



Royal College
of Physicians

RCPCH

Royal College of
Paediatrics and Child Health
Leading the way in Children's Health

Every breath we take The lifelong impact of air pollution

Report of a working party
February 2016





Royal College
of Physicians

RCPCH

Royal College of
Paediatrics and Child Health

Leading the way in Children's Health

Every breath we take: the lifelong impact of air pollution

Report of a working party
February 2016



The Royal College of Physicians

The Royal College of Physicians (RCP) plays a leading role in the delivery of high-quality patient care by setting standards of medical practice and promoting clinical excellence. The RCP provides physicians in over 30 medical specialties with education, training and support throughout their careers. As an independent charity representing 32,000 fellows and members worldwide, the RCP advises and works with government, patients, allied healthcare professionals and the public to improve health and healthcare.

Citation for this document: Royal College of Physicians. *Every breath we take: the lifelong impact of air pollution*. Report of a working party. London: RCP, 2016.

Copyright

All rights reserved. No part of this publication may be reproduced in any form (including photocopying or storing it in any medium by electronic means and whether or not transiently or incidentally to some other use of this publication) without the written permission of the copyright owner. Applications for the copyright owner's written permission to reproduce any part of this publication should be addressed to the publisher.

Copyright © Royal College of Physicians 2016

ISBN 978-1-86016-567-2
eISBN 978-1-86016-568-9

Royal College of Physicians
11 St Andrews Place
Regent's Park
London NW1 4LE

www.rcplondon.ac.uk

Registered charity no 210508

Typeset by Cambrian Typesetters, Camberley, Surrey
Printed and bound in Great Britain by The Lavenham Press, Suffolk

Contents

Foreword	iv
Foreword – European perspective	v
Preface	vii
Members of the working party	viii
Conflicts of interest	x
Executive summary	xii
Recommendations for action and research	xiv
Definitions	xvii
Introduction	1
Chapter 1: Summary	2
Chapter 1: Air pollution in our changing world	4
Chapter 2: Summary	16
Chapter 2: The air we breathe	18
Chapter 3: Summary	34
Chapter 3: In the beginning: protecting our future generations	36
Chapter 4: Summary	50
Chapter 4: Health effects of air pollution over our lifetime	52
Chapter 5: Summary	66
Chapter 5: Our vulnerable groups	68
Chapter 6: Summary	78
Chapter 6: The heavy cost of air pollution	80
Chapter 7: Summary	92
Chapter 7: Changing our future	94
Methodology	103
Acknowledgements	106

Foreword

I am very pleased to introduce this report from the Royal College of Physicians (RCP) and the Royal College of Paediatrics and Child Health (RCPCH), which takes a ‘cradle to grave’ approach to considering the impact of air pollution on our health.

Air pollution, to which we are all exposed to a lesser or greater extent, has a significant public health burden. In 2010, the Department of Health’s Committee on the Medical Effects of Air Pollutants (COMEAP) reported that long-term exposure to outdoor air pollution caused the equivalent of approximately 29,000 deaths in 2008 in the UK. Current work by the committee suggests that the effect might be even greater.

Some health effects associated with air pollution are well recognised, such as increases in hospital admissions and deaths from cardiovascular diseases, respiratory diseases and lung cancer. We know that those with pre-existing cardiovascular and respiratory diseases and older people are particularly at risk. However, researchers are finding that air pollution may be associated with a much wider range of health conditions. For example, the report considers the evidence for effects of air pollution on diabetes and neurological disease, as well as how exposure during pregnancy may be associated with low birth weight and pre-term births. More research is needed to characterise the impacts, but there is no doubt that the health effects of air pollution are significant.

The report also explores how the sources of air pollution have changed and that it is not just an outdoor problem; we spend much of our time indoors, where we can be exposed to numerous pollutants from a variety of sources. It considers what we can expect in the future with an increasing and ageing population and climate change, and the pressures that these put on our changing society.

While air quality improvements have been made since the early 20th century, it is clear that we must address the ongoing problem of air pollution and we all have a role to play, however big or small. Individuals can take steps to reduce their exposure to air pollution and reduce their impact on air quality and the environment, for example by considering the transport they use and the routes they travel. This type of action alone is not sufficient to help those living in the most deprived areas, where levels of air pollution may be greater and where the death rates from cancer and cardiovascular diseases can be higher. If we are to make significant progress, collective action at population level is needed. Such action will not just reduce air pollution, but can also help to address other important public health and environmental issues such as health inequalities, physical activity levels and climate change mitigation.



Professor Dame Sally Davies
Chief medical officer for England

Foreword – European perspective

It is now hard to imagine that, when I started my career in environmental health some 35 years ago, air pollution in western Europe was not seen as much of a public health problem. The great sulphurous smogs of the 1950s and 1960s were a thing of the past, and our summer weather was considered too lousy to produce much in the way of ozone, which was wreaking havoc in the Los Angeles basin at the time.

As the *Every breath we take* report testifies, this has changed enormously over the past decades. It is a bit of a paradox – most air pollutants have been strongly reduced over time, and health benefits of these downward changes have been convincingly shown in quite a few studies.

So why are we still (or again) concerned? Partly, this stems from the fact that, when concentrations are high, the research effort naturally focuses on what's going on at these high exposures. So, without the success of the air pollution abatement programmes, studies of effects at low or lower levels were simply not possible. The many studies now available at lower concentrations document serious effects on population health, which, as this report forcefully argues, cannot be ignored. Partly, it is also a matter of improved research methodology: the application of time-series analyses (developed in econometrics) in air pollution studies has enabled very detailed ascertainment of shapes of acute concentration–response relationships down to very low levels of pollution, seen on the cleanest days only; advances in air pollution exposure modelling have made it possible to study effects of long-term air pollution exposure in large cohort studies, often designed for other purposes; and related to this, studies have now demonstrated that air pollution is involved in much more than symptom exacerbation and early death in older and frail bronchitis patients. Air pollution affects us at just about every stage of life, starting in the womb, continuing through childhood, adolescence and young adulthood into old age. We now see a 'lifecourse' epidemiology of air pollution effects on population health emerging, and this *Every breath we take* report is perhaps the first one to emphasise this perspective in a concise and easy-to-follow format.

It often quotes results from the European Study of Cohorts for Air Pollution Effects (ESCAPE), a recent pan-European effort to use data from over 30 different cohorts, spanning all ages and a multitude of diseases. I had the great privilege of serving as the coordinator of an incredibly dedicated group of talented colleagues in this project, and it is rewarding to see the results being put to some good use in this report.

Although air pollution abatement really has been an environmental and public health success story, this and other reports show that there is still a lot to be gained. Even major additional clean-up efforts cost far less than the monetarised health benefits that they produce. This report, by emphasising that air pollution harms us in all phases of life, provides powerful arguments to support cleaning up the air we breathe every minute, day, year and decade of our lives.



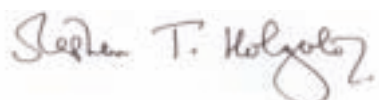
Professor Bert Brunekreef
Chair of the European Respiratory Society Task Force on Air Pollution

Preface

For us all, life on Earth depends upon the air we breathe and our ability to extract oxygen from it for energy creation. During a lifetime, a person breathes about 250 million litres of air, weighing about 300,000 kg. However, unknown by many, the air we breathe contains more than natural gases, as amply demonstrated on 5–9 December 1952, when London was engulfed by a dense fog in which air pollution from the burning of coal causing a massive increase in severe lung disease and death. As pointed out in the first RCP report on air pollution and health in 1972,¹ the recognition of how serious a public health issue this was led to the Clean Air Acts of 1956² and 1968,³ in which measures were introduced to dramatically reduce industrial and domestic fossil fuel emissions with great effect. Following this, everyone thought that the problem of air pollution was over. But how wrong we all were. Over the next half century a different, more insidious form of air pollution appeared, linked to the emissions from the ever-increasing number of motor vehicles and other forms of transport on our roads, rail and seas that are dependent upon combustion of petrol and diesel fuels. Added to this chemical onslaught is the effect of indoor air pollution from workplaces and the fittings, furnishings, heating and cooking in our homes.

Contemporary ambient air pollution in the UK largely comprises small and ultra-fine particles, oxides of nitrogen and ozone, and is largely invisible apart from episodes when particles and oxides of nitrogen cause a brown haze which, in other regions of the world such as China and South-East Asia, is becoming a regular feature. However, although pollution often cannot be seen or smelled, its effects are insidious and dangerous. Population-based studies as well as modern biological science have revealed highly potent toxic effects of chronic exposure to ‘modern-day pollutants’, not only on the lungs but also on the heart and broader cardiovascular system. We are further recognising that the systemic effects of pollutants extend beyond the cardiopulmonary system to affect many other organs, increasing the risk of disease that begins from conception and persists across the lifecourse. Added to this are the multiplying effects of urbanisation and climate change, both of which are driving air pollution in the wrong direction.⁴

The Clean Air Acts of the last century, as well as recent tobacco smoking legislation, put the onus on the polluter to reduce emissions for the greater good of those around them. In the case of modern air pollution, it is currently the public at large that has to take responsibility for avoiding exposure, irrespective of their role in generating pollution, in contrast to the recently introduced tobacco smoking in public places legislation. The evidence base summarised in this report emphasises that the time has now arrived to take air pollution, as currently encountered in the UK, much more seriously than has been the case. It should be considered a major public health problem deserving of multiple measures to drive down exposure in as many ways as possible. It is our view that this requires urgent, determined and multidisciplinary action that is long overdue. Indeed, if we do not act now, our children and generations to follow will be those who suffer from our failure to act.



Professor Stephen Holgate
Chair of the RCP/RCPCH working party on air pollution

1 www.rcplondon.ac.uk/projects/outputs/air-pollution-and-health-1972

2 www.legislation.gov.uk/ukpga/1956/52/pdfs/ukpga_19560052_en.pdf

3 www.legislation.gov.uk/ukpga/1968/62/pdfs/ukpga_19680062_en.pdf

4 www.eea.europa.eu/publications/air-quality-in-europe-2015

Members of the working party

Name	Representation
Professor Stephen Holgate CBE (chair) MRC clinical professor of immunopharmacology, Faculty of Medicine, Southampton General Hospital Honorary consultant physician, Southampton University Hospital Trust	
Professor Jonathan Grigg (vice chair) Professor of paediatric respiratory and environmental medicine, Barts and The London School of Medicine and Dentistry, Queen Mary University of London	Royal College of Paediatrics and Child Health (RCPCH)
Professor Raymond Agius Professor of occupational and environmental medicine, University of Manchester	Faculty of Occupational Medicine (FOM)
Professor John R Ashton CBE President of the Faculty of Public Health of the UK Royal Colleges of Physicians	RCP council adviser
Professor Paul Cullinan Professor in occupational and environmental respiratory disease, National Heart and Lung Institute, Imperial College London	
Dr Karen Exley Senior environmental public health scientist, Centre for Radiation, Chemical and Environmental Hazards, Public Health England (PHE)	Committee on the Medical Effects of Air Pollutants COMEAP) (Secretariat)
Professor David Fishwick Honorary professor of occupational and environmental respiratory medicine, University of Sheffield	British Thoracic Society (BTS)
Dr Gary Fuller Senior lecturer in air quality measurement, MRC PHE Centre for Environment and Health, King's College London	
Mr Nikhil Gokani Deputy director, Law and Non-Communicable Diseases Unit, School of Law and Social Justice, University of Liverpool	RCP Patient and Carer Network
Professor Chris Griffiths Professor of primary care, Blizard Institute, Queen Mary University of London	

Professor Paul Harrison

Owner/director of PTCH Consultancy Ltd, and visiting professor of environment and health, Cranfield University

Professor John Henderson

Professor of paediatric respiratory medicine, Faculty of Health Sciences, University of Bristol

Dr Mike Holland

Consultant, Ecometrics Research and Consulting, and honorary research fellow, Imperial College London

Professor John Holloway

Professor of allergy and respiratory genetics, associate dean (research), University of Southampton Faculty of Medicine

Professor George Morris

Honorary professor, European Centre for Environment and Human Health, University of Exeter Medical School

Professor Patrick Saunders

Visiting professor of public health, University of Staffordshire

Faculty of Public Health

Dr Samantha Walker

Deputy chief executive and director of research and policy, Asthma UK

Asthma UK

Professor Dafydd Walters

Emeritus professor of child health, St George's University of London

Professor Jane Dacre

President, Royal College of Physicians (RCP)

Dr Andrew Goddard

Registrar, RCP

Supported by:

Kieli Bibbings

Claire Cantle

Stephen Deed

Kate Eisenstein

Morgan Evans

Colleen Shannon

Christina Smith

Pauline Whitelaw

Natalie Wilder

Illustrator

Communications project officer, RCP

Reader services librarian, RCP

Deputy head of strategy and policy, RCP

Senior communications adviser, RCP

Healthcare writer

Committee manager, RCP

Committee manager, RCP

Managing editor, RCP

Conflicts of interest

All working party members have been asked to complete a Declaration of Interests form. The following potential conflicts of interest have been declared.

Professor Stephen Holgate is a trustee of the British Lung Foundation (BLF), chair of the BLF's Scientific Committee, a trustee of Cancer Research UK, chair of Defra's Hazardous Substances Advisory Committee and the chair of the European Respiratory Society (ERS) Science Council.

Professor Jonathan Grigg provides scientific advice to the charity Change London (www.changelondon.org/about.php). He is also secretary of the Paediatric Assembly of the ERS.

