years of age died from exposure to unhealthy levels of AAP (see Annex 2). Ambient air is polluted from many different sources, both anthropogenic and natural, which differ in urban to rural areas. In urban settings, the main sources are fossil fuel combustion for energy production, transport, residential cooking, heating and waste incineration. Rural communities in LMICs are exposed to pollution emitted primarily from household burning of kerosene, biomass and coal for cooking, heating and lighting, from agricultural waste incineration and from certain agroforestry activities (7). These processes produce complex mixtures of pollutants that can interact chemically. They typically include carbon monoxide (CO), nitrogen oxides (NO_x), lead, arsenic, mercury, sulfur dioxide (SO₂), polycyclic aromatic hydrocarbons (PAHs) and particulate matter (PM). The last affects more people than any other air pollutant, and it is commonly used as a proxy indicator of air pollution more broadly.

Addressing AAP is a high priority for governments and multilateral agencies around the world. Many proven solutions are available to reduce emissions of dangerous pollutants, including cleaner transport, cleaner cooking and heating fuels and technologies, energy-efficient housing and urban planning, low- or zero-emission power generation, cleaner, safer industrial technologies and better municipal waste management (8). The WHO air quality guidelines (8) provide recommended thresholds and limits for key ambient air pollutants that must be met in order to protect health; an updated version will be published in 2020.

Household air pollution: health toll, sources and solutions

HAP – the single largest environmental health risk factor worldwide – is produced mainly by the incomplete combustion of polluting fuels for cooking, heating and lighting (3, 7). In 2016, WHO estimated that about three billion people – 41% of the world’s population –used polluting cooking sources, most of them in LMICs (3). This number has remained largely unchanged for the past three decades. The damage to health caused by such widespread dependence on polluting energy sources is severe and extensive: in 2016, HAP from solid fuel use resulted in an estimated 3.8 million premature deaths. This toll is equivalent to 6.7% of global mortality, greater than that from malaria, tuberculosis (TB) and HIV/AIDS combined. Of these deaths, 403 000 were among children under 5 years of age (4). HAP is also an important source of AAP, as residential cooking contributes as much as 12% of global PM_{2.5} to ambient air (7).

In many parts of the world, children are especially vulnerable to HAP because they spend a great deal of time in the home and with their mothers as the latter tend the hearth. Smoke emitted from burning biomass, coal, charcoal and kerosene to meet the basic needs of cooking, heating and lighting is the primary contributor to HAP (3). Burning these fuels in inefficient devices produces complex mixtures of contaminants. In dwellings with poor ventilation, emissions of fine particulate matter and other pollutants from stoves can reach 100 times the maximum exposure level recommended by WHO (7).

In 2014 WHO issued *Guidelines for indoor air quality: household fuel combustion* (9), the first guidelines to define fuels and technologies for cooking, heating and lighting that are clean for health at the point of use, including electricity, liquefied petroleum gas (LPG), biogas, ethanol and solar stoves, as well as some high-performing biomass stoves. The guidelines discourage household use of kerosene and unprocessed coal because of the serious associated health hazards. Unfortunately, kerosene is still used for lighting by many of the about one billion people who lack access to electricity. Achieving universal access to clean, safe household energy is a top priority on the global sustainable development agenda, reflected in SDG 7: “ensure access to affordable, reliable, sustainable and modern energy for all”.

Other indoor sources

Many other air pollutants that are risks to health are beyond the scope of this report. These include volatile organic compounds from household products and building supplies, asbestos, pesticides, mercury (e.g. from broken thermometers), radon and biological pollutants. Tobacco smoke is another significant source of indoor air pollution and a health risk for children; the health effects of tobacco smoke have been reviewed extensively in other WHO documents.

Social determinants of children’s health

Poverty is strongly correlated with exposure to air pollution. Children in LMICs and in low-income communities within HICs disproportionately suffer the effects of air pollution. Poverty causes people to rely on polluting energy sources for their basic needs, and poverty compounds the health risks associated with their use. Poverty also limits people’s capacity to improve the environment in which they raise their children. Air pollution is often a chronic problem in poor-quality housing and temporary settlements. The exposure of people living in refugee camps can be particularly high, as they are forced to scavenge for nearby wood and other fuels or to rely on kerosene stoves for heating and cooking.

Women and girls are the primary users and procurers of household energy around the world. Dependence on the energy sources that produce the most HAP (e.g. wood and other solid fuels) used in inefficient stoves also poses other important health and safety risks. In many LMICs, children have the daily or weekly task of fuel collection, often walking long distances with heavy loads of wood and other fuels. A WHO analysis of survey data from 16 African countries in 2016 found that girls in households that used polluting fuels and technologies spent about 18 hours each week collecting wood or water, whereas girls in households in which clean fuels and technologies were used primarily spent 5 hours each week in those tasks (7). This work robs children of time spent for playing and studying. It also leads to musculoskeletal disorders and can expose children, particularly girls, to higher risk of violence as they venture far from their household (7, 10).

Health effects

There is compelling evidence that exposure to air pollution damages the health of children in numerous ways. The evidence summarized in this report is based on a scoping review of relevant studies published within the past 10 years and input from dozens of experts around the world. It covers adverse birth outcomes, infant mortality, neurodevelopmental disorders, childhood obesity, lung function, ALRI, asthma, otitis media and childhood cancers.

Adverse birth outcomes. Numerous studies have shown a significant association between exposure to AAP and adverse birth outcomes, especially exposure to PM, SO₂, NO_x, O₃ and CO. There is strong evidence that exposure to ambient PM is associated with low birth weight. There is also growing evidence that maternal exposure, especially to fine PM, increases the risk of preterm birth. There is emerging evidence for associations between exposure to air pollution and other outcomes, such as stillbirth and infants born small for gestational age.

Infant mortality. There is compelling evidence of an association between air pollution and infant mortality. Most studies to date have focused on acute exposure and AAP. As pollution levels increase, so too does the risk of infant mortality, particularly from exposure to PM and toxic gases.

Neurodevelopment. A growing body of research suggests that both prenatal and postnatal exposure to air pollution can negatively influence neurodevelopment, lead to lower cognitive test outcomes and influence the development of behavioural disorders such as autism spectrum disorders and attention deficit hyperactivity disorder. There is strong evidence that exposure to AAP can negatively affect children's mental and motor development.

Childhood obesity. A limited number of studies have identified a potential association between exposure to AAP and certain adverse metabolic outcomes in children. The findings include positive associations between exposure to air pollution in utero and postnatal weight gain or attained body-mass index (BMI) for age, and an association has been reported between traffic-related air pollution and insulin resistance in children.

Lung function. There is robust evidence that exposure to air pollution damages children's lung function and impedes their lung function growth, even at lower levels of exposure. Studies have found compelling evidence that prenatal exposure to air pollution is associated with impairment of lung development and lung function in childhood. Conversely, there is evidence that children experience better lung function growth in areas in which ambient air quality has improved.

ALRI, including pneumonia. Numerous studies offer compelling evidence that exposure to AAP and HAP increases the risk of ALRI in children. There is robust evidence that exposure to air pollutants such as PM_{2.5}, nitrogen dioxide (NO₂) and O₃ is associated with pneumonia and other respiratory infections in young children. Growing evidence suggests that PM has an especially strong effect.

Asthma. There is substantial evidence that exposure to AAP increases the risk of children for developing asthma and that breathing pollutants exacerbates childhood asthma as well. While relevant there are fewer studies on HAP, there is suggestive evidence that exposure to HAP from use of polluting household fuels and technologies is associated with the development and exacerbation of asthma in children.

Otitis media. There is clear, consistent evidence of an association between AAP exposure and the occurrence of otitis media in children. Although relatively few studies have examined the association between non-tobacco smoke HAP and otitis media, there is suggestive evidence that combustion-derived HAP may increase the risk of otitis media.

Childhood cancers. There is substantial evidence that exposure to traffic-related air pollution is associated with increased risk of childhood leukaemia. Several studies have found associations between prenatal exposure to AAP and higher risk of retinoblastomas and leukaemia in children. While relatively few studies have focused on HAP and cancer risk in children, HAP is strongly associated with several types of cancer in adults and typically contains many known classified carcinogens.

Relation between early exposure and later health outcomes. Children exposed to air pollution prenatally and in early life is more likely to experience adverse health outcomes as they mature and through adulthood. Exposure to air pollution early in life can impair lung development, reduce lung

function and increase the risk of chronic lung disease in adulthood. Evidence suggests that prenatal exposure to air pollution can predispose individuals to cardiovascular disease later in life.

Altogether, there is clear, compelling evidence of significant associations between exposure to air pollution and a range of adverse health outcomes. The evidence suggests that the early years, starting in pregnancy, are the best time to invest in a child's health, through action to improve their environment and reduce their exposure to pollutants. This window of time offers, in effect, a great opportunity: precisely because children are most vulnerable and sensitive to environmental influences in their earliest years, action taken during this critical phase can yield immense health benefits.

Recommended actions for health professionals

The scientific evidence outlined above suggests many clear, concrete steps that can be taken now to reduce the exposure of pregnant women, children and adolescents to air pollution.

Health professionals are trusted sources of information and guidance. They play an important role not only in treating ill health caused by air pollution but also in educating families and patients about risks and solutions and communicating with the broader public and decision-makers (Fig. 5). The role of health professionals in the management of childhood exposure to air pollution must be amplified, through improved methods of care and prevention and collective action. Health professionals can provide evidence to shape public health policy and advocate for effective policies to reduce children's exposure to air pollution. The broader health sector must become more engaged in developing a comprehensive approach to addressing this crisis.

Fig. 5. Critical role of health professionals

- Be informed.
- Recognize exposure and related health conditions.
- Research, publish and disseminate knowledge.
- Prescribe solutions and educate families and communities.
- Educate colleagues and students.
- Advocate solutions to other sectors, policy- and decision-makers.

Be informed. All health professionals should consider air pollution a major risk factor for their patients and understand the sources of environmental exposure in the communities they serve. They should be informed about existing and emerging evidence on the ways in which air pollution may affect children's health.

Recognize exposure and health related conditions. Health professionals have an important role in identifying causative risk factors in order to prevent disease. A health care provider can identify air pollution-related risk factors by asking pertinent questions about the child's or pregnant mother's environment.

Research, publish and disseminate knowledge. Health professionals can conduct research on the effects of air pollution on children's health. They can conduct and publish studies of the causes, mechanisms and effects of environmental exposure of children, as well as potential treatment, prevention and management. They can also use this evidence to inform social and behaviour change communication strategies.

Prescribe solutions and educate families and communities. Health professionals can "prescribe" solutions to air pollution-related problems, such as switching to clean household fuels and devices. In contexts in which there are significant barriers to adopting clean household energy, health care professionals can recommend "transitional" solutions that offer some incremental health benefit, and

they can provide resources and information on relevant government and non-profit programmes to help reduce exposure.

Educate colleagues and students. By training others in the health and education fields, health professionals can increase the reach of their messages on the health risks of air pollution and strategies to reduce exposure. Health professionals can engage their colleagues in their workplace, local health care centres, conferences and professional associations. They can support the inclusion of children's environmental health in curricula in post-secondary institutions and particularly in medical, nursing and midwifery schools.

Advocate solutions to other sectors, policy- and decision-makers. Health professionals are well positioned to share their knowledge with decision-makers, including members of local governments and school boards and other community leaders. Health professionals can accurately convey the health burden of air pollution to decision-makers, conduct health-based assessments, support improved standards and policies to reduce harmful exposures, advocate for monitoring and emphasize the need to protect children at risk.

The need for collective action, equity and access

Low-income families have limited options to improve the air quality in their homes. Because of market and other forces beyond their control, clean fuels and technologies may not be affordable, available or accessible. Outside the household, individuals and families have even less control over what is emitted into the air that surrounds them. Individual protective measures such as use of clean stoves for cooking may mitigate HAP and improve the health of the whole family; however, reducing AAP requires wider action, as individual protective measures are not only insufficient, but neither sustainable nor equitable. To reduce and prevent exposure to both HAP and AAP, public policy is essential.

Lifting lifelong burdens: Exposure to air pollution can alter children's trajectory through life, pushing them onto a path of suffering, illness and challenge. But this is preventable. Informed action by health professionals can help reduce the tremendous burden of disease in children caused by the exposure to air pollution.

Air pollutants do not recognize political borders but travel wherever the wind and prevailing weather patterns take them. Therefore, regional and international cooperative approaches are necessary to achieve meaningful reductions in children's exposure. Approaches to preventing exposure must be

complementary and mutually reinforcing, on every scale: houses, clinics, health care institutions, municipalities, national governments and the global community. Health care professionals can push together for strong action from decision-makers to protect the most vulnerable, voiceless citizens: children who have little or no control over the air they breathe. Individual efforts can add up to collective action that changes minds, changes policies and changes the quality of the air around us. Such actions would go far towards ensuring that children can breathe freely, without the terrible burdens imposed by air pollution.

References – executive summary

1. Ambient air pollution: a global assessment of exposure and burden of disease, second edition. Geneva: World Health Organization (in press).
2. Burden of disease from ambient air pollution for 2016. Version 2 May 2018. Summary of results. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/en/>, accessed September 2018).
3. Exposure to household air pollution for 2016. Version 5 April 2018. Summary of results. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/cities/en/>, accessed August 2018).
4. Burden of disease from household air pollution for 2016. Version 3 April 2018 Summary of results. Geneva: World Health Organization; 2018. (<http://www.who.int/airpollution/data/en/>, accessed August 2018).

5. WHO's global ambient air quality database – update 2018. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/cities>, accessed August 2018).
6. Global Health Observatory (GHO) data. Causes of child mortality, 2016. Geneva: World Health Organization; 2018 (http://www.who.int/gho/child_health/mortality/causes/en/, accessed August 2018).
7. Burning opportunity: clean household energy for health, sustainable development, and wellbeing of women and children. Geneva: World Health Organization; 2016 (<http://www.who.int/iris/handle/10665/204717>, accessed August 2018).
8. WHO air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Global update 2005. Summary of risk assessment. Geneva: World Health Organization; 2006 (<http://www.who.int/iris/handle/10665/69477>, accessed August 2018).
9. WHO guidelines for indoor air quality: household fuel combustion. Geneva: World Health Organization; 2014 (<http://www.who.int/iris/handle/10665/141496>, accessed August 2018).
10. Inheriting a sustainable world? Atlas on children's health and the environment. Geneva: World Health Organization; 2017 (<http://www.who.int/iris/handle/10665/254677>, accessed August 2018).

1. Introduction

Every day around the world, billions of children are exposed to unsafe levels of air pollution. The result is a global public health emergency. Air pollution, whether encountered outdoors or indoors, poses serious risks to children's health. In 2016, 93% of the global population under 18 years of age – including 630 million children under 5 – were exposed to ambient levels of fine PM (PM_{2.5}) pollution that exceed the annual mean WHO air quality guideline (see Annex 2). About three billion people were exposed to HAP from the use of polluting fuels for cooking in 2016 (1).

The health burden of air pollution on the world's children is immense. Environmental factors are responsible for an estimated 26% of all child deaths worldwide (2). ALRI are the second leading killer of children under 5 years worldwide (3). Together, AAP and HAP cause more than 50% of all ALRI in children under 5 in LMICs (4). In 2016, AAP and HAP were together responsible for approximately 543 000 deaths among children under 5 from ALRI, accounting for almost 10% of all child deaths that year (5). Over 99.9% of those deaths were in LMICs. In 2016, ALRI caused by HAP was responsible for 441 000 deaths and around 40 million DALYs in children under 15 (6). Among

Air pollution causes over half of all deaths from acute lower respiratory infections in children under 5 in low- and middle-income countries.

children aged 5–15 years, 52 000 deaths were attributed to the joint effect of HAP and AAP in 2016 (7). These deaths and lost years of healthy life could largely be prevented by improving the environmental conditions to which children are exposed.

The heaviest burden on the smallest shoulders

Air pollution cuts so many lives short, but it can also lead to health burdens that last a lifetime. Exposure increases the risks of adverse birth outcomes, neurodevelopmental disorders and reduced lung function. In addition to respiratory infections, it is also clearly linked to a higher risk of developing asthma, a major cause of morbidity in children.

The toll is perhaps most severe on the very youngest. Fetuses and infants have long been recognized as especially vulnerable to the effects of exposure to environmental agents such as air pollutants, with possible lifelong consequences (8). The earlier children's exposure, the greater their potential loss of healthy years of life (9). A child exposed in the first months of life can suffer lifelong effects, including increased risks of heart disease, stroke and cancer. A growing body of evidence suggests that air pollution can adversely affect cognitive and behavioural development in children and that early exposure might lead to the development of chronic disease in adulthood (4). Better understanding of the effects of air pollution on fetal and childhood growth and disease development is critically important to inform actions and policies to protect public health.

Children live, learn and grow in various contexts and environments: the home, school, the playground, the neighbourhood, the community, the country and the world at large. In these settings, they encounter pollutants from a wide range of sources, with varying effects on their health. For instance, young children in LMICs often spend much of their time with their mothers around the home and hearth and are thus exposed to high levels of smoke emitted from cooking and heating stoves. In poorly ventilated homes, they may breathe polluted air at levels that far exceed WHO guidelines, while their sensitive airways, lungs and immune systems are still developing (10). Compounding these risks is the fact that children are at the mercy of their environment, with little to no control over their living conditions. Air pollution has not only effects on physical health effects but can add psychological burdens of stress and anxiety (10).

Clear and mounting evidence

There is a large body of research on the effects of air pollution on child health, which is reviewed in this report, including effects on fetal growth and birth outcomes, lung development and function, asthma, respiratory infection and otitis media. The links between air pollution and neurodevelopmental disorders (including autism spectrum disorder and attention deficit hyperactivity disorder) are also reviewed, as are associations between air pollution and obesity or insulin resistance in children (conditions that can develop into metabolic syndrome or diabetes mellitus in later life).

The report focuses on exposure to AAP and HAP from the combustion of polluting fuels. It does not include the evidence for all sources of indoor air pollution, such as second-hand tobacco smoke, which are beyond the scope of this document. The evidence of the harm done by tobacco smoking and second-hand smoke to people of all ages is well established. The WHO Framework Convention on Tobacco Control and other initiatives (e.g. the Tobacco Free Initiative) were created to reduce exposure to tobacco smoke. The final section of this document gives suggestions for actions by clinicians and other health care professionals to address the health effects of air pollution, some of which include actions to reduce exposure to tobacco smoke.

Since publication of the monograph on the effects of air pollution on children's health and development by the WHO Regional Office for Europe in 2005 (11), many studies have been published that strengthen the evidence of links between AAP and HAP and health effects in children. This report summarizes the findings of the latest peer-reviewed research on a number of health effects. As there are many studies, the report is based on systematic reviews, meta-analyses and recent studies (published in the past 10 years).

Evidence of causal links is lacking for many exposures, as epidemiology cannot prove causation. Action is warranted, however, when there is sufficient evidence from epidemiological studies and experimental research that strongly suggests causality. A significant number of studies have established associations between air pollution and various health outcomes. For outcomes for which the evidence of links is inconsistent, "knowledge gaps" and questions for further research are suggested.

For certain health outcomes, studies provide strong evidence of the effects of exposure to AAP, but there are relatively few studies of the links with HAP. As the sources of AAP and HAP often overlap significantly, the evidence for AAP could be considered indirect evidence for the health effects of HAP. Minimizing children's exposure to both forms of pollution, especially during the most sensitive, developmental stages of early life, should take precedence over establishing near-certainty about the full extent of the risk and the mechanisms involved. Preventive strategies could be based on the evidence for AAP on the assumption that HAP has similar effects.

Informing action

Scientific understanding of the serious risks posed by air pollution early in life is robust and growing. This knowledge must be translated into action. Taken together, the body of evidence provides ample support for strong action and effective policy measures. The closing section of this review accordingly suggests specific, concrete actions for paediatricians, obstetricians, health care providers, communities and families responsible for protecting fetuses, infants and children.

Recent WHO publications on environmental risk factors for health reveal the scope of the problem. This document builds on and adds to the evidence in those reports: the atlas on children's health and the environment (12), "Burning opportunity" (10), the guidelines for indoor air quality associated with household fuel combustion (13) and the guidelines for ambient air quality (14).

Scope and purpose of the report

Environmental health – and particularly paediatric environmental health – is experiencing accelerated growth. Health care providers who work daily with children, adolescents and their families and communities are on the front line of assessment, treatment and prevention of environment-related diseases and are in a key position to educate the public and provide guidance to parents of young children on ways to mitigate the health risks from air pollution in their home environment.

This document provides health professionals with a summary of up-to-date evidence based on an extensive literature review on the relations between air pollution and various health outcomes in children, including the impact of exposure during pregnancy, child growth and birth outcomes, lung development and function, asthma, respiratory infections, otitis media, neurodevelopmental disorders and childhood cancers. The review includes not only the best available scientific evidence but also expert input on knowledge gaps and research needs. Case studies of successful policies and cost-effective interventions are highlighted as examples of action that could be taken at various levels. The aim is to provide health professionals with concrete actions to protect children from the health risks of exposure to air pollution.

References – introduction

1. Exposure to household air pollution for 2016. Version 5 April 2018. Summary of results. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/cities/en/>, accessed August 2018).
2. Prüss-Ustün A, Wolf J, Corvalán C, Bos R, Neira M. Preventing disease through healthy environments: a global assessment of the burden of disease from environmental risks. Geneva: World Health Organization; 2016 (<http://www.who.int/iris/handle/10665/204585>, accessed August 2018).
3. Global Health Observatory (GHO) data. Causes of child mortality, 2016. Geneva: World Health Organization; 2018 (http://www.who.int/gho/child_health/mortality/causes/en/, accessed August 2018).
4. “Don’t pollute my future!” The impact of the environment on children’s health. Geneva: World Health Organization; 2017:30 (<http://www.who.int/iris/handle/10665/254678>, accessed August 2018).
5. Ambient air pollution: a global assessment of exposure and burden of disease, second edition. Geneva: World Health Organization; in press.
6. Global Health Observatory (GHO) data. Household air pollution, burden of disease. Geneva: World Health Organization; 2018 (<http://apps.who.int/gho/data/node.main.139?lang=en>, accessed August 2018).
7. Burden of disease from the joint effects of household and ambient air pollution for 2016. Summary of results. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/cities/en/>, accessed August 2018).
8. Perera FP. Multiple threats to child health from fossil fuel combustion: impacts of air pollution and climate change. *Environ Health Perspect.* 2017;125(2):141–8.
9. Kassebaum NJ, Arora M, Barber RM, Bhutta ZA, Brown J, Carter A, et al. Global, regional, and national disability-adjusted life-years (DALYs) for 315 diseases and injuries and healthy life expectancy (HALE), 1990–2015: a systematic analysis for the Global Burden of Disease study 2015. *Lancet.* 2016;388(10053):1603–58.
10. Burning opportunity: clean household energy for health, sustainable development, and wellbeing of women and children. Geneva: World Health Organization; 2016 (<http://www.who.int/iris/handle/10665/204717>, accessed August 2018).
11. Effects of air pollution on children’s health and development: a review of the evidence. Copenhagen: WHO Regional Office for Europe; 2005.
12. Inheriting a sustainable world? Atlas on children’s health and the environment. Geneva: World Health Organization; 2017 (<http://www.who.int/iris/handle/10665/254677>, accessed August 2018).
13. WHO guidelines for indoor air quality: household fuel combustion. Geneva: World Health Organization; 2014 (<http://www.who.int/iris/handle/10665/141496>, accessed August 2018).
14. WHO air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide: global update 2005. Summary of risk assessment. Geneva: World Health Organization; 2006 (<http://www.who.int/iris/handle/10665/69477>, accessed August 2018).

2. Routes of exposure to air pollution

Fetuses, infants and children have unique vulnerability and susceptibility to the risks of exposure to air pollution, including subsequent development of adverse health outcomes. These heightened risks are due to a combination of behavioural, environmental and physiological factors. (Note: detailed information on the sources of ambient and HAP can be found in Section 4 below.)

2.1 Inhalation

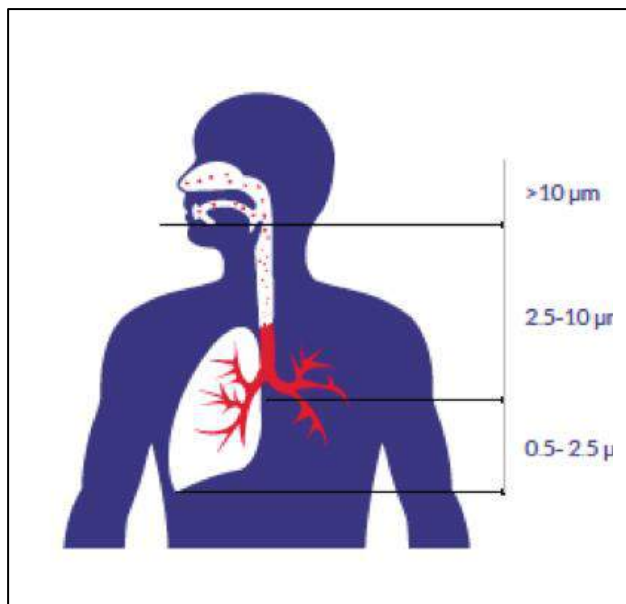
Inhalation is the primary means by which air pollutants enter the human body, through the lungs and alveoli (tiny air-filled sacs located at the end of the bronchioles in the lungs where oxygen exchanges with carbon dioxide in the blood). During the first few years of life, the numbers of alveoli increase rapidly, after which the volume begins to increase; this is therefore a critical window of growth, which may be affected by inhaled pollutants (1). The lungs grow throughout childhood and adolescence and may be exposed to many airborne pollutants that harm growth and function (1). As children breathe at twice the rate that adults do, they inhale larger amounts of air pollutants (2). Particles are also moved through the respiratory system faster, allowing them to reach the lungs, the alveoli and the bloodstream more rapidly (3). In addition, children are more physically active than adults, so that their ventilation is even greater; and they are closer to the ground, where the levels of pollutants are often more concentrated (4). In the case of particulate pollution, the size of the particle

determines how far into the body it penetrates and where it is deposited. Particles $< 10 \mu\text{m}$ in diameter (PM_{10}) are typically filtered out through the nose, whereas smaller particles, such as $\text{PM}_{2.5}$ (particles $< 2.5 \mu\text{m}$ in diameter) can penetrate deeper and reach the lower airways. As children typically breathe through their mouths, the nasal filtration mechanism is often bypassed, so that more particles move into their lungs than is the case for adults (3). During pregnancy, a woman's ventilation rate also increases (5,6), increasing both her own exposure and that of her fetus.

Childhood exposure to air pollution by inhalation is associated with disease later in life. Exposure to various pollutants, including black carbon, NO_2 , $\text{PM}_{2.5}$ and PM_{10} , is linked to the development of asthma in children (7–9), presumably due in large part to the generation of oxidative stress and airway inflammation (10). Research also indicates that PM may cause systemic inflammatory and immunological responses and remodelling in the lung (11).

Other authors have found that ultrafine particles ($< 0.1 \mu\text{m}$ in diameter) can cross the alveoli, enter the bloodstream and cause cardiovascular and cerebrovascular disease (12–15). Research shows that PM_{10} stimulates alveolar macrophages to release the prothrombotic cytokine IL-6, which can cause accelerated arterial thrombosis and increase the risk of cardiovascular events (16).

Fig. 6. Smaller particles of particulate matter penetrate deeper into the lungs



Source: Reference (17)

Inhalation of CO can also have significant health consequences (Boxes 1 and 2). Once absorbed, CO quickly binds the haemoglobin of red blood cells (which have a high affinity for CO), creating carboxyhaemoglobin, which displaces oxygen, potentially leading to tissue hypoxia (18). Environmental exposure to CO has been linked to cardiovascular illness and elevated levels of carboxyhaemoglobin in adults. Evidence suggests that chronic exposure to CO may cause changes to the structure and function of the heart that could leave it more susceptible to stress (19).

Box 1. Dangers of carbon monoxide

CO is a colourless, odourless, tasteless toxic gas produced by incomplete oxidation during the combustion of fuels. It can be emitted from household cooking and heating systems, vehicle exhaust, industrial processes and fires (20). CO inhalation can be deadly. Breathing is the only pathway for CO to enter the body, where it combines with haemoglobin in the blood and reduces oxygen-carrying capacity of the blood. Children are most susceptible to CO toxicity due to their higher metabolic rates (21). Symptoms of CO intoxication can include headaches, irritability, dizziness, fatigue, weakness, drowsiness, nausea, vomiting, loss of consciousness, skin pallor, dyspnoea, palpitations, confusion and irrational behaviour (21).

Low levels of CO can be found in ambient air near roads and parking areas. Common indoor sources of CO vary across high-, low- and middle-income countries. In high-income countries, the main indoor source of CO

is emissions from defective appliances for cooking or heating. If they are not properly maintained, gas burners, wood-burning fireplaces, clogged chimneys and supplementary heaters can all be potential sources of CO. High levels of CO have also been measured in both public and residential garages. In low- and middle-income countries, the most important sources are the burning of biomass fuels, especially in poorly ventilated kitchens, and tobacco smoke (22).

Box 2. Case study: Carbon monoxide: an invisible threat at home

After moving into a new house, a 9-year-old child began experiencing recurring headaches and gastrointestinal discomfort. These symptoms were followed by an episode of loss of consciousness at home. Several medical examinations yielded no conclusive diagnosis. His teachers reported poorer school performance. Some months later, during the winter, the whole family suffered an episode of loss of consciousness. The ultimate diagnosis was CO intoxication. An extended study of the family's house found the source: a wood-burning stove used for heating during winter.

Case report by Amalia Laborde, Professor of Toxicology, University of the Republic, Uruguay

2.2 In utero

The placenta plays a crucial role in the growth and development of the fetus, providing nutrients and oxygen and removing waste and toxicants throughout pregnancy (23–25). Because the placenta is so important in the exchange of substances between the mother and the fetus, it is also a pathway for exposure of the fetus when the mother is exposed to air pollution (26, 27).

Certain inhaled or ingested pollutants that are small enough to penetrate the alveolar wall, including ultrafine PM and heavy metals, can enter the mother's bloodstream (26,28–31). They can then cross the placental barrier and reach the fetus, affecting growth and development by a variety of mechanisms (29,32,33). They can cause oxidative stress, damage DNA and reduce absorption of nutrients by the fetus (30,34). One study suggested that alteration of placental mitochondrial DNA content may be the mediator between exposure to air pollution and low birth weight (35). Exposure to air pollutants in utero can alter the newborn's immune cell population and may predispose children to allergies and asthma (29). Maternal exposure to lead has been linked to increased fetal lead levels and adverse effects on neurodevelopment later in life (36). A recent study also shows that maternal exposure to HAP during pregnancy leads to chronic hypoxia in the placenta; fetal development under these conditions may be associated with adverse pregnancy outcomes (37). Inflammation is another important mechanism, as both maternal and intrauterine inflammation have been observed in response to air pollution, a factor that is believed to play an important role in adverse birth outcomes and poor neurodevelopment (38,39).

The direct consequences of air pollution on maternal health present additional risks to the fetus. For example, both AAP and HAP have been linked to hypertension in pregnancy (40–42). Hypertensive disorders in pregnancy are a leading cause of maternal mortality worldwide (43) and are associated with adverse birth outcomes, including preterm birth and low birth weight (44,45). Children whose mothers experience preeclampsia during pregnancy are also at increased risk of health complications later in life, including endocrine, nutritional, and metabolic diseases (46).

2.3 Ingestion

Air pollutants can settle on surfaces in the home, where an infant or child can ingest them. Because pollutants can persist in the environment for some time after their release, their impact is not always short-lived. Some substances that are released into the air, such as mercury and pesticides, can enter the hydrological cycle as a result of atmospheric dispersion and precipitation and can then be ingested during contact with contaminated water, food, soil or vegetation (20,47).

Breastfeeding is the best source of nutrition for infants, as it provides them with an optimal balance of nutrients while strengthening their immune systems and forming a bond between the mother and the child (48). There is, however, evidence that air pollutants accumulate in breast milk, resulting in exposure of the child. Pollutants from industrial sources, such as pesticides, fossil fuels, chemical by-products, flame retardants, heavy metals and volatile organic compounds, can enter the mother's circulation by inhalation or, more commonly, ingestion before being passed into breast milk (49,50).

For instance, PAHs have been reported at high levels in breast milk samples in the Mediterranean (51). PAHs are classified as carcinogenic, and exposure through breastfeeding may result in adverse developmental outcomes (52,53). Nevertheless, the advantages of breastfeeding still far outweigh any risks from most contaminants. WHO recommends exclusive breastfeeding for up to 6 months of age, with continued breastfeeding and appropriate complementary foods up until 2 years of age (50,54).

Exposure to air pollutants during the pre-conception period may also affect the health of both the mother and the fetus. A recent study showed that maternal exposure to NO_x and SO₂ in the months before pregnancy is associated with an increased risk for gestational diabetes mellitus, a condition that is associated with adverse birth outcomes and risks to maternal health (55). Studies also indicate that exposure to SO₂ before pregnancy may play a role in the formation of orofacial clefts (56). Paternal exposure is also important. A study of persistent organic pollutants indicated that exposure of the mother and/or the father before conception resulted in lower birth weight (57).

References – routes of exposure to air pollution

1. Calogero C, Sly PD. Developmental physiology: lung function during growth and development from birth to old age. *Eur Resp. Monograph*. 2010;1:1–15.
2. Etzel RA. Indoor and outdoor air pollution: tobacco smoke, moulds and diseases in infants and children. *Int J Hyg Environ Health*. 2007;210(5):611–6.
3. Saadeh R, Klaunig J. Child's development and respiratory system toxicity. *J Environ Anal Toxicol*. 2014;4(5):1.
4. Goldizen FC, Sly PD, Knibbs LD. Respiratory effects of air pollution on children. *Pediatr Pulmonol*. 2016;51(1):94–108.
5. Hackley B, Feinstein A, Dixon J. Air pollution: impact on maternal and perinatal health. *J Midwifery Women's Health*. 2007;52(5):435–43.
6. Principles for evaluating health risks in children associated with exposure to chemicals (Environmental Health Criteria 237). Geneva: World Health Organization; 2006.
7. Gehring U, Wijga AH, Hoek G, Bellander T, Berdel D, Brueske I, et al. Exposure to air pollution and development of asthma and rhinoconjunctivitis throughout childhood and adolescence: a population-based birth cohort study. *Lancet Resp Med*. 2015;3(12):933–42.
8. Nishimura KK, Galanter JE, Roth LA, Oh SS, Thakur N, Nguyen EA, et al. Early-life air pollution and asthma risk in minority children. The GALA II and SAGE II studies. *Am J Resp Crit Care Med*. 2013;188(3):309–18.
9. Khreis H, Kelly C, Tate J, Parslow R, Lucas K, Nieuwenhuijsen M. Exposure to traffic-related air pollution and risk of development of childhood asthma: a systematic review and meta-analysis. *Environ Int*. 2017;100:1–31.
10. Esposito S, Tenconi R, Lelii M, Preti V, Nazzari E, Consolo S, et al. Possible molecular mechanisms linking air pollution and asthma in children. *BMC Pulmon Med*. 2014;14(1):31.
11. Grunig G, Marsh LM, Esmail N, Jackson K, Gordon T, Reibman J, et al. Perspective: ambient air pollution: inflammatory response and effects on the lung's vasculature. *Pulm Circ*. 2014;4(1): 25–35.
12. Nemmar A, Hoet PH, Vanquickenborne B, Dinsdale D, Thomeer M, Hoylaerts MF, et al. Passage of inhaled particles into the blood circulation in humans. *Circulation*. 2002;105(4):411–4.
13. Geiser M, Rothen-Rutishauser B, Kapp N, Schürch S, Kreyling W, Schulz H, et al. Ultrafine particles cross cellular membranes by nonphagocytic mechanisms in lungs and in cultured cells. *Environ Health Perspect*. 2005;113(11):1555–60.
14. Ling SH, Van Eeden SF, Hogg J. Particulate matter air pollution exposure: role in the development and exacerbation of chronic obstructive pulmonary disease. *Int J COPD*. 2009;4:233–43.
15. Choi HS, Ashitate Y, Lee JH, Kim SH, Matsui A, Insin N, et al. Rapid translocation of nanoparticles from the lung airspaces to the body. *Nat Biotechnol*. 2011;28(12):1300–3.
16. Mutlu GM, Green D, Bellmeyer A, Baker CM, Burgess Z, Rajamannan N, et al. Ambient particulate matter accelerates coagulation via an IL-6-dependent pathway. *J Clin Investig*. 2007;117(10):2952–61.
17. Burning opportunity: clean household energy for health, sustainable development, and wellbeing of women and children. Geneva: World Health Organization; 2016 (<http://www.who.int/iris/handle/10665/204717>, accessed August 2018).
18. Yang W, Omaye ST. Air pollutants, oxidative stress and human health. *Mutat Res*. 2009;674(1–2):45–54.
19. Reboul C, Thireau J, Meyer G, Andre L, Obert P, Cazorla O, et al. Carbon monoxide exposure in the urban environment: an insidious foe for the heart? *Resp Physiol Neurobiol*. 2012;184(2):204–12.
20. Levy RJ. Carbon monoxide pollution and neurodevelopment: a public health concern. *Neurotoxicol Teratol*. 2015;49:31–40.
21. Etzel RA, Balk SJ, editors. Pediatric environmental health. 3rd edition. Itasca (IL): American Academy of Pediatrics; 2012.
22. WHO guidelines for indoor air quality: selected pollutants. Copenhagen: WHO Regional Office for Europe; 2010 (<http://www.who.int/iris/handle/10665/260127>, accessed March 2018).

23. Sood R, Zehnder JL, Druzin ML, Brown PO. Gene expression patterns in human placenta. *Proc Natl Acad Sci USA*. 2006;103(14):5478–83.
24. Gundacker C, Hengstschläger M. The role of the placenta in fetal exposure to heavy metals. *Wien Med Wochenschr*. 2012;162(9–10):201–6.
25. Moore KL. *The developing human: clinically oriented embryology*. 9th edition. Amsterdam: Elsevier; 2013.
26. Sly PD, Boner AL, Bjorksten B, Bush A, Custovic A, Eigenmann PA, et al. Early identification of atopy in the prediction of persistent asthma in children. *Lancet*. 2008;372(9643):1100–6.
27. Saenen ND, Vrijens K, Janssen BG, Madhloum N, Peusens M, Gyselaers W, et al. Placental nitrosative stress and exposure to ambient air pollution during gestation: a population study. *Am J Epidemiol*. 2016;184(6):442–9.
28. Valavanidis A, Fiotakis K, Vlachogianni T. Airborne particulate matter and human health: toxicological assessment and importance of size and composition of particles for oxidate damage and carcinogenic mechanisms. *J Environ Sci Health C Environ Carcinog Ecotoxicol Rev*. 2008;26(4):339–62.
29. Baiz N, Slama R, Béné MC, Charles MA, Kolopp-Sarda MN, Magnan A, et al. Maternal exposure to air pollution before and during pregnancy related to changes in newborn's cord blood lymphocyte subpopulations. The EDEN study cohort. *BMC Pregnancy Childbirth*. 2011;11(1):87.
30. Smarr MM, Vadillo-Ortega F, Castillo-Castrejon M, O'Neill MS. The use of ultrasound measurements in environmental epidemiological studies of air pollution and fetal growth. *Curr Opin Pediatr*. 2013;25(2):240–6.
31. Morakinyo OM, Mokgobu MI, Mukhola MS. Health outcomes of exposure to biological and chemical components of inhalable and respirable particulate matter. *Int J Environ Res Public Health*. 2016;1:1–22.
32. Slama R, Darrow L, Parker J, Woodruff TJ, Strickland M, Nieuwenhuijsen M, et al. Meeting report: atmospheric pollution and human reproduction. *Environ Health Perspect*. 2008;116(6):791–8.
33. Every breath we take: the lifelong impact of air pollution. Report of a working party. February 2016. London: Royal College of Physicians; 2016.
34. Proietti E, Rösli M, Frey U, Latzin P. Air pollution during pregnancy and neonatal outcome: a review. *J Aerosol Med Pulm Drug Deliver*. 2013;26(1):9–23.
35. Clemente DBP, Casa M, Vilahur N, Beginstain H, Bustamante M, Carsin AE, et al. Prenatal ambient air pollution, placental mitochondrial DNA content, and birth weight in the INMA (Spain) and ENVIRONAGE (Belgium) birth cohorts. *Environ Health Perspect*. 2016;124(5):659–65.
36. Hu H, Téllez-Rojo MM, Bellinger D, Smith D, Ettinger AS, Lamadrid-Figueroa H, et al. Fetal lead exposure at each stage of pregnancy as a predictor of infant mental development. *Environ Health Perspect*. 2006;114(11):1730–5.
37. Dutta A, Khramstova G, Brito K, Alexander D, Mueller A, Chinthala S, et al. Household air pollution and chronic hypoxia in the placenta of pregnant Nigerian women: a randomized controlled ethanol cookstove intervention. *Sci Total Environ*. 2018;619:212–20.
38. Lee PC, Talbott EO, Roberts JM, Catov JM, Sharma RK, Ritz B. Particulate air pollution exposure and C-reactive protein during early pregnancy. *Epidemiology (Cambridge Mass)*. 2011;22(4):524–31.
39. Nachman RM, Mao G, Zhang X, Hong X, Chen Z, Soria CS, et al. Intrauterine inflammation and maternal exposure to ambient PM_{2.5} during preconception and specific periods of pregnancy: the Boston birth cohort. *Environ Health Perspect*. 2016;124(10):1608–15.
40. Hu H, Ha S, Roth P, Kearney G, Talbott EO, Xu X. Ambient air pollution and hypertensive disorders of pregnancy: a systematic review and meta-analysis. *Atmos Environ*. 1994;2014 Nov 1:336–45.
41. Pedersen M, Stayner L, Slama R, Sørensen M, Figueras F, Nieuwenhuijsen MJ, et al. Ambient air pollution and pregnancy-induced hypertensive disorders: a systematic review and meta-analysis. *Hypertension*. 2014;64(3):494–500.
42. Alexander D, Northcross A, Wilson N, Dutta A, Pandya R, Ibigbami T, et al. Randomized controlled ethanol cookstove intervention and blood pressure in pregnant Nigerian women. *Am J Resp Critical Care Med*. 2017;195(12):1629–39.
43. Lo JO, Mission JF, Caughey AB. Hypertensive disease of pregnancy and maternal mortality. *Curr Opin Obstet Gynecol*. 2013;25(2):124–32.
44. Goldenberg RL, Culhane JF, Iams JD, Romero R. Epidemiology and causes of preterm birth. *Lancet*. 2008;371(9606):75–84.
45. Bramham K, Parnell B, Nelson-Piercy C, Seed PT, Poston L, Chappell LC. Chronic hypertension and pregnancy outcomes: systematic review and meta-analysis. *BMJ*. 2014;348(7954):g2301.
46. Wu CS, Nohr EA, Bech BH, Verstergaard M, Catov JM, Olsen J. Health of children born to mothers who had preeclampsia: a population-based cohort study. *Am J Obstet Gynecol*. 2009;201(3):269.
47. Children's health and the environment: a global perspective. Geneva: World Health Organization; 2005 (<http://www.who.int/iris/handle/10665/43162>, accessed February 2018).
48. WHO recommendations on interventions to improve preterm birth outcomes. Geneva: World Health Organization; 2015:98 (<http://www.who.int/iris/handle/10665/183037>, accessed August 2108).
49. Massart F, Harrell JC, Federico G, Saggese G. Human breast milk and xenoestrogen exposure: a possible impact on human health. *J Perinatol*. 2005;25(4):282–8.
50. Massart F, Gherarducci G, Marchi B, Saggese G. Chemical biomarkers of human breast milk pollution.

- Biomarker Insights. 2008(3):159–69.
51. Çok I, Mazmanci B, Mazmanci MA, Turgut C, Henkelmann B, Schramm KW. Analysis of human milk to assess exposure to PAHs, PCBs and organochlorine pesticides in the vicinity Mediterranean city Mersin, Turkey. *Environ Int.* 2012;40(1):63–9.
 52. Perera FP, Rauh V, Whyatt RM, Tang D, Tsai WY, Bernert JT, et al. A summary of recent findings on birth outcomes and developmental effects of prenatal ETS, PAH, and pesticide exposures. *Neurotoxicology.* 2005;26(4):573–87.
 53. Perera FP, Li Z, Whyatt R, Hoepner L, Wang S, Camann D, et al. Prenatal airborne polycyclic aromatic hydrocarbon exposure and child IQ at age 5 years. *Pediatrics.* 2009;124(2):e195–202.
 54. Essential nutrition actions: improving maternal, newborn, infant and young child health and nutrition. Geneva: World Health Organization; 2013 (<http://www.who.int/iris/handle/10665/84409>, accessed February 2018).
 55. Robledo CA, Mendola P, Yeung E, Männistö T, Sundaram R, Liu D, et al. Preconception and early pregnancy air pollution exposures and risk of gestational diabetes mellitus. *Environ Res.* 2015;137:316–22.
 56. Zhu Y, Zhang C, Liu D, Grantz KL, Wallace M, Mendola P. Maternal ambient air pollution exposure preconception and during early gestation and offspring congenital orofacial defects. *Environ Res.* 2015;140:714–20.
 57. Robledo CA, Yeung E, Mendola P, Sundaram R, Maisog J, Sweeney AM, et al. Preconception maternal and paternal exposure to persistent organic pollutants and birth size: the LIFE study. *Environ Health Perspect.* 2015;123(1):88.

3. Vulnerability and susceptibility of children

A number of studies have established that fetuses, infants and children are particularly susceptible and vulnerable² to air pollutants (Fig. 7) (1–9). Children breathe more rapidly than adults, because of their higher resting metabolic rate; as a consequence, they inhale more air – and more air pollutants – relative to their body weight (2,6,10). Children also have a larger lung surface area per kilogram of body weight than adults (1,2). During early life, the respiratory system grows and develops rapidly, and the lung surface area and number of alveoli increase significantly until around 5–8 years of age (11); a higher ratio of lung surface area to volume facilitates absorption of particles. Lung growth continues until about 20 years of age (12). Lung development trajectories are set in early life, so that damage during the prenatal and postnatal stages is potentially irreparable (2,6,10,11,13).

Fig. 7. Fetal development and timing of air pollution risks

² Vulnerable: exposed to the possibility of being harmed by something. Susceptible: being likely or liable to be influenced or harmed by something.

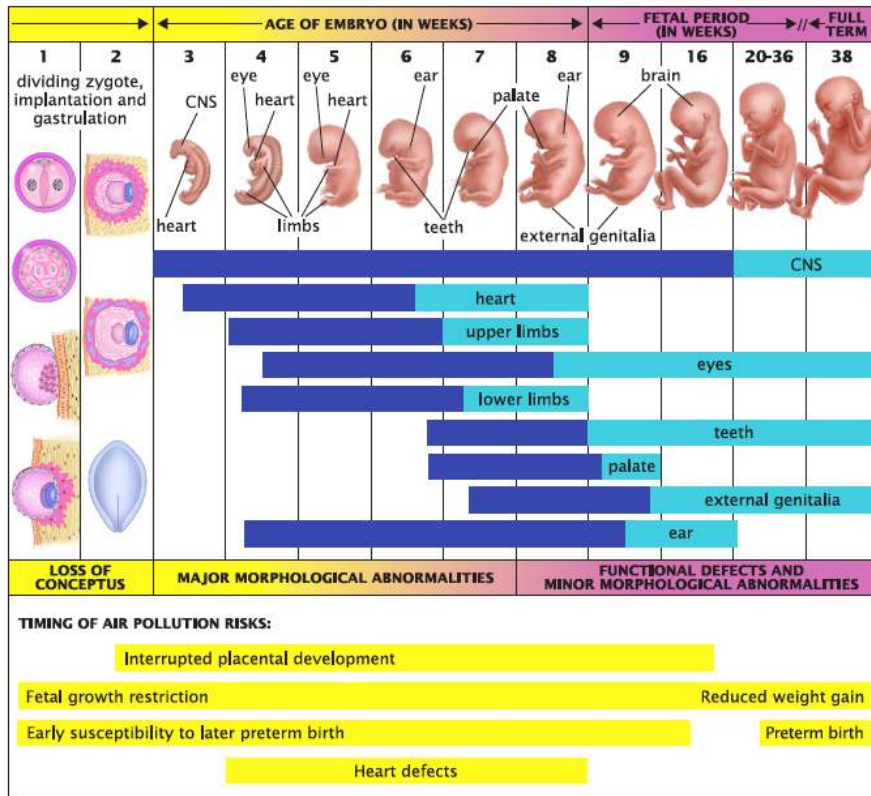


Figure 1. Fetal development and timing of air pollution risks.

Source: Reference (14)

During critical windows of gestation and childhood, when organ systems are developing rapidly, children are more vulnerable to permanent damage. At birth, the immune, respiratory and central nervous systems are immature and highly sensitive to environmental stimuli (15), and many mechanisms have been proposed whereby exposure to air pollutants is linked to health effects in children. Oxidative stress and inflammation are predominant and probably play an important role in perinatal outcomes and childhood asthma (16,17). Air pollutants may also impact endothelial function, coagulation, and maternal hemodynamic responses during pregnancy (16).

Exposure to air pollution during pregnancy, a particularly critical period of development, has been linked to various health outcomes. Exposure before 18 weeks of gestation has been linked to diminished development. Various pollutants are passed from mother to fetus with oxygen and nutrients. A mother's exposure to air pollutants during pregnancy can result in permanent damage to the respiratory and cardiovascular systems, cognitive impairment, intrauterine growth restriction and even compromise the development of vital organ systems (2,18–20). Exposure during this critical period has also been linked to permanent changes in the structure of the lungs, which can have lifelong health consequences (11,21). The timing of environmental exposure during pregnancy determines the effect on the developing fetus: earlier exposures tend to affect development of the airway tree and major pulmonary vessels, whereas later exposure can influence lung volume, alveoli and pulmonary capillaries (11).

Children's airway passages are narrower than those of adults. Thus, any irritation and subsequent inflammation from exposure to air pollutants can result in proportionately greater airway obstruction (2,6,10,22). Exposure to air pollutants can exacerbate existing health conditions in children and cause additional complications (4,23). Children with respiratory or cardiovascular conditions are at particular risk (24).

Children may also be more susceptible than adults to the effects of air pollution because of their behaviour. Children spend their days closer to the floor than adults, and some pollutants in household and ambient air are found in the highest concentrations in this zone, where children breathe and play (25,26). The concentration of nitric oxide has been reported to be significantly higher at children's height near heavy traffic (23,27). In the home, children are often with or near their mothers as they cook, exposing them to additional air pollutants. Infants are often unable to move away from sources

of air pollution, and older children may not recognize the hazards, further compounding the risks (26). Older children spend more time outside, running, playing and breathing hard, and this increased ventilation exposes them to larger doses of AAP (28). Infants are likely to place objects in their mouth, placing them at risk of ingestion of air pollutants (28). Children with pica behaviour, who compulsively put objects in their mouths, are at particular risk.

Children have a longer life expectancy than adults, rendering them more vulnerable to the potential health effects of air pollution in yet another way (8,12,29). They have more time to manifest a disease with a long latency period and will potentially live longer with negative health consequences (15). Thus, the earlier their exposure, the longer the potential chronic illness or disability they will experience. This kind of cumulative exposure to air pollution can become a life sentence, imposed just as life is beginning.

For the same reasons, the early years, starting in pregnancy, are the best time to invest in a child's health by acting to improve their environment and reduce their exposure (30). This is therefore a window of opportunity to improve their lives. Precisely because children are most vulnerable and sensitive to environmental influences in their earliest years, action to protect them during this critical phase can yield immense benefits.

References – vulnerability and susceptibility of children

1. Schwartz J. Air pollution and children's health. *Pediatrics*. 2004;113(4):1037–43.
2. Effects of air pollution on children's health and development: a review of the evidence. Copenhagen: WHO Regional Office for Europe; 2005.
3. Children's health and the environment: a global perspective. Geneva: World Health Organization; 2005.
4. Takenoue Y, Kaneko T, Miyamae T, Mori M, Yokota S. Influence of outdoor NO₂ exposure on asthma in childhood: meta-analysis. *Pediatr Int*. 2012;54(6):762–9.
5. Pedersen M, Giorgis-Allemand L, Bernard C, Aguilera I, Andersen AM, Ballester F, et al. Ambient air pollution and low birthweight: a European cohort study (ESCAPE). *Lancet Resp Med*. 2013;1(9):695–704.
6. Landrigan PJ, Etzel RA. *Textbook of children's environmental health*. New York City (NY): Oxford University Press; 2014.
7. Perera FP. Multiple threats to child health from fossil fuel combustion: impacts of air pollution and climate change. *Environ Health Perspect*. 2017;125(2):141–8.
8. "Don't pollute my future!" The impact of the environment on children's health. Geneva: World Health Organization; 2017:30 (<http://www.who.int/iris/handle/10665/254678>, accessed August 2018).
9. Inheriting a sustainable world? Atlas on children's health and the environment. Geneva: World Health Organization; 2017 (<http://www.who.int/iris/handle/10665/254677>., accessed August 2018).
10. *Pediatric environmental health*. 3rd edition. Washington DC: American Academy of Pediatrics. Council on Environmental Health; 2012.
11. Calogero C, Sly PD. Developmental physiology: lung function during growth and development from birth to old age. *Eur Resp. Monograph*. 2010;1:1–15.
12. Principles for evaluating health risks in children associated with exposure to chemicals (Environmental Health Criteria 237). Geneva: World Health Organization; 2006.
13. Sly PD, Boner AL, Bjorksten B, Bush A, Custovic A, Eigenmann PA, et al. Early identification of atopy in the prediction of persistent asthma in children. *Lancet*. 2008;372(9643):1100–6.
14. Ritz B, Wilhelm M. Air pollution impacts on infants and children. Los Angeles (CA): UCLA Institute of the Environment; 2008 (<https://www.ioes.ucla.edu/publication/air-pollution-impacts-on-infants-and-children/>, accessed August 2018).
15. Sly PD, Flack F. Susceptibility of children to environmental pollutants. *Ann N Y Acad Sci*. 2008;1140(1):163–83.
16. Kannan S, Misra DP, Dvonch JT, Krishnakumar, A. Exposure to airborne particulate matter and adverse perinatal outcomes: a biologically plausible mechanistic framework for exploring potential. *Environ Health Perspect*. 2006;114(11):1636–42.
17. Esposito S, Tenconi R, Lelii M, Preti V, Nazzari E, Consolo S, et al. Possible molecular mechanisms linking air pollution and asthma in children. *BMC Pulmon Med*. 2014;14(1):1–8.
18. Vrijheid M, Martinez D, Manzanares S, Dadvand P, Schembari A, Rankin J, et al. Ambient air pollution and risk of congenital anomalies: a systematic review and meta-analysis. *Environ Health Perspect*. 2011;119(5):598–606.
19. Pedersen M, Stayner L, Slama R, Sørensen M, Figueras F, Nieuwenhuijsen MJ, et al. Ambient air pollution and pregnancy-induced hypertensive disorders: a systematic review and meta-analysis. *Hypertension*. 2014;64(3):494–500.
20. Nachman RM, Mao G, Zhang X, Hong X, Chen Z, Soria CS, et al. Intrauterine inflammation and maternal exposure to ambient PM_{2.5} during preconception and specific periods of pregnancy: the Boston birth

- cohort. *Environ Health Perspect.* 2016;124(10):1608–15.
21. Kajekar R. Environmental factors and developmental outcomes in the lung. *Pharmacol Ther.* 2007;114(2):129–45.
 22. Etzel RA. Indoor and outdoor air pollution: tobacco smoke, moulds and diseases in infants and children. *Int J Hyg Environ Health.* 2007;210(5):611–6.
 23. Gasana J, Dillikar D, Mendy A, Forno E, Ramos Vieira E. Motor vehicle air pollution and asthma in children: a meta-analysis. *Environ Res.* 2012;117:36–45.
 24. Dick S, Doust E, Cowie H, Ayres JG, Turner S. Associations between environmental exposures and asthma control and exacerbations in young children: a systematic review. *BMJ Open* 2014;4(2):1–7.
 25. Gurunathan S, Robson M, Freeman N, Buckley B, Roy A, Meyer R, et al. Accumulation of chlorpyrifos on residential surfaces and toys accessible to children. *Environ Health Perspect.* 1998;106(1):9–16.
 26. Miller MD, Marty MA, Arcus A, Brown J, Morry D, Sandy MS. Differences between children and adults: implications for risk assessment at California EPA. *Int J Toxicol.* 2002;21(5):403–18.
 27. Kenagy HS, Lin C, Wu H, Heal MR. Greater nitrogen dioxide concentrations at child versus adult breathing heights close to urban main road kerbside. *Air Qual Atmos Health.* 2016;9(6):589–95.
 28. Goldizen FC, Sly PD, Knibbs LD. Respiratory effects of air pollution on children. *Pediatr Pulmon.* 2016;51(1):94–108.
 29. Sly PD, Schüepp K. Nanoparticles and children’s lungs: Is there a need for caution? *Paediatr Resp Rev.* 2012;13(2):71–72.
 30. Engle PL, Fernald LCH, Alderman H, Behrman J, O’Gara C, Yousafzai A, et al. Strategies for reducing inequalities and improving developmental outcomes for young children in low-income and middle-income countries. *Lancet.* 2011;378:1339–53.

4. Sources of air pollution

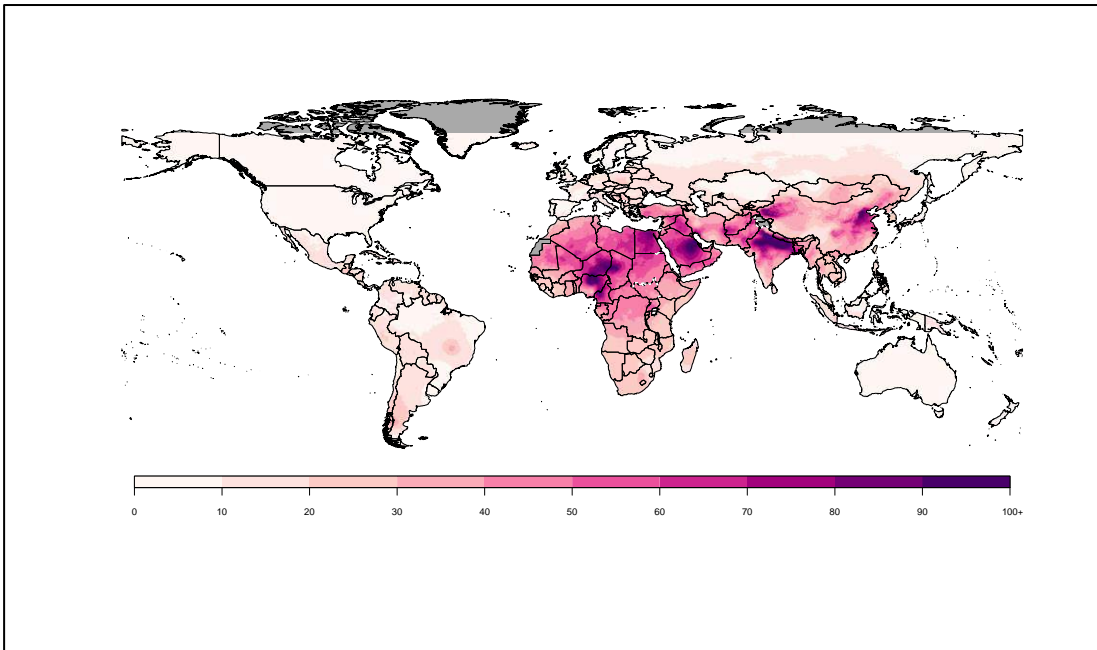
Air pollution from particulate matter is a problem that transcends geographical and political boundaries. It affects people in all countries, in every type of human settlement. It flows between nations and over oceans, from outdoors to indoors to outdoors, from the upwind countryside into the city and then on to the countryside again. Pollutants can be found equally in high, unsafe concentrations in the most remote, rural village and on the busiest, high-traffic urban streets. Children are threatened by this pollution in every region of the world.

HAP and AAP are strongly interconnected: the former is a major, often underestimated source of the latter (1,2). In this section, AAP and HAP are treated separately, as they have been considered distinct phenomena by both scientists and policy-makers. HAP consists of emissions from all household energy, including lighting and heating. Because most of the relevant research to date has focused on cooking and much of the data on energy use are from surveys on cooking fuel and technology, HAP is generally perceived as a risk mostly for rural LMICs (3). While this is broadly true, reliance on polluting household fuels also persists in many urban areas as well. For instance, with growing awareness of the health threat posed by AAP, heating fuels are gaining attention as an important source of air pollution, including in HICs.