Professor John Ashton is a member of the Labour Party.

Professor David Fishwick is a member of the BLF.

Dr Gary Fuller is a member of the Air Quality Committee of Environmental Protection UK (EPUK) and his department holds a corporate membership. EPUK is a member-based non-governmental organisation (NGO) formerly known as the National Society for Clean Air (www.environmental-protection.org.uk/about-us/). He also declared that he has a freelance contract to write a monthly article on pollution in *The Guardian*. The remit of this article is to summarise recent air pollution in the UK. Additionally, he contributes four pieces a year on world air pollution. Each piece appears on the weather pages of the newspaper and online. He is paid £129 per article.

Professor John Henderson receives university departmental funds from Pfizer Inc to support pre-translational research into epigenetic mechanisms in asthma. F Hoffman-La Roche Ltd supports a phase III clinical trial of a pharmaceutical agent in severe asthma through his NHS trust and for which he is the local principal investigator (PI). He has no personal pecuniary interests in either company.

Dr Mike Holland is a subcontractor to a number of commercial consultancy organisations active in the field of environmental regulation, including Ricardo-AEA, AMEC and IIASA. Most work for these organisations is for the European Commission and associated agencies, and for the UK government. He has been consultant to a number of European pressure groups, providing independent assessment of the impacts of industrial facilities and prospective air pollution legislation. These include HEAL and EEB. The purpose of this work is to apply methods agreed with the World Health Organization and the European Commission to cases of interest to the NGOs. The work concludes with presentation of the completed analysis and associated report: he does not participate in any associated lobbying activities should the NGOs wish to use the information in this way. His earnings from these consultancy contracts are variable from year to year, but under €10,000 annually.

Mr Philip Insall is a consultant providing services to clients in the public, private, voluntary and academic sectors, relating to the development of evidence, policy and guidance on transport and public health. At the time he was co-opted onto the RCP/RCPC working party, he was an employee of Sustrans, a charity which, among other activities, campaigns on transport and public health issues including air quality. He has no clients or contracts related to his work with the working party, and has provided his expertise on the project without a fee.

Professor George Morris has received remuneration for work as a consultant in the broad area of environmental public health for the World Health Organization, the Dutch national environment and health agency, and the European Environment Agency. This has occasionally related specifically to air quality issues. He was recently lead author on a paper addressing the 'distal effects of climate change'. This was commissioned by the Natural Environment Research Council as part of the Living with Environmental Change initiative.

Dr Samantha Walker works for Asthma UK.

Executive summary

Why the RCP and the RCPCH are tackling this issue

Each year in the UK, around 40,000 deaths are attributable to exposure to outdoor air pollution, with more linked also to exposure to indoor pollutants.

Air pollution plays a role in many of the major health challenges of our day, and has been linked to cancer, asthma, stroke and heart disease, diabetes, obesity, and changes linked to dementia.

Neither the concentration limits set by government, nor the World Health Organization's air quality guidelines, define levels of exposure that are entirely safe for the whole population.

When our patients are exposed to such a clear and avoidable cause of death, illness and disability, it is our duty as doctors to speak out.

How we approached the task

This report is a joint effort by the Royal College of Paediatrics and Child Health (RCPCH) and the Royal College of Physicians (RCP).

The two colleges assembled experts in medicine and environmental sciences to discuss the evidence and draw up recommendations. We searched the literature and heard detailed evidence from experts and key organisations. A draft of the report was circulated to a wide range of stakeholders for comment.

Full details of the scientific references, evidence heard and stakeholders consulted are available on the RCP website.¹

Effects across a lifetime

This damage occurs across a lifetime, from a baby's first weeks in the womb all the way through to the years of older age.

Gestation, infancy and early childhood are vulnerable times because the young body is growing and developing rapidly. We know that the heart, brain, hormone systems and immunity can all be harmed by air pollution. Research is beginning to point towards effects on growth, intelligence, and development of the brain and coordination.

Harm to babies and children will have an impact that lasts far into the future. For the same reason, any air quality improvements we make now will have long-lasting benefits.

Older people, and adults with long-term conditions, are also vulnerable to the effects of air pollution. Improving air quality will help them to stay independent and well, benefiting individuals and easing the pressure on our NHS and social services.

The most vulnerable suffer the most harm

Air pollution is harmful to everyone. However, some people suffer more than others because they:

- live in deprived areas, which often have higher levels of air pollution
- live, learn or work near busy roads
- are more vulnerable because of their age or existing medical conditions.

Some chemicals in air pollution may be implicated in the development of obesity. It may be a vicious circle, because we also know that obese people are more sensitive to air pollution.

These vulnerabilities are heightened among those living in the most deprived communities. This is due to poor housing and indoor air quality, the stress of living on a low income, and limited access to healthy food and/or green spaces. Moving away from an area of high outdoor air pollution may be unaffordable for local residents. Some people may not want to leave their homes – and they should not have to.

Costs of air pollution

The annual mortality burden in the UK from exposure to outdoor air pollution is equivalent to around 40,000 deaths. To this can be added further impacts from exposure to indoor air pollutants such as radon and second-hand smoke.

The health problems resulting from exposure to air pollution also have a high cost to society and business, our health services, and people who suffer from illness and premature death. In the UK, these costs add up to more than £20 billion every year.

Vulnerable people are prisoners of air pollution, having to stay indoors and limit their activity when pollution levels are high. This is not only unjust; it carries a cost to these individuals and the community from missed work and school, from more health problems due to lack of exercise, and from social isolation.

Taking action will reduce pain, suffering and demands on the NHS, while getting people back to work, learning, and an active life. The value of these benefits far exceeds the cost of reducing emissions.

Air pollution and climate change

Air pollution plays a key role in the process of climate change, which places our food, air and water supplies at risk, and poses a major threat to our health.

Several pollutants that cause this environmental damage are also toxic to our bodies. Therefore, many of the changes that would decrease air pollution to protect our health – especially using energy more efficiently and burning less solid fuel and oil – would also help to slow down the overheating of our planet.

Recommendations for action and research

What must be done

Everyone has some responsibility for reducing air pollution. Real change will only occur when everyone accepts this responsibility, and makes a concerted effort. This includes European, national and local government, business and industry, schools and the NHS, as well as individuals in society at large.

- 1 Act now, think long term.** As a community, we must act now, and with urgency, to protect the health, wellbeing and economic sustainability of today's communities and future generations. Government must empower local authorities and incentivise industry to plan for the long term.
- 2 Educate professionals and the public.** The NHS and patient charities must educate health professionals, policymakers and the public about the serious harm that air pollution causes. Health professionals, in particular, have a duty to inform their patients.
- 3 Promote alternatives to cars fuelled by petrol and diesel.** Government, employers and schools should encourage and facilitate the use of public transport and active travel options like walking and cycling. Active travel also increases physical activity, which will have major health benefits for everyone. Local transport plans, especially in deprived areas, should:
 - expand cycle networks
 - require cycle training at school
 - promote safe alternatives to the 'school run', based on walking, public transport and cycling instead of cars
 - encourage employers to support alternatives to commuting by car
 - promote leisure cycling
 - develop 'islands' of space away from traffic, for safer walking and cycling.

European, national and local policies should also encourage the use of hybrid electrical and hydrogen-powered vehicles.

- 4 Put the onus on the polluters.** Polluters must be required to take responsibility for harming our health. Political leaders at a local, national and EU level must introduce tougher regulations, including reliable emissions testing for cars. They must also enforce regulations vigorously, especially in deprived areas where pollution levels are higher and people are more vulnerable.
- 5 Monitor air pollution effectively.** Air pollution monitoring by central and local government must track exposure to harmful pollutants in major urban areas and near schools. These results should be communicated proactively to the public, in a clear way that everyone can understand. When levels exceed EU limits or World Health Organization guidelines, local authorities must immediately publish serious incident alerts.
- 6 Act to protect the public health when air pollution levels are high.** When these limits are exceeded, local authorities *must* have the power to close or divert roads to reduce the volume of traffic, especially near schools.

- 7 **Tackle inequality.** Our most deprived communities are exposed to some of the worst outdoor and indoor air quality, contributing to the gap in life expectancy of nearly 10 years between the most and the least affluent communities. Regulators, local government and NHS organisations must prioritise improvements in air quality in our most deprived areas, setting high standards of emission control across all sectors of industry.
- 8 **Protect those most at risk.** Children, older people, and people with chronic health problems are among the most vulnerable to air pollution. Public services must take account of this disproportionate harm through local tools such as planning policies for housing and schools, equalities impact assessments, and joint strategic needs assessments. At an individual level, healthcare professionals should help vulnerable patients protect themselves from the worst effects of air pollution.
- 9 **Lead by example in the NHS.** The NHS is one of the largest employers in Europe, contributing 9.1% of the UK's gross domestic product (GDP). The health service must no longer be a major polluter; it must lead by example and set the benchmark for clean air and safe workplaces. In turn, this action will reduce the burden of air-pollution-related illness on the NHS. As pointed out in two earlier reports,^{2,3} the Department of Health, NHS England and the devolved administrations must give commissioners and providers incentives to reduce their emissions, and protect their employees and patients from dangerous pollutants.⁴

What can I do?

As citizens and members of the public, everyone can help by:

- trying alternatives to car travel or preferably taking the active option: bus, train, walking and cycling
- aiming for energy efficiency in our homes
- keeping gas appliances and solid fuel burners in good repair
- asking their local council and MP to take action
- learning more about air quality and staying informed.

The collective effect of actions by a large number of individuals, together with action by local councils and governments, can make a significant difference to pollutant exposure.

1 www.rcplondon.ac.uk/projects/outputs/every-breath-we-take-lifelong-impact-air-pollution

2 www.nihr.ac.uk/policy-and-standards/nihr-carbon-reduction-guidelines.htm

3 www.sduhealth.org.uk/documents/Carbon_Footprint_summary_NHS_update_2013.pdf

4 Tomson C. Reducing the carbon footprint of hospital-based care. *Future Hosp J* 2015;2:57–62.

In many areas, we need more research to improve our understanding of the impact of air pollution on our health, economy and communities.

- 10 **Define the economic impact of air pollution.** Air pollution damages not only our physical health, but also our economic wellbeing. We need further research into the economic impact of air pollution, and the potential economic benefits of well-designed policies to tackle it.
- 11 **Quantify the relationship between indoor air pollution and health.** We must strengthen our understanding of the relationship between indoor air pollution and health, including the key risk factors and effects of poor air quality in our homes, schools and workplaces. A coordinated effort among policymaking bodies will be required to develop and apply any necessary policy changes.
- 12 **Determine how global trends are affecting air quality.** From increased energy production and consumption to global economic development and urbanisation, we need to improve our understanding of how major social and economic trends are affecting air quality and its twin threat, climate change.
- 13 **Develop new technologies to improve air pollution monitoring.** We need better, more accurate and wider-ranging monitoring programmes so that we can track population-level exposure to air pollution. We also need to develop adaptable monitoring techniques to measure emerging new pollutants, and known pollutants that occur below current concentration limits. We must develop practical technology – such as wearable ‘smart’ monitors – that empower individuals to check their exposure and take action to protect their health.
- 14 **Study the effects of air pollution on health.** To appreciate fully the risk to health, we need further research on air pollution’s effects on the body. In addition to lung and cardiovascular disease, research into the adverse health effects of pollution should accommodate systemic effects such as obesity, diabetes, changes linked to dementia, and cancer, as well as effects on the developing fetus and in early childhood.

Definitions

Air pollutants

Black carbon	This is the part of particulate matter that most strongly absorbs light. It is created by the incomplete combustion of fuels and is a major part of soot.
Carbon monoxide (CO)	This gas is produced when carbon-containing fuel burns without an adequate supply of oxygen. Outdoor concentrations of carbon monoxide in the UK are generally low. Malfunctioning indoor gas heating appliances and other fuel-burning devices used indoors may generate high, toxic levels of CO.
Diesel exhaust	Diesel exhaust is composed of <i>gases</i> and <i>particles</i> . Diesel exhaust gases include compounds such as benzene, 1,3-butadiene, formaldehyde and polycyclic aromatic hydrocarbons (PAHs). <i>Diesel exhaust particles (DEPs)</i> have a range of sizes and compositions. Small DEPs often contain elemental carbon (soot), with many toxic compounds on their surface. These include organic compounds, sulphate, nitrate and metals.
Metals and metalloids	These include <i>lead (Pb)</i> , <i>mercury (Hg)</i> , <i>arsenic (As)</i> and <i>nickel (Ni)</i> . <i>Lead</i> air pollution used to be dominated by emissions from road transport, but is now dominated mainly by activities in the iron and steel industry sectors. <i>Mercury</i> emissions are from a number of industrial production processes, but the most important is the manufacture of chlorine from Hg cells. Improved controls and new technologies have reduced emissions in the UK. <i>Arsenic</i> is a metalloid, ie its properties lie somewhere between those of the metals and the non-metals. The largest source of arsenic in the UK is the disposal of treated wood by burning. <i>Nickel</i> is mainly emitted as particles or associated with particles. UK emissions of nickel have declined significantly owing to reductions in the combustion of oil in power stations, industry and for domestic heating.
Nitrogen dioxide (NO ₂)	This gas is a member of a family of compounds called <i>oxides of nitrogen</i> or <i>NOx</i> . Some NO ₂ is produced directly by combustion. Another source of NO ₂ is the oxidation of <i>nitric oxide (NO)</i> in the air; this can happen either slowly by reaction with oxygen or more rapidly by reaction with ozone. NO ₂ levels are higher close to road traffic, or indoors where there is gas cooking. Oxides of nitrogen and particle concentrations are highly linked (correlated) in air samples from city roadside sites and it has been difficult to separate out their effects.

Ozone (O₃)

This is a gas consisting of three oxygen atoms joined together. It is formed by chemical reactions between other air pollutants, in particular the reaction of *oxides of nitrogen* with carbon compounds called *volatile organic compounds (VOCs)* in the presence of sunlight. These reactions take place over periods of several hours or even days. Ozone levels therefore tend to be higher in the countryside than in cities, and greater in summer than winter. Some ozone travels over large distances. Ozone levels fluctuate markedly over time and are highest in hot, bright weather.

Particulate matter (PM)

Particles in the air are a complex mix of materials. The health effects covered by this report mostly relate to particles that are generated by human activity ('anthropogenic' particles), either directly or by chemical reactions in the air. However, some natural dusts (eg soil and sand particles) may also be small enough to be breathed into the lungs. Particle concentrations are usually expressed as mass (micrograms; g) per cubic metre of air (m³).

Historically, concentrations of *particulate matter (PM)* were determined optically by measuring the 'blackness' of a filter after air had been drawn through it. This measurement was called *black smoke* and is practically the same as the *black carbon* measurement, as this is the part of *PM* that most strongly absorbs light. Black carbon is created by the incomplete combustion of fuels and is a major part of soot – for example diesel soot. Besides elemental carbon it contains *polycyclic aromatic hydrocarbons (PAHs)*.

The 'black smoke' metric has been largely replaced by metrics of *PM* based on the *aerodynamic diameter of the particles*. Thus *PM*₁₀ is the amount of particulate matter that is generally less than 10 µm in diameter. *PM*_{2.5} is the smaller fraction of *PM*₁₀ and consists of particles <2.5 µm across (often referred to as 'fine' particles). *Coarse PM* is the difference between *PM*₁₀ and *PM*_{2.5}. *Ultra-fine particles* are the smallest fractions of *PM*_{2.5} and are <0.1 µm (100 nm) in diameter. Differentiation of *PM* into fractions of different sizes is very relevant for health studies because fractions of <10 millionths of a metre (10 µm) in diameter tend not to be filtered out by the nose, and are able to penetrate down into the lungs. In contrast, very small particles (nanoparticles), which range in size from 0.1 to 0.001 µm, are so small that they can pass into the circulation. The surface area:volume ratio of particles increases exponentially as particles become smaller and smaller.

PM consists of *carbon*, *ammonium nitrate* and *ammonium sulphate*, *oxides* and *salts* of many *metals*, and *organic materials* including rather large and complex molecules and fragments of soil. The composition of the *PM* mixture varies from place to place. Near to roads, emissions from vehicles form the majority of *PM*, whereas in rural areas there is

an increased amount of PM produced by chemical reactions in the air including nitrates and sulphates.

Another way of describing PM is by origin. *Primary particles* are those that are emitted directly to the air. These include particles emitted during fossil fuel combustion, brake and tyre wear, and road dust resuspended by vehicles. *Secondary particles* are particles formed by atmospheric processes. These include *ammonium nitrate* and *sulphate*. The origins (precursors) of secondary particles include nitrogen dioxide, sulphur dioxide, ammonia and volatile organic compounds.

Polychlorinated biphenyls (PCBs)

These are synthetic chemicals that do not occur naturally in the environment. The commercial production of PCBs is banned. Sources of PCBs in the air are landfill sites containing electrical equipment such as transformers and capacitors. Incineration of municipal waste may lead to PCB pollution.

Polycyclic aromatic hydrocarbons (PAHs)

These are produced when the burning of fuels is incomplete. PAHs can exist in over 100 different combinations. Examples of PAHs are benzo(a)anthracene and benzo(a)pyrene.

Sulphur dioxide (SO₂)

In the UK, this gas is largely emitted from industrial sources including power stations. The contribution from motor vehicle exhausts has been much reduced in recent years, owing to the use of low-sulphur fuels.

Volatile organic compounds (VOCs)

These are organic compounds with a low boiling point, and therefore a tendency to evaporate. *Benzene* and *1,3-butadiene* are two important VOCs that are emitted from petrol car exhausts and directly from petrol. These emissions from traffic have been reduced through the introduction of three-way catalysts for petrol cars. *Total volatile organic compounds* are also known as *TVOCs*.

Other terms

Association

An association is the statistical relationship between two measured quantities. Air pollution studies that report associations are examining the relationship between concentrations of an air pollutant and a health endpoint. Such an association (or correlation) is not necessarily causal and may be due to chance, bias or some other factor.

COC

Committee on Carcinogenicity

COMEAP

Committee on the Medical Effects of Air Pollutants

COSHH

Control of Substances Hazardous to Health

EC

European Commission

Epidemiological studies	These are studies of the causes of diseases in populations.
ESCAPE	European Study of Cohorts for Air Pollution Effects
EU	European Union
Meta-analysis	This is a statistical technique whereby all data from all available studies are combined. In some cases, meta-analysis reveals statically significant effects by combining individual studies that are not statistically significant. Meta-analysis often uses data that have been identified by a <i>systematic review</i> . A systematic review aims to identify all relevant published and unpublished evidence, select studies for inclusion by an agreed process, assess the quality of each study, interpret the findings and present a balanced and impartial summary of the findings.
SIDS	Sudden infant death syndrome
Spirometry	This technique measures how much air and how fast a person can breathe in and out. Spirometry measurements may be described as the percentage predicted of normal after 'adjusting' for subject height and weight. Thus an individual with an output of spirometry that is 100% of predicted will have the average value expected of a group of healthy subjects of similar height and weight. The <i>forced expiratory volume in 1 second (FEV₁)</i> is an output of spirometry that measures the total amount (in litres) of breath expelled in the first second after first taking in the largest possible breath. Another spirometry output is the <i>forced vital capacity (FVC)</i> , which is the total amount of air (in litres) that can be breathed out by an individual, after taking the largest possible breath in.
WHO	World Health Organization

Introduction

With increasing concerns over the adverse health effects of air pollution on human health, the primary aim of this enquiry by the Royal College of Physicians (RCP) and the Royal College of Paediatrics and Child Health (RCPCH) was to raise awareness of new issues affecting health, across the lifecourse, from indoor and outdoor air pollution and in relation to a changing environment. Recognising that there was already a strong evidence base for the health effects of acute air pollution episodes, on the basis of new evidence, we decided to focus our enquiry on the health impacts of continuous exposure to chronic air pollution over a lifetime, with specific reference to:

- pregnancy and children as well as adults
- indoor as well as outdoor air pollution exposure
- the influence of local, regional and national policy relating to pollution control measures
- examining the influences of climate change.

In addition, during our enquiry we recognised the importance of socio-economic impacts of air pollution and so we added this dimension to our terms of reference.

More specifically, the scope of this report covers:

- the effects of prenatal and childhood exposure to air pollution on susceptibility to chronic disease over the lifecourse; this covers respiratory disease, cardiovascular disease, systemic effects such as diabetes, obesity, central nervous disease and cancer, as well as effects that maternal exposure to air pollution has on the developing fetus, such as miscarriage, stillbirth, premature delivery and low birth weight
- the impact of outdoor pollutants from vehicle exhaust, as well as indoor pollutants (including carbon monoxide (CO), volatile organic compounds (VOCs) and emissions from fossil fuel fires and stoves) on health; the recently introduced tobacco smoking legislation in public places is used as an example of health gains that can be made through exposure reduction
- a predominantly UK focus, but drawing information from research conducted in other countries and, where appropriate, comparisons with rapidly and slowly developing nations
- special attention being paid to vulnerable groups, including deprivation, poor housing and other socio-economic factors, and the overall cost of air pollution to society
- the influence of changing age demographics, urbanisation and climate change on air pollution and associated health risks.

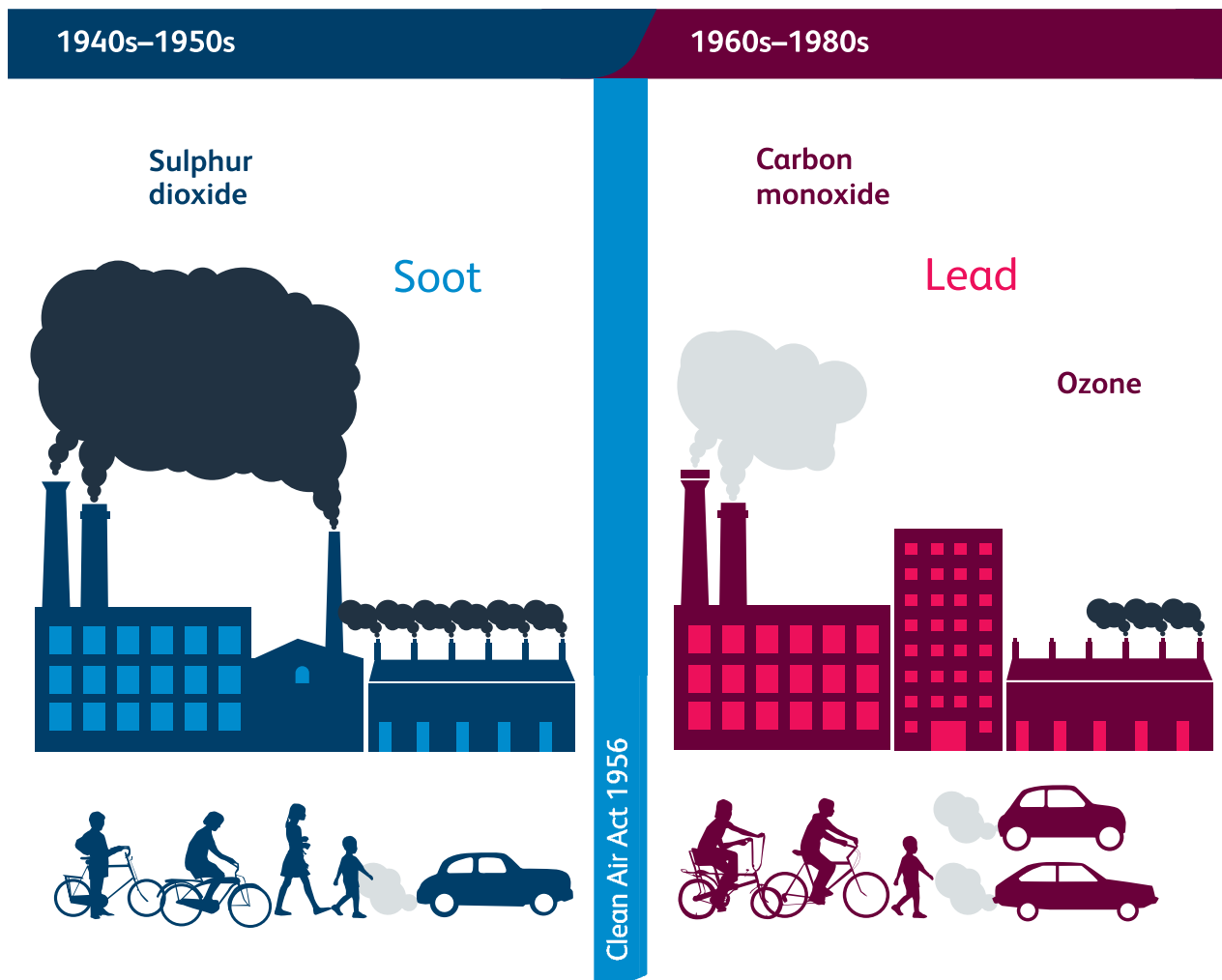
While there is abundant literature on the adverse health effects of air pollution, this report specifically concentrates on the newly recognised, insidious effects of chronic and persistent pollution exposure from conception to old age. It takes account of total pollution exposure sources, both outdoors and indoors, as well as the influence of combinations of pollutants acting together and, finally, viewing air pollution as a stressor that interacts with many other stressors such as diet, socio-economic deprivation and climatic conditions to create reduced health and increased susceptibility to disease. In taking a holistic and multidisciplinary view of the current air pollution problems and trends over time, we have identified this as a major public health problem, which we address in a series of recommendations that mandate urgent and definitive interventions to protect the public, especially those people in society who are most vulnerable.

Chapter 1: Summary

Air pollution is not a new problem in the UK. The London smog of 1952 killed 12,000 people. Since then, changes in the way we live have also changed the air pollution that we breathe. Coal burning has fallen dramatically, but today increased road transport and the failure to control some exhausts from diesel vehicles has led to us being exposed to new air pollutants.

Looking at different generations tells the story. As children, today's grandparents were exposed to soot and sulphur dioxide from coal burning. Those now in middle age breathed in emissions from leaded petrol. Today's children walk and cycle much less, and they inhale nitrogen dioxide and the tiny particulates from diesel-fuelled vehicles.

Around the world, there are many examples where reducing air pollution has improved public health. It now seems likely that childhood exposure to air pollution has a lasting influence on health, so the gains from tackling air pollution today will be felt throughout the decades to come.