4.1 Ambient air pollution

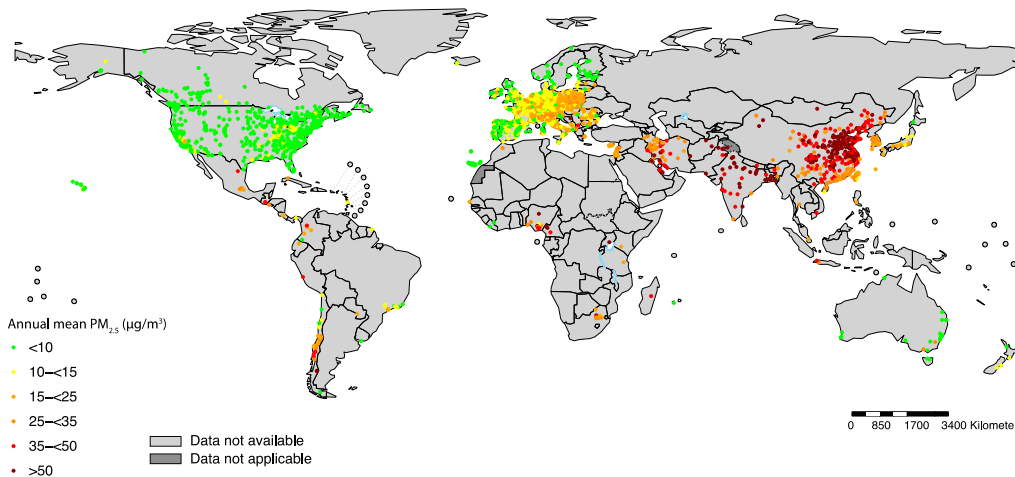
AAP is a global health crisis. The vast majority of people live in places where it is unsafe to breathe the air. Today, 91% of the world’s population – and 93% of its children – are exposed to fine PM at levels that exceed the WHO-recommended limit (Fig. 8 and 9) (4). The distribution of the crisis is also becoming less equal: in more than 4300 settlements monitored by WHO, pollution levels have improved in cities in some HICs but worsened in poorer regions (5).

Fig. 8. Annual average concentrations of ambient PM_{2.5} in µg/m³, 2016.



Source: WHO global ambient air quality database (update 2018). <http://www.who.int/airpollution/data/en/>

Fig. 9. PM_{2.5} concentrations in more than 4300 human settlements, 2010–2016.



Source: WHO global ambient air quality database (update 2018). <http://www.who.int/airpollution/data/cities/en/>
 The map shows sites for which data were available. Few data were available for some areas, including highly polluted settlements.

AAP caused about 4.2 million premature deaths in 2016 (6). In 2016, an estimated 286 000 children under 15 years of age died from exposure to AAP (see Annex 2). The sources of pollution differ in urban and rural contexts (7), the main sources in urban settings being fossil fuel combustion for energy production, transport, residential cooking and heating (household fuel use) and waste incineration (8). Use of polluting fuels persists in some higher-income countries; in some countries in Europe and the Arabian Gulf, for example, coal is still used for household heating, posing a risk of CO poisoning for both children and adults (9). The main source of pollution in rural communities in LMICs, however, is burning of kerosene, biomass and coal for cooking, heating and lighting, agricultural waste incineration and certain agro-forestry activities (8).

Interventions to improve the quality of the air in rural areas must target these and other major sources, including excessive use of agrochemicals like fertilizers and pesticides, deforestation, small-scale industries such as charcoal production and spontaneous forest fires, fog and dust storms (10,11). Geographical and meteorological factors influence the transport and chemistry of air pollutants. Urban areas can affect downwind rural areas, just as rural activities – such as agricultural burning –

can affect air quality in nearby cities. For these reasons, interventions to improve air quality in any locality require cooperation at many levels, including regional and international (1,12).

Poverty is closely associated with high exposure to air pollution. LMICs generally experience higher levels of exposure to PM, particularly in the WHO African, South-East Asian and Western Pacific regions (4), where the annual mean levels of PM_{2.5} are 5–10 times greater than the WHO guideline limit (13). Even in regions in which PM_{2.5} levels are lower and closer to the WHO limit values, such as Europe and Latin America, the levels in LMICs are almost twice those in HICs. It should be noted that national estimates of exposure to PM_{2.5} are averages and that, even within HICs, low-income communities have disproportionately higher exposure to air pollution, as they tend to be located closer to major sources, such as industrial facilities, high-traffic roads and power plants (14). Box 3 gives an example of a successful programme to reduce HAP.

Box 3. Better stoves, better sleep – lessons from a poverty alleviation programme in Peru (15)

Exclusive use of cleaner-burning biomass stoves was linked to better sleep and alleviation of respiratory symptoms in children in a village in Peru.

As a part of the Peruvian Government's "Juntos" national poverty alleviation programme, residents of the small village of Lliupapuquio, Andahuaylas, were given cleaner-burning biomass cooking stoves. These "Inkawasi" stoves have been demonstrated to reduce PM emissions by up to 75% and wood use by 50% as compared with traditional stoves.

Respiratory symptoms in children were assessed while they were sleeping before and after introduction of the stoves. The study subjects were 82 children under 15 years of age who had been exposed to smoke from biomass fuels throughout their lives. During the initial assessment, the population reported a wide prevalence of respiratory symptoms, and more than 33% of the children reported waking during the night, daytime sleepiness and falling asleep at school.

Statistically significant improvements in symptoms were found after introduction of the cleaner-burning stoves; in some cases, the symptoms disappeared. The researchers concluded that exclusive use of the new stoves was probably an important factor in the improvement in health. This study adds to evidence of the health gains made with proper, sustained, exclusive use of clean cooking, as opposed to "fuel stacking", in which a household continues to use polluting energy sources for some tasks.

The sources of AAP vary from manmade to natural, from fossil fuel combustion to crop burning to wildfires, all of which produce a complex mixture of pollutants that can interact chemically. They usually include CO, nitrogen oxides (NO, NO₂, NO_x), lead, arsenic, mercury, SO₂, PAHs and PM (PM_{2.5}, PM₁₀, and ultrafine particulate matter) (16). Air pollution from a combination of agricultural activities, urban emissions and atmospheric conditions contributes to annual periods of extreme air pollution in parts of South-East Asia (2).

Certain pollutants react in the atmosphere and in high temperatures to form secondary pollutants, such as O₃. Ground-level O₃ is created when pollutants such as NO_x and volatile organic compounds react with sunlight (16,17). Whereas O₃ in the upper atmosphere is beneficial, as it blocks incoming ultraviolet radiation, exposure to ground-level O₃ can cause breathing problems, trigger asthma, reduce lung function and cause various lung diseases (16,17,18).

It is difficult to measure the changing components of this complex mixture precisely. Certain pollutants have direct toxic effects and are also used as indicators of total exposure by researchers and national government agencies that set and enforce air quality standards. PM (PM₁₀, PM_{2.5}) is one of the most commonly used markers of exposure to air pollution in general; other common indicators are SO₂, O₃, NO₂, CO and lead (19,20). The WHO air quality guidelines, updated in 2005 (16) were proposed as a basis for regulatory changes to reduce emissions of pollutants and for policy to reduce the health impacts of air pollution. The guidelines propose recommended thresholds and limits for these key ambient air pollutants. Setting science-based air quality standards for important pollutants is one of the most important steps that decision-makers can take to protect the health of their citizens, including children. Governments should adopt the WHO air quality guidelines (e.g. an annual mean threshold of 10 µg/m³ for PM_{2.5}) or set their own stringent emissions limits. Monitoring and identifying areas that exceed the recommended maximum air pollution levels is essential for effective interventions to protect health. Box 4 gives examples of initiatives to help people reduce air pollution.

Box 4. Digital tools to help citizens fight air pollution and improve health

Regional and global initiatives that leverage the trend of increasing digital connectivity in societies around the world provide citizens with access to regularly updated, online data about the quality of the air where they live.

BreatheLife is an initiative of WHO and UN Environment to raise global awareness about the health risks posed by air pollution. Evidence-based information and resources are provided to mobilize individuals and cities to take action to clean up the air they breathe. BreatheLife's network of participating cities is growing, allowing urban decision-makers to demonstrate support for and share lessons about solutions to improving air quality. On the programme's website (www.breathelife2030.org), individuals can access updated information on the pollution levels in their cities and countries and about the related burden of disease and human cost of air pollution.

Country-level initiatives to make data on air quality more accessible and user-friendly include the Air Quality Health Index in Ontario, Canada (www.airqualityontario.com), which rates air quality on a scale from 1 to 10 and is updated daily. The index indicates action people can take to protect themselves when local pollution levels exceed the recommended limits.

Working towards solutions

Addressing AAP is an increasingly important priority for governments and multilateral agencies. Many proven solutions are available to reduce emissions of dangerous pollutants in cities, including cleaner transport, cleaner cooking and heating fuels and technologies, energy-efficient housing and urban planning, low- to zero-emission power generation, cleaner, safer industrial technologies and better municipal waste management (21).

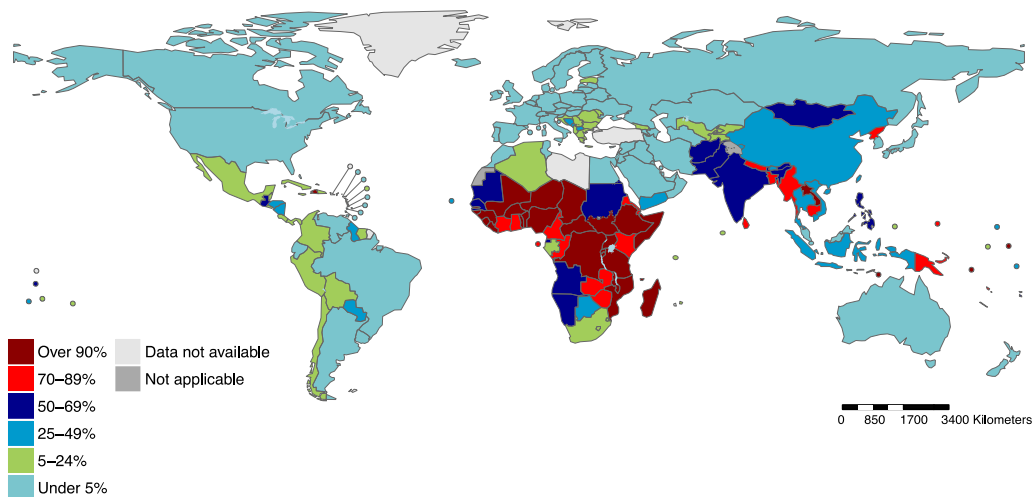
WHO has been publishing reports on air pollution and its health effects since 1958, including work that led to the first air quality guidelines, in the mid-1980s (22). Since the release of the current version of the WHO air quality guidelines in 2005, more evidence has become available on the health effects of ambient air pollutants, even at relatively low concentrations. The guidelines are therefore being revised to reflect the latest available evidence, and an updated version with recommended thresholds for key air pollutants will be published in 2020.

4.2 Household air pollution

Polluted air inside homes, schools, workplaces and recreation facilities – spaces where pregnant women, mothers, infants and children spend much of their time – cause and contribute to a wide range of negative health outcomes. Tragically, excessive air pollution can turn the very places that are meant to shelter and nurture children into places of risk.

HAP is produced mainly by incomplete combustion of polluting fuels used for cooking, heating and lighting and is the single largest environmental health risk factor worldwide (8). HAP is also an important source of AAP (23). In 2016, WHO estimated that about three billion people – 41% of the world's population – still used polluting fuels for cooking, mostly in LMICs (Fig. 10) (24), and this number has remained largely unchanged for the past three decades. The damage to health caused by dependence on polluting energy sources is severe and extensive: in 2016, HAP from solid fuel use resulted in 3.8 million premature deaths, equivalent to 6.7% of global mortality, greater than the toll due to malaria, TB and HIV/AIDS combined. Of these deaths, 403 000 were among children under 5 years of age (8,18). The risks of children are not limited to direct exposure, as there is emerging evidence that pregnant mothers' exposure to high levels of HAP is linked to higher risks of adverse birth outcomes such as low birth weight (25).

Fig. 10. Proportions of households that used polluting fuels for cooking, 2016



Source: Reference (28)

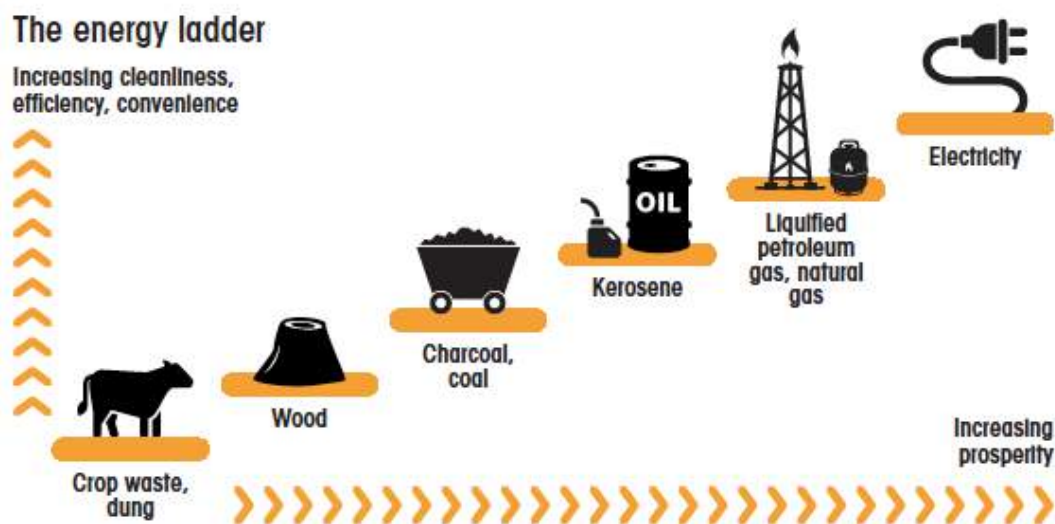
Families' daily acts of survival can undermine the health of their own children. Smoke emitted from burning biomass (wood, dung and crop residues), coal, charcoal and kerosene for cooking, heating and lighting is the primary contributor to HAP; other significant sources are tobacco smoke, candles, incense and mosquito coils (8). Burning produces complex mixtures of contaminants, the composition of which depends on the type of fuel used and on the temperature and phase of combustion (26–29). For instance, emissions from the combustion of coal can contain PM, CO, NO_x, SO₂, benzene, PAHs, carbon and several heavy metals (30). Kerosene combustion can emit CO, NO_x, PM, SO₂, formaldehyde and PAHs (31). Kerosene smoke is also extremely rich in black carbon, a major component of fine PM and a potent climate-warming pollutant (32).

In some regions, children are especially vulnerable to HAP because they spend so much time in the home and with their mothers as they tend the hearth. Women and girls are the main users of household energy around the world. They also spend significant time and effort gathering, transporting, preparing and using fuels like biomass, coal and charcoal to cook food in inefficient stoves or open hearths and to heat their homes. In dwellings with poor ventilation, emissions of fine PM and other pollutants from these stoves can exceed the maximum exposure recommended by WHO by 100 times (8).

Cooking is not the only use of household energy use that poses risks to children's health. The WHO guidelines for household fuel combustion (31) classify kerosene as a polluting fuel and discourage its use as a household fuel. Nevertheless, kerosene is still used for lighting by many of the around one billion people who lack access to electricity. Kerosene lamps are often the only means of lighting houses at night, allowing children and adolescents to study in areas without electricity. Use of kerosene not only pollutes the air inside houses but also increases the risks for fires, burns and CO poisoning.

Although significant numbers of urban dwellers in LMICs still use polluting fuels and devices for cooking, heating and lighting, the vast majority of those who use polluting household energy live in rural areas (Fig. 11) (3). Reliance on polluting fuels is especially prevalent in rural areas of the WHO African, South-East Asian and Western Pacific regions (8). Persistent use of polluting cooking, heating and lighting fuels by more than three billion people – and the resulting health risks – is due largely to lack of access to clean, affordable, convenient alternatives (33). Despite recent progress in all WHO regions in increasing access to clean fuels and technologies (Fig. 12), the number of people who use polluting fuels did not change appreciably between 1990 and 2016 (Fig. 13) (24). Given this trend, better coordinated and concerted action is required to meet the SDG 7 target of achieving universal access to clean energy by 2030.

Fig. 11. The “energy ladder”, showing increasing cleanliness, efficiency and convenience with increasing prosperity

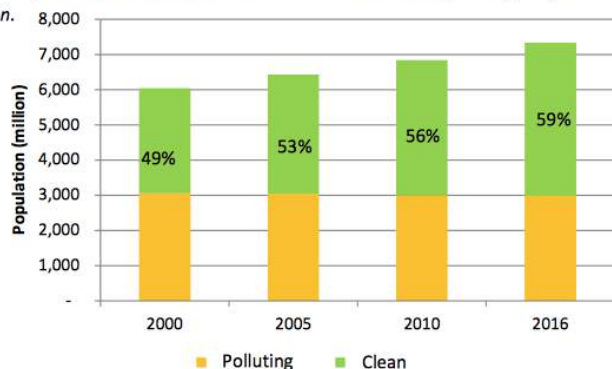


Source: Reference (34).

The combination of fuel and technology determines whether a stove is “clean for health”; e.g., some pellet-burning stoves achieve WHO guideline levels in laboratory testing.

Fig. 12. Population with and without access to clean technologies and fuels for cooking, by year and by region.

Figure 3. Population with and without access to clean technologies and fuels for cooking, by year and by region.

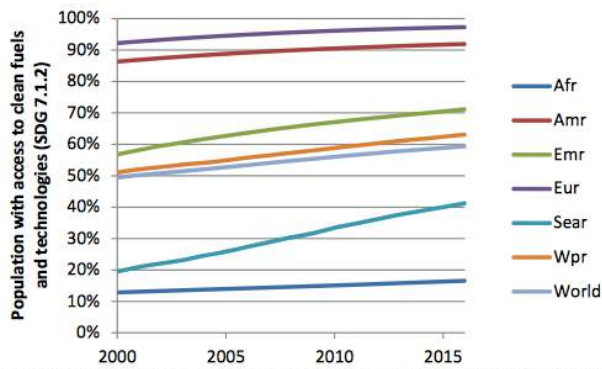


Afr: Africa; Amr: America; Emr: Eastern Mediterranean; Eur: Europe; Sear: South-East Asia; Wpr: Western Pacific; LMIC: Low- and middle-income; HIC: High-income.

Source: Reference (24).

Fig. 13. Trends in access to clean fuels and technologies for cooking, 2000–2016, by region

Figure 2. Trends in access to clean fuels and technologies for cooking for the years 2000-2016, by region.



Afr: Africa; Amr: America; Emr: Eastern Mediterranean; Eur: Europe; Sear: South-East Asia, Wpr: Western Pacific; LMIC: Low- and middle-income; HIC: High-income.

Source: Reference (24).

AFR, African Region; AMR, Region of the Americas; EMR, Eastern Mediterranean Region; SEAR, South-East Asian Region; WPR, Western Pacific Region

HAP and AAP are interconnected: the latter can make its way indoors, and the former contributes to poor air quality outdoors. Globally, an estimated 12% of ambient PM_{2.5} comes from use of solid fuel for cooking (35). This “leakage” of indoor pollutants outdoors is responsible for almost half a million premature deaths due to AAP (36). Reducing cross-contamination between household and ambient air by reducing reliance on polluting fuels in both rural and urban areas throughout the world is an urgent priority for health sector professionals and for those working in energy and sustainable development. Accelerating the transition to clean household energy for billions of people is a critically important means for protecting children’s health.

Working towards solutions

In 2014, WHO issued the first set of guidelines on fuels and technologies for cooking, heating and lighting that are clean for health (31). The guidelines include recommendations on combinations of fuel and technology that are clean for health at the point of use, including electricity, LPG, biogas, ethanol and solar stoves, as well as high-performing biomass stoves that meet the emission rate targets in the guidelines. The guidelines discourage household use of kerosene and unprocessed coal because of the serious health hazards they pose.

Achieving universal access to clean, safe household energy is a high priority on the global sustainable development agenda, reflected in SDG 7 (“ensure access to affordable, reliable, sustainable and modern energy for all”). Box 5 presents the human rights issues involved. WHO has resources to help health professionals and decision-makers to integrate health concerns into planning, programmes and policies on household energy and air quality. These include the Clean Household Energy Solutions Toolkit, which contains modules on needs assessment, standards and testing for household energy devices, monitoring and evaluation and materials, to help the health sector in tackling HAP. (See also Annex 1.)

Box 5. Air pollution is a child rights issue

No group is more vulnerable to environmental harm than children. Yet, a healthy environment is essential for children to fully enjoy their right to health. The Convention on the Rights of the Child (37), the universally ratified human rights treaty, requires States Parties to pursue full implementation of the right to health by appropriate measures that include the provision of nutritious foods and clean drinking-water, taking into consideration the dangers and risks of environmental pollution (Art. 24 (2) (c)). Air pollution in particular jeopardizes children’s right to health. Countless children suffer disease and disability from air pollution, often with lifelong effects, as it can disrupt their physical and cognitive development.

States have heightened their obligations to respect, protect and fulfil the rights of children, who are often unable to protect themselves from environmental harm, including air pollution. The obligations include:

- ensuring that educational programmes increase children’s understanding of environmental issues and strengthen their capacity to respond to environmental challenges;

- assessing the effects of proposed measures on children’s rights before the measures are taken or approved;
- collecting information on sources of environmental harm to children and making the information publicly available and accessible;
- facilitating the participation of children in environmental decision-making and protecting them from reprisals for their participation or otherwise expressing their views on environmental matters; and
- removing barriers to children’s access to justice for environmental harm so that can fully enjoy their rights.

States should adopt and implement environmental standards that are consistent with the best available evidence and relevant international health and safety standards. Thus, States should implement recommendations from expert agencies, such as WHO, on specific measures to protect children’s health and well-being from environmental harm. A good example of concrete guidance is the report of the United Nations Special Rapporteur on Human Rights and the Environment, John H. Knox, on the threat posed to children’s health and well-being in Mongolia by air pollution (38). The Rapporteur noted that, in 2017, the Committee on the Rights of the Child expressed serious concern about the effects of air pollution on Mongolian children, particularly in the capital, Ulaanbaatar. Each winter, the city has some of the most heavily polluted air in the world, as residents of *gers* burn coal in household stoves to stay warm. Knox observed that children are at particular risk, jeopardizing their rights to life and health.

Other sources of indoor air pollution

Many other air pollutants are also important health risks, but fall beyond the scope of this report, which addresses primarily sources of air pollution arising from combustion. Contaminants present in urban and rural indoor air include volatile organic compounds, asbestos, pesticides, mercury (e.g. from broken thermometers), radon and biological pollutants. Volatile organic compounds produce vapours readily at room temperature and are emitted by thousands of household products, including paints, varnishes, solvents, building materials, disinfectants, personal care products, air fresheners, art and hobby supplies and vehicles used in attached garages (20).

Tobacco smoke is a significant source of indoor air pollution and a health risk for children (39). As many as 4000 chemicals may be present in tobacco smoke alone (40). Asbestos is a known occupational carcinogen, and its use in residential and education buildings can contaminate indoor air (30). Pesticides such as insecticides and antimicrobial disinfectants are often sprayed near the ground and can persist in indoor air or settle on surfaces (41). Radon, a radioactive carcinogenic gas naturally present in some soil and rock, can enter houses, buildings and other enclosed spaces (42-44). This document primarily addresses sources of air pollution arising from combustion.

Various biological pollutants are present in indoor air: dust mites, droppings and urine from pests, insects and rodents, pollen from indoor plants and outdoor air, viruses and bacteria and fungi, including mould and mildew, or their by-products (41,44,45). Build-up of certain biological pollutants can trigger asthma or cause allergic reactions (46,47), and others are linked to the spread of infectious diseases (45).

Working toward solutions

WHO has published a series of indoor air quality guidelines (Table 3) (22). There are many solutions to reducing exposure to other air pollutants. In general, indoor environments can be made healthier by a few key actions: avoiding tobacco smoking, improving ventilation and reducing humidity in houses (to reduce mould and mites), storing chemicals safely and avoiding the use of unnecessary chemicals and pesticides. Asbestos and lead paint should no longer be used in building or renovating houses (21,43,48,49).

Table 3. Common air pollutants: sources of exposure and WHO air quality guidelines

Pollutant	Common sources of exposure.	WHO guideline values ^a	Reference
Benzene	Ambient: Building materials, furniture, attached garages. Motor	No safe level of exposure is recommended.	43

	vehicle exhaust. Refineries and petrol stations.	Unit risk of leukaemia per 1 µg/m ³ air concentration is 6 × 10 ⁻⁶ .	
	Household: Heating and cooking with kerosene. Activities such as cleaning, painting, mosquito repellents, photocopying and printing. Tobacco smoke.	The concentrations of airborne benzene associated with excess lifetime risks of 1/10 000, 1/100 000 and 1/1 000 000 are 17, 1.7 and 0.17 µg/m ³ , respectively.	
Carbon monoxide (CO)	Ambient: Incomplete combustion from burning charcoal or biomass and burning fossil fuels in motor vehicles, electric generators and other machinery.	100 mg/m ³ – 15-min 35 mg/m ³ – 1-h 10 mg/m ³ – 8-h 7mg/m ³ – 24-h	43
	Household: Heating and cooking. Tobacco smoke. Vehicle exhausted from attached garages. Electric generators. Incense burning.	Emission rates from household fuel combustion should not exceed CO (unvented) 0.16 (g/min) CO (vented) 0.59 (g/min)	31
Lead	Vehicle and industry emissions, waste incineration, natural processes (e.g. volcanic eruptions)		
Mercury	Vehicle and industry emissions, waste incineration, natural processes (e.g. volcanic eruptions).		
Naphthalene	Ambient air	0.01 mg/m ³ – annual average	43
	Crystalline (pure) naphthalene moth repellents and disinfectants, herbicides, charcoal lighters and hair sprays, unvented kerosene heaters, tobacco smoke, rubber materials Wood smoke, fuel oil and gasoline.		
Nitrogen dioxide (NO ₂)	Ambient: Combustion processes (heating, power generation, and engines in vehicles and ships).	200 µg/m ³ – 1-h average 40 µg/m ³ – annual average	43
	Household: Heating and cooking – gas, wood, oil, kerosene and coal; tobacco smoke Outdoor air. Occupational use of vehicles indoors.		
Ozone (O ₃)	Vehicle and industry emissions, solvents.	100 µg/m ³ – 8-h mean	16
Particulate matter (PM)	Ambient: Motor vehicle emissions. Combustion of fossil fuels and solid fuels. Dust. Various sources.	PM _{2.5} 10 µg/m ³ – annual mean 25 µg/m ³ – 24-h mean PM ₁₀ 20 µg/m ³ – annual mean 50 µg/m ³ – 24-h mean	16, 50
	Household: Combustion of solid fuels in open fires or traditional stoves. Kerosene.	Emission rates of PM _{2.5} from household fuel combustion should not exceed 0.23 mg/min – unvented 0.80 mg/min – vented	31
Polycyclic aromatic hydrocarbons (PAH)	Ambient: Motor vehicles. Burning of coal and oil for electricity and industrial use. Incomplete combustion of	No threshold can be determined, and all indoor exposures are considered deleterious to health.	43

	organic materials. Household: Heating and cooking with dung, wood, agricultural residues, coal. Tobacco smoke. Incense and candles.	Unit risk for lung cancer estimated to be 8.7×10^{-5} per ng/m^3 of benzo(a)pyrene. The corresponding concentrations from lifetime exposure to benzo(a)pyrene that result in excess lifetime cancer risks of 1/10 000, 1/100 000 and 1/1 000 000 are approximately 1.2, 0.12 and 0.012 ng/m^3 , respectively. 20 $\mu\text{g}/\text{m}^3$ – 24-h mean 500 $\mu\text{g}/\text{m}^3$ – 10-min mean	
Sulfur dioxide (SO ₂)	Ambient: Industrial activities, power generation, motor vehicles. Household: Burning of fossil fuels (coal and oil).		16
Volatile organic compounds	Ambient: Petrochemical solvents, vaporization of unburnt fuel, pesticides. Combustion processes and vehicle exhaust. Household: Cooking, solvents, building materials, household products indoors at room temperature.	No guidelines, although there are recommendations for certain volatile organic compounds, such as benzene, which is carcinogenic to humans; there is no safe threshold for exposure to benzene.	20

This list is of important pollutants in indoor air; it is not exhaustive.

^a Some countries and states within countries (e.g. California in the USA) have adopted guideline levels lower than those of WHO.

4.3 Social determinants of exposure and health

Social determinants of health play a central role in the effects of HAP and AAP on health. The circumstances in which we live powerfully influence our lives, beginning at conception (51). Social inequalities negatively affect infant health and are associated with increased rates of infant mortality rates (52). Studies suggest that social status, especially poverty, influences the risk of environmental exposure. Thus, less affluent populations are at greater risk of a variety of exposures; for example, the combined effects of socioeconomic inequality and reduced air quality can contribute to increased infant mortality (53). It has been estimated that, in 2010, 2.7–3.4 million preterm births globally were associated with exposure to PM_{2.5} during gestation (53).

Poverty and pollution are closely linked. Poverty may force people to rely on polluting fuels for their basic needs, and poverty compounds the health risks associated with their use. Poverty also limits people's choices and their ability to improve the environment in which they raise their children; for example, low-income families cannot just decide to move away from a heavily polluting industrial site. Air pollution is often a chronic problem in poor-quality housing and temporary settlements. The exposure of people living in refugee camps can be particularly high, as they have to scavenge for wood and other fuels or rely on kerosene stoves for heating and cooking.

Dependence on the energy sources that produce most HAP – solid fuels such as wood – also contributes to other important health risks. Children, often at the expense of their schooling or playtime, are sometimes given the tasks of cooking on inefficient stoves or gathering fuel. Fuel collection obliges them to walk long distances with heavy loads. This work can lead to musculoskeletal disorders and can put children, particularly girls, at higher risk of violent attack, rape or injury as they venture far from the household (8,34). The risk of attack while gathering fuel is especially high for girls living in refugee camps (54,55). An analysis of survey data in 16 African countries in 2016 showed that girls in households in which polluting fuels were used spent about 18 hours each week collecting wood or water, whereas girls in households in which clean fuels were used primarily spent 5 hours each week in such tasks (8).

Poverty and pollution are closely linked. The poor often have higher exposure to air pollution and more limited access to treatment and interventions.

Gender also determines exposure of children to HAP and AAP. As they move through childhood, their exposure to HAP and AAP may change according to their gender. In some cultures, girls are kept in the kitchen with their mother for longer than boys because of social and cultural attitudes towards the role of women (56). Girls are therefore more exposed to HAP and boys potentially more exposed to AAP (57). This obviously varies, as some parents have a social preference for male children and spending more time looking after them, so that they have greater exposure to HAP (58). The role of gender in exposure to HAP and AAP is complex and depends on social and cultural attitudes.

References – sources of air pollution

1. Amman M, Purohit P, Bhanarkar AD, Bertok I, Borcken-Kleefeld J, Cofola J, et al. Managing future air quality in megacities: a case study for Delhi. *Atmos Environ*. 2017;161:99–111.
2. GBD MAPS Working Group. Burden of disease attributable to major air pollution sources in India. Special Report 21. Boston (MA): Health Effects Institute; 2018.
3. Global Health Observatory (GHO) data: population with primary reliance on clean fuels and technology. Geneva: World Health Organization; 2018 (<http://apps.who.int/gho/data/node.main.SDGFUELS712?lang=en>, accessed August 2018).
4. Exposure to ambient air pollution from particulate matter for 2016. Version 2. Summary of results. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/cities/en/>, accessed August 2018).
5. WHO's ambient air quality database – update 2018. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/cities>, accessed August 2018).
6. Burden of disease from ambient air pollution for 2016. Version 2 May 2018. Summary of results. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/en/>, accessed September 2018).
7. Landrigan PJ, Sly JL, Ruchirawat M, Silva ER, Huo X, Diaz-Barriga F, et al. Health consequences of environmental exposures: changing global patterns of exposure and disease. *Ann Global Health*. 2016;82(1):10–9.
8. Burning opportunity: clean household energy for health, sustainable development, and wellbeing of women and children. Geneva: World Health Organization; 2016 (<http://www.who.int/iris/handle/10665/204717>, accessed August 2018).
9. Yeatts KB, El-Sadig M, Leith D, Kalsbeek W, Al-Maskari F, Couper D, et al. Indoor air pollutants and health in the United Arab Emirates. *Environ Health Perspect*. 2012;120(5):687–94.
10. Lelieveld J, Evans JS, Fnais M, Giannadaki D, Pozzer A. The contribution of outdoor air pollution sources to premature mortality on a global scale. *Nature*. 2015;525(7569):367–71.
11. Reducing global health risks through mitigation of short-lived climate pollutants. Scoping report for policymakers. Geneva: World Health Organization; Paris: Climate and Clean Air Coalition; 2015 (http://apps.who.int/iris/bitstream/10665/189524/1/9789241565080_eng.pdf?ua=1, accessed August 2018).
12. Maas R, Grennfelt P, editors. Towards cleaner air. Scientific assessment report 2016: summary for policymakers. Geneva: United Nations Economic Commission for Europe; 2016.
13. Air pollution levels rising in many of the world's poorest cities. Press release. Geneva: World Health Organization; 2016 (<http://www.who.int/mediacentre/news/releases/2016/air-pollution-rising/en/>, accessed August 2018).
14. Hajat A, Hsia C, O'Neill MS. Socioeconomic disparities and air pollution exposure: a global review. *Curr Environ Health Rep*. 2015;2(4): 440–50.
15. Accinelli RA, Llanos O, López LM, Pino MI, Bravo YA, Salinas V, et al. Adherence to reduced-polluting biomass fuel stoves improves respiratory and sleep symptoms in children. *BMC Pediatr*. 2014;14(1):12.
16. WHO air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide: global update 2005. Geneva: World Health Organization; 2005 (<http://www.who.int/iris/handle/10665/69477>, accessed August 2018).
17. Ozone pollution. Washington DC: Environmental Protection Agency; 2018 (<https://www.epa.gov/ozone-pollution>, accessed July 2017).
18. Burden of disease from household air pollution for 2016. Version 3 April 2018. Summary of results. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/en/>, accessed August 2018).
19. Air quality standards. Brussels: European Commission; 2016 (<http://ec.europa.eu/environment/air/quality/standards.htm>, accessed 26 July 2017).
20. Volatile organic compounds' impact on indoor air quality. Washington DC: Environmental Protection Agency; 2017 (<https://www.epa.gov/indoor-air-quality-iaq/volatile-organic-compounds-impact-indoor-air-quality>, accessed 29 June 2017).
21. Health and environment: draft road map for an enhanced global response to the adverse health effects of air pollution. Geneva: World Health Organization; 2016.

22. Evolution of WHO air quality guidelines: past, present and future. Copenhagen: WHO Regional Office for Europe; 2017.
23. Chafe ZA, Brauer M, Klimont Z, Van Dingenen M, Mehta S, Rao S, et al. Household cooking with solid fuels contributes to ambient PM_{2.5} air pollution and the burden of disease. *Environ Health Perspect.* 2014;122(12):1314–20.
24. Exposure to household air pollution for 2016. Version 5. Summary of results. Geneva: World Health Organization; 2018 (<http://www.who.int/airpollution/data/cities/en/>, accessed August 2018).
25. Balakrishnan K, Ghosh S, Thangavel G, Sambandam S, Mukhopadhyay K, Puttaswamy N, et al. Exposures to fine particulate matter (PM_{2.5}) and birthweight in a rural-urban, mother-child cohort in Tamil Nadu, India. *Environ Res.* 2018;161:524–31.
26. Fullerton DG, Bruce N, Gordon SB. Indoor air pollution from biomass fuel smoke is a major health concern in the developing world. *Trans R Soc Trop Med Hyg.* 2008;102(9):843–51.
27. Lu H, Zhu L, Zhu N. Polycyclic aromatic hydrocarbon emission from straw burning and the influence of combustion parameters. *Atmos Environ.* 2009;43(4):978–83.
28. Clark ML, Peel JL, Balakrishnan K, Breyse PN, Chillrud SN, Naeher LP, et al. Health and household air pollution from solid fuel use: the need for improved exposure assessment. *Environ Health Perspect.* 2013;121(10):1120–8.
29. Mitchell EJS, Lea-Langton AR, Jones JM, Williams A, Layden P, Johnson R. The impact of fuel properties on the emissions from the combustion of biomass and other solid fuels in a fixed bed domestic stove. *Fuel Process Technol.* 2016;142:115–23.
30. Personal habits and indoor combustions. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 100 (part E). Lyon: International Agency for Research on Cancer; 2012.
31. Indoor air quality guidelines: household fuel combustion. Geneva: World Health Organization; 2014 (<http://www.who.int/iris/bitstream/10665/141496>, accessed August 2018).
32. Lam NL, Chen Y, Weyant C, Venkataraman C, Sadavarte P, Johnson MA, et al. Household light makes global heat: high black carbon emissions from kerosene wick lamps. *Environ Sci Technol.* 2012;46(24):13531–8.
33. Foell W, Pachauri S, Spreng D, Zerriffi H. Household cooking fuels and technologies in developing economies. *Energy Policy.* 2011; 39(12): 7487–96.
34. Inheriting a sustainable world? Atlas on children’s health and the environment. Geneva: World Health Organization; 2017 (<http://www.who.int/iris/handle/10665/254677>, accessed August 2018).
35. Smith KR, Bruce N, Balakrishnan K, Adair-Rohani H, Balmes J, Chafe Z, et al. Millions dead: How do we know and what does it mean? Methods used in the comparative risk assessment of household air pollution. *Ann Rev Public Health.* 2014;35(1):185–206.
36. Lim SS, Vos T, Flaxman AD, Dabaei G, Shibuya K, Adair-Rohani H, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease study 2010. *Lancet.* 2012;380(9859):2224–60.
37. Convention on the Rights of the Child, 20 November 1989, United Nations Treaty Series, Vol. 1577. New York City (NY): United Nations; 1989:3 (<http://www.refworld.org/docid/3ae6b38f0.html>, accessed August 2018).
38. Report of the UN Special Rapporteur on Human Rights and the Environment’s recent visit to Mongolia (A/HRC/37/58). New York City (NY): United Nations; 2018 (http://ap.ohchr.org/documents/dpage_e.aspx?si=A/HRC/37/58/Add.2, accessed August 2018).
39. Gilliland FD, Li YF, Peters JM. Effects of maternal smoking during pregnancy and environmental tobacco smoke on asthma and wheezing in children. *Am J Respir Crit Care Med.* 2001;163:429–36.
40. Stout DM, Bradham KD, Egeghy PP, Jones PA, Croghan CW, Ashley PA, et al. American Healthy Homes Survey: a national study of residential pesticides measured from floor wipes. *Environ Sci Technol.* 2009;43(12):4294–300.
41. Man-made mineral fibres and radon. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 43. Lyon: International Agency for Research on Cancer; 1988.
42. Handbook on indoor radon – a public health perspective. Geneva: World Health Organization; 2009 (http://apps.who.int/iris/bitstream/10665/44149/1/9789241547673_eng.pdf, accessed August 2018).
43. WHO guidelines for indoor air quality: selected pollutants. Copenhagen: WHO Regional Office for Europe; 2010.
44. Indoor air quality: biological contaminants: report on a WHO meeting. Copenhagen: WHO Regional Office for Europe; 1988.
45. Biological pollutants’ impact on indoor air quality. Washington DC: Environmental Protection Agency; 2017 (<https://www.epa.gov/indoor-air-quality-iaq/biological-pollutants-impact-indoor-air-quality>, accessed 29 June 2017).
46. Tischer CG, Hohmann C, Thiering E, Herbath O, Müller A, Henderson J, et al. Meta-analysis of mould and dampness exposure on asthma and allergy in eight European birth cohorts: an ENRIECO initiative. *Allergy.* 2011;66(12):1570–9.
47. Dick S, Doust E, Cowie H, Ayres JG, Turner S. Associations between environmental exposures and asthma control and exacerbations in young children: a systematic review.” *BMJ Open.* 2014;4(2):1–7.

48. WHO guidelines for indoor air quality: dampness and mould. Copenhagen: WHO Regional Office for Europe; 2009 (<http://www.who.int/iris/handle/10665/107880>, accessed August 2018).
49. Healthy environments for healthy children, key messages for action 2010. Geneva: World Health Organization; Nairobi: United Nations Environment Programme; 2010.
50. Ambient air pollution: a global assessment of exposure and burden of disease. Geneva: World Health Organization; 2016
51. Appleton AA, Holdsworth EA, Kubzansky LD. A systematic review of the interplay between social determinants and environmental exposures for early-life outcomes. *Curr Environ Health Report*. 2016;3:287–301.
52. Romieu I, Ramírez-Aguilar M, Moreno-Macias H, Barraza-Villarreal A, Miller P, Hernández-Cadena L, et al. Infant mortality and air pollution: modifying effect by social class. *J Occup Environ Med*. 2004;46(12):1210–6.
53. Malley CS, Kuynlenstierna J, Vallack HW, Henze DK, Blencowe H, Ashmore MR. Preterm birth associated with maternal fine particulate matter exposure: a global, regional and national assessment. *Environ Int*. 2017;101:173–82.
54. Beyond firewood: fuel alternatives and protection strategies for displaced women and girls. New York City (NY): Women’s Commission for Refugee Women and Children; 2006.
55. Statistical snapshot: access to improved cookstoves and fuels and its impact on women’s safety in crises. Factsheet. Washington DC: Global Alliance for Clean Cookstoves; 2015 (<http://cleancookstoves.org/resources/353.html>, accessed August 2018).
56. Armstrong JR, Campbell H. Indoor air pollution exposure and lower respiratory infections in young Gambian children. *Int J Epidemiol*. 1991;20(2):424–9.
57. Matinga MN. We grow up with it: an ethnographic study of the experiences, perceptions and responses to the health impacts of energy acquisition and use in rural South Africa. Thesis. Enschede: University of Twente; 2010 (doc.utwente.nl/75414/1/thesis_M_Matinga.pdf, accessed August 2018).
58. Mishra V, Smith KR, Retherford RD. Effects of cooking smoke and environmental tobacco smoke on acute respiratory infections in young Indian children. *Popul Environ*. 2005;26(5):375–96.

5. Effects of air pollution on child health

Since publication of the monograph on the effects of air pollution on children’s health and development by the WHO Regional Office for Europe in 2005 (1), further evidence has been published of the links between AAP and HAP and the health of children.

This document provides a summary of the latest and best available science, to inform and aid healthcare professionals’ efforts to protect children’s health. For each of the 10 sections on health effects below, two experts did an initial scoping review and reviewed the available evidence; because of the extensive relevant published literature, they gave priority to systematic reviews, meta-analyses and recent studies, mainly those published within the past 10 years. A second in-depth review was conducted to identify additional studies, with a round of extensive peer review by a geographically diverse group of experts in air pollution and child health. The document also cites WHO publications on air pollution that are relevant to child health. It should be noted that this document is neither a full systematic review nor a set of official guidelines, and, because of constraints of space and time, the review does not cover every potential effect of air pollution on children’s health.

The health effects discussed are adverse birth outcomes, infant mortality, effects on neurodevelopment, childhood obesity, effects on lung function, acute lower respiratory infection, asthma, otitis media and childhood cancers, demonstrating the diverse range of impacts that air pollution can have on children, often with long-lasting consequences.

5.1 Adverse birth outcomes

Key findings:

- Numerous studies show significant associations between exposure to AAP and adverse birth outcomes, especially in association with the pollutants PM, SO₂, NO_x, O₃ and CO.
- A growing body of evidence shows that air pollution, particularly PM_{2.5}, is associated with low birth weight, and AAP is associated with preterm birth.

- Few studies have examined the role of HAP, but there is moderate evidence of an association between solid fuel combustion and low birth weight.

The health impacts of exposure to air pollution during the prenatal period are often overlooked but can be quite significant. A growing body of research provides evidence of an association between maternal exposure to air pollution and adverse birth outcomes, including stillbirth, preterm birth, low birth weight and being small for gestational age (SGA) (2–19). Exposure before conception can also affect the fetus, with emerging evidence of a link between exposure to air pollution from traffic and other sources of AAP and reproductive disease in women. A systematic literature review provides suggestive evidence of associations between exposure to AAP and the incidence of reproductive and gynaecological diseases, including infertility and endometriosis, although the number of studies to date is limited (20).

WHO defines stillbirth as fetal death occurring at a birth weight of ≥ 1000 g or at ≥ 28 completed weeks of gestation (21). In 2015, an estimated 2.6 million infants were stillborn, and 98% of the deaths occurred in LMICs (22). Preterm birth is defined by WHO as infants born alive before 37 weeks of gestation; an estimated 15 million infants are born preterm worldwide each year (7). Health complications resulting from preterm birth are the leading cause of death among children < 5 years of age, resulting in over one million deaths in 2016 (7, 23, 24).

Low birth weight is a major public health issue worldwide and is associated with a range of short- and long-term health effects. Low birth weight is defined by WHO as a weight at birth of < 2500 g (2). It has been estimated that 15–20% of all infants born worldwide have a low birth weight, corresponding to more than 20 million births per year (3). SGA is commonly defined as having a weight below the 10th percentile of the recommended sex-specific birth weight for gestational age (10). SGA and low birth weight are also associated with preterm birth (18). Thus, preterm birth and SGA can occur independently or together and can result in low birth weight (18, 25). These outcomes have been associated with increased risks for premature death and disability, including cardiovascular morbidity, chronic lung disease, obesity and metabolic syndrome (18, 26). There is also emerging evidence suggestive of an increased risk for developmental delays and poorer cognitive performance (11, 18). Children who had adverse birth outcomes may require more health care after birth, placing demands on health facilities and resources, with wider social impacts (18).

The evidence for links between air pollution and stillbirth, preterm birth, low birth weight and SGA is discussed below.

Stillbirth

Ambient air pollution

Several studies have been published on the effect of AAP, particularly PM_{2.5}, and stillbirth. In a meta-analysis (27), one study on the effect of PM_{2.5} on risk of stillbirth found a statistically nonsignificant increase in risk of stillbirth per 10 $\mu\text{g}/\text{m}^3$ increase in PM_{2.5} (28). Green and colleagues identified a small, statistically significant increase in risk per 10 $\mu\text{g}/\text{m}^3$ increase in PM_{2.5} in a birth cohort in California, USA (29). Other studies reported modest increased risks associated with exposure to PM_{2.5} throughout pregnancy (30) or at specific stages, such as the third trimester (31) or the week before delivery (32). Ebisu and colleagues (33) recommended that the chemical components of PM_{2.5} and the specific cause of stillbirths (fetal growth, maternal complications) be determined to ensure accurate results.

Other studies have reported increased risks of stillbirth with exposure to total AAP (34), PM_{2.5} (30), PM₁₀ (30, 35, 36), CO (28, 30), SO₂ (28, 30, 36), NO₂ (28–30) and O₃ (29, 37). Some publications reported nonsignificant or no associations (38, 39), and some were limited by distance at which air pollution monitors were located from birth address or by failure to adjust for change of address during pregnancy (28). The strongest effect for PM₁₀, SO₂, CO, and O₃ in most studies was in the third trimester (29, 30, 35, 36, 40). As for PM_{2.5}, evidence indicates that exposure to NO₂ throughout pregnancy increases the risk of stillbirth (29, 30).

Acute exposure to AAP in the week before delivery has been the subject of relatively few studies. New evidence suggests that such exposure increases the risk of stillbirth. Faiz and colleagues (32) found an increased risk of stillbirth when mothers were exposed to high levels of CO, SO₂, NO₂ or

PM_{2.5} in the 6 days before delivery. In a retrospective cohort study of 223 375 births in the USA, exposure to O₃ during the week before delivery increased the risk of stillbirth by 13–22% (37).

Household air pollution

Pope et al. (41) identified four studies of the relation between HAP from solid fuel use and stillbirth: three in India (42–44) and one in Pakistan (45). Three of the studies found a significant association between exposure to HAP and increased risk of stillbirth; the fourth found an increased risk, which was not statistically significant. The review (41) found an overall 51% increase in risk of stillbirth with exposure to HAP. The studies differed in design, particularly with respect to the method for exposure assessment. Two of the studies considered kerosene to be a “low pollution” fuel (43, 44), and one considered that exposure to kerosene led to medium exposure to HAP (45), which contradicts current understanding of the adverse health effects of kerosene. WHO guidelines for indoor air quality associated with household fuel combustion (46) discourage use of kerosene in the home. The review of health effects for the guidelines concluded that the findings of these four studies were consistent and that a causal association was possible but could not be confirmed, given the small number of studies. A more recent review (47) included an additional study and found a 29% increase in the risk of stillbirth but found that kerosene was categorized inconsistently in the studies, with one categorizing it as resulting in “high pollution” (48) and two as “low pollution” (43, 49); in two studies, information on kerosene use was not collected. Different classification of kerosene may affect the interpretation of these results and also the findings of studies on other health effects.

Preterm birth

Ambient air pollution

Maternal exposure to individual air pollutants during pregnancy has been linked with preterm birth (8, 9, 14). A number of studies have found a positive association between maternal exposure to PM_{2.5} and preterm birth (50–52), and it has even been estimated that, in 2010, 2.7–3.4 million preterm births globally were associated with exposure to PM_{2.5} during gestation (53). One review found a nonsignificant association between exposure to PM₁₀ and preterm birth (52). Preterm birth has been consistently associated with SO₂ levels (54, 55), while the evidence for associations with CO, NO, NO₂ and O₃ remains inconclusive (50, 53).

It is important to distinguish between very early and later preterm birth (e.g. before 26 weeks, 26–32 weeks and after 32 weeks), because each has different effects on health and probably different causes. Many studies do not distinguish between very early and later preterm birth, although this distinction would be useful, particularly for evaluating the effects of different levels of AAP.

Household air pollution

Very few studies have been undertaken of HAP and preterm birth. A systematic review by Amegah and colleagues (47) covered three studies (44, 49, 56) and found an increased risk of preterm birth with household solid fuel use. One of the studies (44), conducted in India, gave an adjusted odds ratio of 1.43 for preterm birth in houses in which solid fuel was used as compared with those in which LPG or kerosene was used. The literature review for the WHO guidelines on indoor air quality associated with household fuel combustion did not include an assessment of the quality of the evidence for associations between polluting fuel use and preterm birth, because of the small number of studies (46).

Box 6. Air pollution and congenital anomalies

Congenital anomalies, also known as birth defects, are structural or functional abnormalities that occur during intrauterine life (57). They may be identified before birth, at birth or later in life. Congenital anomalies, including metabolic disorders, account for an estimated 11% of global neonatal deaths, 6% of infant deaths and lifelong morbidity (24). Examples of congenital anomalies linked to environmental factors include congenital heart disease, limb reduction, kidney or urinary tract malformation, cleft lip and palate defects, cryptorchidism and hypospadias. While there is evidence that certain birth defects are associated with exposure of nonsmoking pregnant women to second-hand tobacco smoke, the results of studies of AAP and HAP have been inconsistent.

There is a growing body of literature on the relations between AAP and congenital anomalies, and associations of varying strength have been found. A cohort study conducted in Wuhan, China (58), found a significant association between maternal exposure to atmospheric PM_{2.5} during early pregnancy and congenital heart

defects in the offspring. This study of 105 988 live births, stillbirths and fetal deaths was based on 1-week average concentrations from nearby air pollution monitors and showed a significant association with risk of congenital heart defects, which increased monotonically as PM_{2.5} concentration increased. A study of traffic pollutants (PM₁₀ and benzene) in a community in northern Italy showed an association between exposure to PM₁₀ and birth defects (59). A systematic review of 10 epidemiological studies that included a meta-analysis of four studies of associations between the risk of congenital anomalies and concentrations of various air pollutants found statistically significant increased risks of coarctation of the aorta and of tetralogy of Fallot with exposure to NO₂ and SO₂ (60). It also showed an association between exposure to PM₁₀ and an increased risk of atrial septic defects. Another systematic review and meta-analysis showed a significant association between exposure to NO₂ and coarctation of the aorta (61).

Because of the paucity of studies, there is insufficient evidence of an association between congenital anomalies and HAP other than tobacco smoke. A population-based case–control study in Shanxi Province, China (62) on the link between neural tube defects and HAP from coal combustion demonstrated a dose–response trend, whereby the risk of a child having neural tube defects increased with the mother’s exposure to household coal combustion pollutants.

Low birth weight

Ambient air pollution

Evidence has emerged in the past decade that ambient PM affects birth weight (5, 6, 15–17, 63). Several meta-analyses performed between 2012 and 2016 consistently showed positive associations between exposure to PM_{2.5} during pregnancy and low birth weight and suggest that late pregnancy may be a critically vulnerable time (50, 52, 64–66).

Individual chemical elements of PM may be involved in its toxic effect. In a large study of eight pooled European cohorts, an increased amount of sulfur in PM_{2.5} was associated with an increased risk of low birth weight (17). As the chemical components of PM differ widely by source, this may explain some of the inconsistencies in the findings of different studies (64). In an extensive systematic review of studies in China (55), SO₂ was consistently associated with low birth weight. There is less evidence of associations between exposure during pregnancy to PM₁₀, PAH (benzene, toluene, ethyl benzene, M- and p-xylene, and o-xylene) and other elements of AAP, such as CO and NO₂, and low birth weight (4, 52, 65).

Household air pollution

Several studies have tested the association between HAP, particularly from use of solid fuels, and a lower mean birth weight. These studies found an increased risk of low birth weight between 21% and 35% with exposure to HAP (25, 41, 47, 67, 68).

The review of health effects for the 2014 WHO guidelines led to the conclusion that there was moderate evidence of an association between exposure to solid fuel combustion and low birth weight (46). The review comprised seven studies, which had consistent findings (44, 49, 69–73). An earlier review by Pope and colleagues (41) included many of the same studies from Guatemala, India, Pakistan and Zimbabwe, all of which reported higher risks of low birth weight after maternal exposure to solid fuel combustion in the home. The review found that maternal exposure to HAP increased the risk of low birth weight by 38%, for an average reduction in birth weight of 96.6 g. A recent systematic review by Amegah and colleagues (47) found a 35% increase in risk and an average reduction in birth weight of 54 g, after adjustment for publication bias.

Box 7. Air pollution and adverse birth outcomes: new evidence from cohort studies in India

Air pollution is one of the leading risk factors for the national burden of disease in India (74). Exposure to PM_{2.5} in AAP and HAP has been associated with low birth weight in many studies but in few studies in the high-exposure settings that are common in LMICs such as India. Balakrishnan et al. (75) investigated whether exposure to PM_{2.5} during pregnancy was associated with low birth weight in an integrated rural–urban, mother–child cohort in Tamil Nadu. The researchers recruited 1285 women in the first trimester of pregnancy in primary health care centres and urban health posts and followed them until the birth of their child to collect data on maternal health, prenatal care, exposure to air pollution during pregnancy and the birthweight of the child. They found that a 10 µg/m³ increase in exposure to PM_{2.5} during pregnancy was associated with a decrease in birth weight of 4 g and a 2% increase in the prevalence of low birth weight (after adjustment for gestational age, sex, maternal BMI, maternal age, history of a previous low-birth-weight child, birth order and season of

conception). By applying the exposure–response estimates of the median differences in PM_{2.5} concentration (of ~175 µg/m³) between households in which biomass and clean fuel were used, a 70 g decrease in birth weight was estimated to be associated with solid fuel use (76). This study provided some of the first quantitative estimates of the effects of exposure to PM_{2.5} in India to birth weight. It contributed evidence of this association that is relevant for high-exposure settings in LMICs that experience the dual health burdens of AAP and HAP. The findings indicate that maternal exposure to PM_{2.5} should be considered with other risk factors for low birthweight in India. The study also provided baseline information for a new multi-country HAP intervention trial under way in Guatemala, India, Peru and Rwanda on the effects of clean fuel (in this case, LPG) use on maternal and child health (www.hapintrial.org).

Small for gestational age

Ambient air pollution

Few studies have explored the association between exposure to air pollution and infants born small for gestational age (SGA), defined as birth weight below – 2 standard deviations of the mean or below the 10th percentile according to local intrauterine growth charts (77). Maternal exposure to PM_{2.5} and PM₁₀ has been associated with SGA births (15, 50). Le and colleagues (8) investigated the association between SGA at term and exposure to SO₂, CO, NO₂, O₃ and PM₁₀ during the first month and the third trimester of pregnancy. They found an association between SGA at term and exposure to high CO and NO₂ levels in the first month and with exposure to O₃ and PM₁₀ > 35 µg/m³ during the third trimester. Additional evidence confirms the link between elevated NO₂ levels and SGA. A study of 2.5 million births in Canada between 1999 and 2008 found that exposure to NO₂ during pregnancy was significantly associated with infants born SGA at term. The association was independent of PM_{2.5}, and a dose–response relation was found. This information and evidence that NO₂ is a key component of traffic-related air pollution led the authors to suggest that exposure to traffic is an important factor in adverse pregnancy outcomes such as SGA (54).

Social situation strongly affects birth outcome. A population-based study in the USA linked exposure to both O₃ and PM_{2.5} to SGA. The authors noted that more socially disadvantaged populations are at greater risk of infants born SGA, particularly in the case of exposure to PM_{2.5} (12). Overall, there is growing evidence of an association between SGA and ambient air pollution.

Household air pollution

Associations between HAP and infants born SGA have been identified in several studies. In India, infants born to women who used biomass fuels such as wood and/or dung as the primary cooking fuel in the home during pregnancy were more likely to be SGA (44). In a study of pregnant women in Zambia, household air monitors were used to measure exposure to PM_{2.5} and volatile organic compounds during the first trimester (78). The authors found that increasing levels of pollutants were associated with poor birth outcomes, and both PM_{2.5} and VOCs were associated with SGA. The primary sources of pollution identified were biomass fuels (wood, charcoal, crop residues and cow dung) used for cooking. Garbage burning was also common.

Biological mechanisms

It is difficult to determine the association between air pollution and adverse birth outcomes because so many factors can influence the sensitive periods of development. Several plausible mechanisms have been proposed, including oxidative stress, which may affect the embryo directly in its early stages of development or induce DNA damage, pulmonary and placental inflammation, changes in fetal blood coagulation or endothelial function, and altered maternal haemodynamic response (79).

The placenta is central to the health of the fetus, and airborne pollutants that reach the placenta may cause significant damage. PAHs and CO can cross the placenta, triggering a number of effects (63, 80). Researchers found that maternal exposure to fine particulate matter (PM_{2.5}) and CO in household air during pregnancy increases the risk of fetal thrombotic vasculopathy, a disorder characterized by clots on the fetal surface of the placenta that block vascular flow, and also stillbirth and low birth weight (81).

Studies have identified epigenetic changes in the expression of maternal and fetal DNA in cases in which air pollution has been indicated as a factor in preterm birth, suggesting a new mechanism of action (82). Increased methylation of umbilical cord blood and placental DNA has been noted,

although more research is needed (82). Studies in experimental animals showed that high maternal exposure to PM_{2.5} during pregnancy can cause epigenetic changes that interfere with the cerebral development of the embryo (83).

PM can also stimulate maternal inflammatory responses, reduce maternal immunity and increase the risk of infection (84). Infection may cause intrauterine growth restriction or preterm labour (84). Maternal health is critical to fetal health; therefore, if the mother's respiratory health is jeopardized by air pollutants, the transport and delivery of oxygen and nutrients to the fetus may be reduced (85).

Box 8. Clarion call: the FIGO opinion on the effects of exposure to toxic environmental chemicals on reproductive health.

“Exposure to toxic environmental chemicals during pregnancy and breastfeeding is ubiquitous and is a threat to healthy human reproduction.” That is the conclusion of the International Federation of Gynaecology and Obstetrics (FIGO), the leading voice of reproductive health professionals, with member societies in 125 countries and territories. In 2015, FIGO published an opinion by an international group of obstetricians, gynaecologists and scientists, formally endorsed or supported by 12 reproductive health professional organizations worldwide (86), that there is “accumulating, robust evidence” for an association between exposure to environmental chemicals, such as those in air pollution, and reproductive health. “Preventing exposure to environmental chemicals is a priority for reproductive health professionals everywhere”, the opinion states. FIGO recommends that health professionals advocate for policies to prevent exposure to toxic chemicals in the environment, including air pollution.

Conclusions

Pregnancy is a highly vulnerable time. A growing body of evidence shows a link between exposure to air pollution and adverse birth outcomes, which may have lasting health consequences. There is robust evidence that exposure to air pollution, especially ambient PM, is associated with low birth weight. Likewise, there is growing evidence that maternal exposure to AAP, especially to fine PM, increases the risk of preterm birth. While the reported strength of association between stillbirth and exposure to air pollutants (e.g. PM, CO) depends on the individual pollutant, several studies have shown an increased risk of stillbirth linked to higher exposures. There is evidence suggestive of a link between exposure to ambient and household air pollution and infants born SGA. While additional research will advance knowledge, the harmful effects of air pollution on fetal development and birth are clear, and efforts must be made to protect future generations.

Knowledge gaps and research needs

- A substantial number of studies have examined the link between air pollution and various birth outcomes. The studies differ widely in the populations studied, the method and the levels of exposure to air pollution.
- More studies should be conducted on the association between exposure to ultrafine PM and birth outcomes and also on exposure to air pollution and preterm birth.
- As many studies on birth outcomes and air pollution are based on general estimates of exposure, studies should be conducted with state-of-the-art techniques for measuring and modelling air quality to increase the validity of the links between exposure to various air pollutants and birth outcomes and to improve the evidence base for environmental health policies to ensure the health of mothers and children.

References – adverse birth outcomes

1. Effects of air pollution on children' health and development: a review of the evidence. Copenhagen: WHO Regional Office for Europe; 2005.
2. UNICEF, World Health Organization. Low birthweight: country, regional and global estimates, New York City (NY): UNICEF; 2004 (<http://apps.who.int/iris/bitstream/10665/43184/1/9280638327.pdf>, accessed August 2018).
3. Global nutrition targets 2025: low birth weight policy brief (WHO/NMH/NHD/14.5). Geneva: World Health Organization; undated

- (http://www.who.int/nutrition/topics/globaltargets_lowbirthweight_policybrief.pdf, accessed August 2018).
4. Aguilera I, Guxens M, Garcia-Esteban R, Corbella T, Nieuwenhuijsen MJ, Foradada CM, et al. Association between GIS-based exposure to urban air pollution during pregnancy and birth weight in the INMA Sabadell Cohort. *Environ Health Perspect*. 2009;117:1322–7.
 5. Brauer M, Lencar C, Tarnburic L, Koehoorn M, Demers P, Karr C. A cohort study of traffic-related air pollution impacts on birth outcomes. *Environ Health Perspect*. 2008;116:680–6.
 6. Darrow LA, Klein M, Strickland MJ, Mulholland JA, Tolbert PE. Ambient air pollution and birth weight in full-term infants in Atlanta, 1994–2004. *Environ Health Perspect*. 2011;119: 731–7.
 7. Preterm birth. Fact sheet, reviewed November 2016. Geneva: World Health Organization; 2016 (<http://www.who.int/mediacentre/factsheets/fs363/en/>, accessed August 2018).
 8. Le HQ, Batterman SA, Wirth JJ, Wahl RL, Hoggatt KJ, Sadeghnejad A, et al. Air pollutant exposure and preterm and term small-for-gestational-age births in Detroit, Michigan: long-term trends and associations. *Environ Int*. 2012;44:7–17.
 9. Wilhelm M, Ghosh JK, Su J, Cockburn M, Jerrett M, Ritz B. Traffic-related air toxics and preterm birth: a population-based case-control study in Los Angeles County, California. *Environ Health*. 2011;10:89.
 10. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee (WHO Technical Report Series No. 854). Geneva: World Health Organization; 1995.
 11. Lundgren EM, Tuvemo T. Effects of being born small for gestational age on long-term intellectual performance. *Best Pract Res Clin Endocrinol Metab*. 2008;22(3):447–88.
 12. Gray SC, Edwards SE, Schultz BD, Miranda ML. Assessing the impact of race, social factors and air pollution on birth outcomes: a population-based study. *Environ Health*. 2014;13:4.
 13. Vrijheid M, Casas M, Gason M, Valvi, D, Nieuwenhuijsen M. Environmental pollutants and child health – a review of recent concerns. *Int J Hyg Environ Health*. 2016;219:331–42.
 14. Hansen C, Neller A, Williams, G, Simpson R. Maternal exposure to low levels of ambient air pollution and preterm birth in Brisbane, Australia. *Int J Obstet Gynaecol*. 2006;113(8):935–41.
 15. Pereira G, Bracken MB, Bell ML. Particulate air pollution, fetal growth and gestational length: the influence of residential mobility in pregnancy. *Environ Res*. 2016;147:269–74.
 16. Twum C, Zhu J, Wei Y. Maternal exposure to ambient PM_{2.5} and term low birthweight in the State of Georgia. *Int J Environ Health Res*. 2016;26(1):92–100.
 17. Pedersen M, Gehring U, Beelen R, Wang M, Giorgis-Allemand L, Nybo Andersen AM, et al. Elemental constituents of particulate matter and newborn’s size in eight European cohorts. *Environ Health Perspect*. 2016;124(1):141–50.
 18. Etzel RA, Balk SJ, editors. *Pediatric environmental health*. 3rd edition. Itasca (IL): American Academy of Pediatrics Council on Environmental Health; 2012.
 19. Landrigan PJ, Etzel RA. *Textbook of Children’s Environmental Health*. Oxford: Oxford University Press; 2014.
 20. Mahalingaiah S, Lane KJ, Kim C, Cheng JJ, Hart JE. Impacts of air pollution on gynecologic disease: infertility, menstrual irregularity, uterine fibroids, and endometriosis: a systematic review and commentary. *Curr Epidemiol Rep*. 2018;5(3):197–204.
 21. Neonatal and perinatal mortality: country, regional and global estimates. Geneva: World Health Organization; 2006.
 22. Blencowe H, Cousens S, Jassir FB, Say L, Chou D, Mathers C, et al. National, regional, and worldwide estimates of stillbirth rates in 2015, with trends from 2000: a systematic analysis. *Lancet Global Health*. 2016;4(2):e98–108.
 23. Liu L, Oza S, Hogan D, Chu Y, Perin J, Zhu J, et al. Global, regional, and national causes of under-5 mortality in 2000–15: an updated systematic analysis with implications for the Sustainable Development Goals. *Lancet*. 2016;388(10063):3027–35.
 24. WHO-MCEE estimates for child causes of death, 2000–2016. Geneva: World Health Organization; 2018.
 25. Cohen A, Brauer M, Burnett R, Anderson HR, Frostad J, Estep K, et al. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. *Lancet*. 2017;389(10082):1907–18.
 26. Zheng T, Zhang J, Sommer K, Bassig BA, Zhang X, Braun J, et al. Effects of environmental exposures on fetal and childhood growth trajectories. *Ann Glob Health*. 2016;82(1):41–99.
 27. Zhu X, Liu Y, Chen Y, Yao C, Che Z, Cao J. Maternal exposure to fine particulate matter (PM_{2.5}) and pregnancy outcomes: a meta-analysis. *Environ Sci Pollut Res*. 2015;22:3383–96.
 28. Faiz AS, Rhoads GG, Demissie K, Kruse L, Lin Y, Rich DQ. Ambient air pollution and the risk of stillbirth. *Am J Epidemiol*. 2012;176(4):308–16.
 29. Green R, Sarovar V, Malig B, Basu R. Association of stillbirth with ambient air pollution in a California cohort study. *Am J Epidemiol*. 2015;18(11):874–82.
 30. Yang S, Tan Y, Mei H, Wang F, Li N, Zhao J. Ambient air pollution and the risk of stillbirth: a prospective birth cohort study in Wuhan, China. *Int J Hyg Environ Health*. 2018;221(3):502–9.
 31. DeFranco E, Hall E, Hossain M, Chen A, Haynes EN, Jones D, et al. Air pollution and stillbirth risk: exposure to airborne particulate matter during pregnancy is associated with fetal death. *PLoS One*. 2015;10(3):e0120594.

32. Faiz AS, Rhoads GG, Demissie K, Lin Y, Kruse L, Rich DQ. Does ambient air pollution trigger stillbirth? *Epidemiology*. 2013;24(4):538–44.
33. Ebisu K, Malig B, Hasheminassab S, Constantinos S, Basu R. Cause-specific stillbirth and exposure to chemical constituents and sources of fine particulate matter. *Environ Res*. 2018;160:358–64.
34. Pereira LA, Loomis D, Conceição GM, Braga AL, Arcas RM, Kishi HS, et al. Association between air pollution and intrauterine mortality in São Paulo, Brazil. *Environ Health Perspect*. 1998;106(6):325–9.
35. Kim OJ, Ha EH, Kim BM, Seo JH, Park HS, Jung WJ, et al. PM₁₀ and pregnancy outcomes: a hospital-based cohort study of pregnant women in Seoul. *J Occup Environ Med*. 2007;49(12):1394–1402.
36. Hwang BF, Lee YL, Jaakkola JJ. Air pollution and stillbirth: a population-based case-control study in Taiwan. *Environ Health Perspect*. 2011;119(9):1345–9.
37. Mendola P, Ha S, Pollack AZ, Zhu Y, Seeni I, Kim SS, et al. Chronic and acute ozone exposure in the week prior to delivery is associated with the risk of stillbirth. *Int J Environ Res Public Health*. 2017;14(7):731.
38. Bobak M, Leon DA. Pregnancy outcomes and outdoor air pollution: an ecological study in districts of the Czech Republic 1986–8. *Occup Environ Med*. 1999;56(8):539–43.
39. Pearce MS, Glinianaia SV, Rankin J, Rushton S, Charlton M, Parker L, et al. No association between ambient particulate matter exposure during pregnancy and stillbirth risk in the north of England, 1962–1992. *Environ Res*. 2010;110(1):118–22.
40. Siddika N, Balogun HA, Amegah AK, Jaakkola JJ. Prenatal ambient air pollution exposure and the risk of stillbirth: systematic review and meta-analysis of the empirical evidence. *Occup Environ Med*. 2016;73:573–81.
41. Pope DP, Mishra V, Thompson L, Siddiqui AR, Rehfuess EA, Weber M, et al. Risk of low birth weight and stillbirth associated with indoor air pollution from solid fuel use in developing countries. *Epidemiol Rev*. 2010;32:70–81.
42. Mavalankar DV, Trivedi CR, Gray RH. Levels and risk factors for perinatal mortality in Ahmedabad, India. *Bull World Health Organ*. 1991;69(4):435–42.
43. Mishra V, Retherford RD, Smith KR. Cooking smoke and tobacco smoke as risk factors for stillbirth. *Int J Environ Health Res*. 2005;15(6):397–410.
44. Tielsch JM, Katz J, Thulasiraj RD, Coles CL, Sheeladevi S, Yanik EL, et al. Exposure to indoor biomass fuel and tobacco smoke and risk of adverse reproductive outcomes, mortality, respiratory morbidity and growth among newborn infants in South India. *Int J Epidemiol*. 2009;38(5):1351–63.
45. Siddiqui AR, Gold EB, Brown KH, Lee K, Bhutta Z. Preliminary analyses of indoor air pollution and low birth weight (LBW) and stillbirth in Southern Pakistan. In: *Indoor air pollution from solid fuels and risk of low birth weight and stillbirth. Report from a symposium held at the annual conference of the International Society for Environmental Epidemiology (ISEE), Johannesburg, South Africa, 13–16 September 2005* (http://whqlibdoc.who.int/publications/2007/9789241505735_eng.pdf, accessed August 2018).
46. Bruce N, Smith KR, Balmes J, Pope D, Dherani M, Zhang J, et al. WHO Indoor air quality guidelines: household fuel combustion. Review 4: Health effects of household air pollution (HAP) exposure. Geneva: World Health Organization; 2014.
47. Amegah AK, Quansah R, Jaakkola JJK. Household air pollution from solid fuel use and risk of adverse pregnancy outcomes: a systematic review and meta-analysis of the empirical evidence. *PLoS One*. 2014;9(12):e113920.
48. Lakshmi PVM, Viridi NK, Sharma A, Tripathy JP, Smith KR, Bates MN, et al. Household air pollution and stillbirths in India: analysis of the DLHS-II National Survey. *Environ Res*. 2013;121:17–22.
49. Boy E, Bruce N, Delgado H. Birth weight and exposure to kitchen wood smoke during pregnancy in rural Guatemala. *Environ Health Perspect*. 2002;110:109–14.
50. Shah PS, Balkhair T, Knowledge Synthesis Group on Determinants of Preterm/LBW Births. Air pollution and birth outcomes: a systematic review. *Environ Int*. 2011;37:498–516.
51. Sun X, Luo X, Zhao C, Ng RWC, Lim CED, Zhang B, et al. The association between fine particulate matter exposure during pregnancy and preterm birth: a meta-analysis. *BMC Pregnancy Childbirth*. 2015;15:300.
52. Sapkota A, Chelikowsky AP, Nachman KE, Cohen AJ, Ritz B. Exposure to particulate matter and adverse birth outcomes: a comprehensive review and meta-analysis. *Air Qual Atmos Health*. 2012;5(4):369–81.
53. Malley CS, Kuylentierna JC, Vallack HW, Henze DK, Blencowe H, Ashmore MR, et al. Preterm birth associated with maternal fine particulate matter exposure: a global, regional and national assessment. *Environ Int*. 2017;101:173–182.
54. Stieb DM, Luo X, Zhao C, Zhang B, Tao J, Yang Z, et al. A national study of the association between traffic-related air pollution and adverse pregnancy outcomes in Canada, 1999–2008. *Environ Res*. 2016;148:513–26.
55. Jacobs M, Zhang G, Chen S, Mullins B, Bell M, Jin L, et al. The association between ambient air pollution and selected adverse pregnancy outcomes in China: a systematic review. *Sci Total Environ*. 2017;579:1179–92.
56. Yucra S, Tapia V, Steenland K, Naeher LP, Gonzales GF. Association between biofuel exposure and adverse birth outcomes at high altitudes in Peru: a matched case-control study. *Int J Occup Environ*

- Health. 2011;17(4):307–13.
57. Health topics: birth defects. New Delhi: WHO Regional Office for South East Asia; 2018 (http://www.searo.who.int/topics/birth_defects/en/, accessed August 2018).
 58. Zhang B, Liang S, Zhao J, Qian Z, Bassig BA, Yang R. Maternal exposure to air pollutant PM_{2.5} and PM₁₀ during pregnancy and risk of congenital heart defects. *J Exposure Sci Environ Epidemiol*. 2016;26:422–7.
 59. Vinceti M, Malagoli C, Malavolti M, Cherubini A, Maffei G, Rodolfi R, et al. Does maternal exposure to benzene and PM₁₀ during pregnancy increase the risk of congenital anomalies? A population-based case-control study. *Sci Total Environ*. 2016;541:444–51.
 60. Vrijheid M, Martinez D, Manzanares S, Dadvand P, Schembari A, Rankin J, et al. Ambient air pollution and risk of congenital anomalies: a systematic review and meta-analysis. *Environ Health Perspect*. 2011;119(5):598–606.
 61. Chen EKC, Zmirou-Navier D, Padilla C, Deguen S. Effects of air pollution on the risk of congenital anomalies: a systematic review and meta-analysis. *Int J Environ Res Public Health*. 2014;11(8):7642–68.
 62. Li Z, Zhang L, Ye R, Pei L, Liu J, Zheng X, et al. Indoor air pollution from coal combustion and the risk of neural tube defects in a rural population in Shanxi Province, China. *Am J Epidemiol*. 2011;174:4.
 63. Fleischer NL, Merilampi M, van Donkelaar A, Vardillo-Ortega F, Martin RV, Bertran AP, et al. Outdoor air pollution, preterm birth, and low birth weight: analysis of the World Health Organization Global Survey on Maternal and Perinatal Health. *Environ Health Perspect*. 2014;122:425–30.
 64. Sun X, Luo X, Zhao C, Zhang B, Tao J, Yang Z, et al. The associations between birth weight and exposure to fine particulate matter (PM_{2.5}) and its chemical constituents during pregnancy: a meta-analysis. *Environ Pollut*. 2016;211:38–47.
 65. Stieb DM, Chen L, Eshoul M, Judek S. Ambient air pollution, birth weight and preterm birth: a systematic review and meta-analysis. *Environ Res*. 2012;117:100–11.
 66. Li X, Huang S, Jiao A, Yang X, Yun J, Wang Y, et al. Association between ambient fine particulate matter and preterm birth or term low birth weight: an updated systematic review and meta-analysis. *Environ Pollut*. 2017;227:596–605.
 67. Misra P, Srivastava R, Krishnan A, Sreenivaas V, Pandav CS. Indoor air pollution-related acute lower respiratory infections and low birthweight: a systematic review. *J Trop Pediatr*. 2012;58(6): 457–66.
 68. Khan MN, Zhang C, Islam MM, Islam R, Rahman M. Household air pollution from cooking and risk of adverse health and birth outcomes in Bangladesh: a nationwide population based study. *Environ Health*. 2017;16:57.
 69. Abusalah A, Gavana M, Haidich A.B, Smyrnakis E, Papadakis N, Papanikolaou A, et al. Low birth weight and prenatal exposure to indoor pollution from tobacco smoke and wood fuel smoke: a matched case–control study in Gaza Strip. *Matern Child Health J*. 2011;16(8):1718–27.
 70. Mavalankar DV, Gray RH, Trivedi CR. Risk factors for preterm and term low birth weight in Ahmedabad, India. *Int J Epidemiol*. 1992;21:263–72.
 71. Mishra V, Dai X, Smith KR, Mika L. Maternal exposure to biomass smoke and reduced birth weight in Zimbabwe. *Ann Epidemiol*. 2004;14(10):740–7.
 72. Siddiqui AR, Gold EB, Yang X, Lee K, Brown KH, Bhutta ZA. Prenatal exposure to wood fuel smoke and low birth weight. *Environ Health Perspect*. 2008;116(4):543–9.
 73. Thompson LM, Bruce N, Eskenazi B, Diaz A, Pope D, Smith KR. Impact of reduced maternal exposures to wood smoke from an introduced chimney stove on newborn birth weight in rural Guatemala. *Environ Health Perspect*. 2011;119(10):1489–94.
 74. Dandona L, Dandona R, Kumar GA, Shukla DK, Paul VK, Balakrishnan K, et al. Nations within a nation: variations in epidemiological transition across the states of India, 1990–2016 in the Global Burden of Disease Study. *Lancet* 2017;390(10111):2437–2460.
 75. Balakrishnan K, Ghosh S, Thangavel G, Sambandam S, Mukhopadhyay K, Puttaswamy N, et al. Exposures to fine particulate matter (PM_{2.5}) and birthweight in a rural-urban, mother-child cohort in Tamil Nadu, India. *Environ Res*. 2018;161:524–31.
 76. Balakrishnan, Sambandam S, Ramaswamy P, Ghosh S, Venkatesan V, Thangavel G, et al. Establishing integrated rural–urban cohorts to assess air pollution-related health effects in pregnant women, children and adults in southern India: an overview of objectives, design and methods in the Tamil Nadu Air Pollution and Health Effects (TAPHE) study. *BMJ Open*. 2015;5(6):e008090.
 77. ICD-11 for mortality and morbidity statistics 2018. Geneva: World Health Organization; 2018 (<https://icd.who.int/browse11/l-m/en#/http://id.who.int/icd/entity/1786398813>, accessed August 2018).
 78. Mulenga D. Maternal exposure to household air pollution and associated adverse birth outcomes in Ndola and Masaiti, Zambia. *EC Pulmonol Respir Med*. 2018;7:82–97.
 79. Kannan S, Misra DP, Dvonch JT, Krishnakumar A. Exposures to airborne particulate matter and adverse perinatal outcomes: a biologically plausible mechanistic framework for exploring potential. *Cien Saude Colet*. 2007;12(6):1591–602.
 80. Levy RJ. Carbon monoxide pollution and neurodevelopment: a public health concern. *Neurotoxicol Teratol*. 2015;49:31–40.

81. Wylie BJ, Matechi E, Kishashu Y, Fawzi W, Premji Z, Coull BA, et al. Placental pathology associated with household air pollution in a cohort of pregnant women from Dar es Salaam, Tanzania. *Environ Health Perspect.* 2017;125(1):134.
82. Lin VW, Baccarelli AA, Burris HH. Epigenetics – a potential mediator between air pollution and preterm birth. *Environ Epigenet.* 2016;2(1). doi: 10.1093/eep/dvv008.
83. Zhang T, Zheng X, Wang X, Zhao H, Wang T, Zhang H, et al. Maternal exposure to PM_{2.5} during pregnancy induces impaired development of cerebral cortex in mice offspring. *Int J Mol Sci.* 2018;19:257.
84. Slama R, Darrow L, Parker J, Woodruff TJ, Strickland M, Nieuwenhuijsen M, et al. Meeting report: atmospheric pollution and human reproduction. *Environ Health Perspect.* 2008;116(6):791.
85. Ritz B, Wilhelm M. Ambient air pollution and adverse birth outcomes: methodologic issues in an emerging field. *Basic Clin Pharmacol Toxicol.* 2008;102(2):182–90.
86. Di Renzo GC, Conry JA, Blake J, DeFrancesco MS, DeNicola N, Martin JN JR, et al. International Federation of Gynecology and Obstetrics opinion on reproductive health impacts of exposure to toxic environmental chemicals. *Int J Gynecol Obstet.* 2015;131(3):219–25.

5.2 Infant mortality

Key findings:

- While there are few studies, those on the link between exposure to PM and infant mortality have provided compelling evidence of a positive association globally.
- Most studies have addressed acute exposure to AAP with relatively few on the effects of HAP on the risk of infant mortality.

Infant mortality refers to deaths that occur in the first year of life. In 2016, the global infant mortality was 4.2 million, representing 75% of all deaths of children under the age of 5 years (1). While many studies have shown a link between air pollution and mortality in adults, less research has been done on infants. As infants' lungs are highly susceptible to pollutants, they are particularly vulnerable to airborne exposure, and more research is required. The available evidence indicates a link between infant mortality and exposure to HAP and AAP. The links between climate change, air pollution and infant and child health are also increasingly being studied, with growing concern for the health and well-being of future generations (2).

Ambient air pollution

Early studies of the effect of AAP on infant mortality consistently found associations of different strengths. Several studies included in a systematic review in 2005 (3) found strong correlations between air pollution and infant mortality. The studies were conducted in many geographic areas, on a range of pollutants, including total suspended particles: SO₂, O₃, NO₂, NO_x, PM_{2.5} and PM₁₀. A similar review in 2004 found that the results differed by subgroup of infants (4). While inconsistent findings were noted for PM and both total infant mortality and neonatal mortality, the authors found a positive association between exposure to PM and post-neonatal mortality. This was especially pronounced for deaths due to respiratory causes and sudden infant death syndrome. Although the reason for this trend was not clear, the authors suggested links between exposure to particulate air pollution and some causes of infant death.

Several studies examined the effects of AAP on infant mortality in more detail. A large study in Japan was conducted of infant deaths in urban Tokyo between 2002 and 2013 to examine the association with acute exposure to PM_{2.5}, suspended PM (< 7 µm in diameter) and coarse particles (PM_{7-2.5}) (5). The mortality rates associated with increases in the concentration of each pollutant of 10 µg/m³ were compared. Infant mortality was categorized by age at death (infant, neonate and post-neonate) and cause of death. Infant and post-neonatal mortality increased with each 10 µg/m³ increase in PM_{2.5} and was linked with respiratory diseases. An increase in exposure to coarse particles was associated with a 21% increase in the risk of post-neonatal mortality; and the risks of post-neonatal mortality and mortality due to respiratory diseases increased by 10% and 25%, respectively, with increased concentrations of suspended PM. The risk of infant mortality was increased even

when PM concentrations were below the Japanese air quality guideline of a daily average of 35 $\mu\text{g}/\text{m}^3$ for $\text{PM}_{2.5}$. These results highlight the importance of evaluating infants separately from other age groups, as they may be uniquely susceptible to air pollution.

A study with satellite estimates and data from household surveys on the location and timing of almost 1 million births in 30 countries in Africa was conducted to estimate the effect of exposure to ambient $\text{PM}_{2.5}$ on infant mortality (6). The authors found that infant mortality increased by 9% with a 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ and estimated that exposure to $\text{PM}_{2.5}$ above minimum threshold levels was responsible for 22% of infant deaths in those 30 countries in 2015.

In an affluent, densely populated area of Flanders, Belgium in close proximity to traffic, industry and agriculture, researchers investigated acute exposure to PM_{10} and infant mortality between 1998 and 2006 (7). Daily infant mortality, pollutant level and cause of death were recorded. Neonates aged 2–4 weeks were the most vulnerable to air pollution, with an 11% higher risk of death with every increase of 10 $\mu\text{g}/\text{m}^3$ in PM_{10} . When PM_{10} levels exceeded 50 $\mu\text{g}/\text{m}^3$, these neonates were 1.75 times more likely to die. This finding is important, because the WHO guidelines recommend levels < 20 $\mu\text{g}/\text{m}^3$ and daily averages < 50 $\mu\text{g}/\text{m}^3$ on more than 3 days per year (8). Thus, adherence to local pollution standards may significantly affect infant mortality rates. In this study, infant mortality due to perinatal circumstances (e.g. maternal conditions, complications of pregnancy and birth, adverse birth outcomes) and congenital and chromosomal abnormalities also increased with rising daily PM_{10} levels.

Most studies measured exposure to air pollution immediately before an infant's death, and relatively few considered longer exposures, although the extent of exposure to pollutants in the weeks and months before death may be critical. For example, a correlation has been found between the average level of $\text{PM}_{2.5}$ during the time between birth and post-neonatal death (9). In a study in a highly polluted part of California, USA, the average CO, NO_2 , O_3 and PM_{10} levels experienced by infants 2 weeks and 1, 2 and 6 months before death were measured (10). In infants 28 days to 3 months old, the risk of death from respiratory causes increased significantly with rising CO levels in the 2 weeks before death; a moderate increase in risk was seen for infants 4–12 months old with increasing PM_{10} levels in the 2 weeks before death; and, for infants 7–12 months old, the risk more than doubled when they had been exposed to high levels of PM_{10} in the 6 months before death. In another study in the same region, the risk of sudden infant death syndrome increased by 15–19% when average NO_2 levels were elevated during the 2 months before death (11). In a cohort study in the Republic of Korea of the association between long-term exposure, including during pregnancy and postnatally, to PM and infant mortality (12), gestational exposure to increasing levels of PM_{10} , total suspended particulates and $\text{PM}_{2.5}$ increased the risk of infant mortality from all causes and from respiratory causes in infants with a normal birth weight. The first trimester was the only period during which this pattern was found independently, indicating an effect of air pollution in early pregnancy on infant development and mortality.

Policy affects human health and perhaps most significantly that of infants. In a quasi-experiment, Tanaka (13) studied changes in infant mortality in 175 Chinese prefectures before and after the introduction of stringent air pollution regulations. In 1998, the power industry, which was heavily reliant on coal and a major source of emissions (particularly SO_2), dramatically reduced its emissions. This reduction was associated with a 20% decrease in the rate of infant mortality, with a 63% reduction during the neonatal period, particularly from deaths associated with the nervous and circulatory systems. The author proposed that the drastic improvement in neonatal survival with reduced maternal exposure to pollutants benefitted fetal development and increased probability of survival.

Household air pollution

Most of the research on HAP has been on the effects of ambient air pollutants, although infants spend most of their time indoors. In a study in rural India, the risk of infant mortality was 21% higher in households with indoor burning of biomass fuels (wood or dung) than in those in which kerosene or biogas was used (14). In Ecuador, the infant mortality rate increased with the amount of biomass fuels burnt (15). In these studies, households in which biomass fuels were used were compared with those in which fuels considered by the authors as “cleaner”, such as biogas, LPG or kerosene, were used. (As noted above, this categorization contradicts current understanding of the adverse health impacts of kerosene use.) In another study, infants born to women exposed to polluting cooking fuels

(kerosene, charcoal, coal, wood, straw, crop waste and dung) during pregnancy were found to be at increased risk of neonatal mortality within 0–2 days of birth (16), indicating the danger of exposure to HAP both in utero and in the domestic environment.

Biological mechanisms

The biological mechanisms through which air pollution increases infant mortality are not clearly understood. It has been proposed that infants are likely to die when exposed to air pollution because their immature lungs and immune system leave them unable to cope with the reactive inflammation that occurs in response to such exposure (17). Pope et al. (18) proposed that PM damages the lung, resulting in respiratory distress and hypoxaemia. Neonatal rats exposed to PM had reduced cell proliferation and increased oxidative stress in the lungs (19), but toxicological evidence for humans is lacking. CO poisoning is a well-known cause of infant death (20).

The association between exposure to PM and adverse birth outcomes, including low birth weight, preterm delivery and intrauterine growth restriction, is relevant to infant mortality. The possible mechanisms of these outcomes have been studied in more detail. They may include adverse effects on the cardiovascular system, such as oxidative stress, inflammation, impaired coagulation and endothelial function and faulty haemodynamic responses (21). These adverse effects add to the challenges faced by vulnerable infants as they grow and develop during their first year of life.

Conclusion

A correlation has been found between exposure to HAP and AAP and infant mortality, which increases with increasing pollution levels. Infants are at particular risk from exposure to PM and toxic gases. Although inconsistent classification of kerosene in studies of HAP and infant mortality may affect the interpretation of certain findings, HAP is clearly a risk factor for child mortality.

Knowledge gaps and research needs

- Most of the studies addressed acute exposure, and more research is needed to understand the long-term cumulative effects of air pollution, from gestation to death.
- Few studies to date have been conducted on HAP. Better understanding of the effects of indoor pollutants on infant mortality will improve understanding of the interaction between exposures and risk. Studies of the different constituents of air pollution and the biological mechanisms by which they act will improve understanding of how air pollution affects infant mortality.
- Although sufficient data are available to support preventive action, further research on the components of air pollution and their mechanisms of action would provide input for additional preventive policy actions and interventions.

References – infant mortality

1. Levels and trends in child mortality. Report 2017. Estimates developed by the UN Inter-agency Group for Child Mortality Estimation. New York (NY), Geneva and Washington (DC): United Nations Children's Fund, World Health Organization, World Bank and United Nations; 2017.
2. Perera FP. Multiple threats to child health from fossil fuel combustion: impacts of air pollution and climate change. *Environ Health Perspect.* 2017;125(2):141.
3. Šrám RJ, Binková B, Dejmek J, Bobak M. Ambient air pollution and pregnancy outcomes: a review of the literature. *Environ Health Perspect.* 2005;113(4):375–82.
4. Glinianaia SV, Rankin J, Bell R, Pless-Mulloli T, Howel D. Does particulate air pollution contribute to infant death? A systematic review. *Environ Health Perspect.* 2004;112(14):1365–70.
5. Yorifuji T, Kashima S, Doi H. Acute exposure to fine and coarse particulate matter and infant mortality in Tokyo, Japan (2002–2013). *Sci Total Environ.* 2016;551:66–72.
6. Heft-Neal S, Burney J, Bendavid E, Burke M. Robust relationship between air quality and infant mortality in Africa. *Nature.* 2018;559:254–8.
7. Scheers H, Mwalili SM, Faes C, Fierens F, Nemery B, Nawrot TS. Does air pollution trigger infant mortality in western Europe? A case-crossover study. *Environ Health Perspect.* 2011;119(7):1017.
8. WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide: global update 2005. Summary of risk assessment. Geneva: World Health Organization; 2006

(http://apps.who.int/iris/bitstream/handle/10665/69477/WHO_SDE_PHE_OEH_06.02_eng.pdf?sequence=1, accessed August 2018).

9. Carbajal-Arroyo L, Miranda-Soberanis V, Medina-Ramón M, Rojas-Bracho L, Tzintzun G, Solís-Gutiérrez P, et al. Effect of PM₁₀ and O₃ on infant mortality among residents in the Mexico City Metropolitan Area: a case-crossover analysis, 1997–2005. *J Epidemiol Community Health*. 2010;65(8):715–21.
10. Woodruff TJ, Parker JD, Schoendorf KC. Fine particulate matter (PM_{2.5}) air pollution and selected causes of postneonatal infant mortality in California. *Environ Health Perspect*. 2006;114(5):786.
11. Ritz B, Wilhelm M, Zhao Y. Air pollution and infant death in southern California, 1989–2000. *Pediatrics*. 2006;118(2):493–502.
12. Son JY, Bell ML, Lee JT. Survival analysis of long-term exposure to different sizes of airborne particulate matter and risk of infant mortality using a birth cohort in Seoul, Korea. *Environ Health Perspect*. 2011;119(5):725.
13. Tanaka S. Environmental regulations on air pollution in China and their impact on infant mortality. *J Health Econ*. 2015;42:90–103.
14. Tielsch JM, Katz J, Thulasiraj RD, Coles CL, Sheeladevi S, Yanik EL, et al. Exposure to indoor biomass fuel and tobacco smoke and risk of adverse reproductive outcomes, mortality, respiratory morbidity and growth among newborn infants in south India. *Int J Epidemiol*. 2009;38(5):1351–63.
15. Rinne ST, Rodas EJ, Rinne ML, Simpson JM, Glickman LT. Use of biomass fuel is associated with infant mortality and child health in trend analysis. *Am J Trop Med Hyg*. 2007;76(3):585–91.
16. Patel AB, Meleth S, Pasha O, Goudar SS, Esamai F, Garces AL, et al. Impact of exposure to cooking fuels on stillbirths, perinatal, very early and late neonatal mortality-a multicenter prospective cohort study in rural communities in India, Pakistan, Kenya, Zambia and Guatemala. *Mat Health Neonatol Perinatol*. 2015;1(1):18.
17. Ha EH, Lee JT, Kim H, Hong YC, Lee BE, Park HS, et al. Infant susceptibility of mortality to air pollution in Seoul, South Korea. *Pediatrics*. 2003;111(2):284–90.
18. Pope CIII, Arden C, Dockery DW. Health effects of fine particulate air pollution: lines that connect. *J Air Waste Manage Assoc*. 2006;56(6):709–42.
19. Pinkerton KE, Zhou Y, Zhong C, Smith KR, Teague SV, Kennedy IM, et al. Mechanisms of particulate matter toxicity in neonatal and young adult rat lungs. *Res Rep Health Eff Inst*. 2008;135:3–41.
20. Goldstein M. Carbon monoxide poisoning. *J Emerg Nurs*. 2008;34(6):538–42.
21. Kannan S, Misra DP, Dvonch JT, Krishnakumar A. Exposures to airborne particulate matter and adverse perinatal outcomes: a biologically plausible mechanistic framework for exploring potential. *Cienc Saude Colet*. 2007;12(6):1591–602.

5.3 Neurodevelopment

Key findings:

- Exposure to air pollutants can negatively affect neurodevelopment, resulting in lower cognitive test outcomes (such as global intelligence quotient) and the development of behavioural disorders such as autism spectrum and attention deficit hyperactivity disorders.
- Research suggests that both prenatal and postnatal exposure to air pollution represent threats to neurodevelopment.

Overview

Neurodevelopment is a fundamental phase of human growth and development, which begins in the early prenatal period with the proliferation of radial glia and neurons. While neurodevelopment continues well into the second decade of life, the first three years of age are especially important. Various processes occur during this period, including proliferation, migration, differentiation, synaptogenesis, myelination and apoptosis of neuronal cells (1, 2). If neurodevelopment is interrupted or impaired by environmental pollutants, the health consequences for the child can be serious, as this may lead to a number of conditions and symptoms, including cognitive impairment, attention disorders and autism spectrum disorder, which are difficult to diagnose and treat and may have lifelong consequences.

Ambient air pollution

Three systematic reviews concluded that there is an association between exposure to AAP, especially pollutants emitted from vehicles, and impaired neurodevelopment in children (2–4).

Several studies have evaluated the relation between prenatal exposure to air pollution and neurodevelopment in children and suggested that air pollution can negatively affect their mental and

motor development. Lertxundi et al. (5) found that prenatal exposure to PM_{2.5} and NO₂ was associated with significant decreases in cognitive development and motor development in children at the age of 15 months. In a study of birth cohorts in the Republic of Korea, prenatal exposure to PM₁₀ and NO₂ had significant effects on cognitive development and motor development at 6 months of age but not at 12 or 24 months (6).

The findings of studies on the effects of prenatal exposure to air pollution on cognitive function and behaviour have been inconsistent (2). While one meta-analysis of cohort studies in Europe found no association between cognitive development and exposure to NO₂ and PM from traffic-related air pollution, an association was seen between prenatal exposure to NO₂ and deficits in overall psychomotor function in children aged 1–6 years (7, 8).

Prenatal exposure to air pollutants can have various effects on development throughout childhood. In Japan, Yorifuji and colleagues (9) reported an association between prenatal exposure to air pollution and deficits in verbal and fine motor development at the age of 2.5 years. They also found an association with problems of attention, inhibition and impulsivity at 5.5 years. In the same cohort, the risks of attention problems and aggressive behaviour were found to have increased by 8 years of age (10). Other studies indicate that exposure in specific periods during pregnancy is associated with certain stages of neurodevelopmental deficit, with differences by gender. Chiu et al. (11) reported an association between exposure to PM_{2.5} at 31–38 weeks of gestational age and reduced intelligence quotient among boys and an association between exposure to PM_{2.5} at 12–20 weeks of gestational age and decreased general memory index among girls.

Where children live and grow has a powerful effect on their lives. There is increasing evidence that, postnatally, childhood exposure to traffic-related air pollution is linked to neurodevelopmental outcomes such as anxiety and depression (12) and impaired cognitive function (13, 14). In a study of 2715 children aged 7–10 years in Barcelona, Spain, Sunyer and colleagues (15) found that children who attended schools in highly polluted areas had slower growth in cognitive function, measured as working memory, than those in less polluted areas.

In a prospective study of birth cohort, Suglia et al. (16) used black carbon as an indicator of traffic-related air pollution and found that increased exposure was associated with lower scores on intelligence, memory and learning tests in children aged 8–11 years. In a one-year longitudinal study in Spain, Freire et al. (17) observed that high exposure to traffic-related air pollution was associated with a modest decrease in cognitive and motor development. A longitudinal study in Spain showed that students exposed to higher levels of traffic-related NO₂, elemental carbon and ultrafine particles in school classrooms and courtyards had “slower growth in all cognitive measurements” and negative performance on tests of working memory and attentiveness than those exposed to lower levels. In another longitudinal study, Chiu et al. (18) found a nonlinear relation between exposure to air pollution and attention in children aged 7–14. They also found that children in the second and third quartile of exposure to black carbon made more errors and had a slower reaction time on a continuous performance task than those in the lowest quartile, although the association was less strong for those in the highest quartile. Significant associations were found for both boys and girls, but stronger associations were found for boys.

In the first large study of the effect of air pollution on brain morphology, Guxens et al. (19) analysed brain imaging scans and cognitive function tests of 783 children in a Dutch birth cohort. Prenatal exposure to PM_{2.5} was found to cause structural alterations to the cerebral cortex, which partially mediates inhibitory control, of children age 6–10 years. Impaired ability to control impulses at this age may affect educational achievement and increase the risk of mental disorders.

Household air pollution

The review of evidence of health effects for the 2014 WHO guidelines for indoor air quality associated with household fuel combustion (20) identified the association between solid fuel use in houses and neurodevelopment as an emerging area of research. One study in rural Guatemala found an association between exposure to CO during pregnancy and reduced neuropsychological performance in children (21). In another study, memory and building block skills (as indicators of cognitive development) in children aged 3–9 years in Belize, Kenya, Nepal and American Samoa were found to be lower in those who were exposed to open-fire cooking (22). The reviewers concluded that, while research to date suggests a relation between exposure to HAP and impaired cognitive development, no clear association could be concluded from two studies. Indoor exposure to CO from cooking with gas or solid fuels may be independently associated with adverse

neurodevelopmental outcomes in children (23), but this conclusion is also based on a limited number of studies.

Autism spectrum disorders

Autism spectrum disorders (ASD) cover a wide range of conditions, which are usually identified by the age of 5 years. They are characterized by asocial behaviour and difficulties in communication and language (24). WHO has estimated that one in 160 children currently lives with ASD (24).

Ambient air pollution

Several studies have addressed associations between prenatal and postnatal exposure to traffic-related air pollution and ASD in children. Becerra et al. (12) reported an increased risk of ASD with increasing prenatal exposure to NO_x, O₃, and PM_{2.5} in Los Angeles, California, citing traffic as the primary source (12). Three other studies found associations, with an increased risk of ASD and pre- and postnatal exposure to PM_{2.5} (13, 14, 25), PM₁₀, NO₂ and traffic-related air pollution (14). In a prospective cohort study, Jung and colleagues (26) identified an increased risk of ASD with rising levels of CO, NO₂, O₃ and SO₂ in the 1–4 years before diagnosis. In a systematic review of 23 studies, Lam et al. (27) found an increased risk of autism with increased exposure to air pollution but rated the quality of the evidence as moderate, with a low risk of bias. They concluded that there is limited evidence of toxicity.

In contrast, two studies in Europe found no association between autistic traits and prenatal exposure to NO₂, PM_{2.5} or PM₁₀ (28) and no link between pre- and postnatal exposure to NO_x and PM_{2.5} and ASD (28, 29). Furthermore, a study of birth cohorts in Sweden (30) found no association between pre- and postnatal exposure to NO_x and PM₁₀ and ASD or attention-deficit hyperactivity disorder, a brain disorder marked by a continuous pattern of inattention and/or hyperactivity–impulsivity that interferes with functioning or development (31). Lyall et al. (32) suggested that the differences in the results obtained in Europe and the USA were due to differences in exposure measurements, methods for assessing ASD and the age at which assessments were done.

Overall, systematic reviews of studies on ASD have shown relatively consistent evidence of an association between AAP, especially prenatal exposure to PM, and autism (27, 33–36). More research should be conducted to clarify the effects of individual components of AAP. There is inconsistent evidence of an association between the critical period of exposure (pre- or postnatal) and the occurrence of ASD (2, 36, 37).

Household air pollution

The review revealed no published studies on HAP from use of polluting fuels and the development of ASD. ASD were not included in the review of the evidence of health effects for the WHO guidelines on indoor air quality with respect to household fuel combustion in 2014 (20).

Biological mechanisms

Although neurodevelopment is a complex process, studies are beginning to elucidate the mechanisms by which air pollution interferes with the normal physiology. A study with magnetic resonance imaging (MRI) of children aged 7–9 years who had been exposed in utero to PAHs showed a dose–response relation with reductions in white matter surface (38). The changes were found almost exclusively in the left hemisphere of the brain and were associated with specific symptoms, including more severe externalizing behavioural problems, symptoms of attention-deficit hyperactivity disorder and conduct disorders.

Pujol et al. (39) used MRI to document brain structure, membrane metabolites, functional connectivity in major neural networks and activation/deactivation dynamics during a sensory task. Other authors concluded that higher exposure to traffic-related air pollution in childhood slowed brain maturation (13). Other research suggests that exposure during fetal life to high levels of air pollution causes structural changes in the cerebral cortex (19).

MRI also revealed significant differences in white matter volume and cognitive deficits between children living in highly polluted areas and those living in less polluted areas (40). The authors also saw increased serum inflammatory mediators in these children, suggesting a role for neuroinflammation, and proposed that structural brain alterations are a potential response to high

levels of air pollution. Other work showed an association between long-term exposure to air pollution (including ultrafine PM and PM_{2.5}) and neuroinflammation, in addition to an altered innate immune response in children and young adults (41). The authors also noted disruption of the blood–brain barrier, ultrafine particulate deposition and accumulation of amyloid β -42 and α -synuclein, suggesting that long-term exposure to air pollution should be considered a risk factor for degenerative diseases such as Alzheimer and Parkinson diseases.

Conclusions

There is growing evidence of an association between exposure to AAP in the prenatal and postnatal periods and impaired childhood neurodevelopment. There is strong evidence that exposure to AAP can negatively affect children’s mental and motor development. There is suggestive evidence of a link between prenatal exposure to traffic-related air pollution and cognitive and psychomotor function and behavioural problems, but the findings have been inconsistent. Some studies showed an association between exposure to HAP and impaired cognitive development, but further research is needed. Outdoor air pollution has been linked to an increased risk of ASD, especially in studies in the USA in which consistent methods were used.

Knowledge gaps and research needs

- Further research should be conducted on the biological pathways of the effects of air pollution on neurodevelopment. Use of more precise methods for measuring exposure to air pollution, the composition of PM and the sources, long-term evaluations and identification of critical periods of exposure would strengthen the evidence of neurodevelopmental effects.
- Long-term neurobehavioural follow-up of children exposed to air pollution in early life is required to assess the consequences of early exposure later in life in view of the emerging literature on particulate pollution and dementia in adults.

References – neurodevelopment

1. Rice D, Barone SJr. Critical periods of vulnerability for the developing nervous system: evidence from humans and animal models. *Environ Health Perspect.* 2000;108(Suppl 3):511.
2. Suades-González E, Gascon M, Guxens M, Sunyer J. Air pollution and neuropsychological development: a review of the latest evidence. *Endocrinology.* 2015;156(10):3473–82.
3. Vrijheid, M, Casas M, Gascon M, Valvi D, Nieuwenhuijsen M. Environmental pollutants and child health – a review of recent concerns. *Int J Hyg Environ Health.* 2016;219(4–5):331–42.
4. Clifford A, Lang L, Chen R, Anstey KJ, Seaton A. Exposure to air pollution and cognitive functioning across the life course – a systematic literature review. *Environ Res.* 2016;147:383–98.
5. Lertxundi A, Baccini M, Lertxundi N, Fano E, Aranbarri A, Martínez MD, et al., Exposure to fine particle matter, nitrogen dioxide and benzene during pregnancy and cognitive and psychomotor developments in children at 15 months of age. *Environ Int.* 2015;80:33–40.
6. Kim E, Park H, Ha EH, Hong YC, Kim Y, Kim B, et al. Prenatal exposure to PM₁₀ and NO₂ and children’s neurodevelopment from birth to 24 months of age: Mothers and Children’s Environmental Health (MOCEH) study. *Sci Total Environ.* 2014;481:439–45.
7. Guxens M, Sunyer J. A review of epidemiological studies on neuropsychological effects of air pollution. *Swiss Med Wkly.* 2012;141:w13322.
8. Guxens M, Garcia-Esteban R, Giorgis-Allemand L, Fornes J, Badaloni C, Ballester F, et al. Air pollution during pregnancy and childhood cognitive and psychomotor development: six European birth cohorts. *Epidemiology.* 2014;25(5):636–47.
9. Yorifuji T, Kashima S, Higa Diez M, Kado Y, Sanada S, Doi H. Prenatal exposure to traffic-related air pollution and child behavioral development milestone delays in Japan. *Epidemiology.* 2016;27(1):57–65.
10. Yorifuji, T, Kawachi I, Sakamoto T, Doi H. Prenatal exposure to outdoor air pollution and child behavioral problems at school age in Japan. *Environ Int.* 2017;99:192–8.
11. Chiu YHM, Hsu HHL, Coull BA, Bellinger DC, Kloog I, Schwartz J, et al. Prenatal particulate air pollution and neurodevelopment in urban children: examining sensitive windows and sex-specific associations. *Environ Int.* 2016;87:56–65.
12. Becerra TA, Wilhelm M, Olsen J, Cockburn M, Ritz B, et al. Ambient air pollution and autism in Los Angeles County, California. *Environ Health Perspect.* 2013;121(3):380.
13. Raz R, Roberts AL, Lyall K, Hart JE, Just AC, Laden F, et al., Autism spectrum disorder and particulate matter air pollution before, during, and after pregnancy: a nested case–control analysis within the Nurses’ Health Study II cohort. *Environ Health Perspect.* 2015;123(3):264.

14. Volk HE, Lurmann F, Penfold B, Hertz-Picciotto I, McConnell R. Traffic-related air pollution, particulate matter, and autism. *JAMA Psychiatry*. 2013;70(1):71–7.
15. Sunyer J, Esnaola M, Alvarez-Pedrerol M, Fornas J, Rivas I, López-Vicente M, et al. Association between traffic-related air pollution in schools and cognitive development in primary school children: a prospective cohort study. *PLoS Med*. 2015;12(3):e1001792.
16. Suglia SF, Gryparis A, Wright RO, Schwartz J, Wright RJ. Association of black carbon with cognition among children in a prospective birth cohort study. *Am J Epidemiol*. 2007;167(3):280–6.
17. Freire C, Ramos R, Puertas R, Lopez-Espinosa MJ, Julvez J, Aguilera I, et al. Association of traffic-related air pollution with cognitive development in children. *J Epidemiol Community Health*. 2010;64(3):223–8.
18. Chiu YHM, Bellinger DC, Coull BA, Anderson S, Barber R, Wright RO, et al., Associations between traffic-related black carbon exposure and attention in a prospective birth cohort of urban children. *Environ Health Perspect*. 2013;121(7):859.
19. Guxens M, Lubczyńska MJ, Muetzel RL, Dalmau-Bueno A, Jaddoe VWV, Hoek G. Air pollution exposure during fetal life, brain morphology, and cognitive function in school-age children. *Biol Psychiatry*. 2018. doi: 10.1016/j.biopsych.2018.01.016.
20. Bruce N, Smith KR, Balmes J, Pope D, Dherani M, Zhang J, et al. WHO indoor air quality guidelines: household fuel combustion. Review 4: Health effects of household air pollution (HAP) exposure. Geneva: World Health Organization; 2014.
21. Dix-Cooper L, Eskenazi B, Romero C, Balmes J, Smith KR. Neurodevelopmental performance among school age children in rural Guatemala is associated with prenatal and postnatal exposure to carbon monoxide, a marker for exposure to woodsmoke. *J Neurotoxicol*. 2012;33:246–54.
22. Munroe RL, Gauvain M. Exposure to open-fire cooking and cognitive performance in children. *Int J Environ Health Res*. 2011;22(2):156–64.
23. Levy RJ. Carbon monoxide pollution and neurodevelopment: a public health concern. *Neurotoxicol Teratol*. 2015;49:31–40.
24. Autism spectrum disorders. Fact sheet, reviewed April 2017. Geneva: World Health Organization; 2017 (<http://www.who.int/en/news-room/fact-sheets/detail/autism-spectrum-disorders>, accessed August 2018).
25. Talbott EO, Arena VC, Rager JR, Clougherty JE, Michanowicz DR, Sharma RK, et al. Fine particulate matter and the risk of autism spectrum disorder. *Environ Res*. 2015;140:414–20.
26. Jung CR, Lin YT, Hwang BF. Air pollution and newly diagnostic autism spectrum disorders: a population-based cohort study in Taiwan. *PloS One*. 2013;8(9):e75510.
27. Lam J, Sutton P, Kalkbrenner A, Windham G, Halladay A, Koustas E, et al. A systematic review and meta-analysis of multiple airborne pollutants and autism spectrum disorder. *PLoS One*. 11(9):e0161851.
28. Guxens M, Ghassabian A, Gong T, Garcia-Esteban R, Porta M, Giorgis-Allemand L, et al. Air pollution exposure during pregnancy and childhood autistic traits in four European population-based cohort studies: the ESCAPE Project. *Environ Health Perspect*. 2016;124(1):133.
29. Gong T, Dalman C, Wicks S, Dal H, Magnusson C, Lundholm C, et al. Perinatal exposure to traffic-related air pollution and autism spectrum disorders. *Environ Health Perspect*. 2017;125(1):119.
30. Gong T, Almqvist C, Bölte S, Lichtenstein P, Anckarsäter H, Lind T, et al. Exposure to air pollution from traffic and neurodevelopmental disorders in Swedish twins. *Twin Res Human Genet*. 2014;17(6):553–62.
31. Attention deficit hyperactivity disorder. Bethesda (MD): National Institute of Mental Health; 2018 (<https://www.nimh.nih.gov/health/topics/attention-deficit-hyperactivity-disorder-adhd/index.shtml>, accessed August 2018).
32. Lyall K, Croen L, Daniels J, Fallin MD, Ladd-Acosta C, Lee BK. The changing epidemiology of autism spectrum disorders. *Ann Rev Public Health*. 2017;38:81–102.
33. Lyall K, Schmidt RJ, Hertz-Picciotto I. Maternal lifestyle and environmental risk factors for autism spectrum disorders. *Int J Epidemiol*. 2014;43(2):443–64.
34. Rossignol D, Genuis S, Frye R. Environmental toxicants and autism spectrum disorders: a systematic review. *Transl Psychiatry*. 2014;4(2):e360.
35. Ornoy A, Weinstein-Fudim L, Ergaz Z. Prenatal factors associated with autism spectrum disorder (ASD). *Reprod Toxicol*. 2015;56:155–69.
36. Flores-Pajot MC, Ofner M, Minh TD, Lavigne E, Villeneuve P. Childhood autism spectrum disorders and exposure to nitrogen dioxide, and particulate matter air pollution: a review and meta-analysis. *Environ Res*. 2016;151:763–76.
37. Weisskopf MG, Kioumourtoglou MA, Roberts AL. Air pollution and autism spectrum disorders: causal or confounded? *Curr Environ Health Rep*. 2015;2(4):430–9.
38. Peterson BS, Rauh VA, Bansal R, Hao X, Toth Z, Nati G, et al. Effects of prenatal exposure to air pollutants (polycyclic aromatic hydrocarbons) on the development of brain white matter, cognition, and behavior in later childhood. *JAMA Psychiatry*. 2015;72(6):531–40.
39. Pujol J, Martínez-Vilavella G, Macià D, Fenoll R, Alvarez-Pedrerol M, Rivas I, et al. Traffic pollution exposure is associated with altered brain connectivity in school children. *Neuroimage*. 2016;129:175–84.

40. Calderón-Garcidueñas L, Engle R, Mora-Tiscareño A, Styner M, Gómez-Garza G, Zhu H, et al. Exposure to severe urban air pollution influences cognitive outcomes, brain volume and systemic inflammation in clinically healthy children. *Brain Cognition*. 2011;77(3):345–55.
41. Calderón-Garcidueñas L, Solt AC, Henríquez-Roldán C, Torres-Jardón R, Nuse B, Herritt L, et al. Long-term air pollution exposure is associated with neuroinflammation, an altered innate immune response, disruption of the blood-brain barrier, ultrafine particulate deposition, and accumulation of amyloid β -42 and α -synuclein in children and young adults. *Toxicol Pathol*. 2008;36(2):289–310.

5.4 Overweight and obesity

Key findings:

- Some studies suggest an association between exposure to air pollution in utero and postnatal weight gain or attained BMI for age. Other studies suggest an association between traffic-related air pollution and insulin resistance in children.
- Air pollution may disrupt the normal development of children, resulting in increased weight-for-length gain and mean BMI and differences in attained BMI at specific ages. Potential mechanisms for these effects include regulation of lipid metabolism, fat storage and appetite.

“Overweight” and “obesity” are defined as abnormal or excessive fat accumulation that may impair health, with weight-for-height greater than two or three standard deviations above the median WHO child growth standard (1). In practical terms, this means that overweight or obese children are too heavy for their height.

Childhood obesity is increasing worldwide and is now recognized as a major public health challenge (2, 3). The prevalence of obesity among young people is high in many countries, but the rate of obesity is increasing at a faster rate in developing than in developed countries (3, 4). The problem has reached the proportions of an epidemic: in 2017, 38.3 million children < 5 years and 340 million aged 5–19 years worldwide were overweight or obese (4, 5). Childhood obesity is likely to continue into adulthood, with increased risks of cardiovascular or metabolic disorders, including diabetes and heart disease. Increasingly, obese children are presenting with these diseases early in life. The rapid global rise in obesity is due to a variety of factors, including overconsumption of energy-dense foods and less physical activity. The environment in which children are raised can also strongly influence their risk of becoming overweight or obese (6, 7). The effects of the mother’s environment during pregnancy must also be considered. The Commission on Ending Childhood Obesity has recognized the importance of ensuring that pregnant women are protected from environmental hazards to reduce the risk of childhood obesity (3). There is growing interest among researchers and policy-makers in determining the effects on childhood obesity of environmental conditions such as air pollution (2).

Ambient air pollution

As indicated in section 5.1, exposure to air pollution in utero may affect birth weight through placental damage, epigenetic changes and maternal inflammatory responses. Longer-term effects on energy balance, weight-for-length gain and BMI for age in early childhood have also been identified in some studies. In a study of children in the USA, Rundle et al. (8) reported that prenatal exposure to PAHs in AAP was associated with increased BMI and obesity in childhood. Pregnant women in this study wore personal air monitoring devices for 2 days during the third trimester of pregnancy. In comparison with the group with lowest exposure, children born to mothers most heavily exposed to PAH had a higher BMI at 5 and 7 years of age and relative risks of obesity of 1.79 and 2.26, respectively. Adjustment was made for several potentially confounding variables, such as the child’s sex, ethnicity and birth weight, but did not account for physical activity. Fleisch et al. (9) measured the weight and length of infants in the Project Viva cohort at birth and at 6 months and determined the association between prenatal exposure to PM_{2.5} and black carbon and fetal growth and infant weight gain. Infants in the highest quartile of exposure to black carbon in the third trimester had less fetal growth than those in the lowest quartile, and an association was found between exposure to black carbon or PM_{2.5} and weight-for-length gain between 0 and 6 months. Account was taken of

potentially confounding variables, including weight gain, maternal smoking and abnormal glucose tolerance.

There is some evidence that air pollution affects different stages of gestation separately. A cohort study was conducted to determine sensitive periods of exposure and sex-specific effects by modelling the day and week of exposure to PM_{2.5} during pregnancy (10). Exposure to PM_{2.5} during 10–29 weeks of gestation resulted in increased waist–hip ratios in girls at the age of 4 years. Exposure in weeks 8–17 and 15–22 of gestation increased the BMI z score and fat mass in boys at the age of 4 years. Studies in experimental animals also indicate specific differences. A study in rodents found significant sex-specific differences in weight gain after exposure to diesel exhaust in utero (11).

Maternal health plays an important role in infant and child health and can modify the effects of environmental exposure. In a study of a cohort of children in the USA, maternal body mass before pregnancy and exposure to ambient PM_{2.5} during pregnancy were measured. Children born to mothers with a high pre-pregnancy BMI and who were exposed to PM_{2.5} during pregnancy and in the first 2 years of life had a higher risk of being overweight or obese between 2 and 9 years of age. In addition, children whose mothers were exposed to PM_{2.5} at levels above the median were at higher risk of being overweight or obese, regardless of their mother’s pre-pregnancy BMI (12).

Although there have been few epidemiological studies on the link between exposure to air pollution and childhood obesity, seven studies were identified in a recent review in which an association was found between AAP and obesity and metabolic outcomes in children (13). Two prospective birth cohort studies on air pollution and insulin resistance in children found positive associations between exposure to traffic-related NO₂ and PM₁₀ and insulin resistance in 10-year-old children (14). An increase in proximity to the nearest major road by 500 metres increased insulin resistance by 7.2%. Obese or overweight children are thus at increased risk for insulin resistance and other metabolic complications. Insulin resistance is associated with risks for type 2 diabetes and cardiovascular disease, which can have profound lifelong effects. In a 4-year longitudinal study, Jerrett et al. (15) investigated the relation between exposure to traffic-related air pollution and changes in BMI in children aged 5–11 years in 13 communities in southern California. NO_x levels were used as an indicator of traffic-related air pollution (which can contain black carbon, ultrafine particles and many PM components). Exposure had a significant effect on BMI growth and BMI level at the age of 10 years. The average annual rate of BMI growth was associated with exposure to NO_x in children with the highest exposure. In another longitudinal study in California (16), children exposed to higher levels of NO_x from traffic-related air pollution had significantly increased BMI growth over 8 years and a higher attained BMI at 18 years of age as compared with the group with lower exposure.

In a cohort study in Italy (17), no association was found between exposure to NO₂, NO_x, PM₁₀, PM_{2.5}, coarse PM or total traffic load within 100 metres of the residence and characteristics including BMI at the age of 4 years, cholesterol levels and waist circumference at 8 years of age.

Household air pollution

There has been no peer-reviewed publication on a link between sources of HAP and childhood overweight or obesity.

Biological mechanisms

While air pollution’s relation to childhood obesity is a relatively new area of research, some studies have identified mechanisms by which air pollution may influence childhood obesity and metabolic dysfunction. Air pollution may influence metabolic development prenatally, as elevated levels of leptin and adiponectin were found in the umbilical cord blood of infants whose mothers were exposed to NO₂, PM_{2.5} and NO during pregnancy (18, 19), and these adipokines have been linked to obesity-related outcomes in childhood (18). In another study, increased levels of leptin and adiponectin were associated with increased weight gain in infant girls at 6 months of age, suggesting a pathway for air pollution-mediated risk of obesity in children (19).

The causes of metabolic dysfunction in childhood are complex. In a metabolic profiling study of overweight or obese young people aged 8–18 living in a highly polluted urban environment, air pollution was associated with higher insulin resistance and secretion and higher glycaemia (20), indicating air pollution is a risk factor for type 2 diabetes. In another study, it was reported that

children living in Mexico City, where the levels of air pollution (particularly PM_{2.5} and O₃) are high, had altered appetite-regulating peptides, high blood leptin and endothelin-1 and vitamin D deficiency (21). Even when the BMI-for-age of the children was below the cut-off for obesity, the analysis indicated potentially increased risks of insulin resistance, obesity, type 2 diabetes, addiction-like behaviour and premature cardiovascular disease in adulthood.

Box 9 provides information on stunting and air pollution.

Box 9. Air pollution and stunting

A child who has a low height-for-age is considered to be stunted. In 2012, WHO adopted a global target to reduce the number of stunted children under the age of 5 by 40% by 2025 (22). In 2017, 150.8 million children under the age of 5 years were stunted (5). Stunting has both immediate and long-term effects on health and well-being, as, in addition to poor physical growth, stunted children are more susceptible to infections and have an increased risk of neurodevelopmental effects, which can affect their school and work performance (23). Children who are stunted often remain shorter than their peers in adulthood and are at increased risk of becoming overweight as they grow older. Stunted growth is due mainly to prolonged insufficient caloric intake or other nutritional deficiencies, but a link with exposure to air pollution has also been proposed in a growing body of literature on the association between AAP and stunting.