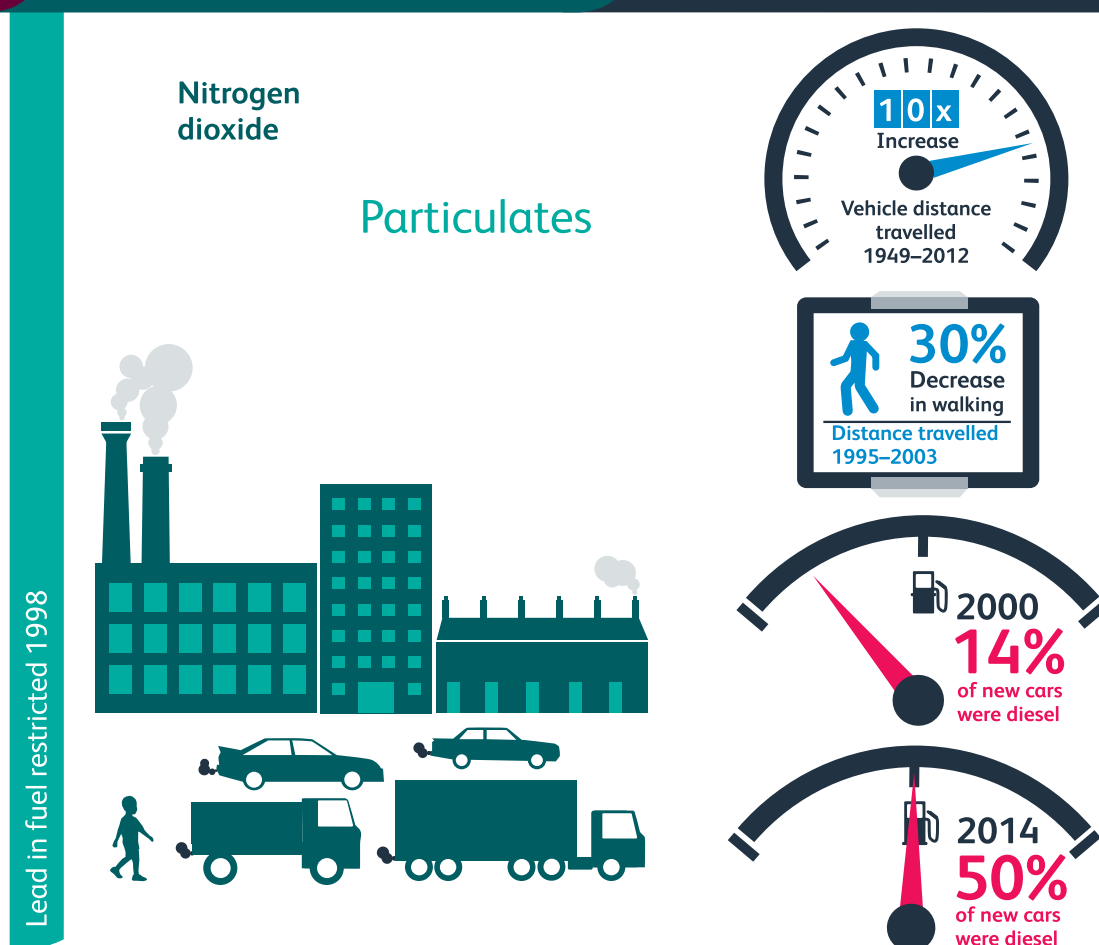


Key facts

- In 2012, road traffic in the UK was ten times higher than in 1949. Total distance walked each year decreased by 30% between 1995 and 2013.
- Growth in pollution has not always been as fast as growth in traffic, thanks to tighter exhaust controls. Modern cars produce very little carbon monoxide and hydrocarbons, and the sulphur and lead in diesel and petrol must meet tight regulations.
- Nitrogen dioxide and particulates from diesel engines have been poorly controlled and these remain a problem. In the UK today, about half of cars run on diesel. This is the trend across Europe, but not in the USA or Japan. Nearly all buses, vans and lorries, forms of water transport, and many trains, use diesel in the UK, along with construction and farm machinery.
- Each year, inhaling particulates causes around 29,000 deaths in the UK, which, on recent evidence, may rise to around 40,000 deaths when also considering nitrogen dioxide exposure.
- Home heating has changed, too. Compared with coal fires, modern gas boilers produce very little particle pollution – but they do give off nitrogen dioxide. Cooking, especially with gas, is also an important source of nitrogen dioxide and particles.
- Air pollution can stay around for days or weeks after it's created. One type of chemical may interact with others in the atmosphere, to cause even more pollution. Air pollution also crosses cities, counties and even countries, so local action is not enough on its own.

1980s–2000s

Across this period in time:



Chapter 1: Air pollution in our changing world

The London smog of 1952, in which up to 12,000 people died,¹ was a defining event in air quality management; it brought about the Clean Air Acts and the start of 3 decades of concerted actions to control public exposure to air pollution. Today, life in the UK is very different from how it was in the 1950s. Much more than legislation has changed since this time, with various social, fuel and technology transitions driving a huge change in outdoor air pollution – changing old pollution sources and introducing new hazards into our breathed environment. Concerns about black smoke and air acidification (sulphur dioxide, SO₂) from coal burning have been replaced by new concerns about particle pollution and nitrogen dioxide (NO₂) from transport, and the air pollution that forms through chemical reactions between other pollutants in the atmosphere. With new knowledge that pollutants can remain in the air for days or even weeks, air pollution has moved from being a local problem to one that requires source control at city, regional and even international scales. Each year in the UK the equivalent of around 40,000 deaths can be attributed to outdoor air pollution linked to exposure to fine particulates and NO₂ (see Chapter 6).^{2,3}

Box 1: Six US cities that changed our knowledge of modern air pollution

By the 1990s, the pollution and mortality peaks linked to wintertime smog seemed consigned to history; the Clean Air Acts and the advent of natural gas heating had displaced solid fuel from our cities. In 1974, US scientists⁴ began following the lives of 8,111 people across six US cities. Of these people, 1,490 had died by 1991, but these deaths were not spread evenly. Having accounted for factors including smoking rates, education level, diabetes and workplace pollution exposure, the worst-affected city had a death rate around 30% greater than that of the least-affected city. Across the six cities, the difference in death rates was found to be associated with airborne particle pollution; this was not from air pollution experienced during short smog episodes, but rather from long-term exposure to everyday pollution concentrations. Although the pollution mixture had changed, long-term exposure to modern air pollution was exerting a heavy health burden, leading to the conclusion that modern pollutants needed better control, not just during smog episodes but every day.

Returning to the same people 8 years later, researchers⁵ were able to repeat and confirm the earlier findings with respect to particle pollution and survival, but with one important difference: those cities with improved air pollution saw improved survival rates, suggesting that at least part of the air pollution health effects might be reversible and that improvements in air pollution can lead to better city-wide health outcomes.

1.1 Changes to transport

Changes to transport systems and habits were a defining movement of the last half of the 20th century. In the 1950s, car ownership was beyond the reach of the majority of families; today, many families regard car ownership as an essential part of life, providing mobility and access to jobs, shops and leisure.

Between 1949 and 2012, a massive investment in roads and road vehicles led to a tenfold increase in the distance travelled in the UK (Fig 1). This 'freedom' has not been universally accepted as a good thing.

Increased car use has affected pollution emissions; it has also changed our air pollution exposure patterns, with more transport time spent in cars in polluted road environments. The charity Sustrans suggests that bias in favour of investment in road building and motorised transport has led to a ‘windscreen perspective’, ie viewing transport issues from the driver’s perspective only. The Royal Commission on Environmental Pollution⁶ pointed to the dominance of road traffic in many urban areas being linked to a web of environmental and social causes, with outcomes including air pollution, urban severance for those without car access, a decline in walking, and closure of local shops.

The types of traffic on our roads have changed; freight transport has seen massive growth, especially with respect to light goods traffic and vans, which have grown in number by over 60% since the turn of the century. Road traffic growth has slowed in recent years. The distance driven on UK roads grew by 50% during the 1980s, by 14% during the 1990s and then by 6% between 2000 and 2009. Road miles peaked in 2007, followed by three consecutive years of decrease: the first consecutive annual falls since traffic records began.⁷ However, transport planners dismiss the notion that we might have achieved peak car use; the current hiatus in road traffic growth is expected to be temporary. Traffic is expected to grow by a further 19–55% between 2010 and 2040, and is being reinforced by the rapid growth of small packet road transport.⁸

Growth in traffic since the 1950s does not necessarily mean a proportionate growth in air pollution. For over 20 years, ever-tightening standards for exhaust emissions have been applied to new vehicles sold across Europe, although concerns are being expressed about how suitable such testing is under realistic driving conditions (<http://theicct.org/nox-control-technologies-euro-6-diesel-passenger-cars/>). There also remains concern about emissions when starting from cold; however, overall the abatement of air pollution from petrol vehicles has been very effective – a new petrol car emits less than one-twentieth of

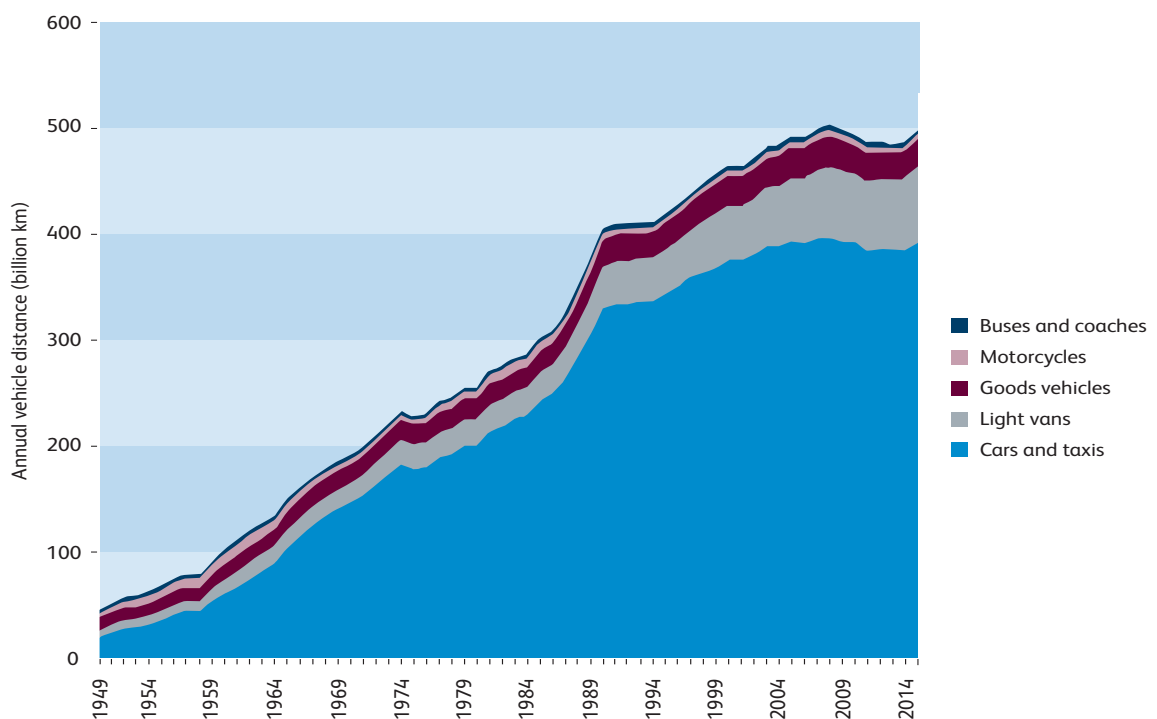


Fig 1. Annual distance travelled by road in the UK⁷

the levels of nitrogen oxides emitted by cars made in 1992, before catalytic converters were required. There have also been huge cuts in CO and hydrocarbons (VOCs) in petrol exhausts. In contrast to petrol engines, the technology applied to clean up diesel vehicles has not yielded significant improvements in emissions of nitrogen oxides, and the proportion of NO₂ in diesel exhaust has actually increased.⁹

The types of fuel used to power road transport have changed over the past 20 years. Although heavy vehicles such as lorries and buses have always been powered by diesel, this was not the case for smaller vehicles that predominantly ran on petrol. In 2000, just 14% of new cars were diesel powered, but today this figure has risen to 50%, and almost all light goods vehicles and vans are now powered by diesel too.¹⁰ This increase in diesel vehicles is very much a European phenomenon. Indeed, diesels play almost no role in car markets in the USA or Japan. Although diesel vehicles are marketed on their low carbon dioxide (CO₂) emissions, the Japanese car market has reduced CO₂ emission further and faster than Europe by investment in petrol engine technology.¹¹ The combination of an increased number of diesel vehicles and the technological difficulties in abating their real-world emissions has meant that urban concentrations of airborne particulate matter (PM) and NO₂ have not improved, as had been hoped. European limits for NO₂ were set in the late 1990s, to be met by 2010, but today busy roads in UK urban areas still fall a long way short of meeting these limits; in places, they are being exceeded by up to threefold.¹² In addition to arterial roads, the main problem areas are urban centres that are dominated by diesel vehicles; these include many shopping streets where traffic overwhelmingly comprises buses, delivery vans and taxis. Although tighter approval tests are expected to result in new diesel vehicles producing less pollution, it

Box 2: Better fuel can mean less pollution

In the latter part of the 20th century, petrol was the main source of lead in urban air. Its use as a fuel additive peaked in the 1970s and 1980s. By this time, lead contamination in the environment had reached global proportions. Around the world analysis of ice sheets, lake and marine sediments and peat deposits showed increased concentrations compared with pre-industrial levels.¹³ The Royal Commission on Environmental Pollution's 1983 report on lead in the environment¹⁴ was a seminal moment in the recognition of the harm to the population arising from lead exposure. The commission called for a phased end to leaded petrol within 6 years. Today, lead is no longer used in petrol in the UK, but it is disappointing that the UK was one of the last countries in Europe to completely remove lead additive from fuel (in 1999). Since the reduction and eventual ban of its use as a petrol additive, there have been clear changes in lead in children's blood. This has decreased from concentrations in the 1970s that were considered harmful to fetuses and small children.¹⁵

Many improvements have been made to oil-based fuels in recent decades. In addition to the phasing out of lead additives in petrol, the other major change has been the regulated decrease of sulphur in fuels, prompted by the need to control gaseous SO₂ and particulate sulphate pollution, and to enable catalysts and other exhaust-control technologies. The transition to ultra-low sulphur diesel in the UK in 2007 caused a decrease of 30–60% in particle number concentration,¹⁶ a measure of airborne particles that had previously been linked to cardiac hospital admissions and deaths in London.¹⁷ The decrease in the maximum allowed sulphur content in marine fuel in most European waters in 2006 caused a notable improvement in air quality, for example, in the Port of Dover. In Hong Kong, restriction on the sulphur content of heavy fuel oil in 1990 was associated with decreases in all-cause, cardiac and respiratory death rates (by 2–4%).¹⁸

will be many years before the vehicle fleet is completely replaced. Recent concerns over software that allows cars to cheat in emissions tests, making them appear cleaner than they actually are, has not added to public confidence in air pollution control.

The composition of transport fuel also has undergone marked changes in the UK and across Europe. Lead is no longer used as an additive in petrol, and sulphur impurities are now removed before fuel is sold. The introduction of ultra-low sulphur diesel in 2007 caused an almost-immediate decrease of up to 60% in the level of sub-micrometre particle pollution in the air that we breathe.¹⁶ This was probably the most rapid improvement in UK urban air quality ever seen. Pollution from road use is not limited to exhaust pipe emissions. It is now recognised that particle pollution also comes from the wear of brakes, tyres and the road itself. Evidence suggests that particles from these sources are rich in transition metals, inhalation of which is associated with toxicological effects. In contrast to exhaust emissions, no regulations exist to control these sources of particles and, with the trend towards heavier vehicles, they look set to increase. Pollution from tyre, brake and road wear also means that even electric and alternatively fuelled vehicles can never be emission free at the point of use.

Box 3: Do low emission zones work?

Excluding the most polluting traffic is a popular way for European cities to try to clean up their air. Progressively tighter emissions standards mean that newer cars should emit less pollution than older ones. By banning higher-polluting vehicles or charging their owners, cities can reduce their traffic emissions faster than waiting for the natural rate of vehicle replacement.

London has Europe's largest low emission zone (LEZ), but this only applies to medium and large vehicles and not to cars and small vans. Other LEZs in Oxford and Brighton ban only the most polluting buses. Elsewhere in Europe, over 200 schemes operate in 12 countries, with the majority being in Italy and Germany. Many of these include passenger cars.¹⁹

Evidence that LEZs work is mixed. Sadly, progressively tighter tests for new vehicles have not delivered the hoped-for reductions for some pollutants in real-world driving. This is especially the case for nitrogen oxides (NO_x) from diesel vehicles, meaning that the increasing popularity of diesel vehicles can undo the positive benefits from other policies to decrease air pollution.¹⁹ Another difficulty is the diversity in types of LEZ and other local policies, which makes it hard to compare cities.

In London, the UK's only city-wide LEZ, links have been found between NO_x exposure and clinically significant impairment of children's lung growth. Three years after the introduction of the London scheme, there was no evidence of improvement in air quality or in children's respiratory health, leading to the conclusion that more aggressive pollution control measures are needed (Mudway *et al*, unpublished data). In Germany a national framework means that all vehicles, including cars, are required to display a red, yellow or green sticker according to their exhaust pollution. The 'cleaner' stickers (yellow and green) are harder to obtain for diesel vehicles owing to their greater emissions compared with petrol. Consistent design means that results from over 70 German cities with LEZs can be viewed together, increasing the power to detect any beneficial effect. Here, air pollution in areas with LEZs has improved faster than in those cities without zones.^{20,21} Additionally, 'dirty vehicles' did not simply move into the areas outside the zone; instead, drivers and especially businesses around the LEZs upgraded their vehicles or bought newer ones.²⁰

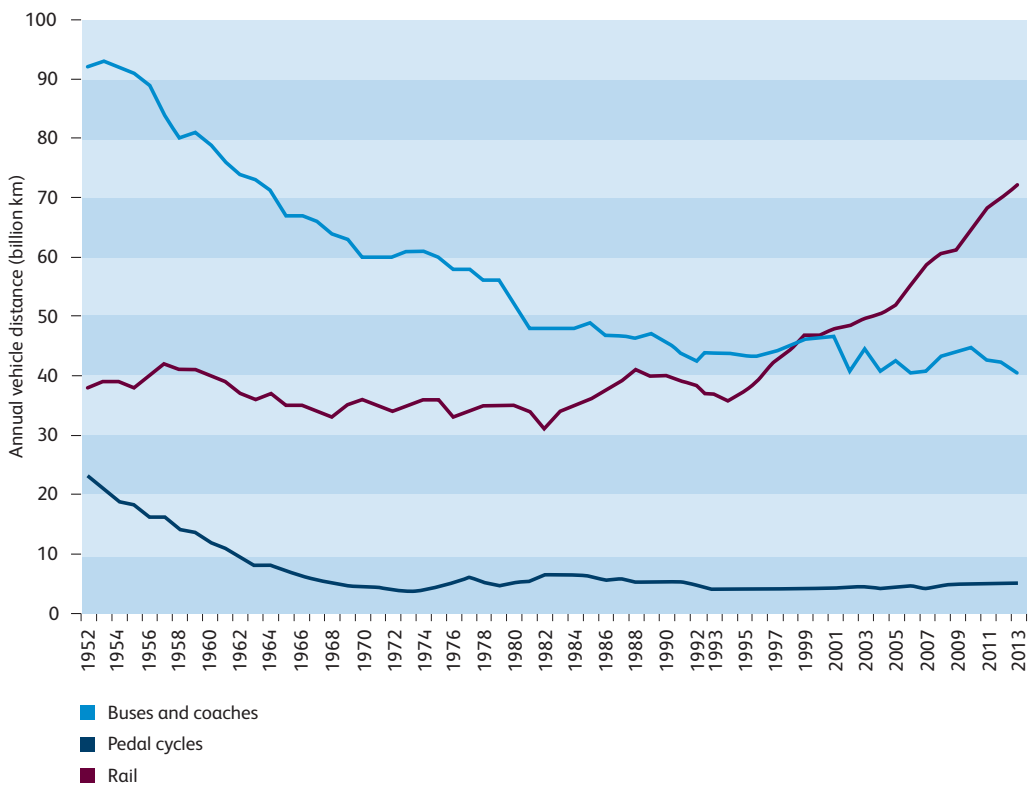
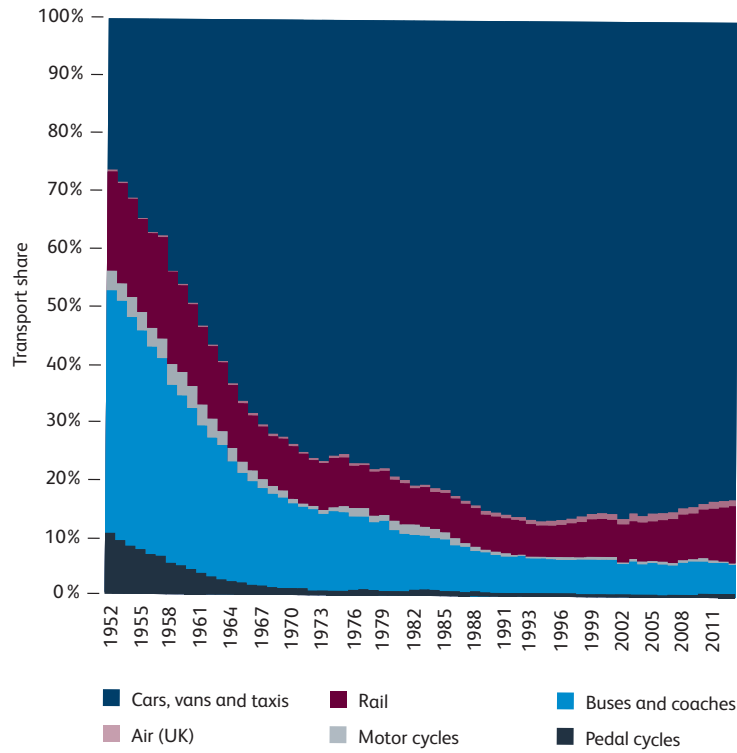


Fig 2. The upper panel shows transport share by mode. The lower panel shows distance travelled by pedal cycle, rail and bus/coach.⁷

In contrast to the growth in motorised traffic (Fig 2), active transport such as walking and cycling has declined progressively since the 1950s. The total distance walked each year declined by 30% between 1995 and 2013,²² and the distance cycled in England and Wales in 2012 was just 20% of that in 1952.⁶ However, there are some signs of a reversal; trends over the last decade show a slow return to cycling. Many studies have underlined the public health benefits of increased active travel by both cycling and walking, with the benefits outweighing the increased risks from accidents and air pollution exposure by a factor of at least ten.²³ Continued focus on controlling urban air pollution through technical measures to abate vehicle exhaust provides less benefit for public health than focusing on measures that increase active travel and public transport (where active travel is often part of the journey).

Looking at other modes of transport, the globalisation of manufacturing and growth in international trade have led to large increases in both air travel and shipping since the 1950s. Air pollution from shipping spreads beyond ports and makes an important contribution to airborne particle pollution across Europe. The greatest air pollution from shipping occurs in coastal countries, including the UK.²⁴ Airports also impact on the communities around them; for example, Heathrow makes a well-recognised contribution to NO₂ concentrations across suburban west London.²⁵

1.2 Heating our homes

The automated central heating systems that most of us enjoy today are a far cry from the daily labour of cleaning and making fires that featured through most of our history. Today, the vast majority of homes are heated by gas or electric systems that provide warmth at the touch of a button or the click of a timer switch. For most UK cities, smoky home fires are a thing of the past. Compared with the sulphur, soot and particle pollution emitted from open coal fires, gas boilers produce very little particle pollution, but they do contribute to urban NO₂.

There are, however, some important differences in the nature of emissions and prevailing pollution levels across the UK. Notably, in Northern Ireland, outside Belfast the gas distribution grid is less developed. As a result, small towns can still experience high levels of airborne particles and polyaromatic hydrocarbons (PAHs) from local coal and oil burning.

Box 4: How banning coal improved the health of Dubliners

Although the availability of natural gas transformed urban heating in the UK, this was not the case in Ireland. In Dublin, increased oil prices brought about an increase in coal heating in the 1980s, with associated wintertime smogs that could be linked to respiratory deaths. On 1 September 1990, the Irish government banned the sale and distribution of bituminous coals in the city. The change in air pollution was immediate, with black smoke decreasing by 70%. Respiratory deaths in Dublin decreased by 17% after the ban, and there was a 9% decrease in Cork when the ban was extended in 1995.²⁶

We must, however, be cautious before consigning solid fuel burning to history. The increasing popularity of wood burning for heating, in part due to policies to reduce CO₂ emissions, risks undoing some of the air quality improvements that have resulted from widespread adoption of gas for domestic heating. Particles from wood burning can now be found each winter in our urban air, mainly at weekends, with wood burning accounting for between 7 and 9% of London's wintertime particle pollution.²⁷ Studies

have shown that smoke from wood heating enters neighbouring homes, providing a clear exposure pathway.^{28,29}

The outdoor environment is not the sole source of the pollution that we experience indoors. Being indoors can offer some protection against outdoor air pollution, but it can also expose us to other air pollution sources. There is now good awareness of the risks from badly maintained gas appliances, radioactive radon gas and second-hand tobacco smoke, but indoors we can also be exposed to NO₂ from gas cooking and solvents that slowly seep from plastics, paints and furnishings. The lemon and pine scents that we use to make our homes smell fresh can react chemically to generate air pollutants, and ozone-based air fresheners can also cause indoor air pollution.

Box 5: The effects of indoor smoking bans can be seen in health data

There is a large body of research into the effects of smoke-free legislation. Various bans are now in place in 92 countries. As a tool to improve indoor air pollution, smoke-free legislation has been very effective; for example, PM_{2.5} (particulate matter that is generally <2.5 µm in diameter) decreased by 86% in Scottish bars.³⁰ However, it is the decrease in heart attacks across the whole population that is most striking. In 2014, a review of 37 studies concluded that indoor smoking bans led to an average 12% decrease in acute coronary events.³¹ Other studies have shown that indoor smoking bans were followed by decreases in hospital admissions for childhood asthma (15%)³² and decreases in the incidence of stroke.³³ Despite fears, smoking bans have not led to more smoking in cars or homes; in Scotland, there is evidence that people with children stopped smoking inside their own homes as well.³⁴

1.3 Powering our homes and industry

The iconic smoke stacks of Battersea Power Station and those that now make Tate Modern a London landmark are a reminder of the extent to which coal and heavy oil used to be burned to generate electricity in the very hearts of our cities. The consequential impacts on urban air pollution can be clearly seen in the 1952 maps of London's air pollution³⁵ and were very obvious in the black staining of the stonework of buildings at the time.