A study of maternal exposure to AAP in Bangladesh showed a strong link between AAP and child stunting (24). The study was based on outcome data from four waves of the nationally representative Bangladesh Demographic and Health Survey, conducted between 2004 and 2014. Maternal exposure to AAP (PM_{2.5}) was estimated from high-resolution satellite data. Over half of all children in the study were exposed to an annual ambient PM_{2.5} level > 46 µg/m³, which is over four times the WHO air quality guideline value of 10 µg/m³. These children were significantly more likely to be stunted. It was concluded that reducing AAP in Bangladesh could significantly reduce child stunting.

HAP is also strongly linked to stunting. A population-based cohort study of exposure to indoor biomass fuel and tobacco smoke and the risks of various adverse health outcomes in newborn infants in south India (25) found that infants exposed to HAP were at a 30% higher risk of being stunted at 6 months of age. The link between HAP and child stunting is further supported by the results of a systematic review and meta-analysis of the link between HAP and various adverse health outcomes, including stunting (26). A statistically significant protective association was found between reduced exposure to HAP and stunting in children under 5 years. The authors suggested that switching from polluting to clean fuels could substantially reduce the risk of child stunting and other adverse health outcomes.

Conclusions

Some studies indicate a potential association between exposure to AAP and certain adverse metabolic outcomes in children. They support the plausibility of the “obesogen” hypothesis, which posits that exposure to chemical compounds during development can increase susceptibility to gaining weight, and also the links between childhood obesity, insulin resistance and exposure to air pollution. Because of the limited number of epidemiological studies of exposure to air pollution and obesity and insulin resistance, it would be premature to draw conclusions about causality.

Knowledge gaps and research needs

- A review of the literature suggests an association between exposure to AAP and childhood obesity or insulin resistance, but relatively little research has been done on the associations between childhood overweight and obesity, insulin resistance and air pollution.
- The effect of HAP on childhood overweight and obesity has not been studied, even though children spend a significant amount of time indoors.

References – overweight and obesity

1. Obesity and overweight. Fact sheet no. 311. Geneva: World Health Organization; 2015.
2. Maziak W, Ward KD, Stockton MB. Childhood obesity: are we missing the big picture? *Obes Rev.* 2008;9(1):35–42.
3. WHO Commission on Ending Childhood Obesity (ECHO). Facts and figures on childhood obesity. Geneva: World Health Organization; 2017 (<http://www.who.int/end-childhood-obesity/facts/en/>, accessed August 2018).

4. NCD Risk Factor Collaboration. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. *Lancet*. 2017;390:2627–42.
5. Joint child malnutrition estimates – 2018 edition. New York City (NY): UNICEF; Geneva: World Health Organization; Washington DC: The World Bank Group; 2018 (<http://www.who.int/nutgrowthdb/estimates2017/en/>).
6. Hill JO, Peters JC. Environmental contributions to the obesity epidemic. *Science*. 1998;280(5368):1371–4.
7. Trasande L, Cronk C, Durkin M, Weiss M, Schoeller DA, Gall EA. Environment and obesity in the National Children’s Study. *Environ Health Perspect*. 2009;117(2):159–66.
8. Rundle A, Hoepner L, Hassoun A, Oberfield S, Freyer G, Holmes D, et al. Association of childhood obesity with maternal exposure to ambient air polycyclic aromatic hydrocarbons during pregnancy. *Am J Epidemiol*. 2012;175(11):1163–72.
9. Fleisch AF, Rifas-Shiman SL, Koutrakis P, Schwartz JD, Kloog I, Melly S, et al. Prenatal exposure to traffic pollution: associations with reduced fetal growth and rapid infant weight gain. *Epidemiology*. 2015;26(1):43–50.
10. Chiu YH, Hsua HHL, Wilson A, Coull BA, Pendof MP, Baccarellig A, et al. Prenatal particulate air pollution exposure and body composition in urban preschool children: examining sensitive windows and sex-specific associations. *Environ Res*. 2017;158:798–805.
11. Bolton JL, Smith SH, Huff NC, Gilmour MI, Foster WM, Auten RL, et al. Prenatal air pollution exposure induces neuroinflammation and predisposes offspring to weight gain in adulthood in a sex-specific manner. *FASEB J*. 2012;26:4743–54.
12. Mao G, Nachman RM, Sun Q, Zhang X, Koehler K, Chen Z, et al. Individual and joint effects of early-life ambient exposure and maternal prepregnancy obesity on childhood overweight or Obesity. *Environ Health Perspect*. 2017;125(6):067005.
13. Vrijheid, M, Casas M, Gascon M, Valvi D, Nieuwenhuijsen M. Environmental pollutants and child health – a review of recent concerns. *Int J Hyg Environ Health*. 2016;219(4–5):331–42.
14. Thiering E, Cyrys J, Kratzsch J, Meisinger C, Hoffmann B, Berdel D, et al. Long-term exposure to traffic-related air pollution and insulin resistance in children: results from the GINIplus and LISApplus birth cohorts. *Diabetologia*. 2013;56(8):1696–704.
15. Jerrett M, McConnell R, Wolch J, Chang R, Lam C, Dunton G, et al. Traffic-related air pollution and obesity formation in children: a longitudinal, multilevel analysis. *Environ Health*. 2014;13:49.
16. McConnell R, Shen E, Gilliland FD, Jerrett M, Wolch J, Chang CC, et al. A longitudinal cohort study of body mass index and childhood exposure to secondhand tobacco smoke and air pollution: the Southern California Children's Health Study. *Environ Health Perspect*. 2015;123(4):360–6.
17. Fioravanti S, Cesaroni G, Badaloni C, Michelozzi P, Forastiere F, Porta D. Traffic-related air pollution and childhood obesity in an Italian birth cohort. *Environ Res*. 2018;160:479–86.
18. Lavigne E, Ashley-Martin J, Dodds L, Arbuckle TE, Hystad P, Johnson M, et al. Air pollution exposure during pregnancy and fetal markers of metabolic function: the MIREC Study. *Am J Epidemiol*. 2016;183(9):842–51.
19. Alderete TL, Song AY, Bastain T, Habre R, Toledo-Corral CM, Salam MT, et al. Prenatal traffic-related air pollution exposures, cord blood adipokines and infant weight. *Pediatric Obesity*. 2018;13(6):348–56.
20. Toledo-Corral CM, Alderete TL, Habre R, Berhane K, Lurmann FW, Weigensberg MJ, et al. Effects of air pollution exposure on glucose metabolism in Los Angeles minority children. *Pediatr Obes*. 2018;13(1):54–62.
21. Calderón-Garcidueñas L, Franco-Lira M, D’angiulli A, Rodríguez-Díaz J, Blaurock-Busch E, Busch Y, et al. Mexico City normal weight children exposed to high concentrations of ambient PM_{2.5} show high blood leptin and endothelin-1, vitamin D deficiency, and food reward hormone dysregulation versus low pollution controls. Relevance for obesity and Alzheimer disease. *Environ Res*. 2015;140:579–92.
22. de Onis M, Dewey KG, Borghi E, Onyango AW, Blössner M, Daelmans B, et al. The World Health Organization’s global target for reducing childhood stunting by 2025: rationale and proposed actions. *Matern Child Nutr*. 2013;9(Suppl 2):6–26.
23. Stewart CP, Iannotti L, Dewey KG, Michaelsen KF, Onyango AW. Contextualising complementary feeding in a broader framework for stunting prevention. *Matern Child Nutr*. 2013;9(S2):27–45.
24. Goyal N, Canning D. Exposure to ambient fine particulate air pollution in utero as a risk factor for child stunting in Bangladesh. *Int J Environ Res Public Health*. 2018;15(1):22.
25. Tielsch JM, Katz J, Thulasiraj RD, Coles CL, Sheeladevi S, Yanik EL, et al. Exposure to indoor biomass fuel and tobacco smoke and risk of adverse reproductive outcomes, mortality, respiratory morbidity and growth among newborn infants in South India. *Int J Epidemiol*. 2009;38(5):1351–63.
26. Bruce NG, Dherani MK, Das JK, Balakrishnan K, Adair-Rohani H, Bhutta ZA, et al. (2013). Control of household air pollution for child survival: estimates for intervention impacts. *BMC Public Health*. 2013;13(Suppl 3):S8.

5.5 Respiratory effects

Lung function

Key findings:

- Elevated AAP, particularly traffic-related pollution, impairs lung function and lung function development in children, even at exposures below United States national ambient air quality standards.
- Prenatal exposure to air pollution is associated with impairment of lung development and lung function in childhood.
- There is evidence that lung function development improves in children in urban areas where ambient air quality has been improved.

Lung function is a measure of how effectively the lungs move air in and out of the body in order to exchange oxygen with the blood and remove CO₂. More simply, lung function indicates how well a person breathes. The lungs go through dramatic changes during the embryonic and fetal stages and continue to develop after birth, until late adolescence. Anything that affects the structure of children's still-maturing lungs can affect their lung function later in life. Children exposed to air pollution in utero or in early childhood are thus at risk of compromised lung function for the rest of their lives.

Ambient air pollution

Many studies have shown that exposure to air pollution has negative effects on lung function, although the effects vary. In the European Study of Cohorts for Air Pollution Effects (ESCAPE), Gehring et al. (1) found an association between PM_{2.5} levels at the current address and a small decrease in lung function in children aged 6–8 years. A prospective cohort study of children in Taiwan showed that increased exposure to ambient PM_{2.5} was associated with lower rates of development in some measures of lung function, including forced vital capacity (FVC) and forced expiration volume in 1s (FEV1) and with reduced development of FVC (2). Reduced lung function was also seen in schoolchildren in Hong Kong who had long-term exposure to higher levels of AAP (3). In four cities in China, Chongqing, Guangzhou, Lanzhou and Wuhan, exposure to ambient PM was associated with decreased development of lung function in children (4).

The magnitude of the effects on lung function differs by study, perhaps because of spatial differences in the mass, number and composition of PM. In a study of five European birth cohorts, Eeftens et al. (5) found a more consistent association between increased PM mass and reduced lung function than with individual components of PM. They also found small adverse effects associated with exposure to nickel and sulfur in PM. Overall, these findings suggest that PM mass, rather than specific components, is more useful for assessing risks from exposure to air pollution to lung development and function in children.

Traffic-related pollution is a subject of widespread concern. NO₂ is commonly used as a reliable marker of traffic-related air pollution. In a meta-analysis of 13 studies, increased levels of NO₂ were associated with a higher prevalence of children with abnormal lung function (measured in terms of FEV1) (6). In the Children's Health Study cohort, independent negative associations were found between regional and traffic-related air pollution and lung function (7). These authors suggested that the differences in the strength of the associations reported among studies was due to differences in the exposure assessment methods used, which included roadway proximity, traffic count and density measures instead of validated methods.

Some studies reported adverse effects on lung function of pollution at levels below the national ambient air quality standards of the Environmental Protection Agency in the USA. In a study of the effect of relatively low exposure to pollution on childhood lung function (8), 614 mother–child pairs in the Boston area were studied. Long-term exposure to AAP (fine particulate matter and black carbon) during pregnancy was associated with lower lung function in mid-childhood.

There is evidence that children with asthma are more vulnerable to the effects of air pollution. In some subgroups of asthmatic children, prenatal and early-life exposure to CO, PM₁₀ and NO₂ had negative effects on pulmonary function (9). A study of children with asthma in two cities in Canada

(10) showed that exposure to air pollution was linked to elevated airway oxidative stress and reduced small airway function. In another study in North America (11), exposure of children with asthma to air pollution had adverse effects on both lung function and methacholine responsiveness, which is a measure used to evaluate the degree of bronchial response to external stimulation.

Prenatal exposure to air pollution can impair organogenesis and lung growth, leading to long-term complications (12). Newborns whose mothers were exposed to high levels of PM₁₀ during pregnancy had increased minute ventilation and higher respiratory rate and tidal breathing flow (13). Jedrychowski et al. (14) found that children aged 5 years whose mothers had been exposed to high levels of PM_{2.5} during pregnancy had reduced FVC and FEV1. Morales and colleagues (15) reported lung function deficits in preschool-age Spanish children who had been exposed to traffic-related NO₂ and benzene during the second trimester of pregnancy.

In southern California, pollution levels have been decreasing steadily over the past several decades as a result of air pollution control policies, and there are indications these long-term reductions may improve the respiratory health of children. A study of lung function measured annually in 2120 children in three cohorts in three separate periods (1994–1998, 1997–2001 and 2007–2011) indicated an association between reduced exposure to NO₂, PM_{2.5} and PM₁₀ and improved lung function (measured as FEV1 and FVC) over 4 years (16). The study also showed that the proportion of children with low FEV1 values decreased as air pollution levels fell. In a study of the same cohort (17), children who moved from the study area to areas with higher air pollution had lower lung function growth at follow-up, and children who moved to areas with lower pollution had increased lung function growth.

Household air pollution

Fewer studies have been published on HAP and lung function or lung function development in children. A randomized controlled trial was performed in rural Guatemala in households with pregnant women or infants to measure the effects of an intervention to improve indoor air quality on childhood respiratory health (18). Households in which cooking was traditionally done over an open fire were randomly selected to receive a chimney stove to improve ventilation of combustion products from cooking, at the beginning or at the end of the 18-month trial. At the end of the trial, children in houses that had received the chimney stove had significantly lower longitudinal peak expiratory flow growth and a large but non-significant decrease in FEV1 growth. Box 10 describes an intervention in Nigeria to improve the respiratory health of women and children by the introduction of improved cookstoves.

Box 10. Cleaner stoves, easier breaths (19)

In Nigeria, more than 70% of the population uses solid fuel stoves for cooking. Most household cooking is done by women, often in poorly ventilated kitchens. This results in high exposure of both women and children to HAP. A community-based pilot study was conducted in which low-emission stoves were substituted for traditional biomass stoves in three rural communities: Ajibade, Eruwa and Olorisaoko. Assessments were conducted before and 1 year after the intervention in households with a mother aged 20–60 years and one or more children aged 5–17 years. Before substitution of the stove, the PM_{2.5} levels were found in several cases to be 60 times greater than the WHO standard, and almost half the mothers and children had diminished respiratory function. After the intervention, a remarkable decrease was found in the frequency of exposure-related respiratory symptoms, such as cough, chest tightness, difficulty in breathing and rhinitis, as well as headaches, fever and dizziness.

In cohorts of children in Chongqing, Guangzhou, Lanzhou and Wuhan, use of coal in houses without appropriate ventilation was associated with deficits in lung function growth (20). The FVC and FEV1 of exposed children were 27% and 61%, respectively, below the average annual growth levels of the cohort.

In a small study of lung function in women and children aged 7–15 years in Ecuador, who were exposed to biomass fuel smoke in the home (21), exposed children had reduced FVC and FEV1.

Biological mechanisms

Inhaled particles can be deposited in the bronchioles and the alveoli, where they may affect gas exchange (22). Small particles, particularly PM_{2.5}, are of interest because their size allows them to penetrate deep into the lungs, where they cause irritation and induce oxidative stress and inflammation, damaging lung cells (23). PM is a mixture of physical and chemical components (e.g. nitrates, sulfates, ammonium, PAHs, allergens, microbial compounds, metals) that can contribute to lung dysfunction (22, 24).

Prenatal exposure to air pollution can alter lung function and development by various plausible mechanisms, by causing epigenetic changes in the fetus and negatively affecting the mother's respiratory health (25). A prospective birth cohort study of children exposed prenatally to PAHs indicated that lung function was better when antihistamine medication was used (26), which supports the theory that the mechanism of fetal PAH-induced alterations in lung function is initiated by the allergic inflammatory response to pollutants in the lungs.

Conclusion

There is robust evidence that exposure to air pollution damages children's lung function and impedes their lung function growth. Even at lower levels of exposure, children – whose lungs are still maturing and therefore especially vulnerable to pollution – can have lasting deficits in their lung function. There is also compelling evidence that policies and interventions to improve ambient or household air quality can lead to improvements in children's lung function. Compromised lung function negatively affects quality of life and is associated with long-lasting chronic conditions such as asthma and chronic obstructive pulmonary disease (27–29).

Knowledge gaps and research needs

- Even at exposures to levels below local recommended guidelines, air pollutants significantly reduced children's lung function in some studies. More research is required to identify the levels of pollutants that adversely affect lung function in order to influence policies on air quality.
- More studies are required on the effect of HAP on childhood lung function.

Acute lower respiratory infections, including pneumonia

Key findings:

- Air pollutants such as PM_{2.5}, NO₂ and O₃ increase the risk of pneumonia and other respiratory infections in young children.
- Household use of biomass use increases the risk of acute lower respiratory infection, including pneumonia, in children.
- HAP is the leading cause of acute lower respiratory infection in children under 5 years.

Lower respiratory infections, including pneumonia, bronchitis, bronchiolitis and other acute respiratory diseases, are the second leading cause of child mortality worldwide. Lower respiratory infections caused 878 829 deaths in children under 5 years in 2016, accounting for 15.55% of all child deaths (30, 31). HAP from cooking with solid fuels, AAP and second-hand tobacco smoke were the causes of 57% of the burden of disease (in DALYs) from lower respiratory infections in children under 5 years in 2012 (32–34). HAP is the leading risk factor for lower respiratory infections in children in LMICs, and 13% of lower respiratory infections are attributable to HAP and AAP in HICs, where the levels of exposure are lower (32).

Pneumonia is an acute respiratory infection caused by viruses, bacteria, fungi or chemicals and is characterized by inflammation of the air sacs of the lungs (31, 35). While the major environmental risk factors for pneumonia in children are HAP, AAP and second-hand smoke (35), different pollutants contribute to respiratory infections in various ways.

Ambient air pollution

Short-term exposure to AAP exacerbates acute respiratory infections. Nhung et al. (36) conducted a meta-analysis of 17 studies on the acute effects of AAP on childhood pneumonia and concluded that short-term increases in AAP are significantly associated with increased hospital admissions for pneumonia. Positive associations were found with PM₁₀, PM_{2.5}, SO₂, O₃ and NO₂ in studies conducted in many countries. Darrow and colleagues (37) investigated the association between short-term changes in ambient air pollutant concentrations and visits to emergency departments for respiratory infections. They found that exposure to air pollutants such as PM_{2.5}, NO₂ and O₃ exacerbates upper respiratory infections and pneumonia in children under 5 years.

Long-term exposure to AAP may also increase the risk for pneumonia in early life. In a meta-analysis of 10 European birth cohorts, MacIntyre et al. (38) found an association between long-term exposure to traffic-related air pollution and the incidence of pneumonia. Vehicle traffic is one of the main sources of exposure to AAP. Rice et al. (39) examined the association between prenatal exposure to traffic-related air pollution in Boston, USA, and the risk of respiratory infection (including pneumonia, bronchiolitis and croup) in early life. Reduced distance from roadways and higher traffic density were correlated with a higher risk of respiratory infection, suggesting that living close to a major road during pregnancy heightens the risk for respiratory infections in early life.

There is increasing evidence that exposure to PM plays a significant role in acute respiratory infections. Jedrychowski et al. (40) assessed the effect of prenatal exposure to PM_{2.5} on the occurrence of acute bronchitis and pneumonia between birth and 7 years and found that the incidence of recurrent pulmonary infections was significantly correlated with prenatal PM_{2.5} exposure in a dose-dependent manner. Fuertes et al. (41) combined the results for seven birth cohorts to investigate the effects of various components of PM on the development of pneumonia in early childhood. All the components (iron, potassium, copper, nickel, sulfur, silicon, vanadium) except zinc from PM₁₀ were associated with a higher risk of pneumonia in early life.

Household air pollution

HAP is not only the largest environmental health risk factor worldwide but is also the leading cause of acute lower respiratory infection, particularly pneumonia, in children (42). Systematic reviews show consistent evidence of an association between exposure to HAP and ALRI, especially pneumonia, in children. A meta-analysis of published observational studies showed that the rate of ALRI in young children exposed to smoke from household biomass fuel was twice that of children who were not exposed or who lived in households in which cleaner fuels were used (43). Bruce and colleagues (44, 45) reviewed 26 studies on non-fatal, severe and fatal ALRI and found an association with exposure to HAP.

Other systematic reviews have reported higher risks associated with exposure to solid fuel emissions. These include a comparative risk assessment by Smith et al. (46), a meta-analysis of 10 studies by Misra et al. (47) and a review of six studies of deaths among children with ALRI in LMICs by Sonogo et al. (48). In a study of acute respiratory infection and ALRI, Po and colleagues (49) reported a strong association with exposure to solid biofuel in rural children. Although many reviews noted that the heterogeneity of the studies included was a limitation, the consistency of the findings suggests an association. Box 11 describes a randomized controlled trial on a clean cookstove intervention in Guatemala.

A systematic review prepared for the 2014 WHO guidelines for indoor air quality associated with household fuel combustion (31) concluded that there is substantial evidence that solid-fuel HAP increases the risk of ALRI and that the risk of severe and fatal ALRI may be more than doubled. The authors concluded, however, there is relatively limited evidence on the mechanisms by which HAP causes pneumonia in children.

Box 11. Breathing lessons: a randomized control trial in Guatemala yields insights on clean cooking and children's health

The RESPIRE study, the first randomized controlled trial on the health effects of cooking interventions, was conducted in the rural highlands of San Marcos in Guatemala between October 2002 and December 2004. The region's inhabitants typically used open wood fires for cooking and heating, resulting in long-term exposure to HAP, particularly for women and children. The aim of the study was to determine whether reducing smoke

from a *plancha* chimney stove would reduce the risk of pneumonia in children < 18 months of age. After visiting more than 5000 households, those in which there was a pregnant woman or children under 4 months of age were selected for the trial. The intervention consisted of providing some households with a closed chimney *plancha*. Levels of CO in the home were monitored, and the children were followed for 18 months at periodic visits by field workers and underwent medical examinations, which included monitoring of hypoxaemia and chest X-rays if pneumonia was diagnosed. The researchers found a 33% reduction in diagnosed cases of severe pneumonia in children living in households with the improved stoves, suggesting that exposure to pollution from household fuel combustion plays a role in the pathogenesis of pneumonia and that substitution of clean fuels and devices for cooking and heating may reduce the incidence of pneumonia in children (50).

Several studies in the same cohort were performed in parallel. One found that exposure in utero to smoke from open wood stoves increased the risk of adverse neurodevelopment outcomes over that of children in households with a stove that had an enclosed combustion chamber, such as a *plancha* (51). Another found a lower incidence of children born with low birth weight to mothers living in households with a *plancha* (52).

Biological mechanisms

Pollutants contribute to respiratory infections by several mechanisms. Inhaled PM can damage the normal defence mechanisms of the respiratory tract by causing inflammation and oxidative stress, and breathing NO₂, which is a free radical, can also damage and inflame the respiratory tract. There is some evidence that combustion-derived PM interferes with alveolar macrophages, which have an essential role in the response of the immune system to viruses and bacterial infections, therefore increasing the susceptibility of individuals to infections. Laboratory analysis of human macrophages exposed to HAP showed impaired ability to phagocytose *Streptococcus pneumoniae* and *Mycobacterium tuberculosis* and a lower oxidative burst capacity, suggesting reduced host defence against infection (53). Box 12 discusses the association between air pollution and TB.

A laboratory study showed that exposure to black carbon alters the biofilm structure, composition and function of *Staphylococcus aureus* and *Streptococcus pneumoniae* and their tolerance to proteolytic degradation and response to antibiotics (54). Furthermore, black carbon caused *S. pneumoniae* to spread from the nasopharynx to the lungs in an animal model. These results have important implications for the pathways by which air pollution may cause lung infections in children.

Box 12. Air pollution and tuberculosis

TB is an infectious disease caused by *Mycobacterium tuberculosis*. It affects mainly the lungs but can also spread to other body systems. It is transmitted from person to person through the air. Disease progression may be influenced by environmental factors, and air pollution has been identified as a potential risk factor for active TB in adults in several studies. There is a growing scientific evidence that both AAP and HAP are associated with TB in children, but no systematic reviews specifically on studies in children have been published, and the risk of childhood TB associated with exposure to AAP has not yet been evaluated. An ecological study conducted in North Carolina, USA, showed a significant association between pulmonary TB and long-term exposure to PM. Children and adolescents aged 0–24 years were included in the study, but their risk was not independently assessed (55). Another study in adults in California, USA, also found a relation between residential exposure to PM_{2.5} and the presence of smear-positive acid-fast bacilli (56).

The association between exposure to HAP from solid fuel combustion and TB has been evaluated in a few studies. Exposure to biomass fuel combustion exhaust was found to prevent macrophages in the lung from functioning correctly (57). As macrophages have a key role in the immune response to infection, these changes may increase the vulnerability of individuals to TB and other respiratory infections (58). A meta-analysis of studies in children and adults performed in 2014 (59) showed a relation between HAP from solid fuel combustion and the risk of TB. Two of the studies included in the review were specifically of children, and both found positive but nonsignificant associations. A case–control study in India showed a correlation between exposure to HAP from solid fuel combustion and the risk of contracting TB in children < 5 years (60). The evidence suggests a positive association between exposure to air pollution and TB infection in children, although further research is needed.

Conclusions

Many studies offer consistent, compelling evidence that exposure to AAP or HAP is a major risk factor for ALRI in children. It is clear that exposure to air pollution increases the incidence of ALRI,

including pneumonia. While a range of pollutants has been found to exacerbate respiratory infections, there is growing evidence that PM has an especially strong effect.

Knowledge gaps and research needs

- There is a lack of studies on associations between exposure to specific chemical components of ambient particulate matter and ALRI such as pneumonia in young children.
- Research on the mechanisms through which air pollution induces lung infection will aid in identification of treatments and preventive measures to protect children from serious, life-threatening illness.

Asthma

Key findings:

- There is evidence of a causal relationship between exposure to AAP and the development and exacerbation of childhood asthma.
- There is suggestive evidence of a causal effect of exposure to HAP and the development and exacerbation of asthma in children.

Asthma affects an estimated 250 million people worldwide and is a common chronic illness in children (61). Both AAP and HAP have long been suspected of contributing to childhood asthma, and a growing body of research suggests that exposure to air pollution both causes and exacerbates the condition. As children have narrower airways and higher breathing rates than adults, they are particularly vulnerable to airborne pollution. Furthermore, children tend to spend much time doing physical activity outdoors and breathe through their mouths more frequently than adults, allowing more unfiltered air pollutants to affect their still-developing lungs.

Asthma development

Ambient air pollution

In a meta-analysis of 19 studies conducted between 1996 and 2012, Gasana et al. (62) observed a positive association between exposure to NO₂ and the incidence of asthma and between exposure to PM and higher incidence of wheeze in children. In addition, they found a higher prevalence of wheeze in children exposed to SO₂ and a higher prevalence of asthma associated with exposure to NO₂, nitrous oxide and CO. A systematic review and meta-analysis by Khreis and colleagues (63) of 41 studies indicated significant associations between increased exposure to PM_{2.5}, PM₁₀, NO₂ and black carbon and the risk of asthma.

The longitudinal association between early childhood exposure to AAP and future asthma incidence has been evaluated in several cohort studies. In a meta-analysis of published birth cohort studies, Bowatte et al. (64) reported significant associations between long-term exposure to black carbon and PM_{2.5} and the risk of asthma in childhood. They also reported an association between exposure to traffic-related air pollution in early childhood and a higher risk of developing asthma up to 12 years of age. Gehring et al. (65) evaluated the longitudinal association between prenatal exposure to air pollution and development of asthma throughout childhood and adolescence in four prospective birth cohort studies in Europe. They found that increased exposure to NO₂ and PM_{2.5} at the birth address was associated with an increased risk for asthma throughout childhood and adolescence. Sbihi et al. (66) analysed a population-based birth cohort of 65,254 children in Vancouver, Canada, and found positive association between perinatal exposure to air pollution and asthma incidence during pre-school years.

Two systematic reviews reached similar conclusions on the role of long-term exposure to AAP in asthma development, further strengthening evidence of an association. A systematic review of 18 studies (67) found evidence of a significant link between prenatal exposure to NO₂, SO₂ and PM₁₀ and the development of asthma. The authors found insufficient evidence that exposure to black carbon, CO or O₃ during pregnancy was associated with asthma in childhood. Andersen and colleagues (68) reviewed 17 cohort studies and found that 12 showed positive associations between exposure to air pollution and the incidence of asthma.

Household air pollution

Several studies found positive associations between indoor cooking with polluting fuels and asthma development in children. In a meta-analysis of 41 studies published before 2013, Lin et al. (69) found a positive association between gas cooking, exposure to NO₂ and childhood asthma or wheeze. In a study of over 512 000 children in primary and secondary schools in 47 countries, Wong et al. (70) found a link between cooking on an open fire and the risk of reported asthma in both boys and girls. Studies in India (71) and Nepal (72) also found statistically significant increases in the risk of asthma with indoor use of biomass fuel stoves, especially in the absence of appropriate ventilation. In contrast, a study in Malaysia found no association between exposure to household wood stoves and a first hospitalization for asthma of children aged 1 month to 5 years (73). The authors also found no association with other factors, such as use of kerosene stoves, aerosol mosquito repellent or crowding.

Asthma exacerbation

Ambient air pollution

Numerous studies have found that exposure to PM₁, PM_{2.5} and PM₁₀ exacerbates asthma, aggravating the symptoms of wheeze and shortness of breath (74, 75). A meta-analysis of 26 studies conducted in Australia, Canada, China, Denmark, Finland, Turkey and the USA (76) found a 4.8% increase in the risk of asthma-associated emergency department visits and admissions among children exposed to short-term increases in PM_{2.5} of 10 µg/m³. The effect was greater in studies conducted in Europe and North America than in Asia. Other meta-analyses have found similar results. A review revealed a 3.6% increase in risk of emergency visits and admissions for asthma of children per 10 µg/m³ increase in ambient PM_{2.5} (77). Zheng and colleagues (78) also reported associations between exposure to PM_{2.5}, O₃, CO, NO₂, SO₂ and PM₁₀, and hospital admissions in 50 studies of children. Meta-analyses by Weinmayr and colleagues (79) showed associations with PM₁₀, and Orellano and colleagues (80) found associations with NO₂, SO₂ and PM_{2.5}.

Zhang et al. (81) conducted a meta-analysis of 26 studies conducted in the East Asian region and found associations between exposure to ambient NO₂, SO₂, CO, and PM₁₀ and asthma-related use of general and emergency hospitals. The association between exposure to air pollution and asthma morbidity was generally stronger in children < 15 years than in other age groups. The results of studies in southern California, USA, where the levels of air pollution have been decreasing for several decades as a result of air pollution controls, show that decreases in AAP levels were associated with statistically significant decreases in bronchitic symptoms among children with asthma (82). These meta-analyses together provide compelling evidence that exposure to a range of ambient air pollutants places children at higher risk of asthma-related hospitalization.

Box 13. Case study: Identifying AAP as a trigger for asthma exacerbation³

Megan, a 9-year-old living in Alberta, Canada, was diagnosed with asthma 5 years ago. Since then, her symptoms have been well controlled by a common treatment: inhaled steroids combined with a long-acting beta agonist, and a short-acting beta agonist as needed. In the past 2 weeks, however, she had had several episodes of breathlessness while training outside with the school track team, which she joined recently. On days spent at home, her symptoms diminished. She had no allergies, and spring had not yet arrived in the region. Her home was in a quiet suburban neighbourhood, with no highways or industrial areas nearby, and close to a park. Her parents didn't smoke or keep pets, and the home did not have a solid-fuel stove.

Her school, however, was on a busy street. Drivers often left their engines running while picking up or dropping off passengers. The school playground was regularly sprayed with herbicides. Her paediatrician concluded that the exacerbation of her asthma was probably related to her exposure to herbicides in the school playground and to AAP from motor vehicles. The paediatrician recommended that the family check the Air Quality Health Index, Canada's local air quality monitoring system, daily basis before Megan participated in outdoor activities. The doctor also advised that Megan be given an extra dose of her regular asthma medication

³ Presented at Workshop 6: Protect the children! What you can do to prevent environmental hazards from harming children. 28th International Congress of Pediatrics, 17–22 August 2016, Vancouver, Canada.

before she engaged in sports. The school principal was notified and decided to implement a “no-idling” policy for vehicles. Parents advocated to stop herbicide being sprayed in the playground and discussed with teachers the possibility of training inside on days when AAP levels were higher than recommended for outdoor activities.

Asthma is a multifaceted condition. In Megan’s case, the exacerbation appeared to be related to poor air quality due to heavy traffic, use of herbicides and idling of vehicles near the training area. After a few weeks of alternating indoor–outdoor training when pollution levels were high, cessation of herbicide use and the no-idling policy, Megan’s episodes of breathlessness diminished, and her extra treatment for asthma was no longer necessary.

Household air pollution

Although the relation between exposure to AAP and exacerbation of childhood asthma has been well documented, there is less evidence on exposure to HAP from incomplete combustion of polluting fuels. The review for the WHO guidelines for indoor air quality associated with household fuel combustion (31) concluded that there is suggestive evidence for a causal effect of exposure to HAP and exacerbation of asthma in children. A cross-sectional study in rural Nigeria of 1690 school-age children (19) found that living in a household in which biomass fuel was used for cooking increased the risk of severe asthma symptoms. Schei and colleagues (83) concluded that use of open fires increased the risk of asthma symptoms in children aged 4–6 years living in an indigenous Maya community in Guatemala.

Box 14 describes a community activity for managing asthma in children.

Biological mechanisms

Many pathways have been studied through which air pollution may contribute to childhood asthma. Inflammation and oxidative stress are known harmful effects of air pollution. In a study of children with asthma living in a highly polluted environment, elevated levels of SO₂, NO₂ and benzene were associated with increased bronchial inflammation and biological markers of oxidative damage and asthma symptoms (84). Research also indicates that epigenetic modification of DNA plays a role in the association between childhood asthma and air pollution (85). The patterns of DNA methylation that contributes to lung damage in children with asthma in response to air pollution have been identified (86), and another study indicated that DNA methylation associated with prenatal exposure to PAHs may contribute to the development of childhood asthma by altering gene expression early in life (84), but more research is needed.

Box 14. “You can control asthma now”.

An award-winning initiative demonstrates effective community engagement in managing asthma in children, at the Children’s Hospital of Richmond, at Virginia Commonwealth University in the USA (84). The hospital formulated a promising programme called “You can control asthma now” in response to the disproportionately high burden of disease attributed to asthma, compounded by poverty, in its region.

When a child is first diagnosed with asthma, the family is directed to the unit by their general practitioner or the emergency room. The unit is staffed by a pulmonologist, a nurse and social workers, who use a multidisciplinary approach for clinical assessments, education and providing support and resources to address barriers to treatment. Practitioners follow up families over the long term by text or phone communications. The home environment is assessed by the City of Richmond Health District, and families can be referred to a medical legal partnership programme to help them resolve any environmental problems in the home that might affect their child’s asthma. The programme offers extensive practical information to families on the effect of environmental exposures on childhood asthma and how to make changes.

Since 2015, the programme has assisted more than 344 patients with family-focused management techniques. The programme has also made the region’s health care system more cost-effective, saving US\$ 691 per patient by fewer hospitalizations and emergency room visits, which adds up to a total cost reduction of US\$ 163 958 since the programme began. The initiative was awarded the “Asthma award” of the Environmental Protection Agency in 2017 in recognition of a successful asthma management intervention that is integrated into health care services. The programme shows that, through collaboration and engagement with the community and the development of specific resources, families can reduce environmental exposures to protect the health of children.

Conclusion

The relation between air pollution and childhood asthma is clear. Many studies provide consistent, robust evidence of an association between exposure to air pollution and the risk of developing asthma in childhood. There is also ample evidence that breathing pollutants exacerbates asthma in children. Although the mechanisms are not as well understood, long-term exposure to PM and other pollutants can increase the probability that a child will develop asthma, with serious long-term implications for health and quality of life. While there are fewer studies on HAP, they provide sufficient evidence to support proactive approaches to limit children's exposure to both AAP and HAP to protect them from developing and exacerbating asthma.

Knowledge gaps and research needs

- Various epidemiological studies support the conclusion that AAP and traffic-related air pollution are related to exacerbation and development of asthma in children. The vulnerable period of exposure for childhood asthma remains to be defined, and more long-term birth cohort studies with regular, repeated follow-up are needed.
- Although there is suggestive evidence of an effect of exposure to HAP on asthma development and exacerbation, additional studies with consistent methods and exposure assessment and intervention studies are necessary to confirm a causal relation.

References – respiratory effects

1. Gehring U, Gruziova O, Agius RM, Beelen R, Custovic A, Cyrus J, et al. Air pollution exposure and lung function in children: the ESCAPE project. *Environ Health Perspect.* 2013;121(11–12):1357–64.
2. Hwang BF, Chen YH, Lin YT, Wu XT, Leo Lee Y. Relationship between exposure to fine particulates and ozone and reduced lung function in children. *Environ Res.* 2015;137:382–90.
3. Gao Y, Chan EYY, Li L, Lau PWC, Wong TW. Chronic effects of ambient air pollution on lung function among Chinese children. *Arch Dis Child.* 2013;98(2):128–35.
4. Roy A, Hu W, Wei F, Korn L, Chapman RS, Zhang J. Ambient particulate matter and lung function growth in Chinese children. *Epidemiology.* 2012;23(3):464–72.
5. Eeftens M, Hoek G, Gruziova O, Mölter A, Agius R, Beelen R, et al. Elemental composition of particulate matter and the association with lung function. *Epidemiology.* 2014;25(5):648–57.
6. Barone-Adesi F, Dent JE, Dajnak D, Beevers S, Anderson HR, Kelly FJ, et al. Long-term exposure to primary traffic pollutants and lung function in children: cross-sectional study and meta-analysis. *PLoS One.* 2015;10(11):e0142565.
7. Urman R, McConnell R, Islam T, Avol EL, Lurmann FW, Vora H, et al. Associations of children's lung function with ambient air pollution: joint effects of regional and near-roadway pollutants. *Thorax.* 2014;69:540–7.
8. Rice MB, Rifas-Shiman SL, Litonjua AA, Oken E, Gillman MW, Kloog I, et al. Lifetime exposure to ambient pollution and lung function in children. *Am J Resp Crit Care Med.* 2016;193(8):881–8.
9. Mortimer, K, Neugebauer R, Lurmann F, Alcorn S, Balmes J, Tager I. Air pollution and pulmonary function in asthmatic children: effects of prenatal and lifetime exposures. *Epidemiology.* 2008;19(4):550–7; discussion 561–2.
10. Liu L, Poon R, Chen L, Frescura AM, Montuschi P, Ciabattini G, et al. Acute effects of air pollution on pulmonary function, airway inflammation, and oxidative stress in asthmatic children. *Environ Health Perspect.* 2009;117(4):668–74.
11. Jerodiakonou D, Zanobetti A, Coull BA, Melly S, Postma DS, Boezen HM, Vonk JM, et al. Ambient air pollution, lung function, and airway responsiveness in asthmatic children. *J Allergy Clin Immunol.* 2016;137(2):390–9.
12. Korten I, Ramsey K, Latzin P. Air pollution during pregnancy and lung development in the child. *Paediatr Resp Rev.*, 2017;21:38–46.
13. Latzin P, Röösli M, Huss A, Kuehni CE, Frey U. Air pollution during pregnancy and lung function in newborns: a birth cohort study. *Eur Resp J.* 2009;33(3):594–603.
14. Jedrychowski WA, Perera FP, Maugeri U, Mroz E, Klimaszewska-Rembiasz M, Flak E, et al. Effect of prenatal exposure to fine particulate matter on ventilatory lung function of preschool children of non-smoking mothers. *Paediatr Perinat Epidemiol.* 2010;24(5):492–501.
15. Morales E, Garcia-Esteban R, de la Cruz OA, Basterrechea M, Lertxundi A, de Dicastillo MD, et al. Intrauterine and early postnatal exposure to outdoor air pollution and lung function at preschool age. *Thorax.* 2015;70:64–73.
16. Gauderman WJ, Urman R, Avol E, Berhane K, McConnell R, Rappaport E, et al. Association of improved air quality with lung development in children. *N Engl J Med.* 2015;372(10):905–13.

17. Avol EL, Gauderman WJ, Tan SM, London SJ, Peters JM. Respiratory effects of relocating to areas of differing air pollution levels. *Am J Respir Crit Care Med.* 2001;164:2067–72.
18. Heinzerling AP, Guarnieri MJ, Mann JK, Diaz JV, Thompson LM, Diaz A, et al. Lung function in woodsmoke-exposed Guatemalan children following a chimney stove intervention. *Thorax.* 2016;71(5):421–8.
19. Oluwole O, Ana GR, Arinola G, Wiskel T, Falusi A, Huo D, et al. Effect of stove intervention on household air pollution and the respiratory health of women and children in rural Nigeria. *Air Quality Atmos Health.* 2013;6(3):553–61.
20. Roy A, Chapman RS, Hu W, Wei F, Liu X, Zhang J. Indoor air pollution and lung function growth among children in four Chinese cities. *Indoor Air.* 2012;22(1):3–11.
21. Rinne ST, Rodas EJ, Bender BS, Rinne ML, Simpson JM, Galer-Unti R, et al. Relationship of pulmonary function among women and children to indoor air pollution from biomass use in rural Ecuador. *Respir Med.* 2006;100(7):1208–15.
22. Kim KH, Kabir E, Kabir S. A review on the human health impact of airborne particulate matter. *Environ Int.* 2015;74:136–43.
23. Xing YF, Xu YH, Shi MH, Lian YX. The impact of PM_{2.5} on the human respiratory system. *J Thoracic Dis.* 2016;8(1):E69.
24. Health effects of particulate matter. Policy implications for countries in eastern Europe, Caucasus and central Asia. Copenhagen: WHO Regional Office for Europe; 2013 (http://www.euro.who.int/__data/assets/pdf_file/0006/189051/Health-effects-of-particulate-matter-final-Eng.pdf, accessed 10 May 2018).
25. Veras MM, de Oliveira Alves N, Fajersztajn L, Saldiva P. Before the first breath: prenatal exposures to air pollution and lung development. *Cell Tissue Res.* 2017;367(3):445–55.
26. Jedrychowski WA, Perera FP, Maugeri U, Majewska R, Spengler J, Mroz E, et al. Antihistamine medication may alleviate negative effects of prenatal exposure to polycyclic aromatic hydrocarbons (PAH) on lung function in children. Birth cohort prospective study. *Pediatr Pulmonol.* 2015;50(5):469–78.
27. Bui DS, Lodge CJ, Burgess JA, Lowe AJ, Perret J, Bui MQ, et al. Childhood predictors of lung function trajectories and future COPD risk: a prospective cohort study from the first to the sixth decade of life. *Lancet Respir Med.* 2018;6(7):535–44.
28. Svanes C, Sunyer J, Plana E, Dharmage S, Heinrich J, Jarvis D, et al. Early life origins of chronic obstructive pulmonary disease. *Thorax.* 2010;65(1):14–20.
29. Martinez FD. Early-life origins of chronic obstructive pulmonary disease. *N Engl J Med* 2016;375:871–8.
30. WHO-MCEE estimates for child causes of death, 2000–2016. Geneva: World Health Organization; 2018 (http://www.who.int/healthinfo/global_burden_disease/estimates/en/index3.html, accessed August 2018).
31. Bruce N, Smith KR, Balmes J, Pope D, Dherani M, Zhang J, et al. Indoor air quality guidelines: household fuel combustion. Review 4: Health effects of household air pollution (HAP) exposure. Geneva: World Health Organization; 2014.
32. Don't pollute my future! The impact of the environment on children's health. Geneva: World Health Organization; 2017.
33. Ambient air pollution: a global assessment of exposure and burden of disease. Geneva: World Health Organization; 2016.
34. Global Health Observatory (GHO) data: Child health. Geneva: World Health Organization; 2016 (http://www.who.int/gho/child_health/en/, accessed 21 October 2016).
35. Pneumonia. Geneva: World Health Organization; 2016 (<http://www.who.int/mediacentre/factsheets/fs331/en/>, accessed August 2018).
36. Nhung NTT, Amini H, Schindler C, Kutlar Joss M, Dien TM, Probst-Hensch N, et al., Short-term association between ambient air pollution and pneumonia in children: a systematic review and meta-analysis of time-series and case-crossover studies. *Environ Pollut.* 2017;230:1000–8.
37. Darrow LA, Klein M, Flanders WD, Mulholland JA, Tolbert PE, Strickland MJ. Air pollution and acute respiratory infections among children 0–4 years of age: an 18-year time-series study. *Am J Epidemiol.* 2014;180(10):968–77.
38. MacIntyre EA, Gehring U, Mölter A, Fuertes E, Klümper C, Krämer U, Quass U, et al. Air pollution and respiratory infections during early childhood: an analysis of 10 European birth cohorts within the ESCAPE Project. *Environ Health Perspect.* 2014;122(1):107.
39. Rice MB, Rifas-Shiman SL, Oken E, Gillman MW, Ljungman PL, Litonjua AA, et al. Exposure to traffic and early life respiratory infection: a cohort study. *Pediatr Pulmonol.* 2015;50(3):252–9.
40. Jedrychowski WA, Perera FP, Spengler JD, Mroz E, Stigter L, Flaka E, et al. Intrauterine exposure to fine particulate matter as a risk factor for increased susceptibility to acute broncho-pulmonary infections in early childhood. *Int J Hyg Environ Health.* 2013;216(4):395–401.
41. Fuertes E, MacIntyre E, Agius R, Beelen R, Brunekreef B, Bucci S, Cesaroni G, et al. Associations between particulate matter elements and early-life pneumonia in seven birth cohorts: results from the ESCAPE and TRANSPHORM projects. *Int J Hyg Environ Health.* 2014;217(8):819–29.
42. Burning opportunity: clean household energy for health, sustainable development, and wellbeing of women and children. Geneva: World Health Organization; 2016.

43. Desai MA, Mehta S, Smith KR. Indoor smoke from solid fuels: assessing the environmental burden of disease at national and local levels Geneva: World Health Organization; 2004 (<http://www.who.int/iris/handle/10665/42885>, accessed August 2018).
44. Dherani M, Pope D, Mascarenhas M, Smith KR, Weber M, Bruce N. Indoor air pollution from unprocessed solid fuel use and pneumonia risk in children aged under five years: a systematic review and meta-analysis. *Bull World Health Organ.* 2008;86(5):390–401.
45. Bruce NG, Dherani MK, Das JK, Balakrishnan K, Adair-Rohani H, et al. Control of household air pollution for child survival: estimates for intervention impacts. *BMC Public Health.* 2013;13(Suppl 3):S8.
46. Smith KR, Mehta S, Feuz M. Indoor air pollution from household use of solid fuels. In: Ezzati M, ed. *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors.* Geneva: World Health Organization; 2004.
47. Misra P, Srivastava R, Krishnan A, Sreenivaas V, Pandav CS. Indoor air pollution-related acute lower respiratory infections and low birthweight: a systematic review. *J Trop Pediatr.* 2012;58(6):457–66.
48. Sonogo M, Pellegrin MC, Becker G, Lazzarini M. Risk factors for mortality from acute lower respiratory infections (ALRI) in children under five years of age in low and middle-income countries: a systematic review and meta-analysis of observational studies. *PLoS One.* 2015;10(1):e0116380.
49. Po JYT, FitzGerald JM, Carlsten C. Respiratory disease associated with solid biomass fuel exposure in rural women and children: systematic review and meta-analysis. *Thorax.* 2011;66:232–9.
50. Smith KR, McCracken JP, Weber MW, Hubbard A, Jenny A, Thompson LM, et al., Effect of reduction in household air pollution on childhood pneumonia in Guatemala (RESPIRE): a randomised controlled trial. *Lancet.* 2011;378(9804):1717–26.
51. Dix-Cooper L, Eskenazi B, Romero C, Balmes J, Smith KR. Neurodevelopmental performance among school age children in rural Guatemala is associated with prenatal and postnatal exposure to carbon monoxide, a marker for exposure to woodsmoke. *Neurotoxicology.* 2012;33(2):246–54.
52. Thompson LM, Bruce N, Eskenazi B, Diaz A, Pope D, Smith KR. Impact of reduced maternal exposures to wood smoke from an introduced chimney stove on newborn birth weight in rural Guatemala. *Environ Health Perspect.* 2011;119(10):1489–94.
53. Rylance J, Fullerton DG, Scriven J, Aljurayyan AN, Mzinza D, Barrett S, et al. Household air pollution causes dose-dependent inflammation and altered phagocytosis in human macrophages. *Am J Resp Cell Mol Biol.* 2015;52(5):584–93.
54. Hussey S, Purves J, Allcock N, Fernandes VE, Monks PS, Ketley JM, et al. Air pollution alters *Staphylococcus aureus* and *Streptococcus pneumoniae* biofilms, antibiotic tolerance and colonisation. *Environ Microbiol.* 2017;19(5):1868–80.
55. Smith GS, Schoenbach VJ, Richardson DB, Gammon MD. Particulate air pollution and susceptibility to the development of pulmonary tuberculosis disease in North Carolina: an ecological study. *Int J Environ Health Res.* 2014;24(2):103–12.
56. Jassal MS, Bakman I, Jones B. Correlation of ambient pollution levels and heavily-trafficked roadway proximity on the prevalence of smear-positive tuberculosis. *Public Health.* 2013;127:268–74.
57. Aam BB, Fonnum F. Carbon black particles increase reactive oxygen species formation in rat alveolar macrophages in vitro. *Arch Toxicol.* 2007;81:441–6.
58. Fullerton DG, Bruce N, Gordon SB. Indoor air pollution from biomass fuel smoke is a major health concern in the developing world. *Trans R Soc Trop Med Hyg.* 2008;102(9):843–51.
59. Kurmi OP, Sadhra CS, Ayres JG, Sadhra SS. Tuberculosis risk from exposure to solid fuel smoke: a systematic review and meta-analysis. *J Epidemiol Community Health.* 2014;68:1112–8.
60. Jubulis J, Kinikar A, Ithape M, Khandave M, Dixit S, Hotalkar S, et al. Modifiable risk factors associated with tuberculosis disease in children in Pune, India. *Int J Tuberc Lung Dis.* 2014;18:198–204.
61. GBD 2015 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet.* 2016;388:1545–602.
62. Gasana J, Dillikar D, Mendy A, Forno E, Ramos Vieira E. Motor vehicle air pollution and asthma in children: a meta-analysis. *Environ Res.* 2012;117:36–45.
63. Khreis H, Kelly C, Tate J, Parslow R, Lucas K, Nieuwenhuijsen M. Exposure to traffic-related air pollution and risk of development of childhood asthma: a systematic review and meta-analysis. *Environ Int.* 2017;100:1–31.
64. Bowatte G, Lodge C, Lowe AJ, Erbas B, Perret J, Abramson MJ, et al. The influence of childhood traffic-related air pollution exposure on asthma, allergy and sensitization: a systematic review and a meta-analysis of birth cohort studies. *Allergy.* 2015;70(3):245–56.
65. Gehring U, Wijga AH, Hoek G, Bellander T, Berdel D, Brüska I, et al., Exposure to air pollution and development of asthma and rhinoconjunctivitis throughout childhood and adolescence: a population-based birth cohort study. *Lancet Respir Med.* 2015;3(12):933–42.
66. Sbihi H, Tamburic L, Koehoorn M, Brauer M. Perinatal air pollution exposure and development of asthma from birth to age 10 years. *Eur Resp J.* 2016:ERJ-00746-2015.
67. Hehua Z, Qing C, Shanyan G, Qijun W, Yuhong Z. The impact of prenatal exposure to air pollution on childhood wheezing and asthma: a systematic review. *Environ Res.* 2017;159:519–30.
68. Anderson HR, Favarato G, Atkinson RW. Long-term exposure to air pollution and the incidence of

- asthma: meta-analysis of cohort studies. *Air Qual Atmos Health*. 2013;6:47–56.
69. Lin W, Brunekreef B, Gehring U. Meta-analysis of the effects of indoor nitrogen dioxide and gas cooking on asthma and wheeze in children. *Int J Epidemiol*. 2013;42(6):1724–37.
 70. Wong GW, Brunekreef B, Ellwood P, Anderson HR, Asher MI, Crane J, et al. Cooking fuels and prevalence of asthma: a global analysis of phase three of the International Study of Asthma and Allergies in Childhood (ISAAC). *Lancet Resp Med*. 2013;1(5):386–94.
 71. Padhi BK, Padhy PK. Domestic fuels, indoor air pollution, and children's health. *Ann N Y Acad Sci*. 2008;1140:209–17.
 72. Melsom T, Brinch L, Hessen JO, Schei MA, Kolstrup N, Jacobsen BK, et al. Asthma and indoor environment in Nepal. *Thorax*. 2001;56:477–81.
 73. Azizi BH, Zulkifli HI, Kasim S. Indoor air pollution and asthma in hospitalized children in a tropical environment. *J Asthma*. 1995;32:413–8.
 74. Yu O, Sheppard L, Lumley T, Koenig JQ, Shapiro GG. Effects of ambient air pollution on symptoms of asthma in Seattle-area children enrolled in the CAMP study. *Environ Health Perspect*. 2000;108:1209–14.
 75. Slaughter JC, Lumley T, Sheppard L, Koenig JQ, Shapiro GG. Effects of ambient air pollution on symptom severity and medication use in children with asthma. *Ann Allergy Asthma Immunol*. 2003;91:346–53.
 76. Lim H, Kwon HJ, Lim JA, Choi JH, Ha M, Hwang S, et al. Short-term effect of fine particulate matter on children's hospital admissions and emergency department visits for asthma: a systematic review and meta-analysis. *J Prev Med Public Health*. 2016;49:205–19.
 77. Fan J, Li S, Fan S, Bai Z, Yang K. The impact of PM_{2.5} on asthma emergency department visits: a systematic review and meta-analysis. *Environ Sci Pollut Res*. 2016;23:843–50.
 78. Zheng XY, Ding H, Jiang LN, Chen SW, Zheng JP, Qiu M, et al. Association between air pollutants and asthma emergency room visits and hospital admissions in time series studies: a systematic review and meta-analysis. *PLoS One*. 2015;10(9):e0138146.
 79. Weinmayr G, Romeo E, Sario MD, Weiland SK, Forastiere F. Short-term effects of PM₁₀ and NO₂ on respiratory health among children with asthma or asthma-like symptoms: a systematic review and meta-analysis. *Environ Health Perspect*. 2010;118(4):449–57.
 80. Orellano P, Quaranta N, Reynoso J, Balbi B, Vasquez J. Effect of outdoor air pollution on asthma exacerbations in children and adults: systematic review and multilevel metaanalysis. *PLoS One*. 2017;12(3):e0174050.
 81. Zhang S, Li G, Tian L, Guo Q, Pan X. Short-term exposure to air pollution and morbidity of COPD and asthma in East Asian area: a systematic review and meta-analysis. *Environ Res*. 2016;148:15–23.
 82. Berthane K, Chang CC, McConnell R, Gauderman WJ, Avol E, Rapaport E, et al. Association of changes in air quality with bronchitic symptoms in children in California, 1993–2012. *JAMA*. 2016;315:1491–501.
 83. Schei MA, Hessen JO, Smith KR, et al: Childhood asthma and indoor woodsmoke from cooking in Guatemala. *J Expo Anal Environ Epidemiol* 2004; 14: pp. S110–S117
 84. Asthma Community Network. Richmond (VA): Children's Hospital of Richmond at Virginia Commonwealth University (<http://www.asthmacommunitynetwork.org/node/16557>, accessed August 2018).
 85. Rusconi F, Catelan D, Accetta G, Peluso M, Pistelli R, Barbone F, et al. Asthma symptoms, lung function, and markers of oxidative stress and inflammation in children exposed to oil refinery pollution. *J Asthma*. 2011;48(1):84–90.
 86. Gruzjeva O, Xu CJ, Breton CV, Annesi-Maesano I, Antó JM, Auffray C, et al. Epigenome-wide meta-analysis of methylation in children related to prenatal NO₂ air pollution exposure. *Environ Health Perspect*. 2017;125:104–10.
 87. Esposito S, Tenconi R, Lelii M, Preti V, Nazzari E, Consolo S, et al. Possible molecular mechanisms linking air pollution and asthma in children. *BMC Pulmon Med*. 2014;14(1):31.

5.6 Otitis media

Key findings:

- An increasing number of epidemiological studies indicate an association between exposure to AAP and the occurrence of otitis media in children.
- HAP from combustion may increase the risk of otitis media.

Otitis media, inflammation of the middle ear, is a common childhood infection (1). Viral and bacterial ear infections are the primary causes of otitis media, which often occurs with upper respiratory tract infections (2). Environmental exposures also play a role. Exposure to second-hand tobacco smoke is a known risk factor (3, 4), and evidence suggests that exposure to AAP and HAP

may have a similar effect on the development of otitis media. A child with recurrent otitis media can have long-term consequences, such as hearing loss, and potential difficulties in learning and communication (5, 6).

Ambient air pollution

AAP has been strongly linked to otitis media in children. A review (2) of five cross-sectional, two time-series and three cohort studies found a higher prevalence of otitis media in children living in areas with high levels of AAP (7–10). Traffic-related air pollution was associated with risk for otitis media (2) and with higher risks for ear, nose and throat infections (11). In a study of over 7000 children in Germany (12), the prevalence of otitis media decreased over a 7-year period in areas in which air quality improved. Strong conclusions could not be drawn from the review because of the limited number of studies.

A systematic review of 24 studies (1) found limited but increasing evidence of a link between exposure to AAP and otitis media in children. All the studies found evidence of a positive association with AAP, but the results were inconsistent for most pollutants, except NO₂.

Other studies have reported a higher incidence of otitis media among children exposed to air pollutants, especially NO₂ and PM_{2.5}. Brauer et al. (13) observed an association between exposure to traffic-related air pollutants (NO₂, PM_{2.5} and elemental carbon) and the incidence of otitis media in the first 2 years of life in two large birth cohorts in Germany and the Netherlands. Zemak and colleagues (3) analysed emergency department visits by children aged 1–3 years for otitis media over 10 years in Canada and found an association with exposure to CO and NO₂. In another study in Canada (14), 42,413 children born in British Columbia were followed until 2 years of age. The authors found that the average levels of exposure to pollutants (NO, CO, PM_{2.5} and wood smoke) in their residence 2 months before hospital visits were associated with the occurrence of otitis media. In a study in Spain, Aguilera et al. (15) reported a significant association between otitis media in early childhood and exposure to NO₂ and benzene during pregnancy. A meta-analysis of 10 European birth cohort studies of the effects of traffic-related air pollution (NO₂, NO_x, PM_{2.5}, PM₁₀ and PM_{2.5-10}) on otitis media (16) showed an association with the annual average ambient NO₂ concentration during the first year of life.

Time-series and case-crossover studies have also reported positive associations between exposure to AAP and visits to an emergency department for otitis media. In an analysis of 4815 such visits by children aged < 3 years in Ontario, Canada (17), the number of visits increased in the days after an increase in the ambient levels of O₃ and PM. A study of 422 268 emergency department visits for otitis media between 2002 and 2008 in Georgia, USA, found associations with exposure to CO, NO₂, O₃, PM₁₀, PM_{2.5}, element carbon, organic carbon ammonium and SO₄²⁻ (18).

The evidence indicates a consistent association between exposure to air pollution and otitis media.

Household air pollution

An association between parental tobacco smoking and otitis media in young children has been well documented (4, 19–21); however, few studies have been conducted on the associations with other sources of HAP, particularly in low-income countries (20).

A systematic review of risk factors for chronic and recurrent otitis media (20) identified only one study on HAP (22), in which indoor cooking was associated with chronic suppurative otitis media. Older studies in high-income countries gave inconsistent results. In a case–control study of 125 otitis media patients and 237 controls in a private paediatric practice in New York, USA, between October 1986 and May 1987, exposure to a wood-burning stove was associated with otitis media (23). A study of more than 900 infants in two states in the USA (24) found no significant association between otitis media and secondary heating sources (fireplace, wood stove, kerosene heater and air conditioning).

Among the few studies from LMICs is a case–control study in Maputo, Mozambique (25), which showed an association between use of wood and charcoal as household fuels and the occurrence of otitis media. A study of 189 children living in urban areas in two Nigerian states (26) indicated that indoor cooking was significantly associated with the occurrence of chronic suppurative otitis media; however, the type of fuel used was not specified.

Multiple sources of exposure

It is important to consider the timing of exposure to household and ambient air pollutants. Deng et al. (26) conducted a retrospective cohort study of 1617 children aged 3–4 years in Changsha, China. The lifetime prevalence of otitis media in preschool children was associated with prenatal exposure to an industrial air pollutant (SO₂) and postnatal exposure to indoor renovations. Both AAP from industrial activities and HAP from renovations were associated with development of early childhood ear infection.

Biological mechanisms

The biological pathways through which air pollution contributes to otitis media in children are not clear. Epithelial cells of the middle ear had significantly altered gene expression in response to exposure to PM (27), and the authors noted that some of the genes affected are involved in cellular processes, including generation of reactive oxygen species, apoptosis, cell proliferation, cell differentiation and inflammatory response. These may therefore be triggered by exposure to PM.

In another study, increased mucin gene expression (which can contribute to chronic infection), decreased cell viability and an increased inflammatory response were observed as a result of exposure to diesel exhaust particles (28). These findings were supported by the results of a study in experimental animals. Further research will help to confirm whether these processes play a role in the development of otitis media in children.

Conclusions

There is consistent evidence of an association between exposure to AAP and otitis media in children. The findings on the effects of individual pollutants are not consistent, and few studies of HAP are available.