Today, electricity comes from larger power stations that are subject to modern emission controls and are predominantly located outside our cities. Although a quarter of our electricity still comes from coal-powered generation, this is set to fall still further. Less-polluting sources such as gas, nuclear and renewables now dominate electricity generation in the UK. Even in the past 20 years, controls on emissions from large power stations have brought an end to the SO₂ problems from power station plumes across Yorkshire, London and other urban areas each summer and winter.

A new focus on decreasing energy consumption should progressively reduce air pollution from power generation and industry. Looking to the future, the closure of further parts of the UK's coal-fired generating capacity and growth in other fuelled sources and renewables will lead to more air quality improvements. However, a proposed return to small-scale urban energy production through combined heat and power systems needs to be properly controlled and managed to avoid additional contributions to local air pollution.³⁶

1.4 Increased urbanisation

Globally, over half of the world's population now lives in cities. Urban dwelling brings people into close proximity to transport infrastructure, along with the pollution from buildings. Managing the air quality consequences of growing urbanisation is a world problem. Within the UK, the proportion of the population living in urban areas has been relatively stable at 80%, with only 2% growth since 1980.³⁷ However, absolute numbers are increasing: between 2001 and 2011, the urban population of England and Wales increased by 8.1%. London stands out, with a decadal growth of 14%³⁸ and a projected additional 1.4–1.7 million people by 2031,³⁹ many of these being children and young people. New urban dwellers will require places to live, schools and amenities, along with increased requirements from transportation: the delivery of goods, services and personal travel. Therefore, we need to ensure that the air pollution exposure of the new urban population does not lead to an additional public health burden, especially recognising relentless growth in the population. To date, little attention has been paid in urban or rural planning to the air pollution exposure in housing and schools due to their proximity to roads.

Through the 1990s, the average urban car journey increased by 20% owing to growing long-distance commuting and non-work travel. Compact mixed-use developments (residential, educational, leisure and business) can reduce the need for travel, whereas the separation of housing, employment, leisure and services can make lengthy car journeys a necessity rather than a choice, and increase social exclusion for those without access to a car.⁵ Even within our current cities, there is considerable scope for active travel for short journeys: in 2013, 23% of car trips in England were shorter than 2 miles.⁴⁰

1.5 New knowledge about the spatial scale of air pollution problems

UK measurements of air pollution in the 1950s through to the 1970s focused on the perceived priorities of black smoke (soot) and air acidification through SO_2 , which both came from local coal burning. This focus meant that many other pollutants were overlooked, including those from traffic and those that form secondarily in the atmosphere from other pollutants (especially O_3 , and ammonium nitrate and sulphate particles). During the mid-1990s, new measurements of PM_{10} (mass of particles $<10 \mu\text{m}$ in diameter) led to the recognition that air pollution over the UK could be substantially influenced by emissions from outside the country⁴¹ from distant industry, traffic and agriculture. Similarly, measurements initiated during the 1970s⁴² found that the UK experienced problems with summertime smog: specifically ground-level O_3 , the pollutant synonymous with the Los Angeles smogs of the 1950s and 1960s. This smog can take days to form, as a result of atmospheric chemistry catalysed by sunlight (photochemical pollution), during which time air can slowly move over hundreds of miles. While it is recognised that air pollution from a factory can affect people and the environment downwind, there is less recognition of the impacts of cities on their surrounding regions. Control of modern air pollution must therefore take place at different spatial scales, from the local busy road to the urban scale. There is an urgent need for cities to collaborate to reduce the impact of the pollution that they emit into the air that they share.

1.6 Taking control of our air

Air pollution management has evolved in tandem with our increasing knowledge of the health burden caused by poor air. The first attempts to control urban air pollution were directed towards single sources: medieval London's prohibitions on the burning of sea coal and the Victorian Alkali Acts, which focused on local impacts from the early chemical industry. The death toll from the 1952 smog led to an evolution in air quality management, with the focus on point sources being supplemented by area-wide actions on

smoke control mandated by the Clean Air Acts. Today, air pollution management has moved beyond source control to the setting of internationally agreed limits on the quality of outdoor air. It takes a multi-source and multi-pollutant approach, guided by analysis of cost-effectiveness and cost–benefit ratio. Such analyses show that the cost of air pollution is so high that far greater investments in tackling the problem would still yield a positive benefit to society. Despite this, few people consider pollution risks in their everyday lives. In its 2011 report,⁴³ the parliamentary Environmental Audit Committee called for ‘[a] public awareness campaign to drive air quality up the political agenda and inform people about the positive action they could take to reduce emissions and their exposure’. In contrast, current information – such as the advice given during periods of high pollution – focuses on individuals, who are advised to restrict their lives, rather than requiring emission reductions by polluters.

With research telling us that air pollution is still harmful even below current limits, and a lack of evidence of a threshold where no effects exist for many pollutants, further control policies should seek to decrease pollution exposure, even where limits are met.

1.7 Conclusions

Since the smogs of the 1950s, numerous transitions in our society, the way in which we lead our everyday lives, our home heating and our travel, along with legislative changes on air pollution emissions, have brought about huge changes in the nature and amount of air pollution to which we are exposed. Not all of these changes have been expected or intended through policy. Research has also changed our perspective on the health risks of air pollution; an emphasis on controlling short pollution peaks from solid fuel burning has been replaced by concerns about long-term exposure to pollution from transport sources.

Looking across the generations alive today, we can see contrasting lifetime pollution exposures. Many of today’s children grow up in urban environments with low levels of active travel and a diesel-dominated transport environment. In contrast, the childhood exposures of those now in middle age will have included lead and emissions from petrol vehicles, along with peaks in summertime smog, while those in retirement had childhoods when winter air was dominated by ‘pea-souper’ smogs.

Many studies have demonstrated the public health burden of air pollution. Viewed from a different perspective, better management of air pollution and our exposure to it presents a substantial opportunity for public health improvement, cost savings and increases in quality of life for many. Numerous case studies have shown how successes in reducing air pollution can lead to public health improvements in the short to medium term. If, as now seems likely, childhood and lifecourse exposures to air pollution have lasting influences, then the gains will be even larger, and the benefits from tackling air pollution today will be felt through the decades to come.

1.8 References

- 1 Bell ML, Davis DL. Reassessment of the lethal London fog of 1952: novel indicators of acute and chronic consequences of acute exposure to air pollution. *Environ Health Perspect* 2001;109 (Suppl 3):389–94.
- 2 Committee on the Medical Effects of Air Pollutants. *The mortality effects of long-term exposure to particulate air pollution in the United Kingdom*. London: Health Protection Agency, 2010. www.gov.uk/government/publications/comeap-mortality-effects-of-long-term-exposure-to-particulate-air-pollution-in-the-uk [Accessed 30 November 2015].

- 3 Committee on the Medical Effects of Air Pollutants. *Nitrogen dioxide: interim view on long-term average concentrations and mortality*. London: Health Protection Agency, 2015. www.gov.uk/government/publications/nitrogen-dioxide-interim-view-on-long-term-average-concentrations-and-mortality [Accessed 17 December 2015].
- 4 Dockery DW, Pope CA 3rd, Xu X *et al*. An association between air pollution and mortality in six U.S. cities. *N Engl J Med* 1993;329:1753–9.
- 5 Laden F, Schwartz J, Speizer FE, Dockery DW. Reduction in fine particulate air pollution and mortality: extended follow-up of the Harvard Six Cities study. *Am J Respir Crit Care Med* 2006;173:667–72.
- 6 Royal Commission on Environmental Pollution. Twenty-sixth report. *The urban environment*. London: HMSO, 2007. www.rcep.org.uk/reports/26-urban/documents/urban-environment.pdf [Accessed 30 November 2015].
- 7 Department for Transport. *Annual road traffic estimates: Great Britain 2012*. London: DfT, 2013. www.gov.uk/government/uploads/system/uploads/attachment_data/file/255742/road-traffic-statistics-2012.pdf [Accessed 30 November 2015].
- 8 Department for Transport. *Road traffic forecasts 2015*. London: DfT, 2015. www.gov.uk/government/publications/road-traffic-forecasts-2015 [Accessed 30 November 2015].
- 9 Carslaw DC, Rhys-Tyler G. New insights from comprehensive on-road measurements of NO_x, NO₂ and NH₃ from vehicle emission remote sensing in London, UK. *Atmos Environ* 2013;81:339–47.
- 10 Society of Motor Manufacturers and Traders. *New car CO₂ report 2014*. The 13th report. London: SMMT, 2014. www.smmt.co.uk/2014/03/new-car-co2-report-2014/ [Accessed 30 November 2015].
- 11 Cames M, Helmers E. Critical evaluation of the European diesel car boom – global comparison, environmental effects and various national strategies. *Environ Sci Eur* 2013;25:15.
- 12 Department for Environment Food and Rural Affairs. *Air pollution in the UK 2014*. London: Defra, 2014. <http://uk-air.defra.gov.uk/library/annualreport/index> [Accessed 21 December 2015].
- 13 Noble SR, Horstwood MSA, Davy P *et al*. Evolving Pb isotope signatures of London airborne particulate matter (PM₁₀) – constraints from on-filter and solution-mode MC-ICP-MS. *J Environ Monit* 2008;10:830–6, and references therein.
- 14 Royal Commission on Environmental Pollution. Ninth report. *Lead in the environment*. London: HMSO, 1983. www.rcep.org.uk/reports/09-lead/1983-09lead.pdf [Accessed 1 December 2015].
- 15 European Environment Agency. *Late lessons from early warnings: science, precaution, innovation*. Copenhagen: EEA, 2013. www.eea.europa.eu/publications/late-lessons-2 [Accessed 1 December 2015].
- 16 Jones AM, Harrison RM, Barratt B, Fuller, G. A large reduction in airborne particle number concentrations at the time of the introduction of ‘sulphur free’ diesel and the London Low Emission Zone. *Atmos Environ* 2012;50:129–38.
- 17 Atkinson RW, Fuller GW, Anderson HR, Harrison RM, Armstrong B. Urban ambient particle metrics and health: a time-series analysis. *Epidemiology* 2010;21:501–11.
- 18 Hedley AJ, Wong CM, Thach TQ *et al*. Cardiorespiratory and all-cause mortality after restrictions on sulphur content of fuel in Hong Kong: an intervention study. *Lancet* 2002;360:1646–52.
- 19 Holman C, Harrison R, Querol X. Review of the efficacy of low emission zones to improve urban air quality in European cities. *Atmos Environ* 2015;111:161–9.
- 20 Wolff H. Keep your clunker in the suburb: low emission zones and adoption of green vehicles. *Econ J* 2014;124:F481–F512.
- 21 Malina C, Scheffler F. The impact of low emission zones on particulate matter concentration and public health. *Transp Res Part A Policy Pract* 2015;77:372–85.
- 22 Department for Transport. *National travel survey: England 2013*. London: DfT, 2014. www.gov.uk/government/statistics/national-travel-survey-2013 [Accessed 30 November 2015].
- 23 Rabl A, De Nazelle A. Benefits of shift from car to active transport. *Transp Policy* 2012;19:121–31.
- 24 Viana M, Hammings P, Colette A *et al*. Impact of maritime transport emissions on coastal air quality in Europe. *Atmos Environ* 2014;90:96–105.
- 25 Carslaw DC, Beevers SD, Ropkins K, Bell MC. Detecting and quantifying aircraft and other on-airport contributions to ambient nitrogen oxides in the vicinity of a large international airport. *Atmos Environ* 2006;40:5424–34.
- 26 Dockery DW, Rich DQ, Goodman PG *et al*. Effect of air pollution control on mortality and hospital admissions in Ireland. *Res Rep Health Eff Inst* 2013;176:3–109.
- 27 Fuller GW, Tremper AH, Baker TD, Yttri KE, Butterfield D. Contribution of wood burning to PM₁₀ in London. *Atmos Environ* 2014;87:87–94.

- 28 Ries FJ, Marshall JD, Brauer M. Intake fraction of urban wood smoke. *Environ Sci Technol* 2009;43:4701–6.
- 29 Fuller GW, Sciare J, Lutz M, Moukhtar S, Wagener S. New Directions: Time to tackle urban wood burning? *Atmos Environ* 2013;68:295–6.
- 30 Semple S, Creely KS, Naji A, Miller BG, Ayres JG. Secondhand smoke levels in Scottish pubs: the effect of smoke-free legislation. *Tob Control* 2007;16:127–32.
- 31 Callinan JE, Clarke A, Doherty K, Kelleher C. Legislative smoking bans for reducing secondhand smoke exposure, smoking prevalence and tobacco consumption. *Cochrane Database Syst Rev* 2010;4:CD005992.
- 32 Mackay D, Haw S, Ayres JG, Fischbacher C, Pell JP. Smoke-free legislation and hospitalizations for childhood asthma. *N Engl J Med* 2010;363:1139–45.
- 33 Tan CE, Glantz SA. Association between smoke-free legislation and hospitalizations for cardiac, cerebrovascular, and respiratory diseases: a meta-analysis. *Circulation* 2012;126:2177–83.
- 34 Akhtar PC, Haw SJ, Currie DB, Zachary R, Currie CE. Smoking restrictions in the home and secondhand smoke exposure among primary schoolchildren before and after introduction of the Scottish smoke-free legislation. *Tob Control* 2009;18:409–15.
- 35 Wilkins ET. Air pollution aspects of the London fog of December 1952. *Q J R Meteorol Soc* 1954;80:267–71.
- 36 Clegg A, Wiatr A, Haigh C, McHugh C. *Biomass and CHP emission standards*. Report for Greater London Authority, Air Quality Support. London: Amec, 2013.
- 37 World Bank, 2014. <http://data.worldbank.org/indicator/SP.URB.TOTL.IN.ZS/countries?display=default> [Accessed 28 April 2015].
- 38 Office for National Statistics. 2011 census analysis – comparing rural and urban areas of England and Wales. ONS, 2013. www.ons.gov.uk/ons/rel/census/2011-census-analysis/rural-urban-analysis/comparing-rural-and-urban-areas-of-england-and-wales.html [Accessed 1 December 2015].
- 39 GLA Intelligence. *GLA 2012 round population projections: intelligence update 05-2013*. London: Greater London Authority, 2013. <http://data.london.gov.uk/dataset/2012-round-population-projections/resource/50a548be-8da7-4310-8a2a-34518281f271> [Accessed 1 December 2015].
- 40 Department for Transport. *National travel survey: 2013*. London: DfT, 2014. www.gov.uk/government/statistics/national-travel-survey-2013 [Accessed 17 June 2015].
- 41 Air Particle Expert Group (APEG). Source apportionment of airborne particulate matter in the United Kingdom. London: Department for the Environment, Transport and the Regions, 1999. http://uk-air.defra.gov.uk/library/reports?report_id=32 [Accessed 1 December 2015].
- 42 Cox RA, Eggleton AEJ, Derwent RG, Lovelock JE, Pack DH. Long-range transport of photochemical ozone in north-western Europe. *Nature* 1975;255:118–21.
- 43 Environmental Audit Committee. *Air quality: a follow up report*. London: TSO, 2011. www.publications.parliament.uk/pa/cm201012/cmselect/cmenvaud/1024/1024.pdf [Accessed 7 December 2015].

Chapter 2: Summary

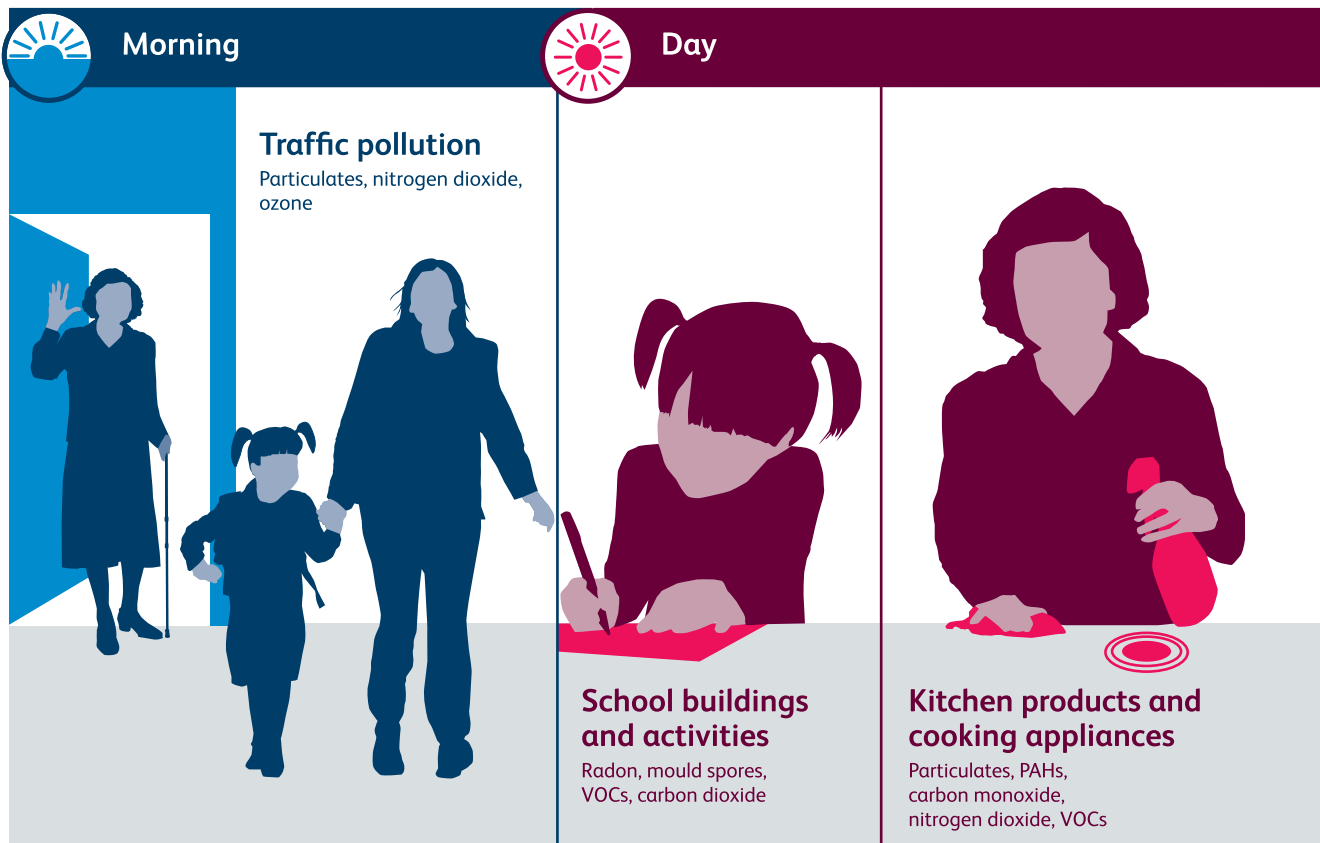
We tend to think of the environment as the wide outdoor world. But it includes indoor spaces too. Each day we move through a series of micro-environments as we make journeys, go to work or school, or stay in our homes. The air we breathe is different in each place.

Outdoors, we are exposed to a range of pollutants, many of which come from vehicles. These include particulates (mostly soot particles from diesel engines) and nitrogen oxides (exhaust gases). We also breathe in ozone, which is produced by chemical reactions in the atmosphere.

The quality of air indoors is important too, because we spend so much time inside. So we need to consider things we use every day, from our gas cookers and cleaning and personal care products, to materials for DIY. Pets and insects can also affect some people, as can damp and mould. A few substances, such as cigarette smoke and carbon monoxide, are very serious hazards.

Key facts

- The most important chemical pollutants in our outdoor air are:
 - **particulates** – small specks of matter such as soot, which can be natural but are primarily from traffic (especially diesel engines)
 - **nitrogen oxides** – gases generated by vehicles, or by chemical reactions in the atmosphere
 - **ozone** – this gas is formed when other pollutants react in the atmosphere.



- Regulations to control outdoor air pollution work in two ways. They may target the source (such as requiring cleaner cars and transport vehicles), or set concentration limits for the pollutants in our air – although it is difficult to say what levels are really safe.
- Increased road traffic, and higher energy use to heat and cool our buildings because of climate change, could make the problem worse.
- According to 2012 figures, indoor air pollution may have caused or contributed to 99,000 deaths in Europe.
- There are few regulatory controls on indoor pollution, apart from building regulations. The drive to reduce energy costs, by creating homes with tighter ventilation, could be making the situation worse.
- Indoors, tobacco smoke is probably the most serious cause of harm.
- Carbon monoxide from faulty boilers and heaters can be fatal.
- Volatile organic compounds (VOCs) are chemicals that start off as solids or liquids, but readily evaporate. They can arise from many common items, including air fresheners and some personal care, DIY and cleaning products. Although they are very common in the air, their health effects are generally minor.
- Formaldehyde vapour can be emitted by certain furniture, furnishings, fabrics, glues and insulation, and can cause irritation of the lungs.
- Asbestos was used as a building material in the 20th century, peaking in the 1960s. It can cause serious damage to the lungs if it is disturbed, which is most likely to happen during maintenance work.
- Particulates and nitrogen oxides from heating and cooking appliances can damage the lungs and/or heart.
- Biological materials that can harm health include house-dust mites, mould and animal dander.



Evening/night



Personal care products
VOCs



Flowers

Pollen, mould spores from soil

Open fire

Carbon monoxide and nitrogen dioxide



Cigarette smoke

Hundreds of hazardous compounds



Cat

Dander

Carpets and sofa cushions

House-dust mites, VOCs

Vapours from chipboard furniture

Formaldehyde

Chapter 2: The air we breathe

Our day-to-day world comprises a range of micro-environments through which we, as individuals, uniquely move, live and breathe through the course of a day – and indeed throughout our lifetime. It includes, of course, the outdoor environment, which is affected by a wide range of factors. Outdoor air, in turn, influences the quality of the air in indoor environments. But indoor environments also have their own sources of contaminants, so that consideration of particular exposures in the home, public places, schools/colleges, hospitals, workplaces and transport is very important in assessing impacts of the breathed environment on our health and wellbeing.

As will be demonstrated in this chapter, there are often ‘unintended consequences’ of actions and policies that impact the breathed environment, including problems relating to the design and/or formulation of products and materials, and the health consequences of exposure to their ingredients or components.

2.1 The air outside

2.1.1 *What are the main pollutants, where do they come from and what effects do they have?*

Outdoor air contains a range of pollutants from a variety of sources, both natural and man-made. As outlined in Chapter 1 of this report, the principal anthropogenic pollution sources are transport vehicles (petrol and diesel engine emissions, along with products from tyre and brake wear), power stations and factories. The atmosphere also contains dust from geological sources and compounds that are the product of chemical reactions between individual substances in the air, as well as a wide variety of gases and particles that originate from natural and biological sources, ranging from volcanic activity to natural ecosystems, agriculture and forestry.

The key pollutants in outdoor air are generally regarded to be particles (measured as PM₁₀ and PM_{2.5}), oxides of nitrogen (principally NO₂) and ozone (O₃), with sulphur dioxide (SO₂), carbon monoxide (CO), hydrocarbons (including benzene, 1,3-butadiene and PAHs) and metals also being significant from a health perspective.

The health effects of all these substances have been intensively studied, and comprehensive assessments of their individual impacts on health are available elsewhere.^{1–3} Chapter 6 shows that their effects are considerable, and that these pollutants are all subject to national and/or international ambient concentration limits. Pollutants with the greatest current impact on public health are considered to be PM_{2.5}, O₃ and NO₂. However, in most cases legislated concentration limits do not represent a ‘safe’ level for the population as a whole, but are often talked about as levels considered to not pose a ‘significant risk’ to health. This begs the question of the meaning of ‘significant’ – given, for example, that impacts of exposure to fine particles have been observed at very low concentrations and that there is no evidence for a threshold for exposure at the population level. The Committee on the Medical Effects of Air Pollutants (COMEAP) estimates 29,000 ‘equivalent’ deaths annually from exposure to PM_{2.5} in the UK,⁴ with only a small fraction of that figure relating to exposures to concentrations in excess of legal limits. This figure increases to around 40,000 if the recently described effects of NO₂ are taken into account.^{5,6} The reality is that agreed standards often incorporate considerations of practicality, ie by how much is it economically reasonable to reduce emissions? For this reason, World Health Organization (WHO) guidelines – based solely on health concerns – are considerably lower in some important cases than national or EU limit values, and even these are not totally protective.⁷

It is known that road traffic emissions – especially of NO₂ and PM – are very significant in terms of total pollutant loading of the outdoor air, and that diesel vehicles emit significantly higher levels of these substances than their petrol-driven equivalents. Thus, diesel vehicles are a valid target for further pollution-reduction measures.

*2.1.2 The control and regulation of outdoor air pollutants**

There are many regulations and initiatives aimed at reducing the concentration of pollutants in the outdoor air. Some of these directly limit point-source emissions (such as those from factories, power stations, vehicles, ships etc), while others aim at managing the total input of pollutants into the local environment. Some options for emission controls will lead to a reduction in several air pollutants simultaneously. For example, improved vehicle efficiency (and more reliable emission monitoring and testing), and the promotion of active transport, should reduce all tailpipe exhaust emissions. Some options, such as requirements for particle traps on diesel vehicles, are more specific.

2.1.3 Possible future impacts of climate change and related factors[†]

Despite technological advances, for example in reducing emissions from individual motor vehicles, the growth in the number of cars, buses and commercial transport vehicles using the roads creates real problems for any authority charged with the task of controlling the levels of key pollutants in outdoor air – especially around busy roads in built-up areas. Such problems are likely to remain with us for many years. In addition, there may be new issues associated with climate change and society's adaptations to these changes (see Chapter 7). For example, in the UK the greatest energy use is by buildings, and if winters were to become colder and/or summers become hotter, as has been forecast with climate change, there may be marked increases in energy demand for heating and air conditioning, which will require increased energy output from power plants. In turn, this may lead to increased combustion of fossil fuels and concomitant rises in pollution emissions from these sources. The growing popularity of wood burning in fires and stoves is a further concern, as this liberates significant amounts of particulate pollution into the outdoor air – as do forest fires. Similarly, the current proliferation of small commercial plants designed to burn biofuels (for heating industrial estates, for example) is of potential concern and may need to be regulated.

Given that fossil fuel combustion is a major source of both greenhouse gases and local air pollutants, if action is taken to address climate change there could be major improvements in outdoor air quality as a result of decarbonisation of power and transport systems, and improved efficiency of energy use. Indeed, the economic benefits of improved health resulting from reduced exposure to fine particles and other local and regional air pollutants as a consequence of climate policies have been estimated to be sufficient, on their own (ie without reference to climate benefits), to justify a range of climate actions being adopted (see Chapter 7).