Knowledge gaps and research needs

- Prospective observational epidemiological studies on the association between AAP exposure and otitis media occurrence should be undertaken.
- Evidence from studies of HAP is limited. As infants and children spend much of their time in the home, more studies should be conducted, with detailed measurements of HAP.
- Studies on the mechanisms by which air pollution contributes to the development of otitis media in children should be undertaken.

References – otitis media

1. Bowatte G, Tham R, Perret JL, Bloom MS, Dong G, Waidyatillake N, et al. Air pollution and otitis media in children: a systematic review of literature. *Int J Environ Res Public Health*. 2018;15:257.
2. Heinrich J, Raghuyamshi VS. Air pollution and otitis media: a review of evidence from epidemiologic studies. *Curr Allergy Asthma Rep*. 2004;4(4):302–9.
3. Zemek R, Szyszkowicz M, Rowe BH. Air pollution and emergency department visits for otitis media: a case-crossover study in Edmonton, Canada. *Environ Health Perspect*. 2010;118(11):1631.
4. The health consequences of involuntary smoking. Washington DC: Department of Health and Human Services; 1986.
5. Qureish A, Lee Y, Belfield K, Birchall JP, Daniel M. Update on otitis media – prevention and treatment. *Infect Drug Resist*. 2014;7:15–24.
6. Williams CJ, Jacobs AM. The impact of otitis media on cognitive and educational outcomes. *Med J Aust*. 2009;191(9 Suppl):S69–72.
7. Dostal M, Hertz-Picciotto I, James R, Keller J, Dejmeck J, Selevan S, et al. Childhood morbidity and air pollution in the Teplice program. *Cas Lek Ces*. 2001;140(21):658–61.
8. Cáceres Udina M, Alvarez Martínez JA, Argente del Castillo J, Chumilla Valderas MA, Fernández Alvarez E, Garrido Romera A, et al. Incidencia, contaminación ambiental y factores de riesgo de otitis

- media aguda en el primer año de vida: estudio prospectivo [Incidence, air pollution and risk factors of acute otitis media in the first year of life: a prospective study]. *An Pediatr (Barc)*. 2004;60(2):113–205.
9. Holtby I, Elliott K, Kumar U. Is there a relationship between proximity to industry and the occurrence of otitis media with effusion in school entrant children? *Public Health*. 1997;111(2):89–91.
 10. Ribeiro H, Cardoso MRA. Air pollution and children's health in São Paulo (1986–1998). *Soc Sci Med*. 2003;57(11):2013–22.
 11. Brauer M, Hoek G, Van Vliet P, Meliefste K, Fischer PH, Wijga A, et al., Air pollution from traffic and the development of respiratory infections and asthmatic and allergic symptoms in children. *Am J Resp Crit Care Med*. 2002;166(8):1092–8.
 12. Heinrich J, Hoelscher B, Frye C, Meyer I, Pitz M, Cyrus J, et al. Improved air quality in reunified Germany and decreases in respiratory symptoms. *Epidemiology*. 2002;13(4):394–401.
 13. Brauer M, Gehring U, Brunekreef B, de Jongste J, Gerritsen J, Rovers M, et al. Traffic-related air pollution and otitis media. *Environ Health Perspect*. 2006;114(9):1414.
 14. MacIntyre EA, Karr CJ, Koehoorn M, Demers PA, Tamburic L, Lencar C, et al., Residential air pollution and otitis media during the first two years of life. *Epidemiology*. 2011;22(1):81–9.
 15. Aguilera I, Pedersen M, Garcia-Esteban R, Ballester F, Basterrechea M, Esplugues A, et al. Early-life exposure to outdoor air pollution and respiratory health, ear infections, and eczema in infants from the INMA study. *Environ Health Perspect*. 2013;121(3):387.
 16. MacIntyre EA, Gehring U, Mölter A, Fuertes E, Klümper C, Krämer U, et al. Air pollution and respiratory infections during early childhood: an analysis of 10 European birth cohorts within the ESCAPE Project. *Environ Health Perspect*. 2014;122(1):107.
 17. Kousha T, Castner J. The air quality health index and emergency department visits for otitis media. *J Nurs Scholarsh*. 2016;48(2):163–71.
 18. Xiao Q, Liu Y, Mulholland JA, Russell AG, Darrow LA, Tolbert PE, et al. Pediatric emergency department visits and ambient air pollution in the US state of Georgia: a case-crossover study. *Environ Health*. 2016;15(1):115.
 19. Jones LL, Hassanién A, Cook DG, Britton J, Leonardi-Bee J. Parental smoking and the risk of middle ear disease in children: a systematic review and meta-analysis. *Arch Pediatr Adolesc Med*. 2012;166:18–27.
 20. Zhang Y, Xu M, Zhang J, Zeng L, Wang Y, Zheng QY. Risk factors for chronic recurrent otitis media – a meta-analysis. *PLoS One*. 2014;9(1):e86397.
 21. Nandasena S, Wickremasinghe AR, Sathiakumar N. Indoor air pollution and respiratory health of children in the developing world. *World J Clin Pediatr*. 2013;2(2):6–15.
 22. Lasisi AO, Olaniyan FA, Muibi SA, Azeez IA, Abdulwasiiu KG, Lasisi TJ, et al. Clinical and demographic risk factors associated with chronic suppurative otitis media. *Int J Pediatr Otorhinolaryngol*. 2007;71(10):1549–54.
 23. Daigler GE, Markello SJ, Cummings KM. The effect of indoor air pollutants on otitis media and asthma in children. *Laryngoscope*. 1991;101(3):293–6.
 24. Pettigrew MM, Gent JF, Triche EW, Belanger KD, Bracken MB, Leaderer BP, et al., Infant otitis media and the use of secondary heating sources. *Epidemiology*. 2004;15(1):13–20.
 25. da Costa JL, Navarro A, Branco Neves J, Martin M. Household wood and charcoal smoke increases risk of otitis media in childhood in Maputo. *Int J Epidemiol*. 2004;33(3):573–8.
 26. Deng Q, Lu C, Jiang W, Zhao J, Deng L, Xiang Y. Association of outdoor air pollution and indoor renovation with early childhood ear infection in China. *Chemosphere*. 2017;169:288–96.
 27. Song JJ, Kwon JY, Park MK, Seo YR. Microarray analysis of gene expression alteration in human middle ear epithelial cells induced by micro particle. *Int J Pediatr Otorhinolaryngol*. 2013;77(10):1760–4.
 28. Park MK, Chae SW, Kim HB, Cho JG, Song JJ. Middle ear inflammation of rat induced by urban particles. *Int J Pediatr Otorhinolaryngol*. 2014;78:2193–7.

5.7 Cancer

Key findings:

- There is substantial evidence that exposure to traffic-related air pollution is associated with childhood leukaemia.
- Several studies have found associations between prenatal exposure to AAP and higher risks of retinoblastoma and leukaemia in children.
- Relatively few studies have been conducted on HAP and cancer risk in children. Nevertheless, HAP is strongly associated with several types of cancer in adults and commonly contains a variety of classified carcinogens.

The incidence of cancer in children is increasing, as shown by data from 68 countries and over 100 population-based registries published by the International Agency for Research on Cancer (IARC) (1). In the period 1990–2017, an average of 215 000 cases of cancers per year were diagnosed in

children under 15 years of age, and 85 000 new cases were diagnosed among those aged 15–19 years (1). In view of the lack of cancer registries in several low-income countries, however, these statistics may be underestimates of the actual incidence (2).

The most prevalent types of cancer are different in children and adults (3). Leukaemia and lymphoma are the most common in children, accounting for almost half of all childhood cancers, followed by central nervous system tumours and tumours originating in embryonic tissues, such as neuroblastoma, retinoblastoma and nephroblastoma. Children also develop carcinomas, but the incidence is low (2).

Ambient air pollution

Children are exposed to a wide range of cancer-causing pollutants in ambient air. Diesel exhaust, AAP and particulate matter have been classified by working groups convened by IARC as Group 1 carcinogens. Nitroarenes, which are derived from diesel engine emissions, have been classified as Group 2 carcinogens, and gasoline exhaust has been classified as a Group 2B carcinogen (possibly carcinogenic to humans) (4). Traffic exhaust also contains harmful contaminants, such as CO, PAHs, benzene, NO_x and PM (5). Benzene has been classified in Group 1 (carcinogenic to humans) (5, 6).

Leukaemia is the most frequent childhood cancer (2). Although the etiology of at least 90% of cases of leukaemia remains unknown in (7), many studies have shown that exposure to traffic-related pollution (including diesel and gasoline exhaust) is associated with childhood leukaemia (4, 8, 9). A meta-analysis indicated that the development of leukaemia in early childhood is associated with exposure to traffic during the postnatal period, with a risk increased by 1.5 times (9). In addition, exposure to PM₁₀ was independently associated with the risk for leukaemia. In a meta-analysis on the role of benzene in the pathogenesis of childhood leukaemia, traffic-related exposure to benzene increased the risk for acute myeloid leukaemia by a factor of 2.07 and the risk for acute lymphoblastic leukaemia by 1.49 (10).

The relation between proximity to highways, urban AAP and childhood cancer has also been assessed. A study in a nationwide cohort in Switzerland found that the risk for leukaemia of children who lived < 100 M from a highway was 1.43 times greater than that of children who lived > 500 M away, especially for those < 5 years of age (11). Box 14 illustrates differences in childhood cancer risk according to residence.

Box 14. Location matters: variations in air pollution and childhood cancer risk in a city in Turkey

A study in Turkey was conducted to assess the relation between exposure to benzene, toluene, ethyl benzene, xylenes, NO₂ and O₃ and childhood cancer risk in two areas of the city of Eskisehir (12). Students at two schools participated: one in an urban area known to have high levels of air pollutants and one in a suburban location with lower levels of pollution. Benzene, toluene, ethyl benzene and xylenes are volatile organic compounds considered to be hazardous air pollutants; benzene is a Group 1 human carcinogen (5).

Personal air sampling and indoor (school and home) and outdoor air sampling was conducted over a 24-h period. Children who lived in smoking and in non-smoking homes were identified. An activity diary was given to each child, with a questionnaire on socioeconomic status, family activities and house characteristics (e.g. floor type, renovations). Potential sources of pollutants at residences and schools were identified from a checklist. Personal, exposure to indoor and outdoor concentrations of all air pollutants except O₃ was higher for children in the urban school than at the suburban site. Personal concentrations were also strongly correlated with indoor concentrations (except for O₃). The responses to the questionnaire indicated that interactions with tobacco smoke, solvent-based products and proximity to petrol stations increased exposure to pollutants. The authors found a higher risk of cancer in the urban school group, particularly for children whose parents smoked, than in children in the suburban location.

The risk assessment in this study focused on chronic exposure to pollutants rather than acute toxic effects. The findings show that levels of pollution can differ significantly in different parts of a city, as can the health effects of ambient and indoor exposure on children in different areas.

A number of studies on prenatal exposure to air pollutants indicate associations with cancer. A study in California, USA, of more than 3000 children with various types of cancer (13) found a clear relation between exposure to traffic pollution during gestation and the first year of life and the risk of cancer by the age of 6 years, not only for acute lymphoblastic leukaemia but also for germ-cell

tumours and retinoblastoma. A study in Texas, USA, indicated an increased risk of embryonal tumours in children whose mothers lived < 500 M from a major roadway during pregnancy (14). The strongest association was found with retinoblastoma, the risk for which was increased 2.57 times. In a study of more than two million children followed-up from birth to 4 years of age in Canada (15), prenatal exposure to AAP, particularly during the first trimester of pregnancy, was associated with increased risks of astrocytoma and acute lymphatic leukaemia. In another study in California, USA (16), each 25 parts per billion increase in average maternal exposure to NO_x during pregnancy increased the risk for leukaemia in their offspring by 23%. Bilateral retinoblastoma was associated with exposure to NO_x during the second and third trimesters of pregnancy. Exposure to PAHs during pregnancy was associated with a 1.44-times increase in risk of medulloblastoma in early childhood (17).

The exposures of both parents must be taken into account in assessing the risk of childhood cancer, in addition to exposure in utero (4). In a case–control study in Australia (18), both maternal exposure during pregnancy and paternal pre-birth occupational exposure to diesel and petrol exhaust were associated with an increased risk of acute childhood lymphoblastic leukaemia. A study in the United Kingdom found a small but statistically significant increased risk of leukaemia in children whose fathers were occupationally exposed to vehicle exhaust fumes and particulate hydrocarbons around the time of conception (19).

Household air pollution

Emissions from household combustion of coal have been classified by IARC as a Group 1 carcinogen (20), emissions from household combustion of biomass fuel, in particular wood, are probably carcinogenic, and the combustion of wood and other biomass fuels can produce toxicants including CO, PAHs, aldehydes and free radicals that are classified as Group 2A carcinogens (probably carcinogenic to humans). Metal compounds present in solid fuel emissions, such as arsenic and nickel, have also been classified as Group 1 carcinogens (21). In some rural areas of China, up to the 60% of the population under 30 years of age are exposed to arsenic from household coal combustion, which may account for the higher incidence of cancer in these populations (22).

HAP has been strongly associated with several types of cancer in adults, including lung cancer, upper aerodigestive tract cancer, kidney and cervical cancer (20, 23–27), but few studies have been conducted on HAP and cancer risk in children. A study in Australia (28) found increased risks of childhood leukaemia by 1.41 times in association with use of a wood burner to heat the home during pregnancy and by 1.25 times when used after birth. A case–control study in California, USA, provided evidence of an association between lung cancer and exposure to coal-burning during childhood and adolescence (29). Given the susceptibility of children, the known cancer risks of adults and the longer time available for cancer to develop in children, further research should be conducted on the risk of cancer associated with exposure to HAP during childhood.

Biological mechanisms

The pathogenesis of childhood cancer is complex, as it involves many genetic and environmental factors. In most cases, the primary causes remain unknown, although most of the scientific literature suggests that the immune system plays a role. A prominent hypothesis is that faulty functioning of the immune system in response to infections and allergies is the primary cause (8), and this is supported by some studies that suggest that environmental exposure to certain chemicals and pollutants that are known to alter the immune system can lead to this aberrant response and, therefore, to the outcome of leukaemia (30).

It has also been proposed that air pollutants contribute to carcinogenesis by damaging DNA. Particulate matter contains several genotoxic and mutagenic chemicals that cause single-strand breaks, micronuclei, sister chromatid exchange and oxidative DNA damage mediated by reactive oxygen species (31). DNA adducts and micronuclei have been identified in the cord blood of women exposed to air pollution during pregnancy; these are important biomarkers of DNA damage that can result in mutations leading to cancer (32). Furthermore, several studies have found that certain genes in xenobiotics pathways (e.g. *CYP2E1*, *GSTM1*, *NQO1*, *NAT2* and *MDR1*) increase the risk of leukaemia by themselves or in association with exposure to chemicals (33).

Conclusion

The rising rate of cancer in children worldwide is deeply concerning. There is ample evidence that both prenatal and childhood exposure to AAP is associated with increased risk of leukaemia and other cancers. There is robust evidence of an increased risk for cancer in adults exposed to HAP, but few studies have examined the association between HAP and childhood cancers.

Knowledge gaps and research needs

- Few studies have been conducted on the association between exposure to HAP from polluting fuels and childhood cancers.

References – cancer

1. Steliarova-Foucher ECM, Ries LAG, Hesselning P, Moreno F, Shin HY, Stiller CA. Lyon: International incidence of childhood cancer. Vol. III. Lyon: International Agency for Research on Cancer; 2017.
2. IICC-3, International incidence of childhood cancer. Vol. 3, Results, introduction. Lyon: International Agency for Research on Cancer; 2016 (<http://iicc.iarc.fr/results/>, accessed August 2018).
3. International Childhood Cancer Day: questions & answers. Geneva: World Health Organization; 2016 (http://www.who.int/cancer/media/news/Childhood_cancer_day/en/, accessed 26/3/2018).
4. Diesel and gasoline engine exhausts and some nitroarenes. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 105. Lyon: International Agency for Research on Cancer; 2014.
5. Benzene. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 100F. Lyon: International Agency for Research on Cancer; 2012.
6. Benzene. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 120. Lyon: International Agency for Research on Cancer; 2018.
7. Reşitoğlu İA, Altinişik K, Keskin A. The pollutant emissions from diesel-engine vehicles and exhaust aftertreatment systems. *Clean Technol Environ Policy*. 2015;17:15–27.
8. Metayer C, Dahl G, Wiemels J, Miller M. Childhood leukemia: a preventable disease. *Pediatrics*. 2016;138:S45–55.
9. Boothe VL, Boehmer TK, Wendel AM, Yip FY. Residential traffic exposure and childhood leukemia: a systematic review and meta-analysis. *Am J Prev Med*. 2014;46:413–22.
10. Carlos-Wallace FM, Zhang L, Smith MT, Rader G, Steinmaus C. Parental, in utero, and early-life exposure to benzene and the risk of childhood leukemia: a meta-analysis. *Am J Epidemiol*. 2016;183:1–14.
11. Spycher BD, Feller M, Rööslı M, Ammann RA, Diezi M, Egger M, et al. Childhood cancer and residential exposure to highways: a nationwide cohort study. *Eur J Epidemiol*. 2015;30:1263–75.
12. Demirel G, Ozden O, Döğeroğlu Y, Gaga EO. Personal exposure of primary school children to BTEX, NO₂ and ozone in Eskişehir, Turkey: relationship with indoor/outdoor concentrations and risk assessment. *Sci Total Environ*. 2014;473(4):537–48.
13. Heck JE, Wu J, Lombardi C, Qiu J, Meyers TJ, Wilhelm M, et al. Childhood cancer and traffic-related air pollution exposure in pregnancy and early life. *Environ Health Perspect*. 2013;121:1385–91.
14. Kumar SV, Lupo PJ, Pompeii LA, Danysh HE. Maternal residential proximity to major roadways and pediatric embryonal tumors in offspring. *Int J Environ Res Public Health*. 2018;15:505.
15. Lavigne E, Bélair MA, Do MT, Stieb DM, Hystad P, van Donkelaar A, et al. Maternal exposure to ambient air pollution and risk of early childhood cancers: a population-based study in Ontario, Canada. *Environ Int*. 2017;100(3):139–47.
16. Ghosh JKC, Heck JE, Cockburn M, Su J, Jerrett M, Ritz B. Prenatal exposure to traffic-related air pollution and risk of early childhood cancers. *Am J Epidemiol*. 2013;178:1233–9.
17. von Ehrenstein OS, Heck JE, Park AS, Cockburn M, Escobedo L, Ritz B. In utero and early-life exposure to ambient air toxics and childhood brain tumors: a population-based case-control study in California, USA. *Environ Health Perspect*. 2016;124:1093–9.
18. Reid A, Glass DC, Bailey HD, Milne E, Armstrong BK, Alvaro F, et al. Parental occupational exposure to exhausts, solvents, glues and paints, and risk of childhood leukemia. *Cancer Causes Control*. 2011;22:1575.
19. McKinney PA, Fear NT, Stockton D. Parental occupation at periconception: findings from the United Kingdom Childhood Cancer Study. *Occup Environ Med*. 2003;60:901–9.
20. Household use of solid fuels and high-temperature frying. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 95. Lyon: International Agency for Research on Cancer; 2010.
21. Gordon SB, Bruce NG, Grigg J, Hibberd PL, Kurmi OP, Lam KH, et al. Respiratory risks from household air pollution in low and middle income countries. *Lancet Respir Med*. 2014;2:823–60.

22. Millman A, Tang D, Perera FP. Air pollution threatens the health of children in China. *Pediatrics*. 2008;122:620–8.
23. Bruce N, Perez-Padilla R, Albalak R. Indoor air pollution in developing countries: a major environmental and public health challenge. *Bull World Health Organ*. 2000;78(9):1078–92.
24. Kleinerman R, Wang Z, Wang L, Metayer C, Zhang S, Brenner AV, et al. Lung cancer and indoor exposure to coal and biomass in rural China. *J Occup Environ Med*. 2002;44:338–44.
25. Lissowska J, Bardin-Mikolajczak A, Fletcher T, Zaridze D, Szeszenia-Dabrowska N, Rudnai P. Lung cancer and indoor pollution from heating and cooking with solid fuels: the IARC international multicentre case-control study in Eastern/Central Europe and the United Kingdom. *Am J Epidemiol*. 2005;162(4):326–33.
26. Hosgood HD 3rd, Wei H, Sapkota A, Choudhury I, Bruce N, Smith KR, et al. Household coal use and lung cancer: systematic review and meta-analysis of case-control studies, with an emphasis on geographic variation. *Int J Epidemiol*. 2011;40(3):719–28.
27. Duan X, Zhang J, Adair-Rohani H, Bruce N, Solomon H, Smith KR. Indoor air quality guidelines: household fuel combustion. Review 8: Household coal combustion: unique features of exposure to intrinsic toxicants and health effects. Geneva: World Health Organization; 2014.
28. Bailey HD, de Klerk NH, Fritschi L, Attia J, Daubenton JD, Armstrong BK, et al. Refuelling of vehicles, the use of wood burners and the risk of acute lymphoblastic leukaemia in childhood. *Paediatr Perinat Epidemiol*. 2011;25:528–39.
29. Wu AH, Henderson BE, Pike MC, Yu MC. Smoking and other risk factors for lung cancer in women. *J Natl Cancer Inst*. 1985;74:747–51.
30. Wiemels J. Perspectives on the causes of childhood leukemia. *Chem Biol Interact*. 2012;196:59–67.
31. Valavanidis A, Fiotakis K, Vlachogianni T. Airborne particulate matter and human health: toxicological assessment and importance of size and composition of particles for oxidative damage and carcinogenic mechanisms. *J Environ Sci Health C*. 2008;26(4):339–62.
32. Pedersen M, Wichmann J, Autrup H, Dang DA, Decordier I, Hvidberg M, et al. Increased micronuclei and bulky DNA adducts in cord blood after maternal exposures to traffic-related air pollution. *Environ Res*. 2009;109(8):1012–20.
33. Brisson GD, Alves LR, Pombo-de-Oliveira MS. Genetic susceptibility in childhood acute leukaemias: a systematic review. *ecancermedsci*. 2015;9. <https://doi.org/10.3332/ecancer.2015.539>.

5.8 Later health outcomes

Key findings:

- Exposure to air pollution early in life can impair lung development, reduce lung function and raise the risk of chronic lung disease in adulthood.
- Evidence suggests that exposure to air pollution during pregnancy can predispose the offspring to cardiovascular disease later in life.

The life course of children can be significantly affected by exposure to toxic air pollutants. Children who are exposed to air pollution during the prenatal period and early life are more likely to experience adverse health outcomes as they mature and throughout adulthood. Exposure not only has a direct impact on children’s health and development but can also stimulate latent diseases to become evident only in later life. Air pollution can contribute to effects on all of the organs and systems of the human body. Children’s physiological vulnerability and susceptibility to pollutants and the delayed emergence of certain adverse effects are an area of growing scientific interest. Several recent studies have addressed these factors and associated diseases.

Impairment of lung growth and development in childhood is an important risk factor for chronic lung disease in adulthood. A study of two European cohorts (n=12 862, age 28–73 years) (1) showed that exposure in early life was significantly associated with decreased FEV1 in adulthood, and the estimates were almost as large as those for personal smoking. In a study of a Swedish cohort, 2278 children were followed up (2). It was found that exposure to traffic-related air pollution in infancy is associated with a lower FEV1 at the age of 16 years. Factors in early life predicted decreased lung function decades later, suggesting that some mechanisms related to lung ageing may be established in childhood or in utero.

One of the common outcomes of impaired lung function is chronic obstructive pulmonary disease, and the link between HAP and development of this disease has been addressed. A meta-analysis

found that people with long-term exposure to HAP from solid fuel combustion had twice the risk of chronic obstructive pulmonary disease (3). Exposure to AAP and HAP also plays a role in the development of lung cancer, as emissions from the combustion of solid fuels in the home and traffic-related pollution both contain well-known carcinogens (4).

Exposure to high levels of air pollutants during pregnancy can predispose to cardiovascular diseases later in life. Exposure to pollution as the fetal organs develop during pregnancy can trigger susceptibility to weight gain and neuroinflammation in adulthood (5, 6). Early exposure to air pollutants has also been associated with early cardiovascular phenotypes in young adults. Zhang et al. (7) reported an association between higher exposure to PM_{2.5} during the third trimester of pregnancy and high blood pressure in children at 3–9 years of age. Breton et al. (8) found an association between prenatal exposure to ambient pollutants (PM₁₀, PM_{2.5}) and higher carotid arterial stiffness, a biomarker of endothelial function, in a population of university students. Iannuzzi et al. (9) evaluated 52 Italian children and found that those who lived closer to a main road had higher carotid arterial stiffness than those living further away. Thiering et al. (10) concluded that traffic-related air pollution may increase the risk of insulin resistance. In all these studies, confounding variables such as economic status, exposure to environmental tobacco smoke, onset of puberty and height and weight were accounted for.

Honda et al. (11) reported a probable relation between early exposure to air pollutants and development of anaemia later in life. This disorder is highly prevalent in elderly populations and is associated with numerous adverse health outcomes.

Conclusion

There is increasing suggestive evidence that exposure to air pollution early in life can influence the development of chronic lung disease, cardiovascular disease and other adverse health outcomes in adulthood. Early exposure can sow the seeds of serious long-term illness, in addition to heightening the risks of adverse outcomes in childhood. Thus, preventive measures to reduce exposure are likely to be extremely cost-effective in terms of reducing the overall burden of disease in populations.

References – later health outcomes

1. Dratva J, Zemp E, Dharmage SC, Accordini S, Burdet L, Gislason T, et al. Early life origins of lung ageing: early life exposures and lung function decline in adulthood in two European cohorts aged 28–73 years. *PLoS One*. 2016;11(1):e0145127.
2. Schultz ES, Hallberg J, Bellander Y, Bergström A, Bottai M, Chiesa F, et al. Early-life exposure to traffic-related air pollution and lung function in adolescence. *Am J Resp Crit Care Med*. 2015;193(2):171–7.
3. Gordon SB, Bruce NG, Grigg J, Hibberd PL, Kurmi OP, Lam KBH, et al. Respiratory risks from household air pollution in low and middle income countries. *Lancet Resp Med*. 2014;2(10):823–60.
4. Diesel and gasoline engine exhausts and some nitroarenes. IARC Monographs on the Evaluation of Carcinogenic Agents to Humans, Vol. 105. Lyon: International Agency for Research on Cancer; 2014.
5. Bolton JL, Smith SH, Huff NC, Gilmour IM, Foster WM, Auten RL, et al. Prenatal air pollution exposure induces neuroinflammation and predisposes offspring to weight gain in adulthood in a sex-specific manner. *FASEB J*. 2012;26(11):4743–54.
6. Backes CH, Nelin T, Gorr MW, Wold LE. Early life exposure to air pollution: How bad is it? *Toxicol Lett*. 2013;216(1):47–53.
7. Zhang M, Mueller N, Wang H, Hong X, Appel H, Wang X. Maternal exposure to ambient particulate matter $\leq 2.5 \mu\text{m}$ during pregnancy and the risk for high blood pressure in childhood. *Hypertension*. 2018;72(1):194–201.
8. Breton CV, Mack WJ, Yao J, Berhane K, Amadeus M, Lurmann F, et al. Prenatal air pollution exposure and early cardiovascular phenotypes in young adults. *PLoS One*. 2016;11(3): e0150825.
9. Iannuzzi A, Verga MC, Renis M, Schiavo A, Salvatore V, Santoriello C, et al. Air pollution and carotid arterial stiffness in children. *Cardiol Young*. 2010;20(2):186–90.
10. Thiering E, Cyrys J, Kratzsch J, Meisinger C, Hoffmann B, Berdel D, et al. Long-term exposure to traffic-related air pollution and insulin resistance in children: results from the GINIplus and LISAplus birth cohorts. *Diabetologia*. 2013;56(8):1696–704.
11. Honda T, Pun VC, Manjourides J, Suh H. Anemia prevalence and hemoglobin levels are associated with long-term exposure to air pollution in an older population. *Environ Int*. 2017;101(4):125–32.

6. Recommended actions for health professionals

Air pollution is a global problem. Evidence of its negative health effects – which may have both lifelong and generational impacts – is clear and compelling.

The developing fetus and child are particularly vulnerable to the effects of air pollution and are at risk of both short- and long-term health outcomes. As summarized above, numerous studies have linked air pollution to adverse birth outcomes, infant mortality, neurodevelopmental disorders, childhood obesity, compromised lung function, pneumonia, asthma and otitis media, with associations of varying strength. In light of this evidence, major health professional organizations throughout the world are focusing increasingly on the adverse health impacts of air pollution on children. While further research is needed in a number of areas, the scientific evidence is already sufficient for taking clear, concrete steps now to reduce the exposure of pregnant women and children to air pollution.

Health professionals are trusted sources of information and guidance. Paediatricians, family doctors, gynaecologists, obstetricians, midwives, nurses and community health care workers who interact with children can all play significant roles in advocating for policies to reduce childhood exposure to air pollution. Health care professionals commonly treat the effects of exposure-related illness but rarely receive training in identifying and managing the underlying causes and are rarely involved in policy-making. Health professionals should expand their role in the management of childhood exposure to air pollution, with better methods of care and prevention and collective action.

The broader health sector must develop a comprehensive approach to this problem. Preventing the health impacts of air pollution on children requires action by both decision-makers and individual health care professionals, who are best positioned to educate both the public and policy-makers about the dangers of air pollution and to suggest the most promising solutions (Fig. 14).

Fig. 14. Critical roles of health professionals

- Be informed.
- Recognize exposure and the associated health conditions.
- Conduct research and publish and disseminate knowledge.
- Prescribe solutions and educate families and communities.
- Educate colleagues and students.
- Advocate to policy- and decision-makers.

6.1 Be informed.

Health professionals should be aware of the sources and patterns of air pollution in their communities and any tools that can be used to monitor air quality. Regulatory levels of air pollutants are established in almost all major cities. When these regulatory levels are exceeded, health professionals should be prepared and know what action to take to protect the health of their patients. All health professionals should understand the sources of environmental exposure in the communities they serve and should consider air pollution a major risk factor for their patients. They should remain aware of the existing and emerging evidence on the ways in which air pollution can affect children's health.

6.2 Recognize exposure and associated health conditions.

Health professionals are trained to prevent, detect, diagnose and treat health conditions. They also have an important role in identifying causative risk factors in order to prevent disease. Training in the prevention of early childhood exposure will reduce not only common childhood morbidity but also adult mortality. A health care provider can identify air pollution-related risk factors by asking pertinent questions about the child's or pregnant mother's environment. Primary health and community workers can take the opportunity to observe and assess exposure during home visits or when providing advice on infant feeding and during visits to schools and community centres.

Questions can be asked during a medical visit to evaluate the risk of exposure to hazardous air pollutants. Box 16 provides examples of questions that could be asked. For more specific guidance on evaluating environmental risks associated with air quality, see *Children's health and the environment: a global perspective (1)*. Primary health and community workers can take the opportunity to ask questions about the child's environment during Integrated Management of

Childhood Illnesses. Alternatively, a comprehensive environmental risk assessment can be conducted during consultations with pregnant women or children who present with air pollution-related health effects, to assess and understand their current exposure and prevent further exposure. A concise version of taking a paediatric environmental history has been prepared and has been field-tested in Argentina; it is available with guidance materials on the WHO website (<http://www.who.int/ceh/capacity/paedenvhistory/en/>). The more questions that are asked about the child's environment, the more valuable the information collected, as it allows health professionals to identify causative risk factors for acute, recurrent and chronic conditions and helps them educate families on preventing further exposure. Box 17 gives examples of questions that can be asked to determine the risk of AAP.

Box 16. Examples of clinical questions for determining household air pollution risk (2–4)

Cooking

1. What fuel does this household use for cooking (including cooking food, making tea/coffee and boiling drinking-water)? *Please circle all cookstoves or devices used. If any technologies are used that are associated with health risk, explain that these stoves produce high levels of pollution that is harmful to health.*

No cooking done in household.....	0	SKIP to Q.5
Electric stove.....	1	CLEAN FOR HEALTH SKIP to Q.5
Solar cooker.....	2	CLEAN FOR HEALTH SKIP to Q.5
Piped natural gas stove.....	3	CLEAN FOR HEALTH SKIP to Q.5
Biogas stove.....	4	CLEAN FOR HEALTH SKIP to Q.5
Liquefied petroleum gas (LPG)/ cooking gas stove.....	5	CLEAN FOR HEALTH SKIP to Q.5
Liquid fuel stove:		
... Using alcohol / ethanol.....	6	CLEAN FOR HEALTH SKIP to Q.5
... Using gasoline / diesel.....	7	HEALTH RISK
... Using kerosene/paraffin.....	8	HEALTH RISK
Manufactured/artisanal solid fuel stove that meets standards for “advanced” (ISO Tier 4 or 5).....	9	CLEAN FOR HEALTH SKIP to Q.5
Manufactured / artisanal solid fuel stove (ISO Tier 0–3).....	10	HEALTH RISK
Traditional solid fuel stove.....	11	HEALTH RISK
Three stone stove/open fire.....	12	HEALTH RISK

If the household uses polluting fuels or stoves for cooking (options with a HEALTH RISK), ask these follow-up questions:

2. Where is cooking usually done? *The exposure of the cook and others is greatest when cooking is done in the main house. Cooking with polluting fuels or stoves can release high concentrations of air pollution. Cooking outdoors or in areas with good ventilation may reduce exposure to air pollution.*

In main house: no separate room.....	1
In main house: separate room.....	2
Outside main house: in a separate room.....	3
Outside main house: in open air.....	4
On veranda or covered porch.....	5
3. Does the cookstove have a chimney or a hood? *If yes, this can reduce the air pollution from cooking or heating.*

Yes.....	1	CAN REDUCE EXPOSURE
No.....	2	
Don't know.....	3	
4. Does your child / do your children spend time around the cookstove or fire? *If yes, the child can be exposed to high levels of harmful air pollution. It is suggested that children minimize the time spent in areas where cooking is done if polluting stoves or fuels are used.*

Yes.....	1	HEALTH RISK
No.....	2	CAN REDUCE EXPOSURE
Don't know.....	3	

Space-heating and other energy uses

5. What fuel does this household use for heating? For example, do you use a space heater(s) or your cookstove for warmth? *Please circle all space-heating devices used. If any of them are associated with a health risk, explain that these devices produce high levels of pollution that is harmful to health.*
- No space heating in house..... 0
 Central heating..... 1 **CLEAN FOR HEALTH**
 Heat pump..... 2 **CLEAN FOR HEALTH**
 Manufactured space heater..... 3 **POSSIBLY CLEAN FOR HEALTH**
 Traditional space heater or cookstove..... 4 **HEALTH RISK**
 Open fire..... 5 **HEALTH RISK**
 Moveable heating pan..... 6 **HEALTH RISK**
 Three-stone stove or open fire..... 7 **HEALTH RISK**
6. Does your household burn wood, coal, charcoal, dung, kerosene or agricultural residues for cooking, heating, lighting or other purposes in or near the home? For example, do you use kerosene lamps, biomass to cook food for animals or burn crop residues to keep flies away from your animals?
If yes: explain that burning these fuels around the home releases high concentrations of air pollution that can be harmful to the health of children.

Box 17. Examples of clinical questions for determining risk of ambient air pollution (2,4–8)

1. Do you identify or perceive sources of smoke, fog or dust close to your household? Examples include fires from burning garbage or other residues, smoke, smog or dust from surrounding industrial or agricultural activities.

- usually see, smell, perceive smoke, dust or mist around the house..... **Health risk**
 .. burning areas from dumps and landfills..... **Health risk**
industrial or agricultural area **Can reduce exposure**
 Do not perceive smells, mist, smoke or dust around the house..... **Clean for health**

If any identified ambient air pollution is associated with a health risk, advocate for local monitoring and control and suggest that parents minimize the time their children spend outside while pollution is present.

2. Does your child/your children live or spend time in an area with heavy traffic or a traffic-congested area, such as a road with frequent blocked traffic, slower speeds and long queues?

- rural or urban area with light traffic..... **Clean for health**
 .. urban with heavy traffic and common traffic congestion.... **Health risk**

If traffic represents a health risk, advocate for local monitoring and control, and suggest that parents keep the windows closed and minimize the time their children spend outside while traffic is congested.

3. How often do lorries pass through the street where you live, on weekdays?

- Never.....
 Seldom.....
 Frequently.....
 Almost the entire day.....
 Any other response.....

4. What is the distance (in meters) from your house to the nearest busy road with frequent traffic?

5. For how many hours a day does your child play outdoors?

6.3 Conduct research and publish and disseminate knowledge.

Environmental health literacy is increasing rapidly. Health professionals have a wealth of knowledge available to them and are themselves trusted sources of synthesized knowledge. With growing interest in air pollution and its health effects, health professionals are in a unique position to identify causative risk factors, educate patients on prevention and advocate for protective interventions. As noted throughout this publication, many gaps in research remain. It is well established, however, that children are the most vulnerable to environmental exposure because they have a lifetime ahead of them for development of the associated illnesses. In order to better protect children from the consequences of air pollution, better understanding is needed of the different sources of pollution, how they enter the body and their biological effects. Health professionals must build collaborations,

work closely with affected communities and identify and evaluate potential interventions. The health sector is well positioned to take the lead in narrowing the knowledge gaps.

Health professionals play a critical role in advancing research on the effects of air pollution on children's health, as they observe the effects in their daily work. Both independently and in collaboration with researchers, health professionals can conduct and publish investigations of the causes, mechanisms and effects of environmental exposure that affects children, as well as potential treatment, prevention and management options. By publishing articles and submitting reports of unique cases, they can help other practitioners to identify signs of air pollution-related health outcomes and raise awareness of potential exposure pathways. Health professionals can also play a valuable role in recruiting patients for large studies, because of their relationships with patients. They also contribute to identification of public health concerns by reporting sentinel cases and clusters of air pollution-related diseases to government authorities and can assist in monitoring and identifying sources of pollution. Health professionals are encouraged to consider interventions that may improve the lives of children exposed to air pollution and design pilot studies to determine their effectiveness and efficiency in use of resources. It is also important that they use this evidence to inform social and behaviour change communication strategies for public health promotion and prevention of exposure of children to air pollution. Box 18 lists the priorities for research on air pollution and effects on health.

Box 18. Research priorities

- Studies of the efficacy of personal protective devices (e.g. facemasks) have shown mixed results. Further studies are required.
- Research on the effect of HAP on children's health is limited. Few sources have been investigated, and the available evidence does not provide a detailed assessment of health outcomes. The types of pollutants and their effects both prenatally and during childhood periods should be evaluated.
- Not only epidemiological studies but also large intervention studies and implementation research are required to assess the efficacy of interventions and their potential deployment on a larger scale.
- Although many studies have evaluated the effects of chemical mixtures in air pollution on children, investigations of associations between chemical components and health effects will clarify which pollutants are most dangerous and how they should be regulated.
- Long-term studies of the effects of air pollution on children over time are necessary to determine the lasting effects of exposure. Children are vulnerable to environmental exposures partly because they have a lifetime to develop illness. More research on health status in adulthood after childhood exposure to air pollutants will indicate whether there is a link with chronic illness.
- There are few studies of interventions. As more becomes known about the effects of air pollution on children's health, studies of protective policies and patient treatments and interventions will be critical.
- There is increasing recognition that exposure to air pollution in early life can cause epigenetic changes. More research is needed on the long-term consequences of such changes and their role in the biological mechanisms for a wide range of health outcomes.

6.4 Prescribe solutions and educate families and communities.

Health professionals can “prescribe” solutions to problems related to air pollution, such as switching to clean household fuels and devices to reduce exposure (Boxes 19 and 20). When it is difficult to change to clean household energy, health care professionals can recommend “transitional” solutions that offer some health benefit. Information could be collected on the availability, accessibility and affordability of clean household energy alternatives and on the obstacles and also on resources and information available in government and other programmes to help reduce exposure. Education and individual protective measures, such as using clean stoves for cooking and clean-burning space heaters, could mitigate HAP, often improving the health of the whole family. Reducing AAP, however, requires action throughout the community: individual protective measures at family and household level are important but often not enough. As entire communities are affected by AAP – which in turn is determined by regional sources and meteorological patterns – policy interventions are necessary. (See Boxes 20 and 21 for examples.)

Box 19. Advice that clinicians could give to patients.

Note: These are not official guidelines but suggested actions that patients can take to improve the quality of the air and health and safety in their home environments.

- Use only clean household energy for cooking, heating and lighting.
- Use the cleanest possible solutions. As the transition to truly clean energy sources can take time, technologies and fuels that reduce exposure the most should be used (e.g. low-emission biomass cookstoves).
- To reduce the exposure of children to hazardous HAP, minimize the time children spend around smoky fires and kerosene lamps.
- Increasing ventilation by opening windows or doors or installing a chimney with regular maintenance can reduce exposure in the indoor environment.
- To reduce the risk of burns and scalds, ensure that technologies and fuels are used in such a way that it is unlikely that they can be pushed over, dropped, handled or touched by children.
- To minimize the risk of poisoning, do not store liquid fuels in water bottles or similar beverage containers, and keep them out of the reach of children.
- Ensure that stove and fuel combinations have appropriate safety controls and mechanisms (e.g. safety valves) and are regularly maintained.
- Avoid tobacco smoking indoors.
- To reduce exposure during acute episodes of AAP, minimize children's outdoor physical exertion and the time they spend outdoors, especially in areas with heavy traffic. During these events, families should seek advice from a medical doctor before letting their children participate in outdoor sports and other physical activity.

Box 20. Messages for families and communities.

Ambient air pollution

AAP is a risk factor for respiratory diseases in children, including reduced lung function, exacerbated respiratory symptoms and increased severity or frequency of asthma attacks.

For families:

- Respect advisories on local air pollution. In some cities, air pollution can be so severe that people may be advised to limit their activities and mobility, and schools might close. In many countries and regions, advisories are becoming more interactive, with display boards in some locations that show the current level of air pollution or indicate local air quality. Increasingly, mobile phones apps are used to forecast local air quality.
- Be aware of the environment. Families can identify signs and symptoms in their child that may be associated with local air pollutants, bring them to the attention of their health provider and promote investigation.
- Work with the community. Families are encouraged to collaborate with other community members, health providers and the government to identify air pollution in their area, take action to protect children from exposure and contribute to policy-making.

For communities:

- Be aware of the effects that local activities and natural events have on air quality and the potential impact on children's health. Rural areas are affected by outdoor air pollution primarily from burning debris on agricultural land and forest fires. With increasing desertification, dust storms may contribute substantially to outdoor air pollution. Air pollution is influenced by regional wind and weather conditions and may be transported over long distances.
- Take action to improve air quality whenever possible. Reduced exposure decreases the risk of health effects. Communities should reduce health risks by identifying and reducing emissions from local sources of pollution. For example, in urban areas, a significant proportion of air pollution is generated by old vehicles, poor vehicle maintenance and low fuel quality. Communities may advocate to phase out these vehicles.
- Become aware of new measures to reduce air pollution. Cost-effective strategies to reduce pollution include better integration of transport and land use (e.g. high-capacity, dedicated busways and pedestrian and cycle networks), use of cleaner (lead-free, low-sulfur) fuels, cleaner vehicle standards and technologies, monitoring of air quality and warnings.

- Actions to reduce air pollution will benefit child health, not only by avoiding direct effects but also by reducing emissions of certain greenhouse gases and thus mitigating climate change and its effects on health (5).

Household air pollution from use of polluting fuels

Use of polluting fuels for cooking and heating poses a serious threat to children's health. HAP is associated with adverse birth outcomes, increased infant mortality, deficits in childhood lung function, asthma and increased risks for lung infection.

For families:

- Switching from wood, dung, coal, charcoal or kerosene to more efficient, less polluting fuels like electricity, solar energy, LPG, biogas and ethanol will reduce exposure to harmful pollutants.
- Kerosene is not a clean fuel. Avoiding use of kerosene for cooking will also prevent burns and poisoning.
- Proper ventilation during cooking and heating may be a partial remedy. Installing eaves spaces and extraction through smoke hoods and opening windows and doors can also reduce indoor air pollution.
- Changing behaviour plays an important role. Newborns and infants are often carried on their mothers' backs while they are cooking or kept close to a warm hearth. Consequently, they spend many hours breathing polluted air during their first years of life when their developing airways and their immature immune systems make them particularly vulnerable. Pregnant women should also keep a distance from such sources (9).
- Quit tobacco smoking or at least avoid smoking in the house (10).

For communities:

- Resources and information on relevant government and other programmes to reduce exposure in the home should be easily accessible. Such programmes are often inexpensive and highly effective.
- Ensure that advice and support to family members to stop tobacco smoking are available to families.

Box 21. Children's environmental health units – a resource for health professionals, communities and families.

“Children's environmental health units” are now established around the world to ensure the care and protection of children exposed to environmental factors that may adversely affect their health. In view of the particular vulnerability of children, the units are accumulating information on the dangers of air pollution and other hazards, responding to public concern, training health professionals and educating communities, governments and other sectors. They are dedicated to the protection of children from environmental threats, management of children with known or suspected exposure to environmental hazards and diagnosis, management and treatment of children with illnesses due to such exposure.

These units, also referred to as “paediatric environmental health units” have been set up in many countries since they were initiated by WHO (11). For example, a unit has been working in Uruguay for more than a decade, and a network of paediatric environmental health specialty units (<https://www.pehsu.net/>) are located in Canada and the USA to promote awareness of environmental health issues, provide advice and guidance to reduce exposure, help families to seek care and aid in the training of health professionals. There are also paediatricians who run activities on children's environmental health in medical centres, although there are still no national policies to support such centres.

If you suspect that your child has symptoms related to environmental exposure or you are concerned about a potential exposure and would like to discuss it with a professional, find a children's environmental health unit near you and talk to your health care provider about possible treatment. If no such unit exists, advocate for one to be created. Units exist in many places, and their creation does not require extensive resources.

6.5 Educate colleagues and students.

By training others in health and education, health professionals can increase the reach of their messages on the health risks of air pollution and on strategies to reduce exposure. Health professionals can:

- educate and engage their colleagues in the workplace, in local health care centres, at conferences and in health professional associations;
- advocate for the inclusion of children's environmental health and the environmental determinants of health in curricula in post-secondary institutions and particularly in medical, nursing and midwifery schools;

- engage student associations in the health and care professions (e.g. medicine and nursing); and
- promote reduction of air pollution in schools and environmental health education for teachers and students.

Exposure to air pollution and the interventions to counteract it depend on the country of residence, urban or rural location, socioeconomic situation and other factors. WHO training materials are available that can be adapted to the location, situation and audience (see Box 22).

Box 22. Training materials for health professionals

To enable health professionals who care for children’s and adolescents’ health to recognize, assess and then manage and prevent diseases linked to environmental factors, WHO and experienced professionals have prepared a training package on children’s environmental health, including “train the trainer” materials and tools (12). WHO promotes its wide use for training.

6.6 Advocate to policy- and decision-makers.

Health professionals around the world recognize that air pollution is a threat to the healthy development of children. They should share their knowledge with decision-makers, including local governments, school boards and community leaders. Health professionals can accurately convey the health burden of air pollution to decision-makers, conduct health assessments, support improved standards and policies to reduce harmful exposures, advocate for monitoring and emphasize the

Health professionals around the world recognize that air pollution is a threat to the healthy development of children.

importance of protecting children at risk. The health sector is promoting this message, coordinating with other groups and aligning their work with government, academia and community to ensure that children are protected. Resources available to support outreach and coordination include the WHO training manual on

health-in-all policies (13) and the BreatheLife campaign (<http://breathelife2030.org/>). Health professionals can also advocate; however, by engaging more comprehensively with the broader health sector, they can extend and increase their influence. Promoting interventions and policies to decrease exposure to air pollution during a child’s early years will contribute to lifelong health and future well-being.

The heightened risks of children should lead health professionals to consider the “precautionary principle”: when there is a likelihood of serious or irreversible damage to health, a lack of full scientific certainty should not preclude the pursuit of effective preventive measures (14). The American Public Health Association (15) and WHO (16) have proposed approaches for using the precautionary principle to protect children from environmental risks such as air pollution. For some of the health outcomes discussed above, there is strong evidence of the effects of AAP on child health effects, but few studies of HAP. As AAP and HAP share many of the same types of combustion sources, minimizing children’s exposure to both forms of pollution, especially during the most sensitive developmental stages of early life, should take precedence over establishing near-certainty about the full extent of the risk and the mechanisms involved (17).

6.7 Benefits of cleaner air for health and the climate

HAP and AAP contribute significantly to global climate change. Household energy is a source of both CO₂ and short-lived climate pollutants such as methane, black carbon and volatile organic compounds. Short-lived climate pollutants are also emitted by diesel-fuelled vehicles and generators, open burning of agriculture waste and livestock production (18). Some of the products of incomplete combustion of biomass or fossil fuels contribute to the formation of O₃, another potent climate pollutant.

Black carbon and O₃ damage children’s health in both the short term, by direct exposure, and the longer term, by increasing food insecurity, extreme weather, water scarcity and infectious disease incidence brought on by global climate change (19, 20). As atmospheric temperatures increase, so will vector-borne disease, diarrhoeal diseases and undernutrition – some of the major killers of young

children. Concentrations of ground-level O₃, pollen, mould and other pollutants will increase as well, exacerbating respiratory illness in children, such as asthma and allergies (21).

The relation between air pollution and climate change is complex: AAP contributes to climate change, and climate change affects air quality. Rising temperatures can result in more frequent, more severe smog and higher annual mean concentrations of PM_{2.5} in certain parts of the world.

The interconnected effects of HAP, AAP and climate change strengthen the benefits of emissions reduction for health and the environment. Changing household energy use to clean options and reducing AAP are critical to both improving children's health and mitigating global climate change. Box 23 gives examples of policy measures for which health professionals could advocate, Box 24 gives an example of a policy that was successful in reducing AAP, and Box 25 describes ways in which households were convinced to change to cleaner fuels.

Box 23. Examples of policy measures for which health professionals could advocate (20)

Each of these measures can be promoted by stressing the health effects of air pollutants in children to policy-makers.

- Accelerate access to clean and efficient household energy for cooking, heating and lighting. Programmes to improve access to cleaner fuels and improved stoves can result in large gains in health and productivity and are highly cost-beneficial. Health professionals can promote national campaigns for improved fuels and technologies that adhere to the WHO guidelines for indoor air quality associated with household fuel combustion (22).
- Reduce the emissions of harmful ambient air pollutants from major sources such as heavy vehicles, through grants and low-cost loan programmes. For example, the Diesel Emissions Reduction Act in the USA provides funds to retrofit or replace diesel buses and other vehicles that emit high levels of NO_x, PM and other pollutants.
- Encourage land-use planning for energy-efficient, compact cities that shorten distances, encourage urban street life and create opportunities for children to play and interact with their community.
- Support investments in “green” and “blue” spaces, such as parks, forests and lakes, so that children can benefit from green areas and clean waters. These can also provide urban ecosystem services, including a cooling effect and a refuge from air and noise pollution.
- Transfer subsidies from polluting fuels such as kerosene to clean household options (e.g. solar, biogas, LPG).
- Design a labelling system for stoves and space heaters that includes rating of emissions (linked to health impacts) and efficiency, perhaps using the ISO standards for voluntary performance targets for clean cookstoves and clean cooking solutions (23).
- Encourage further installation of low-emission and renewable power generation to reduce AAP.
- Support public investment in rapid urban transit with dedicated rights of way and pedestrian and cycle networks. Support use of public transport, bicycles, walking and programmes such as safe routes to school and children's free access to public transport in order to increase children's independent mobility.
- Support the planning and building of energy-efficient housing, clustered in neighbourhoods with schools, shops and services nearby. Upgrade slums by introducing cleaner, safer street networks, larger green spaces and better infrastructure to improve children's physical living conditions and quality of life.
- Discourage urban planning that results in low-density urban sprawl and expanding roads, gated communities and large expanses of concrete, which absorb heat and block sunlight, all of which discourage children's involvement in urban street life.
- Advocate for better waste management practices to reduce incineration and burning of agricultural waste and for phasing out use of agro-chemicals in or near urban areas.
- Promote better land-use management in rural areas to stop deforestation, limit agricultural burning practices and charcoal production and improve control of wildfires.
- Create an emergency alert system that provides information on air pollution resulting from natural disasters such as thunderstorms and wildfires, and advocate for protective measures in such situations.

Box 24. A success story in improving air quality

This case study demonstrates what can be achieved with a coordinated, effective policy. Southern California used to be one of the most polluted regions in the USA. A persistent brown haze was a fact of life for residents of Los Angeles in the 1960s. Over the past few decades, however, California has cleaned up its outdoor air dramatically. The state treated its AAP problem as a public health crisis that demanded strong action. It introduced strict emission controls on almost every source of AAP, with low-emission vehicle programmes, emissions standards for heavy-duty vehicles and diesel vehicles, requirements for emissions reduction by power

plants and refineries and programmes to improve the energy efficiency of and reduce emissions from consumer products and appliances. Through the Clean Air Act and its amendments, the Federal Government also imposed more stringent air pollution standards and effective, concurrent policies to reduce emissions from vehicles and energy-related industries. California's ambitious efforts, however, went beyond the standards set by national legislation and had huge benefits for Californians, as the concentrations of several air pollutants were reduced by 15–65% (24). Children in California now breathe more easily, as the reduction in pollutant levels has resulted in significant improvements in the lung function (25). California lawmakers took action partly in response to strong evidence of the effects of air pollution on health, highlighting the important role of health professionals in fostering change.

Box 25. Smart subsidies: success in transitioning millions of households to clean fuels

The aim of “Cooking for life”, a project of the World LPG Association and the United Nations, is to convince households to change from polluting fuels for cooking primarily to LPG, a fuel that is much cleaner for health. Such a change requires access to affordable fuels and technologies and the financial means to obtain them, and these factors can be influenced by public policy (26).

Indonesia's successful programme for increasing LPG use offers good lessons. In 2006, 48 of 52 million households in Indonesia were using kerosene, a polluting fuel that is also a major risk of poisoning in children, as their main energy source. The Government subsidized the purchase of kerosene, at major expense to the State coffers. In 2007, the Government launched a national programme to switch more than 50 million people from kerosene to LPG. After an extended assessment involving authorities at all levels, conversion packages were distributed, accompanied by communication and education campaigns to increase awareness. Within the first 6 years, LPG packages had been distributed to 54 million households. A large proportion of households have since switched to this cleaner energy source, reducing the cost to the Government of petroleum-related subsidies by more than US\$ 6 billion. The programme resulted in a reduction in CO₂ emissions by 8.4 million tonnes per year and also reduced emissions of other air pollutants, such as PM, methane, CO and hydrocarbons. A survey after implementation of the policy showed that 99.8% of households preferred using LPG to kerosene, citing greater efficiency, speed of cooking and cleanliness (27).

Children's health improved dramatically: the infant mortality rate fell by 30% in the regions that received the intervention, half of the decrease being seen among infants in the early neonatal period. This carefully coordinated nationwide intervention is a compelling example for other countries that wish to accelerate the transition from household use of polluting fuels to benefit the health of their children (27–29).

Further evidence of the importance of targeted subsidies in promoting a transition to clean fuels was found in Latin America and the Caribbean. Use of LPG varies widely across the region, and over 80 million people still rely on polluting fuels as their primary source of household energy. Achieving SDG 7 (“ensure access to affordable, reliable, sustainable and modern energy for all”) would have huge benefits for health in the region, as it would avert an estimated 82 361 premature deaths and 2 327 146 DALYs annually (30). In Latin America and the Caribbean, access and price were identified as the main limitations to substitution of solid fuels by clean fuels. Subsidies for LPG consumption helped Bolivia to lower the proportion of the population that used polluting cooking fuels from 36% in 2005 to 23% in 2013. Other countries, such as Brazil, used cash transfer programmes, which accelerated the transition, from 16% to < 5% in a decade. It has been estimated that subsidies for natural and LPG gas allowed 39% of the rural population and almost the entire urban population to switch from polluting fuels to LPG. Targeted subsidies should therefore be considered a policy option for achieving the SDG7 on clean energy.

Effective programmes to promote adoption of clean fuels can result in meaningful health gains for children. Health professionals can play a role, by justifying the health benefits of such interventions to policy- and decision-makers, drawing on these success stories.

6.8 A perspective on children's health and air pollution: improving equity and access to protect the most vulnerable

While providing information to families and communities is important, it may not remove other barriers to access. The poorest communities often face the greatest environmental risks to their health, and their children are especially vulnerable. Poverty often underlies these disproportionate risks, as those living in poor communities may be unable to afford cleaner household fuels, access care or move to an area with cleaner air. They may also be members of marginalized groups that face systemic barriers to reducing exposure to air pollution. Polluting industries, waste disposal sites, bus depots and trucking routes are often situated in low-income communities, where residents lack the political power to limit or remove these harmful sources of exposure.

In households that rely on polluting fuels for cooking and heating, women and their children often travel long distances to collect fuel and spend much time gathering, processing and cooking with these fuels. Removing or reducing the primary sources of pollution is often the best way to protect children from the damage it can cause. Therefore, low-cost solutions and approaches are likely to result in the most progress in affected communities.

Low-income families are often constrained in their options to improve air quality in their own homes. Because of market and other forces beyond their control, clean fuels and technologies may not be affordable, available or accessible. Beyond the household, individuals and families have even less control over the air they breathe. To reduce and prevent exposure to both HAP and AAP, public policy is essential. Evidence-based air quality standards, monitoring pollutant levels over time and proper enforcement mechanisms can reduce exposure and save lives.

Air pollutants have no political borders; they travel where the wind and prevailing weather patterns take them. Therefore, regional and international cooperation are also necessary to reduce children's exposure. Like the pollutants, actions to protect children should transcend sectoral, geographical and political boundaries. What is needed are approaches to preventing exposure that are complementary and mutually reinforcing, at every scale: in the home, the clinic, the health care institution, the municipality, the national government and the global community. Collectively, health care professionals can push for strong action from decision-makers to protect their most vulnerable and voiceless citizens: children who have little or no control over the quality of the air that surrounds them. Exposure to air pollution can alter children's trajectory through life, pushing them onto a path with suffering, illness and challenge. In some cases, the damage is irreversible – but it is also preventable. The sources of risk and the health effects are diverse and complex, but the solution is clear: we must reduce children's exposure. By working together, we can protect millions of vulnerable young lives from the health effects of air pollution. The benefits would be immeasurable.

References – recommended actions for health professionals

1. Children's health and the environment: a global perspective, a resource manual for the health sector. Geneva: World Health Organization; 2005:232–4 (<http://www.who.int/ceh/publications/handbook/en/>, accessed September 2018).
2. ISAAC questionnaire. Auckland: The International Study of Asthma and Allergies in Childhood; 2012 (<http://isaac.auckland.ac.nz/resources/tools.php?menu=tools1>, accessed September 2018)
3. Zani C, Donato F, Grioni S, Viola GCV, Ceretti E, Feretti D, et al. Feasibility and reliability of a questionnaire for evaluation of the exposure to indoor and outdoor air pollutants, diet and physical activity in 6–8-year-old children. *Ann Ig.* 2015;27(4):646–56.
4. Baseline questionnaire. Healthy Environments for Children Alliance. Mongolia –HECA seed funds for country implementation. to evaluate knowledge, practice and attitudes of population on respiratory diseases among the children that was used in Mongolia. Geneva: World Health Organization; 2018 (<http://www.who.int/heca/activities/mongolia1/en/>, accessed September 2018).
5. Kumari K, Mumar S, Rajagopal V, Khare A, Kumar R. Emission from open burning of municipal solid waste in India. *Environ Technol.* 2017;27 July1–14.
6. Reyna-Bensusan N, Wilson DC, Smith SR. Uncontrolled burning of solid waste by households in Mexico is a significant contributor to climate change in the country. *Environ Res.* 2018;163:280–8.
7. Perez L, Declercq C, Iñiguez C, Aguilera I, Badaloni C, Mallester F, et al. Chronic burden of near-roadway traffic pollution in 10 European cities (APHEKOM network). *Eur Respir J.* 2013;42(3):594–605.
8. Perez L, Lurmann F, Wilson J, Pastor M, Brandt SJ, Künzli N, et al. Near-roadway pollution and childhood asthma: implications for developing “Win-Win” compact urban development and clean vehicle strategies. *Environ Health Perspect.* 2012;120(11):1619–26.
9. WHO, UNEP. Healthy environments for healthy children: key messages for action. Geneva: World Health Organization; 2010 (http://www.who.int/ceh/publications/hehc_booklet_en.pdf?ua=1, accessed September 2018)
10. Strengthening health systems for treating tobacco dependence in primary care. Part III: Training for primary care providers: brief tobacco interventions. Geneva: United World Health Organization; 2013.
11. Children's environmental health units. Geneva: World Health Organization; <http://www.who.int/ceh/publications/units/en/>, accessed September 2018).
12. Children's environmental health. Training modules and instructions for health care providers. Geneva: United World Health Organization; 2017 (<http://www.who.int/ceh/capacity/en/>, accessed September 2018).

13. Health in all policies training manual. Geneva: World Health Organization; 2015 (http://www.who.int/social_determinants/publications/health-policies-manual/en/, accessed September 2018).
14. Rio declaration on environment and development. Nairobi: United Nations Environment Programme; 1992 (<http://www.unep.org/Documents.multilingual/Default.asp? DocumentID=78&ArticleID=1163S>, accessed September 2018).
15. The precautionary principle and children's health. Washington DC: American Public Health Association; 2000 (<https://www.apha.org/policies-and-advocacy/public-health-policy-statements/policy-database/2014/07/14/10/56/the-precautionary-principle-and-childrens-health>, accessed September 2018).
16. Dealing with uncertainty – how can the precautionary principle help protect the future of our children? Working paper for the Fourth Ministerial Conference on Environment and Health, Budapest, Hungary, 23–25 June 2004. Copenhagen: WHO Regional Office for Europe; 2004 (<http://www.euro.who.int/document/hms/edoc11.pdf>, accessed September 2018).
17. Jarosinska D, Gee D. Children's environmental health and the precautionary principle. *Int J Hyg Environ Health*. 2007;210:541–6.
18. Scovronick N. Reducing global health risks through mitigation of short-lived climate pollutants. Scoping report for Policymakers. Geneva: World Health Organization; 2015 (http://apps.who.int/iris/bitstream/10665/189524/1/9789241565080_eng.pdf?ua=1, accessed September 2018).
19. Don't pollute my future! The impact of the environment on children's health. Geneva: World Health Organization; 2017:30 (<http://apps.who.int/iris/bitstream/10665/254678/1/WHO-FWC-IHE-17.01-eng.pdf>, accessed September 2018).
20. Inheriting a sustainable world? Atlas on children's health and the environment. Geneva: World Health Organization; 2017 (<http://www.who.int/ceh/publications/inheriting-a-sustainable-world/en/>, accessed September 2018)
21. Portier CJ, Thigpen Tart K, Carter SR, Dilworth CH, Grambsch AE, Gohlke J, et al. A human health perspective on climate change: a report outlining the research needs on the human health effects of climate change. Research Triangle Park (NC): Environmental Health Perspectives/National Institute of Environmental Health Sciences; 2010. doi:10.1289/ehp.1002272 (www.niehs.nih.gov/climate-report, accessed September 2018).
22. WHO indoor air quality guidelines: household fuel combustion. Geneva: World Health Organization; 2014 (http://apps.who.int/iris/bitstream/10665/141496/1/9789241548885_eng.pdf, accessed September 2018)
23. Clean cookstoves and clean cooking solutions – Harmonized laboratory test protocols – Part 3: Voluntary performance targets for cookstoves based on laboratory testing. Geneva: International Standards Organization; 2018 (<https://www.iso.org/standard/73935.html?browse=tc>, accessed September 2018)
24. Lurmann F, Avol E, Gilliland F. Emissions reduction policies and recent trends in southern California's ambient air quality. *J Air Waste Manag Assoc*. 2015;65(3):324–35.
25. Gauderman WJ, Urman R, Avol E, Berhane K, McConnell R, Rappaport E, et al. Association of improved air quality with lung development in children. *N Engl J Med*. 2015;372(10):905–13.
26. Troncoso K, Soares da Silva A. LPG fuel subsidies in Latin America and the use of solid fuels to cook. *Energy Policy*. 2017;107(C):188–96.
27. Imelda. Fuel switching and infant health: evidence from LPG conversion program in Indonesia. Paper presentation at the 2017 annual meeting of the Agricultural and Applied Economics Association, Chicago (IL) (http://ageconsearch.umn.edu/record/258478/files/Abstracts_17_05_23_18_55_47_34__114_72_228_71_0.pdf, accessed September 2018).
28. Pertamina, World LPG Association. Kerosene to LP gas conversion programme in Indonesia: a case study of domestic energy. Paris: World LPG Association; 2012 (<https://www.wlpga.org/publication/kerosene-to-lp-gas-conversion-programme-in-indonesia/>, accessed September 2018).
29. Kerosene to LP gas conversion programme in Indonesia. Paris: World LPG Association; 2018 (<https://www.wlpga.org/publication/kerosene-to-lp-gas-conversion-programme-in-indonesia/>, accessed 11 April 2018)
30. Global Health Observatory (GHO) data by region: household air pollution burden of disease. Geneva: World Health Organization; 2016 (<http://who.int/gho/>, accessed September 2018).

Annex 1. Global initiatives and organizations working on children's health and air pollution

Sixty-eighth World Health Assembly (2016)

A 4-year plan for responding to the adverse health effects of air pollution that includes four areas of action:

1. Expand the knowledge base, by building and disseminating global evidence and knowledge on the effects of air pollution on health and the effectiveness of interventions and policies to address it.
2. Enhance systems to monitor and report on health trends and progress towards the air pollution-related targets of the Sustainable Development Goals.
3. Leverage health sector leadership and coordinated action at all levels – local, national, regional and global – to raise awareness of air pollution.
4. Enhance the health sector's capacity to address the adverse health effects of air pollution by training, guidelines and national action plans.
<http://www.who.int/mediacentre/news/releases/2016/wha69-27-may-2016/en/>
<http://www.who.int/sustainable-development/news-events/wha69-roadmap-ap/en/>

Sustainable Development Goals (2015)

Health and air pollution are addressed throughout the SDGs. The third SDG, “ensure healthy lives and promote well-being for all at all ages”, emphasizes children's environmental health. Household energy is explicitly addressed in SDG 7: “ensure access to affordable, reliable, sustainable and modern energy for all”. The SDG targets reflect understanding that household energy is a critically important consideration in many facets of human development, from health (SDG 3) to sustainable urban environments (SDG 11) to gender equality (SDG 5) and climate action (SDG 13). SDG 3 includes targets to end preventable deaths of newborns and children under 5 years by 2030, which form the basis of the Global Strategy for Women's, Children's and Adolescent's Health. <http://www.un.org/sustainabledevelopment/sustainable-development-goals/>

WHO's Global Strategy for Women's, Children's and Adolescents' Health (2015)

The strategy has been updated by collaboration among stakeholders, led by WHO. It places women, children and adolescents at the heart of the Sustainable Development Goals. Its aims include reducing premature mortality from noncommunicable diseases in women, children and adolescents by one third by 2030. The Strategy includes targets for extending access to clean household energy and reducing HAP. <http://www.who.int/life-course/partners/global-strategy/en/>

WHO Nurturing Care Framework for Early Childhood Development (2018)

The Framework, launched at the Seventy-first World Health Assembly in May 2018, outlines a whole-of-society approach to ensure effective policies and interventions for a stable, supportive environment and protection from threats to health and well-being. Environmental risks are identified as clear threats to early childhood development, and tools are proposed to translate scientific results into action. The Framework represents the next step in a global movement to prioritize and invest in early childhood development. It provides an opportunity for more action to address air pollution and other environmental risks as part of broader child health programmes and policies. http://www.who.int/maternal_child_adolescent/child/nurturing-care-framework/en/

Every Newborn Action Plan (2014)

The Plan presents evidence-based means to prevent newborn deaths and stillbirths and a plan up to 2020 with global and national milestones and strategic actions that build on commitments made to end preventable newborn deaths and stillbirths.

http://www.who.int/maternal_child_adolescent/newborns/every-newborn/en/

BreatheLife (2016)

A joint campaign led by the WHO, UN Environment and the Climate and Clean Air Coalition to mobilize cities and individuals to protect our health and the planet from the effects of air pollution. <http://breathelife2030.org/>

The Climate and Clean Air Coalition (2012)

The aim of an initiative comprising over 50 governments and 51 civil society and international organizations is to reduce emissions of short-lived climate pollutants. The Coalition catalyses action for rapid introduction of proven technologies and policies to reduce emissions of these pollutants in all sectors.

<http://www.ccacoalition.org/en>

The Global Alliance for Clean Cookstoves (2011)

An initiative launched by the United Nations Foundation to deliver clean cooking solutions to 100 million households by 2020.

<http://cleancookstoves.org>

Sustainable Energy for All (2011)

A partnership co-chaired by the Secretary-General of the United Nations and The World Bank for achieving universal access to modern energy sources for cooking, heating, lighting and other uses. Its “global tracking framework” is an approach for monitoring various tiers of access to energy, “diagnose” situations and form the basis of interventions to move up those tiers towards cleaner, healthier, more reliable energy sources. The WHO global household energy database is used to measure progress toward achieving SDG 7.

<https://www.seforall.org>

UN-Energy (2004)

The mechanism used for inter-agency collaboration in the field of energy to ensure coherence in the multidisciplinary response of the United Nations system to the outcomes of the World Summit on Sustainable Development and to support countries in their transition to sustainable energy.

<http://www.un-energy.org>

Energising Development (EnDev)

A partnership to promote access to energy, which is currently financed by six donor countries: Germany, the Netherlands, Norway, Sweden, Switzerland and the United Kingdom. EnDev promotes sustainable access to modern energy sources that meet the needs of the poor in 25 countries in Africa, Asia and Latin America.

<http://www.who.int/airpollution/household/policy-governance/collaborations/en/>

Global Action Plan for the Prevention and Control of Pneumonia and Diarrhoea (2013)

A plan prepared by WHO and UNICEF that proposes a cohesive approach to end, by 2025, preventable childhood deaths due to pneumonia and diarrhoea by bringing together critical services and interventions to promote good health practices, appropriate treatment and universal vaccination coverage.

http://www.who.int/maternal_child_adolescent/documents/global_action_plan_pneumonia_diarrhoea/en/

Unmask My City

This global campaign involves local health partners and their communities in promoting practical solutions and creating tangible policy changes for a clear downward trend in urban air pollution by 2030. It is an initiative of the Global Climate and Health Alliance and its partners Health Care Without Harm, the Health and Environment Alliance, the US Climate and Health Alliance and the United Kingdom Health Alliance for Climate Change.

<http://unmaskmycity.org/about/>

The World Health Assembly resolution on climate change and health (WHA61.19) (2008) and the Paris Agreement of the United Nations Framework Convention on Climate Change (2015)

These global agreements provide a framework to protect health from risks associated with climate change and to ensure that actions to mitigate climate change also protect and improve people’s

health. The Paris Agreement explicitly states that action to address climate change should include, respect and promote the right to health.

http://www.who.int/globalchange/A61_R19_en.pdf

<https://unfccc.int/process-and-meetings/the-paris-agreement/the-paris-agreement>

<https://unfccc.int/news/climate-change-agreement-is-public-health-agreement>

Annex 2. Additional tables and figures

Table 4. Exposure of children to ambient PM_{2.5} and burden of disease, by country, 2016

Country	Sex	PM _{2.5} _exposure_total (µg/m ³)	PM _{2.5} _exposure_rural (µg/m ³)	PM _{2.5} _exposure_urban (µg/m ³)	No. of DALYs (< 5 years)	DALYs rate per 100 000 (< 5 years)	No. of DALYs (5–14 years)	DALYs rate per 100 000 (5–14 years)	No. of deaths (< 5 years)	Death rate per 100 000 (< 5 years)	No. of deaths (5–14 years)	Death rate per 100 000 (5–14 years)
Afghanistan	Both	53.2	48.7	59.9	386 056.4	7 377.6	27 973.4	280.7	4 245.8	81.1	332.4	3.3
	F				201 830.5	7 933.1	16 339.0	336.4	2 221.6	87.3	194.4	4.0
	M				184 225.9	6 851.9	11 634.3	227.6	2 024.2	75.3	138.0	2.7
Albania	Both	17.9	16.9	18.2	726.6	409.6	119.2	34.9	7.8	4.4	1.4	0.4
	F				344.5	402.0	84.7	51.8	3.7	4.3	1.0	0.6
	M				382.0	416.7	34.5	19.4	4.1	4.5	0.4	0.2
Algeria	Both	35.2	37.6	34.5	77 126.5	1 641.3	3 469.9	49.0	844.5	18.0	40.2	0.6
	F				45 400.9	1 974.4	1 810.1	52.2	497.4	21.6	21.0	0.6
	M				31 725.6	1 322.1	1 659.8	46.0	347.1	14.5	19.2	0.5
Andorra	Both	9.9	9.1	11.5	NA	NA	NA	NA	NA	NA	NA	NA
	F				NA	NA	NA	NA	NA	NA	NA	NA
	M				NA	NA	NA	NA	NA	NA	NA	NA
Angola	Both	27.9	27.7	28.4	353 080.3	6 690.8	17 186.6	208.2	3 884.9	73.6	205.0	2.5
	F				185 045.9	7 043.5	8 587.9	206.1	2 037.8	77.6	102.3	2.5
	M				168 034.3	6 341.2	8 598.7	210.4	1 847.1	69.7	102.6	2.5
Antigua and Barbuda	Both	17.9	14.9	18.0	4.8	59.5	3.0	18.2	0.1	0.6	0.0	0.2
	F				1.9	46.3	0.1	1.2	0.0	0.5	0.0	0.0
	M				2.9	72.5	2.9	35.1	0.0	0.8	0.0	0.4
Argentina	Both	11.8	12.3	11.7	5 940.4	159.0	619.3	8.5	64.6	1.7	7.2	0.1
	F				2 702.2	147.3	321.3	9.0	29.4	1.6	3.8	0.1

	M				3 238.2	170.4	298.0	8.1	35.2	1.9	3.4	0.1
Armenia	Both	30.5	23.7	32.9	1 191.6	589.3	155.0	40.8	13.0	6.4	1.8	0.5
	F				551.1	581.7	70.9	40.0	6.0	6.3	0.8	0.5
	M				640.5	596.0	84.2	41.4	7.0	6.5	1.0	0.5
Australia	Both	7.2	6.1	7.3	228.6	14.7	47.2	1.6	2.4	0.2	0.5	0.0
	F				100.6	13.3	33.4	2.3	1.1	0.1	0.4	0.0
	M				128.0	16.1	13.7	0.9	1.3	0.2	0.1	0.0
Austria	Both	12.4	10.9	13.1	15.4	3.8	36.0	4.4	0.2	0.0	0.4	0.1
	F				7.4	3.7	35.3	8.9	0.1	0.0	0.4	0.1
	M				8.1	3.8	0.7	0.2	0.1	0.0	0.0	0.0
Azerbaijan	Both	18.2	17.8	18.5	9 954.8	1 117.9	911.8	66.9	108.5	12.2	10.7	0.8
	F				5 118.5	1 235.5	410.6	65.0	55.8	13.5	4.8	0.8
	M				4 836.3	1 015.6	501.2	68.6	52.7	11.1	5.8	0.8
Bahamas	Both	17.6	15.6	19.0	124.4	451.8	7.3	13.8	1.4	4.9	0.1	0.2
	F				59.8	446.5	3.1	11.9	0.6	4.8	0.0	0.1
	M				64.6	456.9	4.2	15.6	0.7	5.0	0.0	0.2
Bahrain	Both	69.0	70.7	69.0	110.7	103.6	79.8	43.7	1.2	1.1	0.9	0.5
	F				56.2	108.2	44.2	49.3	0.6	1.1	0.5	0.6
	M				54.5	99.4	35.6	38.3	0.6	1.0	0.4	0.4
Bangladesh	Both	58.3	52.9	58.6	533 035.9	3 498.6	22 646.5	71.1	5 832.3	38.3	266.6	0.8
	F				239 240.0	3 210.1	13 376.9	85.8	2 618.1	35.1	158.3	1.0
	M				293 795.9	3 775.0	9 269.6	57.0	3 214.2	41.3	108.3	0.7
Barbados	Both	22.2	20.3	22.4	18.9	109.0	9.9	26.5	0.2	1.1	0.1	0.3
	F				9.0	105.0	1.6	8.9	0.1	1.1	0.0	0.1
	M				9.9	113.0	8.3	43.1	0.1	1.2	0.1	0.5
Belarus	Both	18.1	16.4	19.3	417.4	72.0	70.9	7.2	4.4	0.8	0.6	0.1
	F				184.2	65.5	34.0	7.1	1.9	0.7	0.3	0.1
	M				233.2	78.2	36.9	7.3	2.5	0.8	0.3	0.1

Belgium	Both	12.9	9.4	13.0	147.6	23.0	22.0	1.7	1.6	0.2	0.2	0.0
	F				62.8	20.0	13.3	2.1	0.7	0.2	0.1	0.0
	M				84.8	25.7	8.6	1.3	0.9	0.3	0.1	0.0
Belize	Both	21.2	21.2	20.9	145.7	361.7	17.7	23.0	1.6	3.9	0.2	0.3
	F				71.8	360.2	7.1	18.8	0.8	3.9	0.1	0.2
	M				73.9	363.1	10.6	27.1	0.8	3.9	0.1	0.3
Benin	Both	33.1	41.3	30.4	138 222.6	7 785.2	14 098.4	488.7	1 520.1	85.6	168.6	5.8
	F				72 869.8	8 336.8	7 239.4	509.0	801.8	91.7	86.5	6.1
	M				65 352.8	7 250.3	6 859.1	468.9	718.2	79.7	82.1	5.6
Bhutan	Both	35.3	35.3	35.4	1 704.3	2 441.1	169.0	116.5	18.6	26.7	2.0	1.4
	F				766.9	2 234.3	92.6	129.7	8.4	24.4	1.1	1.6
	M				937.4	2 641.1	76.4	103.7	10.2	28.9	0.9	1.2
Bolivia (Plurinational State of)	Both	20.2	18.4	23.3	20 868.2	1 755.8	2 703.8	117.6	228.6	19.2	32.4	1.4
	F				9 788.9	1 681.8	1 287.7	114.0	107.2	18.4	15.5	1.4
	M				11 079.3	1 826.9	1 416.1	121.1	121.3	20.0	17.0	1.5
Bosnia and Herzegovina	Both	27.3	24.3	29.7	204.5	130.5	21.9	6.4	2.2	1.4	0.2	0.1
	F				95.5	125.7	8.9	5.4	1.0	1.3	0.1	0.0
	M				109.0	135.0	12.9	7.3	1.1	1.4	0.1	0.1
Botswana	Both	21.2	21.3	20.9	3 767.7	1 453.5	389.3	86.0	41.3	15.9	4.6	1.0
	F				1 818.0	1 417.1	177.2	78.9	19.9	15.5	2.1	0.9
	M				1 949.7	1 489.2	212.1	93.1	21.4	16.3	2.5	1.1
Brazil	Both	11.5	9.5	11.8	40 961.5	274.6	3 745.2	12.1	445.3	3.0	43.0	0.1
	F				18 929.2	259.8	1 786.6	11.7	205.7	2.8	20.4	0.1
	M				22 032.3	288.7	1 958.6	12.4	239.7	3.1	22.6	0.1
Brunei Darussalam	Both	5.8	5.8	5.8	6.8	19.7	3.8	5.9	0.1	0.2	0.0	0.1
	F				3.1	18.4	2.5	8.1	0.0	0.2	0.0	0.1

	M				3.7	20.9	1.3	3.8	0.0	0.2	0.0	0.0
Bulgaria	Both	18.8	17.7	20.8	1 139.7	351.5	218.6	32.1	12.4	3.8	2.5	0.4
	F				509.6	323.6	101.2	30.6	5.5	3.5	1.2	0.4
	M				630.1	377.9	117.4	33.6	6.8	4.1	1.4	0.4
Burkina Faso	Both	36.8	37.2	36.3	184 238.3	5 720.5	23 174.6	442.1	2 025.8	62.9	277.4	5.3
	F				87 392.1	5 524.4	12 790.2	496.9	961.5	60.8	153.0	5.9
	M				96 846.2	5 909.9	10 384.3	389.2	1 064.3	65.0	124.4	4.7
Burundi	Both	35.6	35.2	35.6	113 273.1	5 957.5	19 974.4	708.0	1 244.4	65.4	239.8	8.5
	F				54 101.3	5 728.0	10 650.6	752.5	594.6	62.9	127.8	9.0
	M				59 171.8	6 184.1	9 323.8	663.2	649.8	67.9	112.0	8.0
Cabo Verde	Both	32.0	33.6	31.6	687.7	1 258.9	32.9	29.7	7.5	13.8	0.4	0.4
	F				310.7	1 151.3	15.8	28.7	3.4	12.6	0.2	0.3
	M				377.0	1 363.9	17.2	30.7	4.1	14.9	0.2	0.4
Cambodia	Both	24.0	21.5	24.9	35 159.4	1 996.2	3 230.5	101.4	383.9	21.8	38.0	1.2
	F				15 942.8	1 839.9	1 399.9	89.5	174.2	20.1	16.4	1.1
	M				19 216.6	2 147.7	1 830.6	112.8	209.7	23.4	21.6	1.3
Cameroon	Both	65.3	65.1	65.4	344 937.9	9 067.2	64 364.4	1 029.5	3 790.9	99.7	770.3	12.3
	F				156 001.3	8 284.3	30 891.9	995.8	1 715.1	91.1	369.7	11.9
	M				188 936.6	9 834.5	33 472.5	1 062.6	2 075.8	108.0	400.6	12.7
Canada	Both	6.5	5.3	6.7	157.4	8.2	28.9	0.7	1.7	0.1	0.3	0.0
	F				72.7	7.7	16.9	0.9	0.8	0.1	0.2	0.0
	M				84.7	8.6	12.1	0.6	0.9	0.1	0.1	0.0
Central African Republic	Both	49.5	49.0	51.2	94 519.2	12 939.8	4 449.3	350.8	1 037.9	142.1	53.2	4.2
	F				48 299.8	13 244.6	2 338.6	366.3	530.8	145.5	27.9	4.4
	M				46 219.4	12 635.9	2 110.7	335.1	507.1	138.6	25.2	4.0
Chad	Both	53.0	53.6	50.8	547 683.0	20 540.3	28 618.3	684.4	6 039.4	226.5	343.8	8.2
	F				263 619.5	19 947.5	15 518.3	748.0	2 908.3	220.1	186.5	9.0

	M				284 063.5	21 122.8	13 100.0	621.7	3 131.1	232.8	157.3	7.5
Chile	Both	21.0	17.8	23.1	1 071.5	90.5	83.5	3.3	11.6	1.0	0.9	0.0
	F				480.8	82.8	40.7	3.3	5.2	0.9	0.4	0.0
	M				590.8	97.9	42.8	3.4	6.4	1.1	0.4	0.0
China	Both	49.2	35.7	51.0	611 083.6	709.1	46 469.9	28.5	6 645.8	7.7	515.0	0.3
	F				270 327.0	675.7	26 131.2	34.6	2 930.2	7.3	279.1	0.4
	M				340 756.6	738.0	20 338.7	23.2	3 715.6	8.0	235.9	0.3
Colombia	Both	15.2	13.4	17.2	15 240.6	410.6	1 558.1	19.7	166.5	4.5	18.6	0.2
	F				7 085.8	390.2	674.2	17.4	77.4	4.3	8.0	0.2
	M				8 154.8	430.1	884.0	21.9	89.1	4.7	10.6	0.3
Comoros	Both	18.6	18.4	18.6	5 320.3	4 465.4	294.4	148.0	58.4	49.0	3.5	1.8
	F				2 609.0	4 464.0	142.8	146.1	28.7	49.1	1.7	1.7
	M				2 711.3	4 466.7	151.6	149.8	29.8	49.0	1.8	1.8
Congo	Both	38.7	41.4	36.4	33 752.7	4 094.9	2 446.2	181.4	370.5	45.0	29.4	2.2
	F				16 543.0	4 052.4	1 384.0	206.4	181.8	44.5	16.6	2.5
	M				17 209.8	4 136.6	1 062.2	156.7	188.7	45.4	12.7	1.9
Cook Islands	Both	12.0	12.0	NA	NA	NA	NA	NA	NA	NA	NA	NA
	F				NA	NA	NA	NA	NA	NA	NA	NA
	M				NA	NA	NA	NA	NA	NA	NA	NA
Costa Rica	Both	15.9	12.6	16.7	403.1	116.6	51.7	7.2	4.3	1.3	0.5	0.1
	F				188.1	111.5	28.1	8.0	2.0	1.2	0.3	0.1
	M				215.0	121.5	23.6	6.4	2.3	1.3	0.3	0.1
Côte d'Ivoire	Both	23.7	23.5	23.9	248 406.3	6 434.4	36 550.7	586.9	2 728.3	70.7	439.2	7.1
	F				110 022.3	5 738.8	20 246.4	651.2	1 208.9	63.1	243.3	7.8
	M				138 384.0	7 120.6	16 304.3	522.8	1 519.4	78.2	195.9	6.3
Croatia	Both	17.0	15.8	17.6	106.9	54.5	15.1	3.6	1.1	0.6	0.2	0.0
	F				44.0	46.1	1.0	0.5	0.5	0.5	0.0	0.0
	M				63.0	62.4	14.1	6.5	0.7	0.7	0.2	0.1

Cuba	Both	18.4	15.8	21.6	1 241.4	195.1	93.1	7.6	13.5	2.1	1.0	0.1
	F				571.2	184.5	58.2	9.8	6.2	2.0	0.7	0.1
	M				670.2	205.1	34.9	5.6	7.3	2.2	0.3	0.1
Cyprus	Both	16.8	15.9	17.1	5.7	8.7	0.2	0.1	0.1	0.1	0.0	0.0
	F				2.7	8.6	0.1	0.1	0.0	0.1	0.0	0.0
	M				3.0	8.8	0.1	0.1	0.0	0.1	0.0	0.0
Czechia	Both	15.1	13.6	15.6	296.9	55.6	89.1	8.2	3.2	0.6	1.0	0.1
	F				122.2	47.1	32.9	6.2	1.3	0.5	0.3	0.1
	M				174.7	63.7	56.2	10.1	1.9	0.7	0.6	0.1
Democratic People's Republic of Korea	Both	30.4	29.0	31.0	23 721.3	1 374.0	1 592.9	44.6	258.9	15.0	18.3	0.5
	F				10 458.3	1 240.5	882.3	50.5	114.0	13.5	10.1	0.6
	M				13 263.0	1 501.5	710.6	39.0	144.9	16.4	8.2	0.5
Democratic Republic of the Congo	Both	37.6	37.8	37.4	1 175 790.2	8 112.2	90 407.6	411.0	12 890.7	88.9	1 088.9	5.0
	F				585 953.1	8 166.0	49 954.8	457.5	6 426.4	89.6	601.4	5.5
	M				589 837.2	8 059.6	40 452.8	365.2	6 464.4	88.3	487.5	4.4
Denmark	Both	10.1	9.5	10.3	35.6	12.5	3.9	0.6	0.4	0.1	0.0	0.0
	F				16.7	12.0	3.3	1.0	0.2	0.1	0.0	0.0
	M				19.0	13.0	0.6	0.2	0.2	0.1	0.0	0.0
Djibouti	Both	40.4	40.1	41.0	4 942.5	4 832.2	1 142.5	585.3	54.1	52.9	13.8	7.1
	F				2 281.4	4 524.2	556.6	577.3	25.0	49.5	6.7	7.0
	M				2 661.1	5 131.7	585.9	593.1	29.1	56.1	7.1	7.2
Dominica	Both	18.2	16.9	18.8	NA	NA	NA	NA	NA	NA	NA	NA
	F				NA	NA	NA	NA	NA	NA	NA	NA
	M				NA	NA	NA	NA	NA	NA	NA	NA
Dominican Republic	Both	12.9	11.3	13.3	8 736.8	824.6	466.1	22.2	95.7	9.0	5.4	0.3

	F				3 481.5	671.0	221.4	21.5	38.1	7.3	2.6	0.2
	M				5 255.4	972.0	244.7	23.0	57.6	10.7	2.8	0.3
Ecuador	Both	14.9	14.1	15.5	10 798.2	670.2	866.4	28.0	118.1	7.3	10.3	0.3
	F				4 993.3	634.5	408.9	27.0	54.7	6.9	4.9	0.3
	M				5 804.9	704.3	457.5	29.0	63.5	7.7	5.5	0.3
Egypt	Both	79.3	69.4	79.6	222 583.9	1 728.7	56 687.9	296.3	2 431.9	18.9	675.5	3.5
	F				97 331.7	1 560.2	25 258.7	272.1	1 063.8	17.1	300.1	3.2
	M				125 252.2	1 887.0	31 429.2	319.0	1 368.1	20.6	375.4	3.8
El Salvador	Both	23.4	20.7	23.8	3 664.6	635.7	516.9	43.6	40.0	6.9	6.3	0.5
	F				1 427.1	506.9	186.3	32.1	15.5	5.5	2.2	0.4
	M				2 237.5	758.5	330.6	54.6	24.4	8.3	4.1	0.7
Equatorial Guinea	Both	45.9	45.5	49.1	15 735.2	8 662.9	1 469.4	535.1	172.6	95.0	17.6	6.4
	F				6 747.9	7 527.1	811.9	599.2	74.0	82.6	9.7	7.2
	M				8 987.3	9 769.8	657.5	472.6	98.5	107.1	7.9	5.7
Eritrea	Both	42.4	42.9	41.1	38 460.1	5 171.2	4 830.2	360.5	422.3	56.8	57.6	4.3
	F				17 784.4	4 884.5	2 226.2	338.9	195.3	53.6	26.5	4.0
	M				20 675.7	5 446.1	2 604.0	381.2	227.0	59.8	31.1	4.6
Estonia	Both	6.7	6.2	7.0	9.5	14.0	5.9	4.1	0.1	0.1	0.1	0.0
	F				4.5	13.5	2.3	3.3	0.0	0.1	0.0	0.0
	M				5.0	14.6	3.6	4.9	0.1	0.1	0.0	0.0
eSwatini	Both	16.3	16.4	16.2	5 927.3	3 300.2	582.3	180.5	64.9	36.1	6.9	2.2
	F				2 764.4	3 098.5	302.6	188.2	30.3	33.9	3.6	2.2
	M				3 162.9	3 499.1	279.7	172.8	34.6	38.3	3.3	2.1
Ethiopia	Both	34.4	34.9	34.0	703 519.9	4 635.4	100 742.9	374.4	7 733.5	51.0	1 209.0	4.5
	F				313 661.3	4 195.2	47 635.0	357.8	3 447.9	46.1	571.2	4.3
	M				389 858.5	5 062.7	53 107.9	390.5	4 285.6	55.7	637.7	4.7
Fiji	Both	10.2	9.7	10.5	440.5	508.7	57.6	33.8	4.8	5.5	0.7	0.4
	F				213.3	506.5	29.5	35.8	2.3	5.5	0.3	0.4

	M				227.2	510.8	28.1	31.9	2.5	5.6	0.3	0.4
Finland	Both	5.9	5.5	6.5	11.4	3.9	0.4	0.1	0.1	0.0	0.0	0.0
	F				5.4	3.7	0.2	0.1	0.1	0.0	0.0	0.0
	M				6.0	4.0	0.3	0.1	0.1	0.0	0.0	0.0
France	Both	11.6	9.9	12.4	354.4	9.2	95.0	1.2	3.6	0.1	0.7	0.0
	F				169.4	9.0	44.1	1.1	1.7	0.1	0.3	0.0
	M				185.0	9.4	50.8	1.3	1.9	0.1	0.4	0.0
Gabon	Both	38.5	38.8	37.8	9 337.7	3 412.7	1 320.2	302.5	102.4	37.4	16.2	3.7
	F				4 241.4	3 134.8	635.5	293.9	46.6	34.4	7.8	3.6
	M				5 096.3	3 684.6	684.7	311.1	55.9	40.4	8.4	3.8
Gambia	Both	32.2	31.7	32.3	16 104.3	4 467.4	1 452.4	255.8	177.3	49.2	17.3	3.1
	F				7 641.2	4 282.0	840.0	298.5	84.2	47.2	10.0	3.6
	M				8 463.0	4 649.3	612.4	213.9	93.1	51.2	7.3	2.6
Georgia	Both	21.2	17.9	24.0	662.8	244.4	235.7	49.9	7.2	2.7	2.8	0.6
	F				330.1	252.8	86.9	38.9	3.6	2.7	1.0	0.5
	M				332.7	236.6	148.7	59.6	3.6	2.6	1.8	0.7
Germany	Both	11.7	10.5	11.9	487.6	13.7	137.5	1.9	5.2	0.1	1.5	0.0
	F				221.7	12.8	51.1	1.5	2.4	0.1	0.5	0.0
	M				265.9	14.6	86.4	2.4	2.8	0.2	1.0	0.0
Ghana	Both	31.9	34.0	31.1	132 424.2	3 241.5	13 607.8	199.4	1 454.6	35.6	163.1	2.4
	F				58 899.7	2 944.6	6 977.2	209.1	646.9	32.3	83.6	2.5
	M				73 524.5	3 526.3	6 630.6	190.1	807.7	38.7	79.5	2.3
Greece	Both	15.7	13.5	16.4	209.0	44.1	32.2	2.9	2.3	0.5	0.4	0.0
	F				95.2	41.5	14.6	2.7	1.0	0.5	0.2	0.0
	M				113.8	46.6	17.6	3.0	1.2	0.5	0.2	0.0
Grenada	Both	21.6	20.4	21.8	64.8	656.6	2.1	11.6	0.7	7.1	0.0	0.1
	F				30.5	633.5	0.1	1.7	0.3	6.9	0.0	0.0
	M				34.3	678.5	2.0	21.1	0.4	7.4	0.0	0.2

Guatemala	Both	23.6	21.0	24.2	33 930.6	1 677.5	2 561.5	65.8	371.2	18.4	30.6	0.8
	F				15 724.5	1 589.9	1 272.7	66.8	172.0	17.4	15.2	0.8
	M				18 206.1	1 761.4	1 288.8	64.9	199.2	19.3	15.4	0.8
Guinea	Both	22.4	22.6	22.2	114 271.7	5 762.9	12 485.4	380.5	1 255.6	63.3	149.5	4.6
	F				54 110.4	5 492.7	7 599.1	466.5	594.8	60.4	90.9	5.6
	M				60 161.3	6 029.7	4 886.2	295.8	660.8	66.2	58.6	3.5
Guinea-Bissau	Both	27.1	27.6	26.5	20 053.3	6 895.0	1 125.1	242.1	221.1	76.0	13.5	2.9
	F				9 528.5	6 581.2	587.9	253.1	105.1	72.6	7.0	3.0
	M				10 524.9	7 206.1	537.3	231.1	116.1	79.5	6.4	2.8
Guyana	Both	20.5	17.4	21.6	702.5	919.3	122.6	81.2	7.7	10.0	1.5	1.0
	F				325.6	874.3	53.1	72.2	3.6	9.6	0.6	0.9
	M				376.9	962.0	69.5	89.6	4.1	10.5	0.8	1.1
Haiti	Both	14.6	13.3	14.7	49 247.9	3 992.8	3 714.0	155.8	540.6	43.8	44.4	1.9
	F				22 195.9	3 670.9	1 570.6	133.9	243.7	40.3	18.8	1.6
	M				27 052.0	4 302.3	2 143.4	177.1	296.9	47.2	25.6	2.1
Honduras	Both	20.1	18.9	21.5	6 832.2	718.2	276.6	13.9	74.7	7.8	3.2	0.2
	F				3 055.7	655.5	78.0	8.0	33.4	7.2	0.9	0.1
	M				3 776.4	778.5	198.6	19.6	41.3	8.5	2.4	0.2
Hungary	Both	15.6	14.4	16.3	303.6	69.6	12.8	1.3	3.2	0.7	0.0	0.0
	F				127.2	60.2	6.1	1.3	1.3	0.6	0.0	0.0
	M				176.4	78.6	6.7	1.4	1.9	0.8	0.0	0.0
Iceland	Both	5.9	6.0	5.9	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0
	F				0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0
	M				0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0
India	Both	65.2	55.9	68.0	5 560 430.5	4 633.8	368 945.7	145.6	60 987.2	50.8	4 360.5	1.7
	F				2 996 229.5	5 270.4	205 376.5	171.5	32 889.5	57.9	2 441.7	2.0
	M				2 564 201.0	4 060.6	163 569.2	122.4	28 097.7	44.5	1 918.9	1.4
Indonesia	Both	15.6	13.2	16.4	265 856.9	1 071.1	16 089.2	34.0	2 902.5	11.7	187.5	0.4

	F				130 505.2	1 076.1	7 446.9	32.2	1 425.5	11.8	86.4	0.4
	M				135 351.7	1 066.2	8 642.3	35.6	1 477.0	11.6	101.1	0.4
Iran (Islamic Republic of)	Both	35.1	35.6	34.4	64 015.8	938.3	2 711.3	22.3	698.5	10.2	29.9	0.2
	F				33 860.4	1 014.1	1 344.0	22.5	370.0	11.1	14.8	0.2
	M				30 155.4	865.6	1 367.3	22.0	328.5	9.4	15.1	0.2
Iraq	Both	57.7	54.8	60.1	181 782.9	3 168.3	3 681.5	39.4	1 988.9	34.7	41.6	0.4
	F				59 381.7	2 129.3	1 993.5	43.9	650.6	23.3	22.7	0.5
	M				122 401.2	4 150.9	1 688.0	35.1	1 338.3	45.4	18.9	0.4
Ireland	Both	8.3	7.6	8.7	31.3	9.1	3.4	0.5	0.3	0.1	0.0	0.0
	F				13.5	8.1	2.7	0.8	0.1	0.1	0.0	0.0
	M				17.8	10.1	0.7	0.2	0.2	0.1	0.0	0.0
Israel	Both	19.5	21.1	19.4	148.5	17.7	58.7	4.1	1.6	0.2	0.7	0.0
	F				72.5	17.7	25.5	3.6	0.8	0.2	0.3	0.0
	M				76.0	17.6	33.3	4.5	0.8	0.2	0.4	0.1
Italy	Both	15.3	11.1	15.7	279.8	11.3	63.8	1.1	3.0	0.1	0.7	0.0
	F				125.5	10.4	43.0	1.6	1.3	0.1	0.5	0.0
	M				154.2	12.1	20.8	0.7	1.6	0.1	0.2	0.0
Jamaica	Both	13.3	12.5	13.6	316.6	154.4	48.6	10.6	3.4	1.7	0.5	0.1
	F				144.1	144.9	24.0	10.7	1.5	1.6	0.3	0.1
	M				172.6	163.4	24.6	10.6	1.9	1.8	0.3	0.1
Japan	Both	11.4	9.3	11.8	1 882.6	35.2	527.3	4.7	20.2	0.4	5.4	0.0
	F				885.3	34.1	216.7	4.0	9.5	0.4	2.2	0.0
	M				997.2	36.3	310.6	5.4	10.7	0.4	3.2	0.1
Jordan	Both	32.1	38.0	31.7	8 581.1	699.3	672.2	31.2	93.5	7.6	7.8	0.4
	F				4 112.6	684.8	388.5	36.5	44.8	7.5	4.5	0.4
	M				4 468.5	713.2	283.7	26.0	48.6	7.8	3.2	0.3
Kazakhstan	Both	11.3	10.4	14.5	5 701.0	285.5	390.6	13.3	62.1	3.1	4.4	0.2
	F				2 567.9	264.5	187.5	13.1	28.0	2.9	2.1	0.1

	M				3 133.1	305.3	203.1	13.5	34.1	3.3	2.3	0.2
Kenya	Both	25.9	26.1	25.8	215 951.5	3 074.8	13 977.7	109.3	2 370.7	33.8	167.5	1.3
	F				104 676.1	3 011.9	5 498.3	86.6	1 149.5	33.1	65.7	1.0
	M				111 275.4	3 136.3	8 479.4	131.6	1 221.2	34.4	101.8	1.6
Kiribati	Both	10.5	10.4	10.9	283.7	1 958.7	16.6	65.7	3.1	21.5	0.2	0.8
	F				136.5	1 932.1	4.8	38.6	1.5	21.2	0.1	0.4
	M				147.2	1 984.1	11.9	91.7	1.6	21.8	0.1	1.1
Kuwait	Both	57.2	52.5	58.9	1 046.9	331.2	219.3	41.1	11.4	3.6	2.5	0.5
	F				517.5	334.8	99.8	39.0	5.6	3.6	1.1	0.4
	M				529.4	327.7	119.5	43.0	5.7	3.6	1.4	0.5
Kyrgyzstan	Both	18.1	18.7	17.4	10 768.6	1 416.0	448.8	40.2	117.7	15.5	5.2	0.5
	F				4 973.2	1 344.2	196.4	35.9	54.3	14.7	2.2	0.4
	M				5 795.4	1 484.0	252.4	44.3	63.3	16.2	2.9	0.5
Lao People's Democratic Republic	Both	24.5	23.7	25.5	33 457.6	4 370.1	2 398.2	161.7	366.7	47.9	28.4	1.9
	F				16 173.4	4 314.4	1 085.7	149.4	177.4	47.3	12.8	1.8
	M				17 284.2	4 423.4	1 312.5	173.6	189.3	48.4	15.5	2.1
Latvia	Both	12.7	10.8	14.4	66.6	68.8	3.6	1.8	0.7	0.7	0.0	0.0
	F				31.8	67.7	1.7	1.7	0.3	0.7	0.0	0.0
	M				34.8	69.8	1.9	1.8	0.4	0.7	0.0	0.0
Lebanon	Both	30.7	29.7	30.7	937.8	194.1	69.3	7.4	10.1	2.1	0.6	0.1
	F				516.4	217.0	37.1	7.9	5.6	2.3	0.3	0.1
	M				421.4	171.8	32.1	7.0	4.5	1.9	0.3	0.1
Lesotho	Both	27.8	27.3	28.1	17 410.3	6 089.3	1 225.1	246.4	190.7	66.7	14.6	2.9
	F				8 789.9	6 191.7	681.4	275.5	96.3	67.8	8.2	3.3
	M				8 620.5	5 988.2	543.7	217.7	94.4	65.6	6.5	2.6
Liberia	Both	17.2	17.4	17.0	24 483.1	3 421.9	1 996.1	162.6	268.2	37.5	24.0	2.0
	F				12 191.9	3 482.8	1 088.0	181.1	133.6	38.2	13.1	2.2

	M				12 291.1	3 363.5	908.1	144.9	134.6	36.8	10.9	1.7
Libya	Both	44.2	44.9	41.7	4 146.2	661.8	735.2	63.3	45.3	7.2	8.4	0.7
	F				2 155.4	705.9	354.6	62.6	23.5	7.7	4.0	0.7
	M				1 990.8	619.8	380.6	64.0	21.7	6.8	4.4	0.7
Lithuania	Both	11.5	10.9	12.3	66.7	43.9	3.9	1.4	0.7	0.5	0.0	0.0
	F				34.1	46.0	1.9	1.4	0.4	0.5	0.0	0.0
	M				32.6	41.8	2.0	1.4	0.3	0.4	0.0	0.0
Luxembourg	Both	10.2	8.8	10.4	1.0	3.1	0.1	0.1	0.0	0.0	0.0	0.0
	F				0.4	2.8	0.0	0.1	0.0	0.0	0.0	0.0
	M				0.5	3.3	0.0	0.1	0.0	0.0	0.0	0.0
Madagascar	Both	21.4	20.7	22.5	111 123.1	2 948.7	14 298.0	219.6	1 218.9	32.3	171.2	2.6
	F				54 845.3	2 946.9	6 740.7	208.3	601.8	32.3	80.5	2.5
	M				56 277.8	2 950.4	7 557.3	230.8	617.0	32.3	90.6	2.8
Malawi	Both	22.1	23.3	21.9	78 573.0	2 701.7	4 617.6	90.5	861.8	29.6	55.1	1.1
	F				36 320.7	2 526.5	2 056.0	81.1	398.3	27.7	24.4	1.0
	M				42 252.3	2 873.0	2 561.5	99.9	463.4	31.5	30.7	1.2
Malaysia	Both	16.0	11.6	17.3	3 312.3	126.8	1,170.5	23.1	35.8	1.4	13.8	0.3
	F				1 425.4	113.1	509.3	20.7	15.4	1.2	6.0	0.2
	M				1 886.9	139.6	661.2	25.4	20.4	1.5	7.9	0.3
Maldives	Both	7.6	7.6	7.7	28.2	72.3	4.2	6.8	0.3	0.8	0.0	0.1
	F				15.2	81.3	2.2	7.5	0.2	0.9	0.0	0.1
	M				13.0	64.0	1.9	6.2	0.1	0.7	0.0	0.1
Mali	Both	31.2	32.7	29.0	248 818.1	7 467.3	16 723.2	317.0	2 737.2	82.1	199.4	3.8
	F				126 586.9	7 736.3	9 540.6	367.5	1 393.1	85.1	113.8	4.4
	M				122 231.3	7 207.7	7 182.6	268.0	1 344.2	79.3	85.6	3.2
Malta	Both	14.0	10.1	14.0	9.1	42.2	0.1	0.1	0.1	0.5	0.0	0.0
	F				4.6	43.9	0.0	0.1	0.1	0.5	0.0	0.0
	M				4.5	40.5	0.0	0.1	0.0	0.4	0.0	0.0

Marshall Islands	Both	9.4	9.4	NA	NA	NA	NA	NA	NA	NA	NA	NA
	F				NA	NA	NA	NA	NA	NA	NA	NA
	M				NA	NA	NA	NA	NA	NA	NA	NA
Mauritania	Both	40.8	40.0	41.7	46 817.5	7 146.5	2 088.2	195.2	515.1	78.6	24.8	2.3
	F				19 235.3	5 982.5	1 172.9	222.6	211.7	65.8	13.9	2.6
	M				27 582.2	8 268.4	915.3	168.6	303.5	91.0	10.9	2.0
Mauritius	Both	13.5	14.9	13.5	155.6	228.4	28.0	16.4	1.7	2.5	0.3	0.2
	F				65.9	197.4	16.1	19.2	0.7	2.1	0.2	0.2
	M				89.7	258.2	11.9	13.7	1.0	2.8	0.1	0.2
Mexico	Both	20.1	14.4	20.9	57 023.4	492.4	2 449.4	10.7	623.2	5.4	29.0	0.1
	F				26 372.7	466.0	1 107.7	9.9	288.2	5.1	13.0	0.1
	M				30 650.7	517.6	1 341.7	11.4	334.9	5.7	16.0	0.1
Micronesia (Federated States of)	Both	10.2	10.1	10.5	116.4	999.6	10.7	45.4	1.3	10.9	0.1	0.5
	F				53.4	948.9	4.5	39.3	0.6	10.4	0.1	0.5
	M				63.0	1 047.0	6.2	51.1	0.7	11.5	0.1	0.6
Monaco	Both	12.2	NA	12.2	NA	NA	NA	NA	NA	NA	NA	NA
	F				NA	NA	NA	NA	NA	NA	NA	NA
	M				NA	NA	NA	NA	NA	NA	NA	NA
Mongolia	Both	40.4	36.9	49.5	4 467.1	1 215.3	413.2	79.5	48.7	13.2	4.8	0.9
	F				1 608.8	886.9	170.3	66.3	17.5	9.7	2.0	0.8
	M				2 858.3	1 535.2	242.9	92.5	31.2	16.7	2.8	1.1
Montenegro	Both	20.2	20.6	19.3	19.6	54.1	9.4	11.9	0.2	0.5	0.1	0.1
	F				8.6	49.1	4.8	12.8	0.1	0.5	0.0	0.1
	M				11.0	58.8	4.5	11.1	0.1	0.6	0.0	0.1
Morocco	Both	31.0	30.4	31.1	48 582.1	1 384.7	2 072.4	33.3	531.5	15.1	23.9	0.4
	F				22 119.4	1 296.0	1 070.0	35.3	242.3	14.2	12.4	0.4
	M				26 462.6	1 468.8	1 002.4	31.5	289.2	16.1	11.5	0.4

Mozambique	Both	19.4	20.1	18.4	174 278.2	3 520.8	15 233.8	189.7	1 908.5	38.6	183.5	2.3
	F				85 623.6	3 486.0	7 451.6	186.2	937.8	38.2	89.7	2.2
	M				88 654.6	3 555.1	7 782.2	193.3	970.7	38.9	93.8	2.3
Myanmar	Both	34.7	34.8	34.6	205 998.5	4 539.6	15 630.7	157.3	2 258.5	49.8	185.8	1.9
	F				92 061.5	4 085.8	5 573.1	112.8	1 010.9	44.9	66.1	1.3
	M				113 936.9	4 987.1	10 057.6	201.2	1 247.6	54.6	119.7	2.4
Namibia	Both	22.6	24.0	21.0	9 710.1	2 821.7	884.3	154.9	106.6	31.0	10.6	1.9
	F				4 613.1	2 699.0	395.9	139.0	50.6	29.6	4.7	1.7
	M				5 096.9	2 942.8	488.4	170.9	56.0	32.3	5.9	2.1
Nauru	Both	12.5	10.0	12.5	NA	NA	NA	NA	NA	NA	NA	NA
	F				NA	NA	NA	NA	NA	NA	NA	NA
	M				NA	NA	NA	NA	NA	NA	NA	NA
Nepal	Both	94.3	68.3	99.5	115 948.0	4 206.8	7 958.3	123.9	1 268.4	46.0	93.7	1.5
	F				51 581.9	3 863.0	4 026.1	128.5	564.3	42.3	47.5	1.5
	M				64 366.1	4 530.0	3 932.2	119.4	704.1	49.6	46.2	1.4
Netherlands	Both	12.1	11.0	12.1	106.7	11.9	72.5	3.8	1.2	0.1	0.8	0.0
	F				46.4	10.7	50.6	5.4	0.5	0.1	0.6	0.1
	M				60.3	13.1	21.9	2.2	0.7	0.1	0.2	0.0
New Zealand	Both	5.7	5.2	5.8	82.5	27.1	7.6	1.2	0.9	0.3	0.1	0.0
	F				39.2	26.4	3.5	1.2	0.4	0.3	0.0	0.0
	M				43.3	27.8	4.1	1.3	0.5	0.3	0.0	0.0
Nicaragua	Both	16.9	14.6	19.0	6 290.0	1 053.1	452.9	37.2	68.8	11.5	5.5	0.4
	F				2 417.2	827.6	214.0	36.2	26.4	9.0	2.6	0.4
	M				3 872.8	1 268.9	238.8	38.1	42.4	13.9	2.9	0.5
Niger	Both	70.8	69.7	73.0	545 182.9	12 925.6	77 215.2	1 252.2	6 014.2	142.6	929.3	15.1
	F				277 404.3	13 442.9	40 120.8	1 327.8	3 062.3	148.4	482.8	16.0
	M				267 778.6	12 430.0	37 094.4	1 179.5	2 951.9	137.0	446.5	14.2
Nigeria	Both	48.7	56.5	46.3	4 330 967.5	13 618.7	486 606.3	969.4	47 674.7	149.9	5 810.7	11.6

	F				1 996 787.6	12 877.7	278 996.4	1 135.2	21 994.6	141.8	3 330.2	13.6
	M				2 334 180.0	14 323.9	207 609.9	810.4	25 680.1	157.6	2 480.5	9.7
Niue	Both	11.5	11.5	NA	NA	NA	NA	NA	NA	NA	NA	NA
	F				NA	NA	NA	NA	NA	NA	NA	NA
	M				NA	NA	NA	NA	NA	NA	NA	NA
Norway	Both	7.0	6.4	7.8	15.5	5.1	2.6	0.4	0.2	0.1	0.0	0.0
	F				6.4	4.3	0.5	0.2	0.1	0.0	0.0	0.0
	M				9.1	5.8	2.1	0.7	0.1	0.1	0.0	0.0
Oman	Both	38.2	40.0	36.2	1 380.0	344.9	132.8	23.2	15.0	3.7	1.4	0.2
	F				794.5	408.8	68.1	24.0	8.6	4.4	0.7	0.3
	M				585.6	284.5	64.7	22.3	6.3	3.1	0.7	0.2
Pakistan	Both	55.2	52.0	56.2	1 928 216.0	7 724.4	93 060.9	219.2	21 136.9	84.7	1 110.8	2.6
	F				975 687.2	8 129.1	35 489.6	173.8	10 699.8	89.1	422.3	2.1
	M				952 528.8	7 349.6	57 571.3	261.4	10 437.1	80.5	688.5	3.1
Palau	Both	12.2	12.0	12.4	NA	NA	NA	NA	NA	NA	NA	NA
	F				NA	NA	NA	NA	NA	NA	NA	NA
	M				NA	NA	NA	NA	NA	NA	NA	NA
Panama	Both	11.2	9.4	12.0	1 642.7	423.0	148.3	20.4	17.9	4.6	1.8	0.2
	F				713.3	375.3	65.1	18.3	7.8	4.1	0.8	0.2
	M				929.3	468.7	83.2	22.4	10.1	5.1	1.0	0.3
Papua New Guinea	Both	10.9	10.8	11.5	18 250.9	1 767.3	1 273.6	67.0	199.6	19.3	15.0	0.8
	F				7 956.8	1 595.6	443.8	48.3	87.0	17.4	5.2	0.6
	M				10 294.1	1 927.7	829.9	84.6	112.6	21.1	9.8	1.0
Paraguay	Both	11.2	10.2	11.7	2 865.3	426.6	300.0	22.6	31.2	4.6	3.3	0.3
	F				1 286.8	390.9	144.0	22.1	14.0	4.3	1.6	0.2
	M				1 578.5	460.9	156.0	23.1	17.2	5.0	1.8	0.3
Peru	Both	24.3	18.4	29.0	17 394.2	573.6	5 134.5	89.2	189.7	6.3	61.1	1.1
	F				7 802.9	525.6	2 295.6	81.5	85.1	5.7	27.3	1.0

	M				9 591.3	619.6	2 838.9	96.7	104.6	6.8	33.8	1.2
Philippines	Both	18.4	14.0	18.7	173 752.3	1 506.9	25 630.5	119.2	1 904.2	16.5	308.8	1.4
	F				80 151.2	1 431.7	11 809.2	112.9	878.8	15.7	141.9	1.4
	M				93 601.1	1 577.9	13 821.4	125.2	1 025.5	17.3	166.9	1.5
Poland	Both	20.5	18.0	21.5	1 403.7	77.2	595.6	15.5	15.1	0.8	6.9	0.2
	F				625.5	70.7	281.8	15.1	6.7	0.8	3.3	0.2
	M				778.2	83.3	313.8	15.9	8.4	0.9	3.6	0.2
Portugal	Both	7.9	7.1	8.1	95.0	22.0	9.7	1.0	1.0	0.2	0.1	0.0
	F				43.7	21.0	3.5	0.7	0.5	0.2	0.0	0.0
	M				51.3	23.0	6.3	1.2	0.6	0.3	0.1	0.0
Qatar	Both	90.3	81.3	91.7	385.3	296.5	62.4	27.5	4.2	3.2	0.7	0.3
	F				150.5	236.1	24.9	22.6	1.6	2.5	0.3	0.2
	M				234.8	354.6	37.5	32.3	2.5	3.8	0.4	0.4
Republic of Korea	Both	24.6	23.7	24.7	645.2	29.0	163.6	3.5	6.6	0.3	1.2	0.0
	F				319.2	29.8	72.6	3.2	3.3	0.3	0.5	0.0
	M				326.0	28.3	90.9	3.7	3.3	0.3	0.7	0.0
Republic of Moldova	Both	16.0	15.2	16.5	1 656.5	760.4	109.3	26.0	18.0	8.3	1.2	0.3
	F				709.3	675.2	48.9	24.0	7.7	7.4	0.5	0.3
	M				947.2	839.8	60.3	27.9	10.3	9.1	0.7	0.3
Romania	Both	14.3	12.7	15.4	6 136.0	649.9	623.5	29.9	66.9	7.1	7.2	0.3
	F				2 759.5	600.6	323.4	31.9	30.1	6.5	3.8	0.4
	M				3 376.5	696.6	300.0	28.0	36.8	7.6	3.5	0.3
Russian Federation	Both	13.7	12.2	14.7	11 757.3	123.0	2 015.2	13.1	126.3	1.3	21.4	0.1
	F				5 386.8	115.9	1 006.5	13.5	57.8	1.2	10.8	0.1
	M				6 370.5	129.6	1 008.7	12.8	68.4	1.4	10.6	0.1
Rwanda	Both	40.7	44.0	40.7	44 833.3	2 577.0	8 471.6	274.8	491.3	28.2	101.7	3.3
	F				20 259.5	2 338.0	3 931.9	254.3	222.0	25.6	47.2	3.0

	M				24 573.8	2 814.2	4 539.8	295.4	269.3	30.8	54.5	3.5
Saint Kitts and Nevis	Both	12.3	12.3	12.3	NA	NA	NA	NA	NA	NA	NA	NA
	F				NA	NA	NA	NA	NA	NA	NA	NA
	M				NA	NA	NA	NA	NA	NA	NA	NA
Saint Lucia	Both	21.2	19.0	21.2	37.3	340.6	1.4	5.8	0.4	3.7	0.0	0.0
	F				18.1	334.8	1.1	9.7	0.2	3.6	0.0	0.1
	M				19.2	346.3	0.2	2.0	0.2	3.8	0.0	0.0
Saint Vincent and the Grenadines	Both	21.2	19.7	21.4	56.7	683.0	4.3	24.0	0.6	7.4	0.1	0.3
	F				27.5	669.5	2.2	24.4	0.3	7.3	0.0	0.3
	M				29.3	696.2	2.2	23.6	0.3	7.6	0.0	0.3
Samoa	Both	10.6	10.2	10.9	71.7	305.2	10.2	21.1	0.8	3.3	0.1	0.2
	F				29.1	256.7	4.0	17.3	0.3	2.8	0.0	0.2
	M				42.6	350.5	6.2	24.7	0.5	3.8	0.1	0.3
San Marino	Both	13.4	NA	13.4	NA	NA	NA	NA	NA	NA	NA	NA
	F				NA	NA	NA	NA	NA	NA	NA	NA
	M				NA	NA	NA	NA	NA	NA	NA	NA
Sao Tome and Principe	Both	25.7	26.3	25.2	544.1	1 736.7	122.3	222.4	6.0	19.0	1.4	2.6
	F				225.2	1 450.3	58.9	215.8	2.5	15.9	0.7	2.6
	M				318.9	2 018.1	63.4	228.9	3.5	22.1	0.8	2.7
Saudi Arabia	Both	78.4	75.1	86.7	16 146.4	544.4	1 854.7	35.2	176.7	6.0	20.7	0.4
	F				7 905.3	541.1	1 028.9	39.6	86.5	5.9	11.5	0.4
	M				8 241.1	547.7	825.8	30.8	90.2	6.0	9.1	0.3
Senegal	Both	37.5	35.2	39.7	93 893.9	3 690.3	11 931.3	292.3	1 030.6	40.5	143.3	3.5
	F				42 660.2	3 399.8	6 145.4	304.7	468.6	37.3	73.7	3.7
	M				51 233.7	3 973.0	5 785.9	280.2	562.0	43.6	69.7	3.4
Serbia	Both	24.3	23.0	24.7	388.2	82.8	51.5	5.2	4.1	0.9	0.5	0.1

	F				173.2	75.6	21.6	4.5	1.8	0.8	0.2	-
	M				215.0	89.6	29.8	5.9	2.3	1.0	0.3	0.1
Seychelles	Both	18.7	19.0	18.6	21.4	272.7	10.5	81.6	0.2	3.0	0.1	1.0
	F				8.6	224.8	4.8	74.2	0.1	2.4	0.1	0.9
	M				12.7	318.7	5.8	89.0	0.1	3.5	0.1	1.1
Sierra Leone	Both	20.6	20.7	20.6	63 466.9	5 563.1	7 217.0	361.5	695.0	60.9	86.0	4.3
	F				30 738.8	5 405.9	3 832.8	383.0	336.6	59.2	45.6	4.6
	M				32 728.1	5 719.3	3 384.3	340.0	358.4	62.6	40.3	4.1
Singapore	Both	18.3	12.7	18.3	213.1	80.3	17.9	3.0	2.3	0.9	0.2	0.0
	F				89.0	69.7	10.4	3.6	1.0	0.8	0.1	0.0
	M				124.1	90.1	7.5	2.5	1.3	1.0	0.1	0.0
Slovakia	Both	17.5	16.4	18.0	503.7	179.1	99.6	18.1	5.5	1.9	1.2	0.2
	F				228.7	166.4	54.2	20.1	2.5	1.8	0.6	0.2
	M				275.0	191.1	45.5	16.1	3.0	2.1	0.5	0.2
Slovenia	Both	15.8	14.7	16.4	10.8	10.1	6.0	3.0	0.1	0.1	0.1	0.0
	F				5.4	10.4	0.8	0.9	0.1	0.1	0.0	0.0
	M				5.4	9.8	5.2	5.0	0.0	0.1	0.1	0.0
Solomon Islands	Both	10.7	10.6	11.5	745.1	900.4	39.5	26.0	8.1	9.8	0.5	0.3
	F				365.1	910.9	16.3	22.1	4.0	9.9	0.2	0.3
	M				380.0	890.6	23.2	29.6	4.2	9.7	0.3	0.3
Somalia	Both	29.5	29.9	28.0	385 089.7	14 714.7	22 838.9	564.4	4 241.5	162.1	272.4	6.7
	F				185 858.5	14 323.6	11 821.5	587.0	2 047.4	157.8	140.9	7.0
	M				199 231.2	15 099.3	11 017.3	542.0	2 194.1	166.3	131.5	6.5
South Africa	Both	23.6	20.9	24.3	159 530.4	2 796.4	10 686.9	100.4	1 743.2	30.6	127.7	1.2
	F				74 588.8	2 642.3	4 634.3	87.8	815.1	28.9	55.1	1.0
	M				84 941.6	2 947.3	6 052.6	113.0	928.2	32.2	72.6	1.4
South Sudan	Both	41.1	41.1	40.9	205 630.5	10 681.8	9 267.5	289.9	2 265.4	117.7	110.6	3.5
	F				99 177.9	10 458.9	4 687.2	296.8	1 092.9	115.3	55.9	3.5

	M				106 452.6	10 898.3	4 580.3	283.1	1 172.6	120.0	54.7	3.4
Spain	Both	9.5	8.3	9.8	235.3	11.4	37.1	0.8	2.5	0.1	0.4	0.0
	F				111.5	11.1	24.8	1.1	1.2	0.1	0.3	0.0
	M				123.8	11.6	12.2	0.5	1.3	0.1	0.1	0.0
Sri Lanka	Both	15.2	17.3	15.1	1 913.9	119.5	617.8	17.9	20.7	1.3	7.1	0.2
	F				792.1	100.6	315.9	18.4	8.6	1.1	3.7	0.2
	M				1 121.8	137.8	301.9	17.4	12.1	1.5	3.5	0.2
Sudan	Both	47.9	48.3	46.8	346 987.7	5 841.2	15 068.4	145.6	3 829.3	64.5	178.4	1.7
	F				197 254.8	6 755.9	7 456.8	146.3	2 177.0	74.6	88.4	1.7
	M				149 732.9	4 957.1	7 611.6	144.8	1 652.2	54.7	90.0	1.7
Suriname	Both	23.6	19.4	25.8	250.4	497.6	26.0	26.4	2.7	5.4	0.3	0.3
	F				107.4	442.2	11.9	25.0	1.2	4.8	0.1	0.3
	M				143.0	549.4	14.1	27.6	1.6	6.0	0.2	0.3
Sweden	Both	5.9	5.4	6.1	42.0	7.2	16.6	1.5	0.5	0.1	0.2	0.0
	F				16.8	5.9	4.8	0.9	0.2	0.1	0.1	0.0
	M				25.2	8.4	11.8	2.0	0.3	0.1	0.1	0.0
Switzerland	Both	10.2	8.6	10.4	43.3	10.0	8.4	1.0	0.5	0.1	0.1	0.0
	F				19.4	9.2	6.7	1.7	0.2	0.1	0.1	0.0
	M				23.9	10.7	1.7	0.4	0.3	0.1	0.0	0.0
Syrian Arab Republic	Both	39.4	49.2	37.4	18 592.1	885.5	4 051.8	84.6	203.6	9.7	47.8	1.0
	F				8 281.0	809.8	2 258.2	96.7	90.7	8.9	26.7	1.1
	M				10 311.1	957.4	1 793.6	73.1	112.9	10.5	21.0	0.9
Tajikistan	Both	40.0	37.8	42.8	49 135.8	4 153.5	3 787.6	200.5	536.9	45.4	44.1	2.3
	F				23 175.8	4 024.1	1 867.1	203.1	253.3	44.0	21.8	2.4
	M				25 959.9	4 276.3	1 920.5	198.1	283.6	46.7	22.4	2.3
Thailand	Both	26.2	25.2	26.6	13 971.7	370.8	2 541.5	30.3	152.1	4.0	29.8	0.4
	F				5 898.3	321.9	828.5	20.3	64.3	3.5	9.5	0.2
	M				8 073.4	417.1	1 713.1	39.7	87.9	4.5	20.3	0.5

The former Yugoslav Republic of Macedonia	Both	28.3	24.6	33.0	405.1	343.4	26.1	11.3	4.4	3.7	0.2	0.1
	F				194.7	339.1	2.9	2.5	2.1	3.7	0.0	0.0
	M				210.4	347.4	23.3	19.6	2.3	3.7	0.2	0.2
Timor-Leste	Both	17.9	17.2	18.2	7 316.2	3 549.9	295.4	84.7	80.1	38.8	3.5	1.0
	F				3 747.9	3 710.8	161.7	94.6	41.0	40.6	1.9	1.1
	M				3 568.3	3 395.4	133.7	75.1	39.0	37.1	1.6	0.9
Togo	Both	32.7	35.1	31.2	65 377.8	5 560.0	9 176.9	457.6	718.5	61.1	110.3	5.5
	F				28 670.6	4 891.1	5 227.8	522.8	315.2	53.8	62.8	6.3
	M				36 707.2	6 224.8	3 949.1	392.7	403.3	68.4	47.5	4.7
Tonga	Both	10.1	9.9	10.2	41.9	330.0	5.6	21.4	0.5	3.6	0.1	0.2
	F				23.9	386.3	2.2	17.3	0.3	4.2	0.0	0.2
	M				18.0	276.5	3.4	25.3	0.2	3.0	0.0	0.3
Trinidad and Tobago	Both	22.0	20.9	22.4	464.1	490.8	27.0	14.3	5.0	5.3	0.3	0.2
	F				213.9	459.9	7.0	7.5	2.3	5.0	0.1	0.1
	M				250.2	520.7	20.0	21.0	2.7	5.7	0.2	0.2
Tunisia	Both	35.7	35.5	35.7	5 505.9	523.6	694.4	41.5	60.0	5.7	7.9	0.5
	F				2 825.9	550.1	414.5	50.6	30.8	6.0	4.8	0.6
	M				2 680.0	498.4	280.0	32.7	29.2	5.4	3.1	0.4
Turkey	Both	42.0	43.2	41.2	17 035.1	251.5	2 769.7	20.8	184.7	2.7	31.2	0.2
	F				8 288.6	250.6	1 223.3	18.7	89.9	2.7	13.6	0.2
	M				8 746.5	252.2	1 546.4	22.7	94.7	2.7	17.5	0.3
Turkmenistan	Both	19.0	18.0	24.2	19 238.8	2 712.4	641.9	61.9	210.6	29.7	7.6	0.7
	F				8 186.1	2 343.3	273.3	53.4	89.6	25.7	3.2	0.6
	M				11 052.7	3 070.5	368.7	70.3	121.0	33.6	4.4	0.8
Tuvalu	Both	11.4	11.4	NA	NA	NA	NA	NA	NA	NA	NA	NA
	F				NA	NA	NA	NA	NA	NA	NA	NA

	M				NA	NA	NA	NA	NA	NA	NA	NA
Uganda	Both	48.4	47.1	48.7	405 794.7	5 270.8	54 975.3	450.3	4 456.2	57.9	659.9	5.4
	F				187 496.0	4 917.1	26 635.4	439.5	2 057.7	54.0	319.6	5.3
	M				218 298.8	5 618.0	28 339.8	460.9	2 398.5	61.7	340.3	5.5
Ukraine	Both	18.3	16.7	19.4	5 379.3	230.5	494.5	11.2	58.1	2.5	4.8	0.1
	F				2 864.5	253.2	251.9	11.7	31.0	2.7	2.5	0.1
	M				2 514.7	209.1	242.7	10.6	27.1	2.3	2.3	0.1
United Arab Emirates	Both	39.4	40.2	37.2	826.1	178.1	63.3	7.7	8.8	1.9	0.6	0.1
	F				464.4	204.5	21.0	5.2	5.0	2.2	0.2	0.0
	M				361.7	152.7	42.3	10.1	3.8	1.6	0.4	0.1
United Kingdom	Both	10.5	8.4	10.6	1,201.2	30.0	218.7	2.9	13.1	0.3	2.5	0.0
	F				551.8	28.3	98.1	2.6	6.0	0.3	1.1	0.0
	M				649.4	31.7	120.7	3.1	7.1	0.3	1.4	0.0
United Republic of Tanzania	Both	25.6	26.4	25.1	349 813.8	3 623.0	38 743.3	251.6	3 841.4	39.8	461.5	3.0
	F				170 825.3	3 572.0	19 573.9	255.0	1 875.9	39.2	233.0	3.0
	M				178 988.5	3 673.0	19 169.4	248.2	1 965.5	40.3	228.5	3.0
United States of America	Both	7.4	6.7	7.6	4 106.7	20.9	780.7	1.9	44.0	0.2	7.9	0.0
	F				1 880.0	19.6	359.9	1.8	20.2	0.2	3.6	0.0
	M				2 226.8	22.2	420.8	2.0	23.8	0.2	4.3	0.0
Uruguay	Both	8.6	8.0	8.7	132.5	55.3	10.8	2.2	1.4	0.6	0.1	0.0
	F				57.9	49.3	3.0	1.3	0.6	0.5	0.0	0.0
	M				74.7	61.1	7.8	3.1	0.8	0.7	0.1	0.0
Uzbekistan	Both	25.3	22.3	28.9	42 403.4	1 331.7	6 865.0	122.3	462.4	14.5	81.3	1.4
	F				19 002.0	1 238.2	3 183.6	116.4	207.2	13.5	37.7	1.4
	M				23 401.4	1 418.6	3 681.4	127.9	255.2	15.5	43.6	1.5
Vanuatu	Both	10.3	10.1	11.0	244.9	711.6	18.5	29.2	2.7	7.8	0.2	0.3

	F				115.3	693.7	6.7	22.0	1.3	7.6	0.1	0.3
	M				129.6	728.3	11.8	35.7	1.4	7.9	0.1	0.4
Venezuela (Bolivarian Republic of)	Both	15.8	14.6	16.8	13 427.7	451.5	945.7	16.2	146.6	4.9	11.2	0.2
	F				5 822.2	400.5	399.7	14.0	63.6	4.4	4.7	0.2
	M				7 605.5	500.2	546.0	18.3	83.1	5.5	6.5	0.2
Viet Nam	Both	29.7	26.6	30.1	89 655.6	1 155.2	2 679.9	19.1	979.7	12.6	30.0	0.2
	F				35 201.1	958.1	875.3	13.0	384.5	10.5	9.4	0.1
	M				54 454.5	1 332.5	1 804.7	24.7	595.2	14.6	20.7	0.3
Yemen	Both	45.0	46.5	44.3	221 360.4	5 431.9	6 609.1	94.0	2 427.7	59.6	77.4	1.1
	F				126 048.6	6 323.9	3 465.8	100.6	1 382.6	69.4	40.7	1.2
	M				95 311.9	4 578.0	3 143.4	87.7	1 045.1	50.2	36.7	1.0
Zambia	Both	24.7	25.2	23.8	103 337.7	3 664.3	9 846.4	211.2	1 134.4	40.2	117.7	2.5
	F				49 165.1	3 519.4	4 660.9	201.2	539.9	38.6	55.7	2.4
	M				54 172.6	3 806.6	5 185.5	221.2	594.5	41.8	62.0	2.6
Zimbabwe	Both	19.4	19.5	19.1	69 229.9	2 726.2	9 044.7	218.3	760.2	29.9	108.5	2.6
	F				34 940.5	2 765.4	4 816.1	233.0	383.8	30.4	57.8	2.8
	M				34 289.5	2 687.4	4 228.6	203.6	376.4	29.5	50.7	2.4

NA, not available

F, females; M, males

Table 5. Exposure of children to household PM_{2.5} and burden of disease, by country, 2016

Country	Sex	Households that rely primarily on clean cooking fuels (%)	Households that rely primarily on polluting fuels (%)	No. of DALYs (< 5 years)	DALYs rate per 100 000 (< 5 year)	No. of DALYs (5 - 14 years)	DALYs rate per 100 000 (5 - 14 years)	No. of deaths (< 5 years)	Death rate per 100 000 (< 5 years)	No. of deaths (5 - 14 years)	Death rate per 100 000 (5 - 14 years)
Afghanistan	Both	<5	68	536 072.8	10 244.4	38 386.5	385.1	5 895.7	112.7	456.2	4.6
	F			280 259.1	11 015.8	23 334.5	480.5	3 084.9	121.3	277.7	5.7
	M			255 813.7	9 514.4	15 052.0	294.5	2 810.8	104.5	178.5	3.5
Albania	Both	77	<5	961.1	541.9	157.8	46.2	10.4	5.8	1.8	0.5
	F			455.8	531.8	116.4	71.2	4.9	5.7	1.4	0.8
	M			505.4	551.3	41.4	23.3	5.4	5.9	0.4	0.3
Algeria	Both	93	7	20 107.2	427.9	885.4	12.5	220.2	4.7	10.3	0.1
	F			11 836.2	514.7	493.3	14.2	129.7	5.6	5.7	0.2
	M			8 271.0	344.7	392.1	10.9	90.5	3.8	4.5	0.1
Andorra	Both	>95	<5	NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA
Angola	Both	<5	52	611 101.3	11 580.2	29 091.3	352.5	6 723.9	127.4	346.9	4.2
	F			320 272.3	12 190.6	15 335.7	368.1	3 526.9	134.2	182.8	4.4
	M			290 829.1	10 975.1	13 755.6	336.6	3 197.0	120.6	164.2	4.0
Antigua and Barbuda	Both	>95	<5	0.5	5.8	0.3	1.6	0.0	0.1	0.0	0.0
	F			0.2	4.6	0.0	0.1	0.0	0.0	0.0	0.0
	M			0.3	7.1	0.2	3.0	0.0	0.1	0.0	0.0
Argentina	Both	>95	<5	979.7	26.2	99.4	1.4	10.7	0.3	1.2	0.0
	F			445.7	24.3	55.8	1.6	4.8	0.3	0.7	0.0

	M			534.1	28.1	43.5	1.2	5.8	0.3	0.5	0.0
Armenia	Both	>95	<5	169.1	83.6	21.2	5.6	1.8	0.9	0.2	0.1
	F			78.2	82.6	10.6	6.0	0.9	0.9	0.1	0.1
	M			90.9	84.6	10.6	5.2	1.0	0.9	0.1	0.1
Australia	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Austria	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Azerbaijan	Both	>95	<5	3 262.8	366.4	286.8	21.0	35.6	4.0	3.4	0.2
	F			1 677.6	404.9	140.6	22.3	18.3	4.4	1.6	0.3
	M			1 585.1	332.9	146.2	20.0	17.3	3.6	1.7	0.2
Bahamas	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Bahrain	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Bangladesh	Both	<5	82	791 065.4	5 192.2	33 263.4	104.4	8 655.5	56.8	391.7	1.2
	F			355 050.2	4 764.0	20 364.5	130.7	3 885.5	52.1	241.0	1.5
	M			436 015.2	5 602.4	12 898.9	79.3	4 770.0	61.3	150.8	0.9
Barbados	Both	>95	<5	0.7	4.0	0.3	0.9	0.0	0.0	0.0	0.0
	F			0.3	3.9	0.1	0.3	0.0	0.0	0.0	0.0
	M			0.4	4.2	0.3	1.4	0.0	0.0	0.0	0.0
Belarus	Both	>95	<5	52.1	9.0	8.6	0.9	0.5	0.1	0.1	0.0
	F			23.0	8.2	4.5	0.9	0.2	0.1	0.0	0.0
	M			29.1	9.8	4.1	0.8	0.3	0.1	0.0	0.0

Belgium	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Belize	Both	85	<5	124.2	308.1	14.4	18.8	1.3	3.3	0.2	0.2
	F			61.2	306.9	6.3	16.7	0.7	3.3	0.1	0.2
	M			63.0	309.3	8.1	20.7	0.7	3.4	0.1	0.2
Benin	Both	6	94	285 483.2	16 079.5	28 648.8	993.0	3 139.6	176.8	342.7	11.9
	F			150 504.4	17 218.8	15 313.6	1 076.7	1 656.1	189.5	183.0	12.9
	M			134 978.8	14 974.7	13 335.2	911.7	1 483.5	164.6	159.7	10.9
Bhutan	Both	52	<5	2 332.0	3 340.2	227.3	156.7	25.5	36.5	2.7	1.9
	F			1 049.3	3 057.2	130.9	183.3	11.5	33.4	1.6	2.2
	M			1 282.7	3 613.9	96.5	130.9	14.0	39.5	1.2	1.6
Bolivia (Plurinational State of)	Both	80	<5	23 378.9	1 967.1	2 935.6	127.7	256.1	21.5	35.2	1.5
	F			10 966.6	1 884.1	1 503.4	133.1	120.1	20.6	18.0	1.6
	M			12 412.3	2 046.7	1 432.2	122.5	135.9	22.4	17.2	1.5
Bosnia and Herzegovina	Both	63	<5	291.9	186.3	30.1	8.8	3.1	2.0	0.2	0.1
	F			136.3	179.4	13.2	7.9	1.4	1.9	0.1	0.1
	M			155.6	192.7	16.9	9.6	1.6	2.0	0.1	0.1
Botswana	Both	64	<5	5 980.2	2 307.0	599.3	132.5	65.6	25.3	7.2	1.6
	F			2 885.6	2 249.3	291.2	129.7	31.6	24.7	3.5	1.5
	M			3 094.6	2 363.6	308.1	135.2	34.0	25.9	3.7	1.6
Brazil	Both	>95	<5	19 393.6	130.0	1 711.1	5.5	210.8	1.4	19.6	0.1
	F			8 962.2	123.0	888.5	5.8	97.4	1.3	10.1	0.1
	M			10 431.4	136.7	822.6	5.2	113.5	1.5	9.5	0.1
Brunei Darussalam	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

Bulgaria	Both	89	<5	400.5	123.5	74.2	10.9	4.3	1.3	0.9	0.1
	F			179.1	113.7	37.1	11.2	1.9	1.2	0.4	0.1
	M			221.4	132.8	37.1	10.6	2.4	1.4	0.4	0.1
Burkina Faso	Both	9	91	365 511.5	11 349.0	45 373.5	865.5	4 019.0	124.8	543.0	10.4
	F			173 377.7	10 959.8	25 996.9	1 009.9	1 907.4	120.6	311.0	12.1
	M			192 133.8	11 724.7	19 376.6	726.2	2 111.6	128.9	232.0	8.7
Burundi	Both	<5	>95	253 667.5	13 341.5	44 100.4	1 563.2	2 786.7	146.6	529.3	18.8
	F			121 156.3	12 827.4	24 410.2	1 724.6	1 331.5	141.0	292.9	20.7
	M			132 511.3	13 848.9	19 690.1	1 400.6	1 455.2	152.1	236.4	16.8
Cabo Verde	Both	71	<5	749.1	1,371.2	34.9	31.5	8.2	15.0	0.4	0.4
	F			338.5	1,254.0	17.9	32.5	3.7	13.7	0.2	0.4
	M			410.6	1,485.6	17.0	30.5	4.5	16.2	0.2	0.4
Cambodia	Both	<5	82	85 859.8	4 874.9	7 697.8	241.6	937.4	53.2	90.6	2.8
	F			38 932.6	4 493.1	3 506.9	224.3	425.3	49.1	41.2	2.6
	M			46 927.2	5 244.6	4 190.9	258.2	512.1	57.2	49.4	3.0
Cameroon	Both	<5	77	467 990.2	12 301.8	85 522.5	1 367.9	5 143.3	135.2	1 023.5	16.4
	F			211 652.8	11 239.6	43 030.6	1 387.1	2 327.0	123.6	515.0	16.6
	M			256 337.4	13 342.8	42 491.9	1 348.9	2 816.3	146.6	508.5	16.1
Canada	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Central African Republic	Both	<5	>95	172 005.1	23 547.7	7 977.6	629.0	1 888.8	258.6	95.3	7.5
	F			87 895.5	24 102.4	4 355.6	682.3	965.9	264.9	52.0	8.1
	M			84 109.6	22 994.7	3 622.0	575.0	922.9	252.3	43.3	6.9
Chad	Both	<5	>95	932 652.8	34 978.1	48 074.9	1 149.6	10 284.5	385.7	577.5	13.8
	F			448 919.3	33 968.7	27 053.5	1 303.9	4 952.6	374.7	325.1	15.7
	M			483 733.4	35 970.1	21 021.3	997.7	5 332.0	396.5	252.4	12.0
Chile	Both	92	8	520.2	43.9	39.3	1.6	5.6	0.5	0.4	0.0

	F			233.4	40.2	20.7	1.7	2.5	0.4	0.2	0.0
	M			286.8	47.5	18.6	1.5	3.1	0.5	0.2	0.0
China	Both	59	<5	641 169.7	744.0	47 970.3	29.4	6 973.0	8.1	530.3	0.3
	F			283 636.2	709.0	28 360.7	37.6	3 074.5	7.7	302.9	0.4
	M			357 533.4	774.3	19 609.5	22.4	3 898.6	8.4	227.4	0.3
Colombia	Both	92	8	9 831.5	264.9	964.0	12.2	107.4	2.9	11.5	0.1
	F			4 571.0	251.7	455.5	11.8	49.9	2.7	5.4	0.1
	M			5 260.6	277.5	508.6	12.6	57.5	3.0	6.1	0.2
Comoros	Both	9	91	16 620.7	13 950.0	902.5	453.7	182.6	153.2	10.8	5.4
	F			8 150.6	13 945.8	457.2	467.8	89.6	153.2	5.4	5.6
	M			8 470.1	13 954.1	445.4	440.2	93.0	153.2	5.3	5.3
Congo	Both	<5	76	59 422.8	7 209.3	4 250.9	315.3	652.3	79.1	51.0	3.8
	F			29 124.4	7 134.4	2 502.1	373.1	320.0	78.4	30.0	4.5
	M			30 298.3	7 282.7	1 748.8	258.1	332.3	79.9	21.0	3.1
Cook Islands	Both	84	<5	NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA
Costa Rica	Both	93	7	216.4	62.6	27.1	3.8	2.3	0.7	0.3	0.0
	F			101.0	59.8	15.8	4.5	1.1	0.6	0.2	0.0
	M			115.4	65.3	11.3	3.1	1.2	0.7	0.1	0.0
Côte d'Ivoire	Both	<5	82	615 560.0	15 944.6	89 345.5	1 434.5	6 760.8	175.1	1 073.6	17.2
	F			272 639.3	14 220.9	51 471.6	1 655.4	2 995.7	156.3	618.4	19.9
	M			342 920.7	17 645.1	37 873.9	1 214.4	3 765.2	193.7	455.2	14.6
Croatia	Both	93	7	53.0	27.0	6.7	1.6	0.6	0.3	0.1	0.0
	F			21.8	22.8	0.5	0.3	0.2	0.2	0.0	0.0
	M			31.2	30.9	6.2	2.9	0.3	0.3	0.1	0.0
Cuba	Both	79	<5	1 300.7	204.4	96.7	7.9	14.1	2.2	1.0	0.1
	F			598.4	193.3	63.2	10.7	6.5	2.1	0.7	0.1

	M			702.2	214.9	33.4	5.3	7.6	2.3	0.3	0.1
Cyprus	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Czechia	Both	>95	<5	57.0	10.7	16.1	1.5	0.6	0.1	0.2	0.0
	F			23.5	9.0	6.6	1.3	0.3	0.1	0.1	0.0
	M			33.5	12.2	9.5	1.7	0.4	0.1	0.1	0.0
Democratic People's Republic of Korea	Both	<5	89	51 126.5	2 961.4	3 388.3	95.0	558.0	32.3	39.0	1.1
	F			22 540.8	2 673.5	1 948.9	111.6	245.6	29.1	22.4	1.3
	M			28 585.7	3 236.2	1 439.5	79.0	312.4	35.4	16.6	0.9
Democratic Republic of the Congo	Both	<5	>95	2 447 746.5	16 888.0	185 810.5	844.7	26 835.8	185.2	2 237.9	10.2
	F			1 219 830.4	16 999.8	106 474.9	975.1	13 378.4	186.4	1 281.9	11.7
	M			1 227 916.2	16 778.3	79 335.6	716.2	13 457.4	183.9	956.0	8.6
Denmark	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Djibouti	Both	<5	88	9 180.7	8 975.9	2 082.3	1 066.8	100.4	98.2	25.1	12.9
	F			4 237.7	8 403.8	1 059.7	1 099.0	46.4	91.9	12.8	13.2
	M			4 943.0	9 532.2	1 022.6	1 035.2	54.1	104.3	12.3	12.5
Dominica	Both	91	9	NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA
Dominican Republic	Both	90	<5	7 409.4	699.3	381.7	18.2	81.2	7.7	4.4	0.2
	F			2 952.5	569.0	196.5	19.1	32.3	6.2	2.3	0.2
	M			4 456.9	824.3	185.2	17.4	48.8	9.0	2.1	0.2

Ecuador	Both	>95	<5	4 218.9	261.9	325.8	10.5	46.2	2.9	3.9	0.1
	F			1 950.9	247.9	167.4	11.1	21.4	2.7	2.0	0.1
	M			2 268.0	275.2	158.4	10.0	24.8	3.0	1.9	0.1
Egypt	Both	>95	<5	15 687.4	121.8	3 820.6	20.0	171.4	1.3	45.5	0.2
	F			6 859.8	110.0	1 853.4	20.0	75.0	1.2	22.0	0.2
	M			8 827.6	133.0	1 967.2	20.0	96.4	1.5	23.5	0.2
El Salvador	Both	86	<5	2 740.1	475.3	367.4	31.0	29.9	5.2	4.5	0.4
	F			1 067.1	379.0	145.4	25.0	11.6	4.1	1.8	0.3
	M			1 673.0	567.2	222.0	36.7	18.3	6.2	2.7	0.5
Equatorial Guinea	Both	<5	66	22 068.8	12,149.8	2,029.8	739.2	242.0	133.2	24.3	8.8
	F			9 464.0	10 556.8	1 171.0	864.3	103.9	115.8	14.0	10.3
	M			12 604.9	13 702.3	858.9	617.4	138.2	150.2	10.3	7.4
Eritrea	Both	<5	84	67 043.0	9 014.3	8 238.4	614.8	736.1	99.0	98.2	7.3
	F			31 001.5	8 514.6	3 980.0	605.8	340.4	93.5	47.4	7.2
	M			36 041.5	9 493.6	4 258.3	623.5	395.7	104.2	50.9	7.4
Estonia	Both	93	7	13.1	19.4	7.8	5.4	0.1	0.2	0.1	0.1
	F			6.1	18.6	3.3	4.7	0.1	0.2	0.0	0.0
	M			7.0	20.1	4.5	6.0	0.1	0.2	0.0	0.1
eSwatini	Both	50	50	14 882.5	8 286.2	1 432.7	444.1	163.0	90.8	17.1	5.3
	F			6 941.0	7 780.0	784.2	487.8	76.0	85.2	9.4	5.8
	M			7 941.5	8 785.8	648.4	400.6	87.0	96.2	7.7	4.8
Ethiopia	Both	<5	>95	1 532 998.5	10 100.7	215 306.3	800.1	16 851.7	111.0	2 583.7	9.6
	F			683 480.8	9 141.6	106 266.4	798.3	7 513.1	100.5	1 274.3	9.6
	M			849 517.7	11 031.9	109 039.9	801.8	9 338.6	121.3	1 309.4	9.6
Fiji	Both	<5	60	1 801.2	2 080.3	230.9	135.5	19.6	22.7	2.7	1.6
	F			872.1	2 071.4	124.3	150.9	9.5	22.6	1.5	1.8
	M			929.1	2 088.8	106.5	121.1	10.1	22.8	1.3	1.4
Finland	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
France	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gabon	Both	79	<5	6 915.9	2 527.6	949.1	217.5	75.9	27.7	11.6	2.7
	F			3 141.4	2 321.8	489.8	226.5	34.5	25.5	6.0	2.8
	M			3 774.5	2 729.0	459.4	208.7	41.4	29.9	5.6	2.6
Gambia	Both	<5	>95	36 698.0	10 180.3	3 274.6	576.8	404.1	112.1	39.1	6.9
	F			17 412.6	9 757.6	1 959.7	696.3	191.9	107.5	23.4	8.3
	M			19 285.3	10 594.6	1 315.0	459.4	212.2	116.6	15.7	5.5
Georgia	Both	78	<5	766.2	282.5	260.6	55.1	8.3	3.1	3.1	0.7
	F			381.7	292.2	104.7	46.9	4.1	3.2	1.2	0.5
	M			384.6	273.5	156.0	62.5	4.2	3.0	1.9	0.7
Germany	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ghana	Both	<5	78	272 401.1	6 667.9	27 499.1	403.0	2 992.1	73.2	329.5	4.8
	F			121 158.7	6 057.1	14 732.9	441.5	1 330.6	66.5	176.5	5.3
	M			151 242.3	7 253.8	12 766.2	366.1	1 661.5	79.7	153.0	4.4
Greece	Both	94	6	85.5	18.0	12.8	1.1	0.9	0.2	0.1	0.0
	F			39.0	17.0	6.3	1.1	0.4	0.2	0.1	0.0
	M			46.6	19.0	6.5	1.1	0.5	0.2	0.1	0.0
Grenada	Both	>95	<5	13.7	138.9	0.4	2.2	0.1	1.5	0.0	0.0
	F			6.4	134.0	0.0	0.4	0.1	1.5	0.0	0.0
	M			7.3	143.5	0.4	4.0	0.1	1.6	0.0	0.0
Guatemala	Both	<5	55	71 014.1	3 510.9	5 243.6	134.8	776.9	38.4	62.6	1.6
	F			32 910.2	3 327.5	2 746.5	144.2	360.1	36.4	32.7	1.7

	M			38 103.9	3 686.4	2 497.2	125.7	416.9	40.3	29.9	1.5
Guinea	Both	<5	>95	339 682.9	17 130.7	36 814.8	1 122.0	3 732.4	188.2	440.8	13.4
	F			160 848.1	16 327.5	23 119.8	1 419.1	1 768.1	179.5	276.6	17.0
	M			178 834.9	17 923.7	13 695.0	829.0	1 964.3	196.9	164.2	9.9
Guinea-Bissau	Both	<5	>95	51 497.1	17 706.4	2 846.0	612.3	567.9	195.3	34.1	7.3
	F			24 469.1	16 900.6	1 545.1	665.2	269.8	186.3	18.5	8.0
	M			27 028.0	18 505.3	1 300.8	559.5	298.1	204.1	15.6	6.7
Guyana	Both	74	<5	931.1	1 218.4	156.8	103.8	10.2	13.3	1.9	1.2
	F			431.6	1 158.8	73.2	99.6	4.7	12.7	0.9	1.2
	M			499.5	1 275.0	83.6	107.8	5.5	13.9	1.0	1.3
Haiti	Both	<5	>95	189 093.2	15 330.7	13 926.5	584.4	2 075.7	168.3	166.6	7.0
	F			85 223.7	14 094.7	6 174.6	526.4	935.6	154.7	73.9	6.3
	M			103 869.6	16 519.2	7 751.9	640.5	1 140.1	181.3	92.7	7.7
Honduras	Both	53	<5	14 417.5	1 515.7	556.3	27.9	157.5	16.6	6.4	0.3
	F			6 448.3	1 383.4	169.9	17.4	70.4	15.1	1.9	0.2
	M			7 969.2	1 642.8	386.4	38.1	87.1	18.0	4.6	0.5
Hungary	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Iceland	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
India	Both	<5	59	6 098 660.0	5 082.3	398 523.0	157.3	66 890.5	55.7	4 711.7	1.9
	F			3 286 253.8	5 780.6	232 061.8	193.8	36 073.1	63.5	2 758.9	2.3
	M			2 812 406.5	4 453.7	166 461.2	124.6	30 817.4	48.8	1 952.8	1.5
Indonesia	Both	58	<5	628 573.7	2 532.3	36 982.9	78.1	6 862.4	27.6	430.8	0.9
	F			308 557.5	2 544.4	18 211.6	78.7	3 370.4	27.8	211.2	0.9
	M			320 016.2	2 520.8	18 771.4	77.4	3 492.0	27.5	219.6	0.9

Iran (Islamic Republic of)	Both	>95	<5	4 347.9	63.7	178.3	1.5	47.4	0.7	2.0	0.0
	F			2 299.8	68.9	95.8	1.6	25.1	0.8	1.1	0.0
	M			2 048.1	58.8	82.5	1.3	22.3	0.6	0.9	0.0
Iraq	Both	>95	<5	14 770.1	257.4	292.2	3.1	161.6	2.8	3.3	0.0
	F			4 824.8	173.0	170.5	3.8	52.9	1.9	1.9	0.0
	M			9 945.3	337.3	121.7	2.5	108.7	3.7	1.4	0.0
Ireland	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Israel	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Italy	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Jamaica	Both	90	<5	267.6	130.5	39.8	8.7	2.9	1.4	0.4	0.1
	F			121.7	122.4	21.3	9.5	1.3	1.3	0.2	0.1
	M			145.8	138.1	18.6	8.0	1.6	1.5	0.2	0.1
Japan	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Jordan	Both	>95	<5	392.0	31.9	30.0	1.4	4.3	0.3	0.3	0.0
	F			187.9	31.3	18.6	1.7	2.0	0.3	0.2	0.0
	M			204.1	32.6	11.4	1.0	2.2	0.4	0.1	0.0
Kazakhstan	Both	95	5	2 738.7	137.1	181.1	6.2	29.8	1.5	2.1	0.1
	F			1 233.6	127.1	94.1	6.6	13.4	1.4	1.1	0.1
	M			1 505.1	146.7	87.0	5.8	16.4	1.6	1.0	0.1
Kenya	Both	<5	87	504 366.5	7 181.3	31 760.7	248.3	5 536.8	78.8	380.6	3.0

	F			244 476.8	7 034.4	13 165.4	207.5	2 684.7	77.2	157.4	2.5
	M			259 889.7	7 325.1	18 595.3	288.5	2 852.1	80.4	223.2	3.5
Kiribati	Both	6	94	1 420.1	9 803.2	80.4	317.4	15.6	107.4	0.9	3.7
	F			683.2	9 670.0	24.5	197.6	7.5	105.9	0.3	2.3
	M			736.9	9 929.9	55.9	431.9	8.1	108.9	0.7	5.1
Kuwait	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kyrgyzstan	Both	81	<5	11 263.5	1 481.1	453.0	40.6	123.1	16.2	5.2	0.5
	F			5 201.8	1 406.0	214.0	39.2	56.8	15.4	2.4	0.4
	M			6 061.8	1 552.2	238.9	41.9	66.2	17.0	2.8	0.5
Lao People's Democratic Republic	Both	6	94	90 213.0	11 783.1	6 330.0	426.9	988.7	129.1	74.9	5.1
	F			43 609.0	11 633.1	2 997.9	412.5	478.2	127.6	35.5	4.9
	M			46 604.0	11 927.1	3 332.1	440.7	510.4	130.6	39.5	5.2
Latvia	Both	95	5	30.7	31.7	1.6	0.8	0.3	0.3	0.0	0.0
	F			14.6	31.2	0.8	0.8	0.2	0.3	0.0	0.0
	M			16.0	32.1	0.8	0.7	0.2	0.3	0.0	0.0
Lebanon	Both	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA
Lesotho	Both	<5	64	34 605.3	12 103.2	2 399.1	482.6	379.0	132.5	28.7	5.8
	F			17 471.0	12 306.8	1 393.5	563.4	191.3	134.8	16.7	6.7
	M			17 134.3	11 902.4	1 005.5	402.6	187.6	130.3	12.0	4.8
Liberia	Both	<5	>95	89 766.3	12 546.2	7 222.4	588.4	983.2	137.4	87.0	7.1
	F			44 701.3	12 769.7	4 082.6	679.5	489.7	139.9	49.1	8.2
	M			45 065.0	12 332.1	3 139.7	501.0	493.6	135.1	37.8	6.0
Libya	Both	NA		NA	NA	NA	NA	NA	NA	NA	NA

	F			NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA
Lithuania	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Luxembourg	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Madagascar	Both	<5	>95	335 527.3	8 903.3	42 347.8	650.4	3 680.3	97.7	506.9	7.8
	F			165 601.0	8 898.1	20 830.2	643.6	1 817.2	97.6	248.8	7.7
	M			169 926.2	8 908.3	21 517.6	657.1	1 863.1	97.7	258.1	7.9
Malawi	Both	<5	>95	230 369.8	7 921.2	13 249.1	259.7	2 526.6	86.9	158.2	3.1
	F			106 489.4	7 407.4	6 170.7	243.3	1 167.9	81.2	73.3	2.9
	M			123 880.4	8 423.4	7 078.4	276.0	1 358.7	92.4	84.8	3.3
Malaysia	Both	>95	<5	993.0	38.0	337.5	6.7	10.7	0.4	4.0	0.1
	F			427.3	33.9	160.0	6.5	4.6	0.4	1.9	0.1
	M			565.7	41.8	177.5	6.8	6.1	0.5	2.1	0.1
Maldives	Both	94	6	29.5	75.6	4.2	6.9	0.3	0.8	0.0	0.1
	F			15.9	85.0	2.4	8.2	0.2	0.9	0.0	0.1
	M			13.6	66.9	1.8	5.8	0.1	0.7	0.0	0.1
Mali	Both	<5	>95	554 968.1	16 655.1	36 885.2	699.1	6 105.1	183.2	439.8	8.3
	F			282 341.5	17 255.2	21 778.6	838.9	3 107.1	189.9	259.7	10.0
	M			272 626.7	16 076.1	15 106.6	563.7	2 998.0	176.8	180.1	6.7
Malta	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Marshall Islands	Both	65	<5	NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA

	M			NA	NA	NA	NA	NA	NA	NA	NA
Mauritania	Both	<5	53	67 277.8	10 269.7	2 955.2	276.2	740.3	113.0	35.1	3.3
	F			27 641.6	8 597.0	1 738.2	329.9	304.2	94.6	20.6	3.9
	M			39 636.2	11 881.8	1 217.0	224.1	436.1	130.7	14.4	2.7
Mauritius	Both	93	7	86.9	127.6	15.3	9.0	0.9	1.4	0.2	0.1
	F			36.8	110.3	9.4	11.2	0.4	1.2	0.1	0.1
	M			50.1	144.2	6.0	6.9	0.5	1.6	0.1	0.1
Mexico	Both	85	<5	49 786.2	429.9	2 060.9	9.0	544.1	4.7	24.4	0.1
	F			23 025.6	406.9	1 010.1	9.0	251.7	4.4	11.9	0.1
	M			26 760.7	451.9	1 050.8	9.0	292.4	4.9	12.5	0.1
Micronesia (Federated States of)	Both	<5	88	587.9	5 048.6	52.7	223.7	6.4	55.3	0.6	2.6
	F			269.8	4 792.5	23.1	203.7	3.0	52.5	0.3	2.4
	M			318.1	5 288.2	29.6	242.4	3.5	57.9	0.3	2.9
Monaco	Both	>95	<5	NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA
Mongolia	Both	<5	57	6 965.2	1 894.9	624.9	120.3	75.9	20.6	7.2	1.4
	F			2 508.5	1 382.8	273.6	106.5	27.3	15.1	3.2	1.2
	M			4 456.7	2 393.8	351.2	133.7	48.6	26.1	4.1	1.6
Montenegro	Both	69	<5	29.7	81.8	13.8	17.6	0.3	0.8	0.1	0.2
	F			13.1	74.2	7.5	20.0	0.1	0.7	0.1	0.2
	M			16.6	88.9	6.3	15.4	0.2	0.9	0.1	0.1
Morocco	Both	>95	<5	7 844.1	223.6	325.9	5.2	85.8	2.4	3.8	0.1
	F			3 571.4	209.2	181.6	6.0	39.1	2.3	2.1	0.1
	M			4 272.7	237.1	144.3	4.5	46.7	2.6	1.7	0.1
Mozambique	Both	<5	>95	541 134.6	10 932.0	46 454.2	578.6	5 925.9	119.7	559.7	7.0
	F			265 861.7	10 823.9	23 688.1	591.9	2 912.0	118.6	285.2	7.1

	M			275 272.9	11 038.5	22 766.1	565.4	3,013.9	120.9	274.4	6.8
Myanmar	Both	<5	82	404 645.6	8 917.2	29 751.4	299.4	4 436.5	97.8	353.5	3.6
	F			180 837.7	8 025.9	11 230.6	227.3	1 985.8	88.1	133.2	2.7
	M			223 807.9	9 796.3	18 520.8	370.5	2 450.7	107.3	220.4	4.4
Namibia	Both	<5	58	21 020.8	6 108.6	1 863.9	326.6	230.8	67.1	22.4	3.9
	F			9 986.7	5 842.9	882.9	309.9	109.6	64.1	10.6	3.7
	M			11 034.0	6 370.7	981.0	343.2	121.2	70.0	11.8	4.1
Nauru	Both	91	9	NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA
Nepal	Both	<5	72	130 634.5	4 739.7	8 797.7	136.9	1 429.0	51.8	103.5	1.6
	F			58 115.5	4 352.2	4 660.9	148.8	635.7	47.6	55.0	1.8
	M			72 519.0	5 103.8	4 136.8	125.6	793.3	55.8	48.6	1.5
Netherlands	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
New Zealand	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Nicaragua	Both	52	<5	15 219.7	2 548.2	1 067.6	87.7	166.5	27.9	12.9	1.1
	F			5 848.9	2 002.6	534.8	90.5	64.0	21.9	6.4	1.1
	M			9 370.8	3 070.4	532.9	85.0	102.5	33.6	6.4	1.0
Niger	Both	<5	>95	823 082.0	19 514.2	114 793.9	1 861.6	9 079.8	215.3	1 381.6	22.4
	F			418 807.2	20 295.2	61 999.4	2 051.9	4 623.2	224.0	746.1	24.7
	M			404 274.8	18 766.0	52 794.6	1 678.8	4 456.6	206.9	635.5	20.2
Nigeria	Both	5	95	6 950 066.0	21 854.5	772 212.4	1 538.4	76 505.4	240.6	9 221.0	18.4
	F			3 204 320.0	20 665.3	458 454.4	1 865.4	35 295.5	227.6	5 472.3	22.3
	M			3 745 746.0	22 986.1	313 757.9	1 224.7	41 209.8	252.9	3 748.7	14.6

Niue	Both	93	7	NA	NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA	NA
Norway	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Oman	Both	95	5	250.4	62.6	23.5	4.1	2.7	0.7	0.3	0.0	0.0
	F			144.2	74.2	12.9	4.5	1.6	0.8	0.1	0.0	0.0
	M			106.3	51.6	10.6	3.7	1.1	0.6	0.1	0.0	0.0
Pakistan	Both	<5	57	2 365 748.2	9 477.2	110 327.6	259.9	25 933.0	103.9	1 316.7	3.1	3.1
	F			1 197 080.8	9 973.7	44 879.4	219.7	13 127.7	109.4	534.1	2.6	2.6
	M			1 168 667.5	9 017.3	65 448.2	297.2	12 805.3	98.8	782.7	3.6	3.6
Palau	Both	87	<5	NA	NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA	NA
Panama	Both	89	<5	1 907.1	491.1	165.4	22.8	20.8	5.4	2.0	0.3	0.3
	F			828.2	435.7	79.0	22.2	9.0	4.8	0.9	0.3	0.3
	M			1 079.0	544.1	86.4	23.3	11.8	5.9	1.0	0.3	0.3
Papua New Guinea	Both	<5	87	89 471.3	8 663.9	6 050.7	318.5	978.4	94.7	71.1	3.7	3.7
	F			39 006.5	7 821.9	2 230.1	242.5	426.4	85.5	26.1	2.8	2.8
	M			50 464.8	9 450.2	3 820.6	389.7	552.0	103.4	45.1	4.6	4.6
Paraguay	Both	66	<5	7 976.4	1 187.6	812.4	61.2	86.9	12.9	9.1	0.7	0.7
	F			3 582.2	1 088.3	415.6	63.8	38.9	11.8	4.6	0.7	0.7
	M			4 394.2	1 283.0	396.9	58.7	47.9	14.0	4.5	0.7	0.7
Peru	Both	75	<5	20 398.0	672.6	5,818.1	101.1	222.4	7.3	69.2	1.2	1.2
	F			9 150.4	616.4	2 798.7	99.3	99.8	6.7	33.3	1.2	1.2
	M			11 247.6	726.6	3 019.4	102.8	122.7	7.9	36.0	1.2	1.2
Philippines	Both	<5	57	455 244.2	3 948.2	65 457.6	304.5	4 989.2	43.3	788.6	3.7	3.7

	F			210 002.3	3 751.0	31 886.4	304.9	2 302.4	41.1	383.2	3.7
	M			245 242.0	4 134.3	33 571.1	304.1	2 686.8	45.3	405.4	3.7
Poland	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Portugal	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Qatar	Both	>95	<5	16.9	13.0	2.6	1.2	0.2	0.1	0.0	0.0
	F			6.6	10.4	1.2	1.0	0.1	0.1	0.0	0.0
	M			10.3	15.6	1.5	1.3	0.1	0.2	0.0	0.0
Republic of Korea	Both	>95	<5	119.3	5.4	29.0	0.6	1.2	0.1	0.2	0.0
	F			59.0	5.5	14.0	0.6	0.6	0.1	0.1	0.0
	M			60.3	5.2	15.0	0.6	0.6	0.1	0.1	0.0
Republic of Moldova	Both	92	8	957.3	439.4	60.7	14.4	10.4	4.8	0.7	0.2
	F			409.9	390.2	29.5	14.5	4.5	4.2	0.3	0.2
	M			547.4	485.3	31.2	14.4	6.0	5.3	0.4	0.2
Romania	Both	86	<5	6 372.2	674.9	630.6	30.3	69.4	7.4	7.3	0.4
	F			2 865.7	623.7	350.1	34.5	31.2	6.8	4.1	0.4
	M			3 506.5	723.4	280.6	26.2	38.2	7.9	3.3	0.3
Russian Federation	Both	>95	<5	2 056.0	21.5	340.2	2.2	22.1	0.2	3.6	0.0
	F			942.0	20.3	184.9	2.5	10.1	0.2	2.0	0.0
	M			1 114.0	22.7	155.4	2.0	12.0	0.2	1.6	0.0
Rwanda	Both	<5	>95	93 389.2	5 367.9	17 300.2	561.1	1 023.4	58.8	207.6	6.7
	F			42 201.2	4 870.1	8 381.9	542.1	462.3	53.4	100.5	6.5
	M			51 188.0	5 862.0	8 918.3	580.2	561.0	64.2	107.1	7.0
Saint Kitts and Nevis	Both	>95	<5	NA	NA	NA	NA	NA	NA	NA	NA

	F			NA	NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA	NA
Saint Lucia	Both	>95	<5	6.9	63.1	0.3	1.1	0.1	0.7	0.0	0.0	0.0
	F			3.4	62.0	0.2	1.9	0.0	0.7	0.0	0.0	0.0
	M			3.6	64.2	0.0	0.3	0.0	0.7	0.0	0.0	0.0
Saint Vincent and the Grenadines	Both	>95	<5	14.3	172.7	1.1	5.9	0.2	1.9	0.0	0.1	0.1
	F			6.9	169.3	0.6	6.5	0.1	1.8	0.0	0.1	0.1
	M			7.4	176.1	0.5	5.3	0.1	1.9	0.0	0.1	0.1
Samoa	Both	<5	68	311.1	1 324.6	43.0	88.9	3.4	14.4	0.5	1.0	1.0
	F			126.2	1 113.8	18.0	77.1	1.4	12.1	0.2	0.9	0.9
	M			184.9	1 521.0	25.0	99.8	2.0	16.5	0.3	1.2	1.2
San Marino	Both	>95	<5	NA	NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA	NA
Sao Tome and Principe	Both	<5	83	1 362.7	4 349.7	300.2	545.9	14.9	47.6	3.6	6.5	6.5
	F			564.0	3 632.4	151.3	554.3	6.2	39.7	1.8	6.6	6.6
	M			798.7	5 054.6	148.9	537.7	8.7	55.3	1.8	6.4	6.4
Saudi Arabia	Both	>95	<5	1 917.2	64.6	216.4	4.1	21.0	0.7	2.4	0.0	0.0
	F			938.7	64.2	127.9	4.9	10.3	0.7	1.4	0.1	0.1
	M			978.6	65.0	88.4	3.3	10.7	0.7	1.0	0.0	0.0
Senegal	Both	<5	68	161 812.9	6 359.8	20 185.9	494.5	1 776.0	69.8	242.4	5.9	5.9
	F			73 518.8	5 859.1	10 889.6	539.8	807.5	64.4	130.5	6.5	6.5
	M			88 294.1	6 846.9	9 296.2	450.2	968.6	75.1	111.9	5.4	5.4
Serbia	Both	76	<5	417.4	89.0	53.2	5.4	4.4	0.9	0.5	0.1	0.1
	F			186.2	81.3	24.1	5.0	2.0	0.9	0.2	0.0	0.0
	M			231.1	96.3	29.1	5.7	2.5	1.0	0.3	0.1	0.1
Seychelles	Both	90	<5	11.3	143.7	5.4	41.8	0.1	1.6	0.1	0.5	0.5

	F			4.5	118.5	2.6	40.8	0.0	1.3	0.0	0.5
	M			6.7	167.9	2.8	42.8	0.1	1.8	0.0	0.5
Sierra Leone	Both	<5	>95	203 840.7	17 867.4	22 848.1	1 144.6	2 232.3	195.7	272.1	13.6
	F			98 725.7	17 362.6	12 599.0	1 258.9	1 081.2	190.1	150.0	15.0
	M			105 115.1	18 369.1	10 249.0	1 029.6	1 151.0	201.1	122.2	12.3
Singapore	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Slovakia	Both	>95	<5	84.8	30.1	16.3	3.0	0.9	0.3	0.2	0.0
	F			38.5	28.0	9.5	3.5	0.4	0.3	0.1	0.0
	M			46.3	32.2	6.8	2.4	0.5	0.3	0.1	0.0
Slovenia	Both	>95	<5	3.9	3.6	2.0	1.0	0.0	0.0	0.0	0.0
	F			1.9	3.7	0.3	0.3	0.0	0.0	0.0	0.0
	M			1.9	3.5	1.7	1.6	0.0	0.0	0.0	0.0
Solomon Islands	Both	8	92	3 880.4	4 689.4	200.8	131.9	42.4	51.2	2.3	1.5
	F			1 901.6	4 743.8	87.0	118.1	20.8	51.8	1.0	1.4
	M			1 978.8	4 638.4	113.7	144.8	21.6	50.7	1.3	1.7
Somalia	Both	<5	>95	895 779.6	34 228.7	52 304.9	1 292.6	9 866.4	377.0	623.9	15.4
	F			432 336.2	33 319.0	28 148.6	1 397.7	4 762.6	367.0	335.6	16.7
	M			463 443.4	35 123.4	24 156.4	1 188.5	5 103.7	386.8	288.4	14.2
South Africa	Both	85	<5	126 567.7	2 218.6	8 149.6	76.6	1 383.1	24.2	97.4	0.9
	F			59 177.1	2 096.4	3 836.4	72.7	646.7	22.9	45.6	0.9
	M			67 390.7	2 338.3	4 313.1	80.5	736.4	25.6	51.7	1.0
South Sudan	Both	<5	>95	403 214.0	20 945.7	17 876.2	559.1	4 442.2	230.8	213.3	6.7
	F			194 474.8	20 508.5	9 406.2	595.5	2 143.0	226.0	112.2	7.1
	M			208 739.3	21 370.0	8 470.0	523.6	2 299.3	235.4	101.1	6.3
Spain	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0

	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Sri Lanka	Both	<5	74	7 689.0	480.1	2 436.8	70.6	83.0	5.2	28.1	0.8
	F			3 182.3	404.0	1 303.7	75.9	34.4	4.4	15.1	0.9
	M			4 506.7	553.7	1 133.1	65.5	48.6	6.0	13.0	0.7
Sudan	Both	<5	59	476 142.0	8 015.4	20 232.5	195.4	5 254.6	88.5	239.5	2.3
	F			270 676.2	9 270.5	10 540.7	206.8	2 987.3	102.3	124.9	2.5
	M			205 465.8	6 802.2	9 691.8	184.4	2 267.2	75.1	114.6	2.2
Suriname	Both	90	<5	146.9	291.9	14.7	14.9	1.6	3.2	0.2	0.2
	F			63.0	259.4	7.3	15.4	0.7	2.8	0.1	0.2
	M			83.9	322.3	7.4	14.5	0.9	3.5	0.1	0.2
Sweden	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Switzerland	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Syrian Arab Republic	Both	>95	<5	733.6	34.9	155.3	3.2	8.0	0.4	1.8	0.0
	F			326.8	32.0	92.9	4.0	3.6	0.4	1.1	0.0
	M			406.9	37.8	62.4	2.5	4.5	0.4	0.7	0.0
Tajikistan	Both	80	<5	34 122.4	2 884.4	2 555.6	135.3	372.8	31.5	29.8	1.6
	F			16 094.5	2 794.5	1 350.2	146.9	175.9	30.5	15.7	1.7
	M			18 027.9	2 969.7	1 205.4	124.3	196.9	32.4	14.0	1.4
Thailand	Both	74	<5	15 863.0	421.0	2 744.5	32.7	172.7	4.6	32.1	0.4
	F			6 696.7	365.5	977.7	24.0	73.0	4.0	11.2	0.3
	M			9 166.3	473.6	1 766.8	41.0	99.8	5.2	21.0	0.5
The former Yugoslav Republic of Macedonia	Both	66	<5	534.5	453.0	32.0	13.8	5.8	4.9	0.3	0.1
	F			256.9	447.3	3.9	3.5	2.8	4.8	0.0	0.0

	M			277.6	458.3	28.1	23.7	3.0	4.9	0.3	0.2
Timor-Leste	Both	7	93	23 025.5	11 172.3	917.4	262.9	251.9	122.2	10.9	3.1
	F			11 795.4	11 678.5	521.4	305.0	129.1	127.8	6.2	3.6
	M			11 230.1	10 685.8	395.9	222.5	122.8	116.9	4.7	2.6
Togo	Both	7	93	142 733.2	12 138.6	19 804.5	987.5	1 568.6	133.4	238.1	11.9
	F			62 593.9	10 678.3	11 690.4	1 169.1	688.1	117.4	140.4	14.0
	M			80 139.3	13 590.1	8 114.1	806.9	880.4	149.3	97.6	9.7
Tonga	Both	59	<5	136.4	1 072.9	17.6	67.2	1.5	11.6	0.2	0.8
	F			77.8	1 255.9	7.3	58.1	0.8	13.6	0.1	0.7
	M			58.6	898.9	10.3	75.6	0.6	9.8	0.1	0.9
Trinidad and Tobago	Both	>95	<5	22.9	24.2	1.2	0.7	0.2	0.3	0.0	0.0
	F			10.6	22.7	0.4	0.4	0.1	0.2	0.0	0.0
	M			12.4	25.7	0.9	0.9	0.1	0.3	0.0	0.0
Tunisia	Both	>95	<5	243.2	23.1	30.2	1.8	2.7	0.3	0.3	0.0
	F			124.8	24.3	19.2	2.3	1.4	0.3	0.2	0.0
	M			118.4	22.0	11.0	1.3	1.3	0.2	0.1	0.0
Turkey	Both	NA		NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA
Turkmenistan	Both	>95	<5	1 003.4	141.5	31.8	3.1	11.0	1.5	0.4	0.0
	F			427.0	122.2	14.9	2.9	4.7	1.3	0.2	0.0
	M			576.5	160.1	16.9	3.2	6.3	1.8	0.2	0.0
Tuvalu	Both	50	50	NA	NA	NA	NA	NA	NA	NA	NA
	F			NA	NA	NA	NA	NA	NA	NA	NA
	M			NA	NA	NA	NA	NA	NA	NA	NA
Uganda	Both	<5	>95	735 637.6	9 555.1	97 865.2	801.5	8 078.3	104.9	1 174.7	9.6
	F			339 898.6	8 913.8	49 416.8	815.4	3 730.2	97.8	593.0	9.8
	M			395 738.9	10 184.4	48 448.4	787.9	4 348.1	111.9	581.7	9.5

Ukraine	Both	>95	<5	1 408.3	60.3	125.4	2.8	15.2	0.7	1.2	0.0
	F			750.0	66.3	69.0	3.2	8.1	0.7	0.7	0.0
	M			658.4	54.7	56.4	2.5	7.1	0.6	0.5	0.0
United Arab Emirates	Both	>95	<5	53.1	11.4	3.8	0.5	0.6	0.1	0.0	0.0
	F			29.8	13.1	1.4	0.3	0.3	0.1	0.0	0.0
	M			23.2	9.8	2.4	0.6	0.2	0.1	0.0	0.0
United Kingdom	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
United Republic of Tanzania	Both	<5	>95	885 108.1	9 166.9	96 415.9	626.2	9 719.6	100.7	1 148.4	7.5
	F			432 226.7	9 038.0	50 695.0	660.5	4 746.4	99.2	603.5	7.9
	M			452 881.4	9 293.4	45 720.9	592.1	4 973.2	102.1	544.9	7.1
United States of America	Both	>95	<5	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	F			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
	M			0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Uruguay	Both	>95	<5	41.5	17.3	3.2	0.6	0.4	0.2	0.0	0.0
	F			18.1	15.4	1.0	0.4	0.2	0.2	0.0	0.0
	M			23.4	19.1	2.2	0.9	0.3	0.2	0.0	0.0
Uzbekistan	Both	92	8	16 830.9	528.6	2 626.3	46.8	183.6	5.8	31.1	0.6
	F			7 542.3	491.5	1 324.3	48.4	82.3	5.4	15.7	0.6
	M			9 288.6	563.1	1 302.1	45.2	101.3	6.1	15.4	0.5
Vanuatu	Both	<5	87	1 297.3	3 768.9	94.8	149.9	14.1	41.1	1.1	1.7
	F			610.8	3 674.3	36.1	119.6	6.7	40.1	0.4	1.4
	M			686.5	3 857.3	58.7	177.6	7.5	42.1	0.7	2.1
Venezuela (Bolivarian Republic of)	Both	>95	<5	2 899.1	97.5	195.5	3.4	31.7	1.1	2.3	0.0
	F			1 257.1	86.5	91.0	3.2	13.7	0.9	1.1	0.0

	M			1 642.1	108.0	104.5	3.5	17.9	1.2	1.3	0.0
Viet Nam	Both	67	<5	113 253.8	1 459.3	3 228.0	23.0	1 237.5	15.9	36.1	0.3
	F			44 466.4	1 210.3	1 146.8	17.0	485.7	13.2	12.3	0.2
	M			68 787.5	1 683.2	2 081.2	28.4	751.8	18.4	23.9	0.3
Yemen	Both	65	<5	224 534.9	5 509.8	6 560.1	93.3	2 462.5	60.4	76.8	1.1
	F			127 856.2	6 414.6	3 643.3	105.8	1 402.5	70.4	42.8	1.2
	M			96 678.7	4 643.6	2 916.8	81.3	1 060.1	50.9	34.0	0.9
Zambia	Both	<5	84	258 792.2	9 176.6	24 151.9	518.1	2 840.8	100.7	288.7	6.2
	F			123 126.0	8 813.7	11 972.1	516.8	1 352.0	96.8	143.0	6.2
	M			135 666.2	9 532.9	12 179.8	519.5	1 488.8	104.6	145.7	6.2
Zimbabwe	Both	<5	71	188 041.6	7 404.9	24 163.5	583.2	2 064.8	81.3	289.8	7.0
	F			94 904.9	7 511.4	13 445.1	650.6	1 042.4	82.5	161.3	7.8
	M			93 136.7	7 299.5	10 718.4	516.1	1 022.4	80.1	128.5	6.2

B, both sexes; F, females; M, males

Table 6. Joint effects of exposure of children to ambient and household PM_{2.5} and burden of disease, by country, 2016

Country	Sex	No. of DALYs (< 5 years)	DALYs rate per 100 000	No. of DALYs (5 -14)	DALYs rate per 100 000	No. of deaths (<5 years)	Death rate per 100 000	No. of deaths (5 -14)	Death rate per 100 000
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			(< 5 years)	years)	(5 -14 years)		(<5 years)	years)	(5 -14 years)
Afghanistan	Both	743 587.6	14 210.0	53 573.7	537.5	8 177.9	156.3	636.7	6.4
	F	388 748.0	15 280.0	31 906.4	657.0	4 279.1	168.2	379.7	7.8
	M	354 839.6	13 197.5	21 667.3	424.0	3 898.8	145.0	257.0	5.0
Albania	Both	1 532.4	863.9	251.5	73.7	16.5	9.3	2.9	0.8
	F	726.6	847.8	182.3	111.5	7.8	9.1	2.1	1.3
	M	805.7	878.9	69.1	38.9	8.7	9.5	0.7	0.4
Algeria	Both	91 680.0	1 951.0	4 110.7	58.1	1 003.8	21.4	47.6	0.7
	F	53 967.9	2 346.9	2 167.2	62.5	591.2	25.7	25.1	0.7
	M	37 712.1	1 571.6	1 943.5	53.8	412.6	17.2	22.5	0.6
Andorra	Both	NA	NA	NA	NA	NA	NA	NA	NA
	F	NA	NA	NA	NA	NA	NA	NA	NA
	M	NA	NA	NA	NA	NA	NA	NA	NA
Angola	Both	824 454.4	15 623.2	39 623.8	480.1	9 071.4	171.9	472.5	5.7
	F	432 088.5	16 446.7	20 416.8	490.0	4 758.3	181.1	243.3	5.8
	M	392 365.8	14 806.8	19 206.9	469.9	4 313.2	162.8	229.2	5.6
Antigua and Barbuda	Both	5.2	64.4	3.2	19.5	0.1	0.7	0.0	0.2
	F	2.0	50.1	0.1	1.3	0.0	0.5	0.0	0.0
	M	3.2	78.5	3.1	37.6	0.0	0.8	0.0	0.4
Argentina	Both	6 806.6	182.2	707.2	9.8	74.0	2.0	8.2	0.1
	F	3 096.2	168.7	370.7	10.4	33.7	1.8	4.3	0.1
	M	3 710.4	195.2	336.5	9.1	40.4	2.1	3.9	0.1
Armenia	Both	1 319.8	652.7	171.1	45.0	14.3	7.1	1.9	0.5
	F	610.4	644.3	78.9	44.6	6.6	7.0	0.9	0.5
	M	709.4	660.1	92.2	45.3	7.7	7.2	1.1	0.5
Australia	Both	228.6	14.7	47.2	1.6	2.4	0.2	0.5	0.0
	F	100.6	13.3	33.4	2.3	1.1	0.1	0.4	0.0
	M	128.0	16.1	13.7	0.9	1.3	0.2	0.1	0.0

Austria	Both	15.4	3.8	36.0	4.4	0.2	0.0	0.4	0.1
	F	7.4	3.7	35.3	8.9	0.1	0.0	0.4	0.1
	M	8.1	3.8	0.7	0.2	0.1	0.0	0.0	0.0
Azerbaijan	Both	12 697.7	1 425.9	1 152.9	84.6	138.4	15.5	13.5	1.0
	F	6 528.9	1 575.9	528.8	83.7	71.2	17.2	6.2	1.0
	M	6 168.9	1 295.4	624.1	85.4	67.3	14.1	7.3	1.0
Bahamas	Both	124.4	451.8	7.3	13.8	1.4	4.9	0.1	0.2
	F	59.8	446.5	3.1	11.9	0.6	4.8	0.0	0.1
	M	64.6	456.9	4.2	15.6	0.7	5.0	0.0	0.2
Bahrain	Both	110.7	103.6	79.8	43.7	1.2	1.1	0.9	0.5
	F	56.2	108.2	44.2	49.3	0.6	1.1	0.5	0.6
	M	54.5	99.4	35.6	38.3	0.6	1.0	0.4	0.4
Bangladesh	Both	1 049 873.4	6 890.9	44 378.0	139.3	11 487.3	75.4	522.6	1.6
	F	471 209.8	6 322.6	26 686.1	171.2	5 156.7	69.2	315.8	2.0
	M	578 663.6	7 435.2	17 691.9	108.7	6 330.6	81.3	206.8	1.3
Barbados	Both	19.4	112.3	10.2	27.2	0.2	1.2	0.1	0.3
	F	9.2	108.1	1.7	9.2	0.1	1.1	0.0	0.1
	M	10.2	116.4	8.5	44.3	0.1	1.2	0.1	0.5
Belarus	Both	461.0	79.6	78.1	8.0	4.8	0.8	0.6	0.1
	F	203.5	72.4	37.8	7.9	2.1	0.8	0.3	0.1
	M	257.6	86.3	40.3	8.0	2.7	0.9	0.3	0.1
Belgium	Both	147.6	23.0	22.0	1.7	1.6	0.2	0.2	0.0
	F	62.8	20.0	13.3	2.1	0.7	0.2	0.1	0.0
	M	84.8	25.7	8.6	1.3	0.9	0.3	0.1	0.0
Belize	Both	247.4	614.0	29.5	38.4	2.7	6.7	0.3	0.4
	F	122.0	611.5	12.3	32.5	1.3	6.6	0.1	0.4
	M	125.5	616.4	17.2	44.1	1.4	6.7	0.2	0.5
Benin	Both	348 671.9	19 638.6	35 215.4	1 220.6	3 834.5	216.0	421.2	14.6

	F	183 817.0	21 030.0	18 528.9	1 302.7	2 022.7	231.4	221.5	15.6
	M	164 855.0	18 289.2	16 686.5	1 140.8	1 811.8	201.0	199.8	13.7
Bhutan	Both	3 407.2	4 880.4	335.0	230.9	37.2	53.3	4.0	2.8
	F	1 533.1	4 466.9	188.2	263.5	16.7	48.8	2.3	3.2
	M	1 874.1	5 280.3	146.8	199.3	20.5	57.7	1.8	2.4
Bolivia (Plurinational State of)	Both	40 128.4	3 376.4	5 121.9	222.8	439.5	37.0	61.4	2.7
	F	18 823.5	3 234.0	2 526.0	223.6	206.2	35.4	30.3	2.7
	M	21 304.9	3 513.0	2 595.8	221.9	233.3	38.5	31.1	2.7
Bosnia and Herzegovina	Both	432.5	276.1	45.4	13.2	4.6	2.9	0.4	0.1
	F	201.9	265.9	19.2	11.6	2.1	2.8	0.1	0.1
	M	230.6	285.6	26.2	14.8	2.4	3.0	0.2	0.1
Botswana	Both	8 588.3	3 313.2	872.3	192.8	94.2	36.3	10.4	2.3
	F	4 144.1	3 230.3	411.9	183.5	45.4	35.4	4.9	2.2
	M	4 444.2	3 394.5	460.4	202.0	48.8	37.2	5.5	2.4
Brazil	Both	58 241.8	390.4	5 269.7	17.0	633.2	4.2	60.5	0.2
	F	26 914.8	369.4	2 578.1	16.9	292.4	4.0	29.4	0.2
	M	31 327.0	410.5	2 691.6	17.0	340.8	4.5	31.1	0.2
Brunei Darussalam	Both	6.8	19.7	3.8	5.9	0.1	0.2	0.0	0.1
	F	3.1	18.4	2.5	8.1	0.0	0.2	0.0	0.1
	M	3.7	20.9	1.3	3.8	0.0	0.2	0.0	0.0
Bulgaria	Both	1 471.8	454.0	280.1	41.1	16.0	4.9	3.2	0.5
	F	658.1	417.9	132.0	39.9	7.1	4.5	1.5	0.5
	M	813.7	488.0	148.2	42.3	8.8	5.3	1.7	0.5
Burkina Faso	Both	450 882.7	13 999.8	56 272.2	1 073.4	4 957.7	153.9	673.5	12.8
	F	213 872.9	13 519.7	31 756.9	1 233.7	2 353.0	148.7	379.9	14.8
	M	237 009.8	14 463.2	24 515.4	918.7	2 604.7	159.0	293.6	11.0

Burundi	Both	303 901.0	15 983.5	53 112.4	1 882.6	3 338.6	175.6	637.5	22.6
	F	145 148.7	15 367.7	28 995.1	2 048.5	1 595.2	168.9	347.9	24.6
	M	158 752.3	16 591.4	24 117.2	1 715.5	1 743.4	182.2	289.6	20.6
Cabo Verde	Both	1 256.2	2 299.4	59.4	53.6	13.7	25.1	0.7	0.6
	F	567.6	2 103.0	29.3	53.4	6.2	23.0	0.3	0.6
	M	688.6	2 491.3	30.1	53.9	7.5	27.2	0.4	0.6
Cambodia	Both	103 146.9	5 856.4	9 325.3	292.6	1 126.1	63.9	109.7	3.4
	F	46 771.4	5 397.7	4 176.7	267.1	510.9	59.0	49.0	3.1
	M	56 375.6	6 300.6	5 148.7	317.2	615.2	68.8	60.7	3.7
Cameroon	Both	641 356.5	16 858.9	118 525.0	1 895.7	7 048.7	185.3	1,418.4	22.7
	F	290 059.3	15 403.3	58 157.5	1 874.7	3 189.0	169.4	696.0	22.4
	M	351 297.2	18 285.7	60 367.5	1 916.4	3 859.6	200.9	722.4	22.9
Canada	Both	157.4	8.2	28.9	0.7	1.7	0.1	0.3	0.0
	F	72.7	7.7	16.9	0.9	0.8	0.1	0.2	0.0
	M	84.7	8.6	12.1	0.6	0.9	0.1	0.1	0.0
Central African Republic	Both	213 754.1	29 263.2	9 978.9	786.8	2 347.2	321.3	119.2	9.4
	F	109 229.5	29 952.5	5 358.5	839.4	1 200.4	329.2	64.0	10.0
	M	104 524.6	28 576.0	4 620.3	733.5	1 146.9	313.5	55.2	8.8
Chad	Both	1 177 331.0	44 154.5	61 071.6	1 460.4	12 982.7	486.9	733.7	17.5
	F	566 691.8	42 880.3	33 786.8	1 628.5	6 251.9	473.1	406.1	19.6
	M	610 639.2	45 406.7	27 284.8	1 294.9	6 730.8	500.5	327.6	15.5
Chile	Both	1 499.5	126.7	115.9	4.6	16.2	1.4	1.2	0.0
	F	672.8	115.9	57.8	4.7	7.3	1.3	0.6	0.0
	M	826.7	137.1	58.1	4.6	9.0	1.5	0.6	0.0
China	Both	1 046 128.8	1 213.9	79 016.5	48.4	11 377.2	13.2	874.8	0.5
	F	462 779.3	1 156.8	45 380.3	60.2	5 016.3	12.5	484.7	0.6
	M	583 349.5	1 263.4	33 636.2	38.3	6 360.9	13.8	390.1	0.4
Colombia	Both	23 646.1	637.0	2 382.2	30.1	258.3	7.0	28.4	0.4

	F	10 993.8	605.3	1 063.5	27.5	120.1	6.6	12.6	0.3
	M	12 652.3	667.4	1 318.7	32.7	138.2	7.3	15.8	0.4
Comoros	Both	19 120.9	16 048.4	1 043.8	524.7	210.0	176.3	12.4	6.3
	F	9 376.7	16 043.6	522.4	534.5	103.0	176.3	6.2	6.4
	M	9 744.2	16 053.1	521.4	515.3	107.0	176.3	6.2	6.2
Congo	Both	76 625.6	9 296.3	5 512.9	408.9	841.1	102.0	66.2	4.9
	F	37 555.9	9 199.9	3 189.4	475.6	412.7	101.1	38.3	5.7
	M	39 069.7	9 391.0	2 323.5	342.9	428.5	103.0	27.9	4.1
Cook Islands	Both	NA	NA	NA	NA	NA	NA	NA	NA
	F	NA	NA	NA	NA	NA	NA	NA	NA
	M	NA	NA	NA	NA	NA	NA	NA	NA
Costa Rica	Both	589.1	170.4	75.0	10.4	6.3	1.8	0.8	0.1
	F	274.9	162.9	41.6	11.8	2.9	1.7	0.4	0.1
	M	314.2	177.6	33.3	9.0	3.4	1.9	0.4	0.1
Côte d'Ivoire	Both	738 191.4	19 121.1	107 634.4	1 728.2	8 107.7	210.0	1 293.4	20.8
	F	326 954.3	17 053.9	61 198.6	1 968.2	3 592.5	187.4	735.3	23.6
	M	411 237.1	21 160.3	46 435.8	1488.9	4 515.2	232.3	558.1	17.9
Croatia	Both	151.8	77.3	20.8	4.9	1.6	0.8	0.2	0.0
	F	62.4	65.4	1.5	0.7	0.7	0.7	0.0	0.0
	M	89.4	88.6	19.3	8.9	1.0	0.9	0.2	0.1
Cuba	Both	2 327.2	365.7	173.8	14.2	25.3	4.0	1.9	0.2
	F	1 070.7	345.9	111.0	18.7	11.6	3.8	1.2	0.2
	M	1 256.4	384.4	62.8	10.0	13.7	4.2	0.6	0.1
Cyprus	Both	5.7	8.7	0.2	0.1	0.1	0.1	0.0	0.0
	F	2.7	8.6	0.1	0.1	0.0	0.1	0.0	0.0
	M	3.0	8.8	0.1	0.1	0.0	0.1	0.0	0.0
Czechia	Both	345.7	64.7	102.9	9.5	3.7	0.7	1.1	0.1
	F	142.3	54.8	38.5	7.3	1.5	0.6	0.4	0.1

	M	203.4	74.1	64.4	11.6	2.2	0.8	0.7	0.1
Democratic People's Republic of Korea	Both	62 342.0	3 611.1	4 152.2	116.4	680.4	39.4	47.8	1.3
	F	27 485.5	3 260.0	2 354.5	134.8	299.5	35.5	27.0	1.5
	M	34 856.5	3 946.2	1 797.7	98.7	380.9	43.1	20.8	1.1
Democratic Republic of the Congo	Both	2 977 828.2	20 545.2	227 190.6	1 032.8	32 647.3	225.2	2 736.3	12.4
	F	1 483 995.6	20 681.3	128 347.4	1 175.4	16 275.6	226.8	1 545.2	14.2
	M	1 493 832.5	20 411.8	98 843.2	892.3	16 371.7	223.7	1 191.1	10.8
Denmark	Both	35.6	12.5	3.9	0.6	0.4	0.1	0.0	0.0
	F	16.7	12.0	3.3	1.0	0.2	0.1	0.0	0.0
	M	19.0	13.0	0.6	0.2	0.2	0.1	0.0	0.0
Djibouti	Both	11 510.3	11 253.5	2 632.0	1 348.4	125.9	123.1	31.7	16.2
	F	5 313.0	10 536.2	1 314.8	1 363.6	58.1	115.2	15.8	16.4
	M	6 197.3	11 950.9	1 317.1	1 333.5	67.8	130.8	15.9	16.1
Dominica	Both	NA	NA	NA	NA	NA	NA	NA	NA
	F	NA	NA	NA	NA	NA	NA	NA	NA
	M	NA	NA	NA	NA	NA	NA	NA	NA
Dominican Republic	Both	15 211.5	1 435.7	799.6	38.1	166.6	15.7	9.2	0.4
	F	6 061.5	1 168.2	393.1	38.2	66.4	12.8	4.5	0.4
	M	9 150.0	1 692.3	406.6	38.1	100.3	18.5	4.7	0.4
Ecuador	Both	14 450.1	896.9	1 148.3	37.1	158.1	9.8	13.7	0.4
	F	6 682.0	849.1	553.7	36.6	73.1	9.3	6.6	0.4
	M	7 768.1	942.5	594.6	37.6	85.0	10.3	7.1	0.4
Egypt	Both	231 920.0	1 801.2	58 961.3	308.2	2,533.9	19.7	702.6	3.7
	F	101 414.2	1 625.7	26 362.2	284.0	1 108.4	17.8	313.2	3.4
	M	130 505.8	1 966.2	32 599.1	330.9	1 425.5	21.5	389.3	4.0

El Salvador	Both	5 853.9	1 015.4	810.5	68.3	63.9	11.1	9.9	0.8
	F	2,279.7	809.8	302.5	52.1	24.8	8.8	3.6	0.6
	M	3,574.2	1,211.7	508.0	83.9	39.0	13.2	6.2	1.0
Equatorial Guinea	Both	30 835.4	16 976.2	2 858.1	1 040.8	338.1	186.2	34.2	12.5
	F	13 223.4	14 750.4	1 613.3	1 190.7	145.1	161.9	19.3	14.2
	M	17 612.0	19 145.3	1 244.8	894.9	193.0	209.8	14.9	10.7
Eritrea	Both	85 692.6	11 521.8	10 633.4	793.5	940.9	126.5	126.8	9.5
	F	39 625.4	10 883.1	5 030.6	765.7	435.1	119.5	59.9	9.1
	M	46 067.3	12 134.4	5 602.8	820.3	505.8	133.2	66.9	9.8
Estonia	Both	21.9	32.4	13.3	9.2	0.2	0.3	0.1	0.1
	F	10.3	31.1	5.4	7.7	0.1	0.3	0.1	0.1
	M	11.6	33.6	7.9	10.6	0.1	0.3	0.1	0.1
eSwatini	Both	18 542.3	10 323.9	1 796.5	556.8	203.1	113.1	21.4	6.6
	F	8 647.9	9 693.2	967.2	601.7	94.7	106.2	11.5	7.2
	M	9 894.4	10 946.4	829.3	512.3	108.4	119.9	9.9	6.1
Ethiopia	Both	1 849 426.1	12 185.6	261 665.8	972.3	20 330.0	134.0	3 140.1	11.7
	F	824 558.8	11 028.5	127 071.8	954.6	9 063.9	121.2	1 523.8	11.4
	M	1 024 867.4	13 309.0	134 594.1	989.7	11 266.1	146.3	1 616.2	11.9
Fiji	Both	2 058.4	2 377.5	264.9	155.5	22.4	25.9	3.1	1.8
	F	996.6	2 367.2	141.2	171.3	10.9	25.8	1.7	2.0
	M	1 061.8	2 387.2	123.8	140.6	11.6	26.0	1.5	1.7
Finland	Both	11.4	3.9	0.4	0.1	0.1	0.0	0.0	0.0
	F	5.4	3.7	0.2	0.1	0.1	0.0	0.0	0.0
	M	6.0	4.0	0.3	0.1	0.1	0.0	0.0	0.0
France	Both	354.4	9.2	95.0	1.2	3.6	0.1	0.7	0.0
	F	169.4	9.0	44.1	1.1	1.7	0.1	0.3	0.0
	M	185.0	9.4	50.8	1.3	1.9	0.1	0.4	0.0
Gabon	Both	14 354.1	5 246.1	2 008.6	460.3	157.5	57.6	24.6	5.6

	F	6 520.0	4 818.9	990.8	458.1	71.6	52.9	12.1	5.6
	M	7 834.1	5 664.1	1 017.7	462.4	85.9	62.1	12.5	5.7
Gambia	Both	43 944.2	12 190.4	3 936.4	693.4	483.8	134.2	47.0	8.3
	F	20 850.8	11 684.3	2 326.7	826.7	229.7	128.7	27.8	9.9
	M	23 093.4	12 686.6	1 609.7	562.4	254.1	139.6	19.2	6.7
Georgia	Both	1 287.8	474.8	448.2	94.8	14.0	5.2	5.3	1.1
	F	641.4	491.1	172.3	77.2	7.0	5.3	2.0	0.9
	M	646.4	459.7	275.9	110.6	7.0	5.0	3.3	1.3
Germany	Both	487.6	13.7	137.5	1.9	5.2	0.1	1.5	0.0
	F	221.7	12.8	51.1	1.5	2.4	0.1	0.5	0.0
	M	265.9	14.6	86.4	2.4	2.8	0.2	1.0	0.0
Ghana	Both	339 033.1	8 298.9	34 463.0	505.0	3 724.0	91.2	413.0	6.1
	F	150 795.4	7 538.8	18 151.8	544.0	1 656.1	82.8	217.5	6.5
	M	188 237.7	9 028.1	16 311.2	467.7	2 067.9	99.2	195.5	5.6
Greece	Both	282.4	59.6	43.2	3.8	3.1	0.6	0.5	0.0
	F	128.6	56.1	20.0	3.6	1.4	0.6	0.2	0.0
	M	153.8	62.9	23.2	4.0	1.7	0.7	0.3	0.0
Grenada	Both	75.9	768.8	2.5	13.4	0.8	8.4	0.0	0.1
	F	35.7	741.8	0.2	2.0	0.4	8.1	0.0	0.0
	M	40.2	794.4	2.3	24.3	0.4	8.7	0.0	0.3
Guatemala	Both	91 132.4	4 505.6	6 784.8	174.4	997.0	49.3	81.0	2.1
	F	42 233.7	4 270.1	3 484.7	183.0	462.1	46.7	41.6	2.2
	M	48 898.7	4 730.8	3 300.0	166.1	535.0	51.8	39.5	2.0
Guinea	Both	390 832.7	19 710.2	42 456.6	1 293.9	4 294.4	216.6	508.3	15.5
	F	185 068.8	18 786.1	26 421.0	1 621.8	2 034.3	206.5	316.1	19.4
	M	205 764.0	20 622.7	16 035.6	970.7	2 260.1	226.5	192.2	11.6
Guinea-Bissau	Both	60 451.1	20 785.1	3 357.5	722.4	666.6	229.2	40.2	8.6
	F	28 723.7	19 839.1	1 799.9	774.9	316.7	218.8	21.5	9.3

	M	31 727.4	21 722.9	1 557.6	670.0	349.9	239.6	18.7	8.0
Guyana	Both	1 465.2	1 917.4	251.1	166.2	16.0	21.0	3.0	2.0
	F	679.2	1 823.7	113.1	153.8	7.4	19.9	1.3	1.8
	M	786.1	2 006.5	138.0	177.9	8.6	21.9	1.7	2.2
Haiti	Both	211 679.7	17 161.9	15 676.2	657.8	2 323.7	188.4	187.6	7.9
	F	95 403.3	15 778.3	6 873.8	586.1	1 047.4	173.2	82.3	7.0
	M	116 276.4	18 492.3	8 802.4	727.3	1 276.3	203.0	105.3	8.7
Honduras	Both	18 714.4	1 967.4	735.0	36.9	204.5	21.5	8.5	0.4
	F	8 370.1	1 795.6	218.0	22.3	91.4	19.6	2.4	0.2
	M	10 344.3	2 132.4	517.0	51.0	113.1	23.3	6.1	0.6
Hungary	Both	303.6	69.6	12.8	1.3	3.2	0.7	0.0	0.0
	F	127.2	60.2	6.1	1.3	1.3	0.6	0.0	0.0
	M	176.4	78.6	6.7	1.4	1.9	0.8	0.0	0.0
Iceland	Both	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0
	F	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0
	M	0.0	0.2	0.0	0.1	0.0	0.0	0.0	0.0
India	Both	9 280 411.0	7 733.8	612 021.7	241.6	101 788.2	84.8	7 234.4	2.9
	F	5 000 735.5	8 796.4	346 992.9	289.8	54 893.0	96.6	4 125.3	3.4
	M	4 279 675.5	6 777.2	265 028.8	198.3	46 895.2	74.3	3 109.1	2.3
Indonesia	Both	804 671.2	3 241.8	47 788.0	100.9	8 784.9	35.4	556.7	1.2
	F	395 001.1	3 257.2	23 055.2	99.7	4 314.6	35.6	267.4	1.2
	M	409 670.1	3 227.1	24 732.8	102.0	4 470.3	35.2	289.4	1.2
Iran (Islamic Republic of)	Both	67 188.0	984.8	2 841.3	23.3	733.1	10.7	31.3	0.3
	F	35 538.3	1 064.4	1 413.9	23.7	388.3	11.6	15.5	0.3
	M	31 649.7	908.5	1 427.4	23.0	344.8	9.9	15.8	0.3
Iraq	Both	191 318.0	3 334.5	3 870.2	41.4	2 093.2	36.5	43.7	0.5
	F	62 496.4	2 241.0	2 103.6	46.4	684.7	24.6	23.9	0.5
	M	128 821.5	4 368.6	1 766.5	36.8	1 408.5	47.8	19.8	0.4

Ireland	Both	31.3	9.1	3.4	0.5	0.3	0.1	0.0	0.0
	F	13.5	8.1	2.7	0.8	0.1	0.1	0.0	0.0
	M	17.8	10.1	0.7	0.2	0.2	0.1	0.0	0.0
Israel	Both	148.5	17.7	58.7	4.1	1.6	0.2	0.7	0.0
	F	72.5	17.7	25.5	3.6	0.8	0.2	0.3	0.0
	M	76.0	17.6	33.3	4.5	0.8	0.2	0.4	0.1
Italy	Both	279.8	11.3	63.8	1.1	3.0	0.1	0.7	0.0
	F	125.5	10.4	43.0	1.6	1.3	0.1	0.5	0.0
	M	154.2	12.1	20.8	0.7	1.6	0.1	0.2	0.0
Jamaica	Both	551.7	269.0	83.6	18.3	5.9	2.9	0.9	0.2
	F	251.0	252.4	42.7	19.1	2.7	2.7	0.5	0.2
	M	300.7	284.7	40.9	17.6	3.2	3.1	0.5	0.2
Japan	Both	1 882.6	35.2	527.3	4.7	20.2	0.4	5.4	0.0
	F	885.3	34.1	216.7	4.0	9.5	0.4	2.2	0.0
	M	997.2	36.3	310.6	5.4	10.7	0.4	3.2	0.1
Jordan	Both	8 873.6	723.1	694.6	32.2	96.6	7.9	8.0	0.4
	F	4 252.8	708.1	402.3	37.8	46.4	7.7	4.7	0.4
	M	4 620.8	737.5	292.3	26.8	50.3	8.0	3.3	0.3
Kazakhstan	Both	8 157.7	408.5	553.0	18.9	88.8	4.4	6.3	0.2
	F	3 674.5	378.4	271.9	19.1	40.0	4.1	3.1	0.2
	M	4 483.2	436.9	281.2	18.7	48.8	4.8	3.2	0.2
Kenya	Both	607 742.3	8 653.2	38 646.6	302.2	6 671.7	95.0	463.1	3.6
	F	294 585.2	8 476.2	15 724.9	247.8	3 234.9	93.1	188.0	3.0
	M	313 157.1	8 826.5	22 921.6	355.7	3 436.7	96.9	275.1	4.3
Kiribati	Both	1 551.9	10 713.3	88.5	349.1	17.0	117.4	1.0	4.1
	F	746.6	10 567.8	26.6	215.0	8.2	115.8	0.3	2.5
	M	805.3	10 851.9	61.8	477.4	8.8	119.0	0.7	5.6
Kuwait	Both	1 046.9	331.2	219.3	41.1	11.4	3.6	2.5	0.5

	F	517.5	334.8	99.8	39.0	5.6	3.6	1.1	0.4
	M	529.4	327.7	119.5	43.0	5.7	3.6	1.4	0.5
Kyrgyzstan	Both	20 013.1	2 631.6	820.5	73.5	218.7	28.8	9.4	0.8
	F	9 242.5	2 498.2	372.0	68.1	101.0	27.3	4.2	0.8
	M	10 770.6	2 758.0	448.5	78.7	117.7	30.1	5.2	0.9
Lao People's Democratic Republic	Both	105 545.5	13 785.8	7 456.0	502.8	1 156.7	151.1	88.2	6.0
	F	51 020.7	13 610.3	3 481.1	479.0	559.5	149.3	41.2	5.7
	M	54 524.8	13 954.2	3 974.9	525.8	597.2	152.8	47.1	6.2
Latvia	Both	93.5	96.6	5.0	2.4	1.0	1.0	0.0	0.0
	F	44.6	95.0	2.4	2.4	0.5	1.0	0.0	0.0
	M	48.9	98.0	2.6	2.5	0.5	1.0	0.0	0.0
Lebanon	Both	937.8	194.1	69.3	7.4	10.1	2.1	0.6	0.1
	F	516.4	217.0	37.1	7.9	5.6	2.3	0.3	0.1
	M	421.4	171.8	32.1	7.0	4.5	1.9	0.3	0.1
Lesotho	Both	44 220.4	15 466.0	3 083.5	620.3	484.2	169.4	36.8	7.4
	F	22 325.3	15 726.3	1 761.0	711.9	244.5	172.2	21.1	8.5
	M	21 895.0	15 209.4	1 322.6	529.5	239.7	166.5	15.8	6.3
Liberia	Both	100 750.4	14 081.4	8 131.9	662.5	1 103.5	154.2	97.9	8.0
	F	50 171.1	14 332.2	4 556.3	758.4	549.6	157.0	54.8	9.1
	M	50 579.3	13 841.1	3 575.7	570.6	554.0	151.6	43.1	6.9
Libya	Both	4 146.2	661.8	735.2	63.3	45.3	7.2	8.4	0.7
	F	2 155.4	705.9	354.6	62.6	23.5	7.7	4.0	0.7
	M	1 990.8	619.8	380.6	64.0	21.7	6.8	4.4	0.7
Lithuania	Both	66.7	43.9	3.9	1.4	0.7	0.5	0.0	0.0
	F	34.1	46.0	1.9	1.4	0.4	0.5	0.0	0.0
	M	32.6	41.8	2.0	1.4	0.3	0.4	0.0	0.0
Luxembourg	Both	1.0	3.1	0.1	0.1	0.0	0.0	0.0	0.0

	F	0.4	2.8	0.0	0.1	0.0	0.0	0.0	0.0
	M	0.5	3.3	0.0	0.1	0.0	0.0	0.0	0.0
Madagascar	Both	385 194.1	10 221.2	48 886.6	750.8	4 225.0	112.1	585.2	9.0
	F	190 114.3	10 215.2	23 754.0	734.0	2 086.2	112.1	283.8	8.8
	M	195 079.8	10 227.0	25 132.6	767.4	2 138.9	112.1	301.4	9.2
Malawi	Both	265 781.4	9 138.8	15 383.5	301.6	2 915.0	100.2	183.7	3.6
	F	122 858.6	8 546.0	7 070.1	278.8	1 347.4	93.7	84.0	3.3
	M	142 922.9	9 718.2	8 313.4	324.1	1 567.6	106.6	99.6	3.9
Malaysia	Both	4 161.7	159.3	1 459.2	28.8	45.0	1.7	17.2	0.3
	F	1 790.9	142.1	646.2	26.2	19.4	1.5	7.6	0.3
	M	2 370.8	175.4	813.1	31.3	25.7	1.9	9.7	0.4
Maldives	Both	55.9	143.1	8.1	13.3	0.6	1.6	0.1	0.1
	F	30.2	160.9	4.5	15.1	0.3	1.8	0.1	0.2
	M	25.7	126.7	3.6	11.6	0.3	1.4	0.0	0.1
Mali	Both	665 347.8	19 967.7	44 405.1	841.6	7 319.4	219.7	529.5	10.0
	F	338 497.4	20 687.2	25 887.1	997.2	3 725.1	227.7	308.7	11.9
	M	326 850.4	19 273.6	18 518.1	691.0	3 594.3	211.9	220.8	8.2
Malta	Both	9.1	42.2	0.1	0.1	0.1	0.5	0.0	0.0
	F	4.6	43.9	0.0	0.1	0.1	0.5	0.0	0.0
	M	4.5	40.5	0.0	0.1	0.0	0.4	0.0	0.0
Marshall Islands	Both	NA	NA	NA	NA	NA	NA	NA	NA
	F	NA	NA	NA	NA	NA	NA	NA	NA
	M	NA	NA	NA	NA	NA	NA	NA	NA
Mauritania	Both	95 137.9	14 522.4	4 210.4	393.6	1 046.8	159.8	50.0	4.7
	F	39 088.1	12 157.1	2 421.5	459.6	430.1	133.8	28.8	5.5
	M	56 049.8	16 802.2	1 788.9	329.5	616.7	184.9	21.2	3.9
Mauritius	Both	231.3	339.6	41.4	24.3	2.5	3.7	0.5	0.3
	F	98.0	293.5	24.2	29.0	1.1	3.2	0.3	0.3

	M	133.4	384.0	17.1	19.7	1.5	4.2	0.2	0.2
Mexico	Both	97 907.4	845.4	4 141.5	18.0	1 070.0	9.2	49.1	0.2
	F	45 281.0	800.1	1 937.1	17.3	494.9	8.7	22.8	0.2
	M	52 626.3	888.7	2 204.4	18.8	575.1	9.7	26.3	0.2
Micronesia (Federated States of)	Both	644.3	5,532.5	58.1	246.3	7.1	60.6	0.7	2.9
	F	295.7	5 251.9	25.2	222.2	3.2	57.5	0.3	2.6
	M	348.6	5 795.2	32.8	268.7	3.8	63.4	0.4	3.2
Monaco	Both	NA	NA	NA	NA	NA	NA	NA	NA
	F	NA	NA	NA	NA	NA	NA	NA	NA
	M	NA	NA	NA	NA	NA	NA	NA	NA
Mongolia	Both	9 546.4	2 597.0	868.8	167.2	104.0	28.3	10.1	1.9
	F	3 438.1	1 895.2	369.9	144.0	37.4	20.6	4.3	1.7
	M	6 108.3	3 280.9	499.0	190.0	66.6	35.8	5.8	2.2
Montenegro	Both	44.0	121.1	20.7	26.3	0.4	1.2	0.2	0.2
	F	19.3	109.9	11.0	29.2	0.2	1.1	0.1	0.3
	M	24.6	131.6	9.7	23.7	0.2	1.3	0.1	0.2
Morocco	Both	54 513.0	1 553.8	2 318.8	37.3	596.4	17.0	26.7	0.4
	F	24 819.8	1 454.2	1 207.4	39.8	271.8	15.9	13.9	0.5
	M	29 693.2	1 648.1	1 111.4	34.9	324.6	18.0	12.8	0.4
Mozambique	Both	620 295.6	12 531.2	53 519.6	666.6	6 792.8	137.2	644.8	8.0
	F	304 753.8	12 407.3	26 973.9	674.0	3 338.0	135.9	324.8	8.1
	M	315 541.8	12 653.3	26 545.7	659.3	3 454.8	138.5	320.0	7.9
Myanmar	Both	505 643.2	11 142.9	37 658.4	378.9	5 543.8	122.2	447.5	4.5
	F	225 973.9	10 029.1	13 890.0	281.2	2 481.4	110.1	164.7	3.3
	M	279 669.2	12 241.3	23 768.4	475.5	3 062.4	134.0	282.8	5.7
Namibia	Both	26 643.4	7 742.5	2 385.5	418.0	292.5	85.0	28.6	5.0
	F	12 658.0	7 405.8	1 107.0	388.6	139.0	81.3	13.3	4.7

	M	13 985.4	8 074.7	1 278.5	447.3	153.6	88.7	15.4	5.4
Nauru	Both	NA	NA	NA	NA	NA	NA	NA	NA
	F	NA	NA	NA	NA	NA	NA	NA	NA
	M	NA	NA	NA	NA	NA	NA	NA	NA
Nepal	Both	190 700.7	6 919.0	12 992.0	202.2	2,086.1	75.7	152.9	2.4
	F	84 837.2	6 353.4	6 694.8	213.7	928.0	69.5	79.0	2.5
	M	105 863.4	7 450.5	6 297.2	191.3	1,158.0	81.5	73.9	2.2
Netherlands	Both	106.7	11.9	72.5	3.8	1.2	0.1	0.8	0.0
	F	46.4	10.7	50.6	5.4	0.5	0.1	0.6	0.1
	M	60.3	13.1	21.9	2.2	0.7	0.1	0.2	0.0
New Zealand	Both	82.5	27.1	7.6	1.2	0.9	0.3	0.1	0.0
	F	39.2	26.4	3.5	1.2	0.4	0.3	0.0	0.0
	M	43.3	27.8	4.1	1.3	0.5	0.3	0.0	0.0
Nicaragua	Both	19 161.4	3 208.2	1 355.7	111.4	209.6	35.1	16.3	1.3
	F	7 363.6	2 521.3	666.2	112.8	80.5	27.6	8.0	1.4
	M	11,797.8	3,865.5	689.5	110.0	129.1	42.3	8.3	1.3
Niger	Both	1 064 295.9	25 233.0	149 607.5	2 426.2	11 740.8	278.4	1 800.6	29.2
	F	541 543.6	26 242.9	79 237.6	2 622.4	5 978.1	289.7	953.6	31.6
	M	522 752.3	24 265.7	70 369.9	2 237.6	5 762.7	267.5	847.1	26.9
Nigeria	Both	8 902 806.0	27 994.9	994 548.2	1 981.3	98 000.8	308.2	11 876.0	23.7
	F	4 104 628.2	26 471.5	580 661.6	2 362.6	45 212.4	291.6	6 931.0	28.2
	M	4 798,177.0	29 444.5	413 886.6	1 615.5	52 788.4	323.9	4 945.0	19.3
Niue	Both	NA	NA	NA	NA	NA	NA	NA	NA
	F	NA	NA	NA	NA	NA	NA	NA	NA
	M	NA	NA	NA	NA	NA	NA	NA	NA
Norway	Both	15.5	5.1	2.6	0.4	0.2	0.1	0.0	0.0
	F	6.4	4.3	0.5	0.2	0.1	0.0	0.0	0.0
	M	9.1	5.8	2.1	0.7	0.1	0.1	0.0	0.0

Oman	Both	1 559.4	389.7	149.7	26.1	16.9	4.2	1.6	0.3
	F	897.8	461.9	77.3	27.2	9.8	5.0	0.8	0.3
	M	661.7	321.5	72.3	25.0	7.1	3.5	0.8	0.3
Pakistan	Both	3 489 562.8	13 979.2	165 856.7	390.7	38 252.1	153.2	1 979.6	4.7
	F	1 765 736.8	14 711.6	65 119.0	318.8	19 363.8	161.3	774.9	3.8
	M	1 723 826.1	13 300.8	100 737.8	457.4	18 888.3	145.7	1 204.7	5.5
Palau	Both	NA	NA	NA	NA	NA	NA	NA	NA
	F	NA	NA	NA	NA	NA	NA	NA	NA
	M	NA	NA	NA	NA	NA	NA	NA	NA
Panama	Both	3 354.1	863.6	296.7	40.8	36.6	9.4	3.5	0.5
	F	1 456.5	766.3	136.0	38.2	15.9	8.4	1.6	0.5
	M	1 897.6	956.9	160.7	43.4	20.7	10.5	1.9	0.5
Papua New Guinea	Both	98 364.8	9 525.1	6 691.4	352.2	1 075.6	104.2	78.7	4.1
	F	42 883.8	8 599.4	2 440.3	265.4	468.8	94.0	28.5	3.1
	M	55 481.0	10 389.6	4 251.1	433.6	606.8	113.6	50.1	5.1
Paraguay	Both	9 998.8	1 488.7	1 026.5	77.3	108.9	16.2	11.4	0.9
	F	4 490.5	1 364.2	515.6	79.2	48.8	14.8	5.7	0.9
	M	5 508.3	1 608.4	510.9	75.6	60.1	17.5	5.7	0.8
Peru	Both	33 641.7	1 109.3	9 768.1	169.7	366.8	12.1	116.3	2.0
	F	15 091.4	1 016.5	4 524.6	160.6	164.5	11.1	53.8	1.9
	M	18 550.3	1 198.3	5 243.5	178.5	202.3	13.1	62.5	2.1
Philippines	Both	557 310.0	4 833.4	80 775.9	375.8	6 107.8	53.0	973.2	4.5
	F	257 084.8	4 592.0	38 670.5	369.8	2 818.7	50.3	464.8	4.4
	M	300 225.2	5 061.2	42 105.3	381.5	3 289.2	55.4	508.4	4.6
Poland	Both	1 403.7	77.2	595.6	15.5	15.1	0.8	6.9	0.2
	F	625.5	70.7	281.8	15.1	6.7	0.8	3.3	0.2
	M	778.2	83.3	313.8	15.9	8.4	0.9	3.6	0.2
Portugal	Both	95.0	22.0	9.7	1.0	1.0	0.2	0.1	0.0

	F	43.7	21.0	3.5	0.7	0.5	0.2	0.0	0.0
	M	51.3	23.0	6.3	1.2	0.6	0.3	0.1	0.0
Qatar	Both	395.1	304.0	64.0	28.2	4.3	3.3	0.7	0.3
	F	154.3	242.1	25.6	23.2	1.7	2.6	0.3	0.2
	M	240.8	363.7	38.4	33.0	2.6	3.9	0.4	0.4
Republic of Korea	Both	739.8	33.2	186.5	4.0	7.6	0.3	1.3	0.0
	F	366.0	34.1	83.7	3.7	3.8	0.4	0.6	0.0
	M	373.8	32.4	102.8	4.2	3.8	0.3	0.7	0.0
Republic of Moldova	Both	2 470.4	1 134.1	160.9	38.3	26.9	12.4	1.8	0.4
	F	1 057.9	1 007.0	74.0	36.3	11.5	11.0	0.8	0.4
	M	1 412.6	1 252.5	86.9	40.2	15.4	13.6	1.0	0.5
Romania	Both	11 648.5	1 233.7	1 169.0	56.1	126.9	13.4	13.6	0.7
	F	5 238.6	1 140.2	626.2	61.7	57.1	12.4	7.3	0.7
	M	6 409.9	1 322.4	542.7	50.7	69.8	14.4	6.3	0.6
Russian Federation	Both	13 546.5	141.7	2 311.2	15.1	145.5	1.5	24.6	0.2
	F	6 206.5	133.6	1 167.3	15.6	66.6	1.4	12.5	0.2
	M	7 340.0	149.3	1 143.9	14.6	78.8	1.6	12.1	0.2
Rwanda	Both	113 210.2	6 507.2	21 136.9	685.5	1 240.6	71.3	253.6	8.2
	F	51 158.0	5 903.7	10 069.3	651.2	560.5	64.7	120.8	7.8
	M	62 052.2	7 106.1	11 067.5	720.1	680.1	77.9	132.9	8.6
Saint Kitts and Nevis	Both	NA	NA	NA	NA	NA	NA	NA	NA
	F	NA	NA	NA	NA	NA	NA	NA	NA
	M	NA	NA	NA	NA	NA	NA	NA	NA
Saint Lucia	Both	42.9	391.9	1.6	6.7	0.5	4.3	0.0	0.1
	F	20.8	385.2	1.3	11.3	0.2	4.2	0.0	0.1
	M	22.1	398.4	0.3	2.3	0.2	4.3	0.0	0.0

Saint Vincent and the Grenadines	Both	68.3	822.7	5.2	28.7	0.7	9.0	0.1	0.3
	F	33.1	806.4	2.7	29.6	0.4	8.8	0.0	0.4
	M	35.2	838.5	2.6	27.9	0.4	9.1	0.0	0.3
Samoa	Both	350.5	1 492.2	48.8	100.7	3.8	16.2	0.6	1.2
	F	142.1	1 254.7	20.2	86.3	1.5	13.6	0.2	1.0
	M	208.3	1 713.4	28.6	114.2	2.3	18.6	0.3	1.3
San Marino	Both	NA	NA	NA	NA	NA	NA	NA	NA
	F	NA	NA	NA	NA	NA	NA	NA	NA
	M	NA	NA	NA	NA	NA	NA	NA	NA
Sao Tome and Principe	Both	1 631.9	5 208.9	361.9	658.1	17.9	57.0	4.3	7.8
	F	675.4	4 349.9	179.7	658.2	7.4	47.6	2.1	7.8
	M	956.5	6 053.0	182.2	658.1	10.5	66.2	2.2	7.8
Saudi Arabia	Both	17 303.6	583.4	1 985.3	37.6	189.4	6.4	22.1	0.4
	F	8 471.9	579.8	1 106.2	42.6	92.7	6.3	12.4	0.5
	M	8 831.7	586.9	879.2	32.8	96.7	6.4	9.7	0.4
Senegal	Both	212 158.8	8 338.5	26 683.1	653.7	2 328.6	91.5	320.5	7.9
	F	96 393.2	7 682.1	14 105.1	699.2	1 058.7	84.4	169.1	8.4
	M	115 765.6	8 977.3	12 577.9	609.2	1 269.9	98.5	151.4	7.3
Serbia	Both	719.0	153.3	93.6	9.4	7.6	1.6	0.9	0.1
	F	320.8	140.1	40.7	8.4	3.4	1.5	0.4	0.1
	M	398.2	165.9	52.9	10.4	4.2	1.8	0.5	0.1
Seychelles	Both	30.7	392.2	15.0	116.4	0.3	4.3	0.2	1.4
	F	12.4	323.3	7.0	108.1	0.1	3.5	0.1	1.3
	M	18.3	458.3	8.1	124.5	0.2	5.0	0.1	1.5
Sierra Leone	Both	232 286.8	20 360.9	26 138.2	1 309.4	2 543.8	223.0	311.3	15.6
	F	112 502.9	19 785.5	14 266.1	1 425.4	1 232.1	216.7	169.8	17.0
	M	119 783.9	20 932.5	11 872.1	1 192.7	1 311.7	229.2	141.5	14.2

Singapore	Both	213.1	80.3	17.9	3.0	2.3	0.9	0.2	0.0
	F	89.0	69.7	10.4	3.6	1.0	0.8	0.1	0.0
	M	124.1	90.1	7.5	2.5	1.3	1.0	0.1	0.0
Slovakia	Both	575.4	204.5	113.4	20.6	6.2	2.2	1.3	0.2
	F	261.3	190.1	62.2	23.1	2.8	2.1	0.7	0.3
	M	314.1	218.3	51.2	18.1	3.4	2.4	0.6	0.2
Slovenia	Both	14.1	13.2	7.7	3.8	0.1	0.1	0.1	0.0
	F	7.1	13.6	1.1	1.1	0.1	0.1	0.0	0.0
	M	7.0	12.8	6.6	6.4	0.1	0.1	0.1	0.1
Solomon Islands	Both	4 233.0	5 115.5	220.0	144.5	46.2	55.9	2.5	1.7
	F	2 074.3	5 174.7	94.5	128.3	22.7	56.5	1.1	1.5
	M	2 158.6	5 059.8	125.4	159.7	23.6	55.3	1.5	1.9
Somalia	Both	1 068 103.0	40 813.4	62 716.7	1 549.9	11 764.4	449.5	748.1	18.5
	F	515 505.8	39 728.6	33 284.4	1 652.7	5 678.8	437.7	396.8	19.7
	M	552 597.1	41 880.2	29 432.3	1 448.0	6 085.5	461.2	351.3	17.3
South Africa	Both	260 058.2	4 558.5	17 158.8	161.3	2 841.8	49.8	205.1	1.9
	F	121 590.9	4 307.4	7 681.2	145.5	1 328.7	47.1	91.4	1.7
	M	138 467.4	4 804.5	9 477.6	176.9	1,513.0	52.5	113.7	2.1
South Sudan	Both	494 024.8	25 663.0	22 051.9	689.7	5 442.7	282.7	263.2	8.2
	F	238 273.8	25 127.4	11 415.7	722.7	2 625.6	276.9	136.1	8.6
	M	255 751.0	26 182.9	10 636.2	657.5	2 817.1	288.4	127.0	7.9
Spain	Both	235.3	11.4	37.1	0.8	2.5	0.1	0.4	0.0
	F	111.5	11.1	24.8	1.1	1.2	0.1	0.3	0.0
	M	123.8	11.6	12.2	0.5	1.3	0.1	0.1	0.0
Sri Lanka	Both	8 692.4	542.8	2 765.8	80.2	93.8	5.9	31.9	0.9
	F	3 597.6	456.8	1 465.1	85.3	38.9	4.9	17.0	1.0
	M	5 094.8	626.0	1 300.8	75.1	54.9	6.7	14.9	0.9
Sudan	Both	674 268.1	11 350.7	28 973.5	279.9	7 441.0	125.3	343.0	3.3

	F	383 306.5	13 128.1	14 703.7	288.5	4 230.4	144.9	174.3	3.4
	M	290 961.6	9 632.6	14 269.9	271.5	3 210.7	106.3	168.8	3.2
Suriname	Both	368.5	732.4	37.9	38.4	4.0	8.0	0.4	0.4
	F	158.1	650.8	17.8	37.4	1.7	7.1	0.2	0.4
	M	210.4	808.5	20.1	39.3	2.3	8.8	0.2	0.5
Sweden	Both	42.0	7.2	16.6	1.5	0.5	0.1	0.2	0.0
	F	16.8	5.9	4.8	0.9	0.2	0.1	0.1	0.0
	M	25.2	8.4	11.8	2.0	0.3	0.1	0.1	0.0
Switzerland	Both	43.3	10.0	8.4	1.0	0.5	0.1	0.1	0.0
	F	19.4	9.2	6.7	1.7	0.2	0.1	0.1	0.0
	M	23.9	10.7	1.7	0.4	0.3	0.1	0.0	0.0
Syrian Arab Republic	Both	19 112.1	910.3	4 161.8	86.9	209.3	10.0	49.1	1.0
	F	8 512.6	832.4	2 324.0	99.6	93.3	9.1	27.5	1.2
	M	10 599.5	984.2	1 837.8	74.9	116.0	10.8	21.6	0.9
Tajikistan	Both	73 611.8	6 222.5	5 620.4	297.5	804.3	68.0	65.5	3.5
	F	34 720.4	6 028.6	2 835.7	308.5	379.4	65.9	33.0	3.6
	M	38 891.4	6 406.4	2 784.7	287.2	424.8	70.0	32.4	3.3
Thailand	Both	26 484.1	702.9	4 706.0	56.1	288.4	7.7	55.2	0.7
	F	11 180.5	610.3	1 599.6	39.2	121.8	6.6	18.3	0.4
	M	15 303.6	790.6	3 106.4	72.1	166.6	8.6	36.9	0.9
The former Yugoslav Republic of Macedonia	Both	814.5	690.4	50.6	21.9	8.8	7.5	0.4	0.2
	F	391.5	681.8	5.8	5.2	4.2	7.4	0.0	0.0
	M	423.0	698.5	44.8	37.7	4.6	7.5	0.4	0.4
Timor-Leste	Both	26 423.1	12 820.9	1 056.6	302.8	289.1	140.3	12.5	3.6
	F	13 535.9	13 401.8	594.4	347.6	148.2	146.7	7.0	4.1
	M	12 887.2	12 262.6	462.2	259.8	141.0	134.1	5.5	3.1

Togo	Both	172 757.5	14 691.9	24 074.9	1 200.4	1 898.5	161.5	289.4	14.4
	F	75 760.7	12 924.6	14 022.9	1 402.3	832.9	142.1	168.5	16.8
	M	96 996.9	16 448.8	10 052.0	999.7	1 065.6	180.7	121.0	12.0
Tonga	Both	164.7	1 295.6	21.4	81.9	1.8	14.1	0.2	0.9
	F	93.9	1 516.6	8.8	69.6	1.0	16.5	0.1	0.8
	M	70.7	1 085.5	12.7	93.3	0.8	11.8	0.1	1.1
Trinidad and Tobago	Both	482.7	510.4	28.0	14.8	5.2	5.5	0.3	0.2
	F	222.5	478.3	7.2	7.8	2.4	5.2	0.1	0.1
	M	260.2	541.5	20.7	21.7	2.8	5.9	0.2	0.2
Tunisia	Both	5 683.2	540.5	716.4	42.8	62.0	5.9	8.1	0.5
	F	2 916.9	567.8	428.5	52.3	31.8	6.2	4.9	0.6
	M	2 766.3	514.4	287.9	33.6	30.1	5.6	3.2	0.4
Turkey	Both	17 035.1	251.5	2 769.7	20.8	184.7	2.7	31.2	0.2
	F	8 288.6	250.6	1 223.3	18.7	89.9	2.7	13.6	0.2
	M	8 746.5	252.2	1 546.4	22.7	94.7	2.7	17.5	0.3
Turkmenistan	Both	20 070.6	2 829.6	668.3	64.5	219.7	31.0	7.9	0.8
	F	8 540.0	2 444.6	285.7	55.8	93.5	26.8	3.4	0.7
	M	11 530.5	3 203.2	382.7	73.0	126.2	35.1	4.5	0.9
Tuvalu	Both	NA	NA	NA	NA	NA	NA	NA	NA
	F	NA	NA	NA	NA	NA	NA	NA	NA
	M	NA	NA	NA	NA	NA	NA	NA	NA
Uganda	Both	914 692.1	11 880.8	122 668.0	1 004.7	10 044.5	130.5	1 472.5	12.1
	F	422 630.1	11 083.4	60 827.1	1 003.6	4 638.1	121.6	729.9	12.0
	M	492 062.0	12 663.3	61 840.8	1 005.7	5 406.4	139.1	742.5	12.1
Ukraine	Both	6 546.3	280.5	598.4	13.5	70.7	3.0	5.8	0.1
	F	3 486.0	308.2	309.1	14.4	37.7	3.3	3.1	0.1
	M	3 060.3	254.4	289.3	12.7	33.0	2.7	2.8	0.1
United Arab	Both	864.3	186.3	66.0	8.0	9.2	2.0	0.6	0.1

Emirates									
	F	485.9	213.9	22.0	5.5	5.2	2.3	0.2	0.0
	M	378.4	159.8	44.0	10.5	4.0	1.7	0.5	0.1
United Kingdom	Both	1 201.2	30.0	218.7	2.9	13.1	0.3	2.5	0.0
	F	551.8	28.3	98.1	2.6	6.0	0.3	1.1	0.0
	M	649.4	31.7	120.7	3.1	7.1	0.3	1.4	0.0
United Republic of Tanzania	Both	1 041 731.9	10 789.1	114 108.2	741.1	11 439.5	118.5	1 359.2	8.8
	F	508 711.1	10 637.3	59 202.8	771.3	5 586.3	116.8	704.8	9.2
	M	533 020.8	10 938.0	54 905.4	711.0	5 853.2	120.1	654.4	8.5
United States of America	Both	4 106.7	20.9	780.7	1.9	44.0	0.2	7.9	0.0
	F	1 880.0	19.6	359.9	1.8	20.2	0.2	3.6	0.0
	M	2 226.8	22.2	420.8	2.0	23.8	0.2	4.3	0.0
Uruguay	Both	170.8	71.3	13.7	2.8	1.8	0.8	0.1	0.0
	F	74.6	63.5	4.0	1.6	0.8	0.7	0.0	0.0
	M	96.2	78.7	9.8	3.9	1.0	0.9	0.1	0.0
Uzbekistan	Both	55 718.4	1 749.8	8 942.2	159.3	607.7	19.1	105.9	1.9
	F	24 968.8	1 627.0	4 231.2	154.7	272.3	17.7	50.1	1.8
	M	30 749.6	1 864.1	4 711.1	163.7	335.4	20.3	55.8	1.9
Vanuatu	Both	1 416.2	4 114.2	104.1	164.5	15.4	44.9	1.2	1.9
	F	666.7	4,010.9	39.3	130.0	7.3	43.7	0.5	1.5
	M	749.4	4,210.7	64.8	196.1	8.2	45.9	0.8	2.3
Venezuela (Bolivarian Republic of)	Both	15 909.9	534.9	1 113.0	19.1	173.7	5.8	13.2	0.2
	F	6 898.5	474.6	477.6	16.7	75.3	5.2	5.6	0.2
	M	9 011.4	592.6	635.4	21.4	98.4	6.5	7.6	0.3
Viet Nam	Both	176 592.3	2 275.5	5 157.4	36.7	1 929.6	24.9	57.7	0.4
	F	69 334.7	1 887.1	1 755.6	26.0	757.3	20.6	18.8	0.3

	M	107 257.6	2 624.6	3 401.9	46.5	1 172.3	28.7	39.0	0.5
Yemen	Both	376 758.6	9 245.2	11 148.7	158.6	4 132.0	101.4	130.6	1.9
	F	214 536.5	10 763.4	5 987.9	173.9	2 353.2	118.1	70.3	2.0
	M	162 222.1	7 791.8	5 160.9	143.9	1 778.8	85.4	60.2	1.7
Zambia	Both	309 264.4	10 966.3	29 062.8	623.5	3 394.8	120.4	347.3	7.5
	F	147 139.2	10 532.6	14 186.8	612.4	1 615.6	115.7	169.4	7.3
	M	162 125.2	11 392.1	14 876.1	634.4	1 779.2	125.0	177.9	7.6
Zimbabwe	Both	224 757.2	8 850.7	29 028.2	700.6	2 467.9	97.2	348.2	8.4
	F	113 435.3	8 978.0	15 935.1	771.0	1 245.9	98.6	191.2	9.3
	M	111 321.9	8 724.7	13 093.1	630.5	1 222.0	95.8	157.0	7.6

NA, not available

F, females; M, males

Table 7. Death rate per 100 000 children attributable to the joint effects of household and ambient air pollution in 2016, by WHO region, the world, and income group, by sex

WHO region	Income group	Death rate per 100 000, boys < 5 years	Death rate per 100 000, girls < 5 years	Death rate per 100 000, boys 5–14 years	Death rate per 100 000, girls 5–14 years
African	LMIC	190.5	177.4	11.6	14.2
	HIC	5	3.5	1.5	1.3
Americas	LMIC	15.2	13.2	0.7	0.6
	HIC	0.3	0.3	0	0
Eastern Mediterranean	LMIC	94.6	102.8	3.7	3.5
	HIC	5.2	5.4	0.3	0.4
European	LMIC	9.1	8.4	0.6	0.5
	HIC	0.3	0.2	0	0
South-East Asia	LMIC	68.8	81.8	2.1	2.9
	HIC	NA	NA	NA	NA
Western Pacific	LMIC	21.4	19.5	1	1.1
	HIC	0.4	0.3	0	0
All	LMIC	87.6	89.9	4.1	5
	HIC	0.7	0.6	0	0.1
World		79.6	81.6	3.7	4.5

LMIC, low- and middle-income country; HIC, high-income country
NA, not available

Table 8. Population attributable fraction (PAF) of childhood mortality due to ambient air pollution, by WHO region and income level, 2016

WHO region	Income level	Children < 5 years (%)	Children 5–14 years (%)
African	LMIC	28	29
	HIC	17	17
Americas	LMIC	15	16
	HIC	8	7
Eastern Mediterranean	LMIC	32	33
	HIC	37	38
European	LMIC	20	20
	HIC	12	13
South-East Asia	LMIC	35	35
	HIC	NA	NA
Western Pacific	LMIC	25	22
	HIC	11	11
All	LMIC	30	29
	HIC	17	15
World		30	29

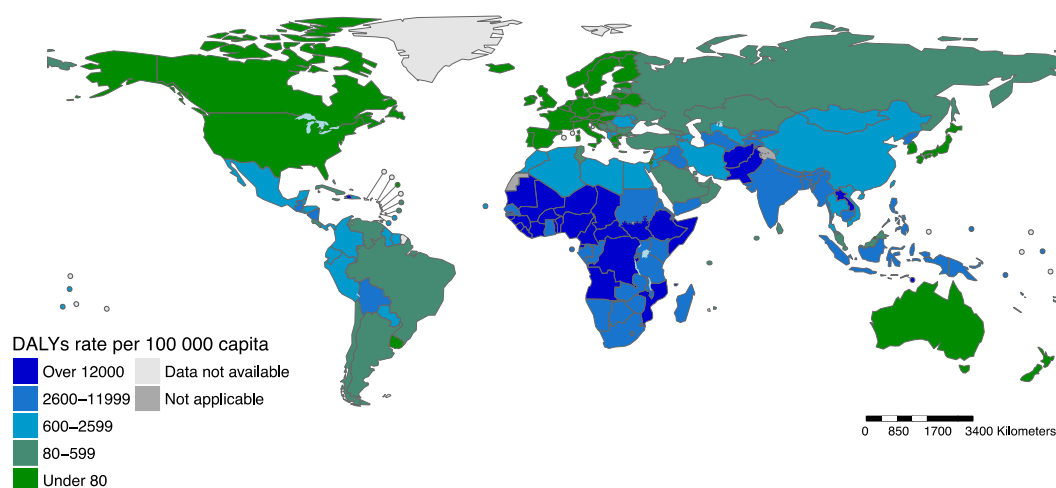
LMIC, low- and middle-income country; HIC, high-income country
NA, not available

Table 9. Population attributable fraction (PAF) of childhood mortality due to household air pollution, by WHO region and income level, 2016

WHO region	Income level	Children < 5 years	Children 5–14
		(%)	years (%)
African	LMIC	53	52
	HIC	9	9
Americas	LMIC	23	22
	HIC	1	0
Eastern Mediterranean	LMIC	38	33
	HIC	4	4
European	LMIC	9	8
	HIC	1	0
South-East Asia	LMIC	43	42
	HIC	NA	NA
Western Pacific	LMIC	38	38
	HIC	0	0
All	LMIC	46	46
	HIC	2	1
World		46	46

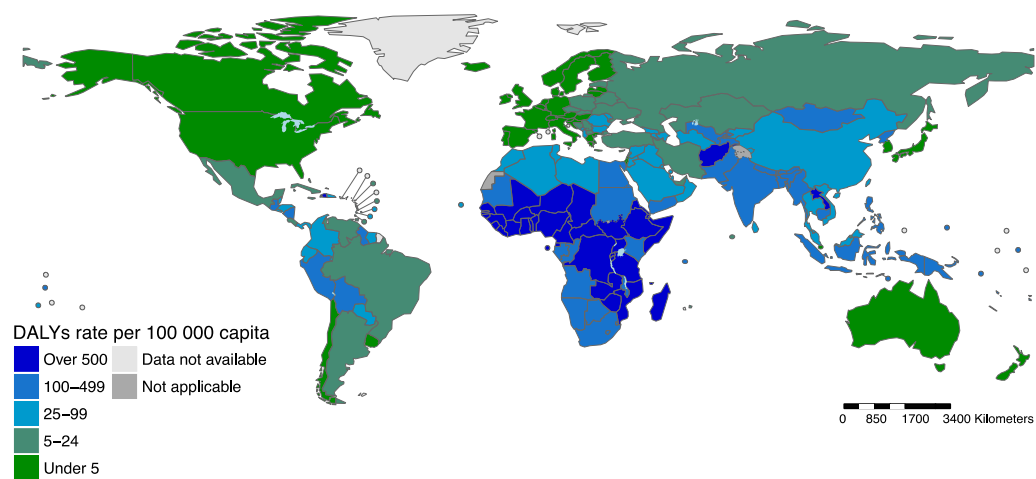
LMIC, low- and middle-income country; HIC, high-income country
 NA, not available

Fig. 15. Disability-adjusted life years (DALYs) per 100 000 capita due to acute lower respiratory infections associated with the joint effects of ambient and household air pollution in children under 5 years of age in 2016



Source: see Annex 2

Fig. 16. Disability-adjusted life years (DALYs) rate per 100 000 due to acute lower respiratory infections associated with the joint effects of ambient and household air pollution in children 5–14 years of age in 2016



Source: see Annex 2