2.1.4 A brief overview

Numerous pollution sources impact the outdoor air, causing a build-up of substances that can adversely affect our health and wellbeing. Some of these pollutants are natural and unavoidable, while others are the clear and direct result of industrialisation and urbanisation. While the impacts of these on any

*See also section 2.4.

[†]See also Chapter 7 of this report.

individual may be indiscernible, effects on whole populations can be significant, as is the case for PM, which affects the heart and lungs, and causes measurable increases in population morbidity and mortality (see Chapter 4).

Principal pressures on outdoor air quality come from vehicles and the burning of fossil fuels for energy. Further expansion of road traffic and possible increased energy consumption in buildings because of climate change further add to the burden of pollutants in outdoor air.

2.2 Indoor air

2.2.1 Indoor sources of pollution

The quality of the air indoors is important, because it is here that we spend the majority of our time – whether that is at home, at work,* at school, in shops or in vehicles. One important source of indoor air pollution is outdoor air, gaining ingress through windows, doors and general building ‘leakiness’. So, ‘clean’ outdoor air will help to ensure high-quality air indoors. But there are many important and sometimes potent sources of pollution that are located inside buildings and other internal spaces. These include both natural and anthropogenic sources.

2.2.2 Natural pollution sources

Natural pollution sources include a number of biological ones. We ourselves release pollutants including a personal particle cloud through normal metabolic processes, and also through use of personal care products. In addition, we often share our indoor environment – both deliberately and unwittingly – with various other living organisms. There are pets of course, dogs, cats and sometimes birds, rabbits and rodents, but also various insects and arachnids that enjoy home comforts, especially including house-dust mites, bacteria and moulds that thrive in warm, moist environments. In addition, cut flowers and potted plants may release pollen into the indoor air.

Another important potential source of indoor air pollutants is the soil and bedrock upon which a house or other dwelling is built – a major determinant of exposure to radon, for example.

2.2.3 Anthropogenic sources – the influence of what we do, what we use, and how and where we live

There are a large number of potential ‘man-made’ pollution sources in indoor environments, especially the home (see Fig 3). Probably top of the list in terms of health consequences is the smoking of cigarettes, cigars, pipes etc, giving rise to so-called ‘second-hand smoke’ containing many noxious substances. In addition, hookahs/shisha smoking, candles, joss sticks and other materials that we burn for recreational purposes emit pollutants into the indoor air. Combustion appliances – cookers, boilers, open fires and portable gas/paraffin heaters (with no flue) – are particularly significant in terms of total emissions.† The building itself, the materials from which it is built and those with which it is decorated are also important potential sources of chemical pollutants – these include the construction materials, as well as paints, glues, furniture, wallpaper and drapery. Cleaning and DIY products, air fresheners and other consumer products such as insecticide sprays that we use in the home are also important. Some

*Workplaces are considered special situations and are dealt with separately in section 2.3.

†It is noteworthy that, in the UK, the use of gas for cooking and heating is particularly prevalent.⁸

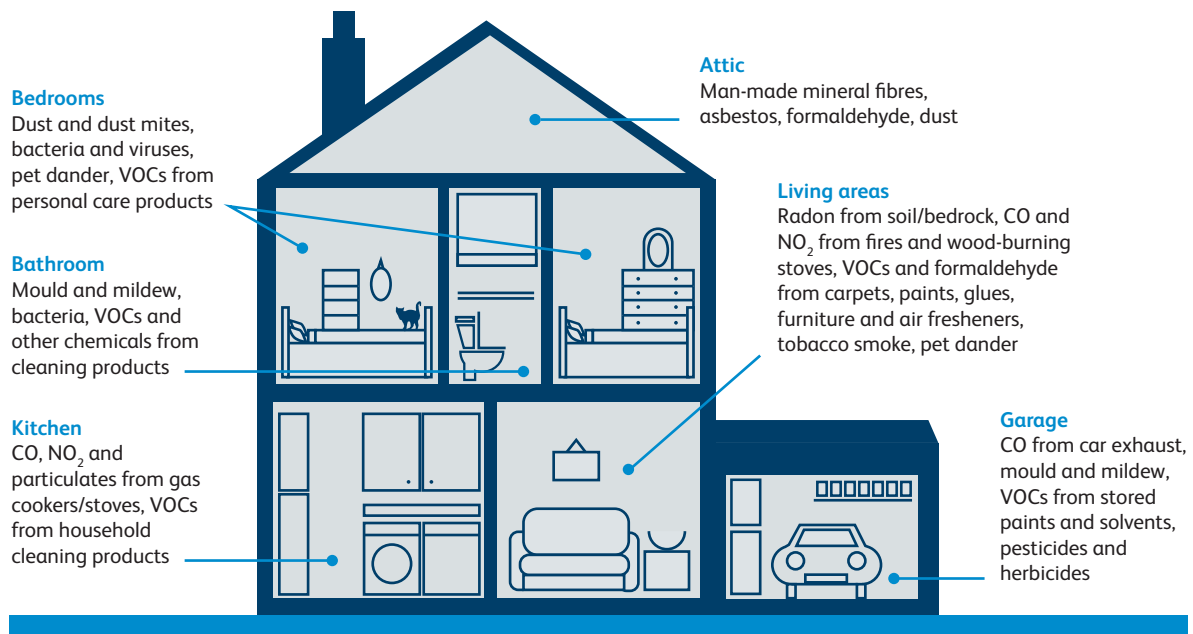


Fig 3. Sources and types of indoor pollution encountered in homes. VOCs = volatile organic compounds. Please note that these lists are not exhaustive and that the actual pollutants present, and their amounts, will vary from household to household.

waterproofing/filling DIY products can contain highly asthmagenic di-isocyanates, for example. It is now known that pieces of electrical equipment, including scanners and photocopiers, also emit pollutants – and for houses with built-in garages, the ingress of vehicle exhaust and vapours from petrol, stored paints and solvents, etc can affect the quality of indoor air in the home. Educational establishments (see Box 7), shops and offices may have additional sources of pollution – relating to activities and/or stored items – including materials that emit VOCs.

The principal pollutants emitted from indoor sources are shown in Box 6. Additional compounds may be generated through chemical reactions between certain pollutants in indoor air – for example, between ozone and VOCs – forming complex new organic substances. Also, water vapour production in homes (and condensation due to poor building design/construction etc) is important because it encourages the growth of moulds and house-dust mites, and because damp homes are known to be unhealthy homes.

The possible health consequences of exposure to the substances listed in Box 6 have been well evaluated elsewhere;^{9,10} potential health impacts include asthma, chronic obstructive pulmonary disease (COPD), respiratory irritation, effects on the heart, and cancer,* as well as non-specific symptoms such as headache, tiredness and loss of concentration (sometimes referred to as ‘sick building syndrome’). According to the European INDEX project,¹¹ the most significant of these pollutants (excluding tobacco smoke – see below) in terms of health impact are formaldehyde, NO₂, CO, benzene and naphthalene. CO is of special concern because it is known that acute exposure can cause collapse and death. It is all the more deadly because there are no warning signs and the early symptoms can resemble those of food poisoning.⁸ Moreover, certain persistent health effects may occur following high-level acute exposure or

*Second-hand tobacco smoke, benzo(a)pyrene, radon, benzene and asbestos are established human carcinogens.

Box 6: Key indoor pollutants

Pollutants emitted from indoor sources include:

- second-hand smoke – from tobacco smoking
- CO (and CO₂) – from combustion appliances, open fires and burned materials/products; faulty or poorly maintained gas heaters and boilers are a particularly important source
- bacteria and viruses – from inhabitants and decaying materials
- biological allergens – from house-dust mites, insects, moulds, and animal dander from pets
- formaldehyde – from composite wood furniture and fittings, fabric, glues, urea–formaldehyde foam insulation
- PAHs, including benzo(a)pyrene – from cooking
- VOCs, including benzene, naphthalene and ‘essential oils’ such as terpenoids – from a wide variety of household, consumer and personal care products
- oxides of nitrogen – from combustion appliances
- ultra-fine particles – from combustion appliances and cooking
- pollen – from plants/flowers
- ozone – from electrical appliances
- phthalates – from plastic materials
- polychlorinated biphenyls (PCBs) and other persistent organic compounds – from old paints, mastics and sealants, plastics and flame retardants
- insecticides – from timber, timber treatment, pesticide sprays
- radon (a radioactive gas) – from soil/bedrock and building stone
- methane – from contaminated ground soil
- lead – in dust from old paintwork
- mineral dusts and fibres (including asbestos) – from building and insulation material.

prolonged low-level exposure to CO. Formaldehyde is important because it is a respiratory irritant and a sensitiser, and is essentially ubiquitous. CO₂ is rarely regarded as a threat to health in indoor environments, but in buildings such as schools it can rise to levels high enough to cause drowsiness,

Box 7: Indoor air quality in schools

The recently completed EU SINPHONIE project¹² has revealed significant problems within school buildings, including:

- concentrations of PM_{2.5} and radon above recommended limits in some schools
- levels of CO₂ above 1,000 ppm in some schools
- exposure of some schoolchildren to levels of benzene above 5 µg/m³
- exposure of some schoolchildren to levels of formaldehyde above 10 µg/m³
- exposure of some children and teachers to high levels of moulds and bacterial endotoxins.

Health outcomes linked with environmental exposure include asthma, allergies and various other nasal/respiratory symptoms.

hence affecting concentration and productivity. Exposure of children generally – and indeed of the developing fetus via maternal exposure – to indoor air pollutants is of particular concern (see Chapter 3).

The presence of asbestos in the fabric of schools¹³ is discussed in section 2.3. The question of whether children are at particular risk from asbestos exposure, as might occur while they are at school, was recently addressed by the Committee on Carcinogenicity.¹⁴ The committee concluded that, from the limited data, it is not possible to say whether children are intrinsically more susceptible than adults to asbestos-related injury (lung fibrosis, cancer and mesothelioma).^{*} However, owing to the increased life expectancy of children compared with adults, there is an increased lifetime risk of mesothelioma as a result of the long latency period of the disease. For a given dose of asbestos, the lifetime risk of developing mesothelioma is predicted to be about three times greater if first exposure occurs as a child (aged 5) rather than as an adult (aged 25).

The health effects of second-hand smoke are now well understood and are considerable (Box 8), and legislation has been put in place to control exposure in public places (see Chapter 1). However, controlling or reducing exposure to second-hand smoke in the domestic environment is more difficult and requires substantial educational campaigning – especially with regard to the exposure of children, and pregnant women and hence the unborn child. The legislation on smoking in vehicles containing children is a further step forward. There are also current concerns about possible adverse consequences of exposure to e-cigarettes, but the body of evidence is not sufficient to allow any firm conclusions or recommendations to be made at this time.

Box 8: Health effects of second-hand smoke¹⁵

Second-hand smoke contains at least 7,000 chemicals. Consequences of exposure include:

- cardiovascular disease – including coronary heart disease and stroke
- lung cancer – non-smokers exposed to second-hand smoke have an estimated 20–30% increased risk of developing lung cancer
- sudden infant death syndrome (SIDS) – risk is increased in infants exposed before and/or after birth
- health problems in children – including wheezing, bronchitis, ear infections and asthma attacks

Because polluted outdoor air can enter buildings and degrade the quality of the indoor air that we breathe, there is much to be gained by building schools, hospitals etc away from heavily polluted roads. A location's postcode can be used as a reasonable surrogate for exposure to certain outdoor pollutants. But for other substances the concentration indoors is as much as ten times higher than that outdoors because of the presence of internal sources. This emphasises the importance of indoor air quality – not only do we spend considerably more time indoors than out, but the range and concentration of pollutants inside buildings are often much greater than those found outdoors.

Useful indicators of indoor air quality are measured levels of CO₂ or of total VOCs (TVOCs). These are not indicators of potential health effects but rather of problems with ventilation that could lead to health effects, and hence are useful for remediation purposes.

^{*}Mesothelioma is a malignant cancer of the tissue that lines the space between the chest and the lung.

2.2.4 *The question of mixtures*

We are constantly exposed to numerous substances from multiple sources, and this is especially true of air pollutants. Most risk assessment approaches and procedures evaluate risks on a substance-by-substance basis and do not consider combined adverse health effects due to exposure to multiple chemicals.¹⁶ It has been asserted that the determination of risk on a single-chemical basis could well underestimate the combined risks of mixtures.¹⁷ The indoor environment is one situation where the issue of simultaneous exposure to multiple substances is of high relevance. The recent publication by De Brouwere *et al*¹⁶ proposes a practical screening method for the evaluation of mixtures in residential indoor air.

Sometimes synergistic (more than additive) interactions between pollutants can occur. While these are mostly unknown, or at least uncharacterised, there is some evidence that such interactions may occur between radon and tobacco smoking in the causation of lung cancer. This is one reason why radon, which is important in itself, is increasingly recognised as a very significant indoor air pollutant. Also, pollutant gases found indoors, such as NO₂ and formaldehyde, can markedly increase the effects of exposure to allergens such as house-dust mites by acting as adjuvants in enhancing allergic sensitisation.

2.2.5 *Out and about – pollutants in transport and public places*

Exposure to air pollutants inside road vehicles is dominated by traffic pollutants drawn into the vehicle. Indeed, air pollution levels inside vehicles are frequently higher than those outside as a result of fans and air conditioning units venting exhaust fumes from tailpipes directly into the vehicle. Other potential pollutants include petrol vapour (including benzene) released during refuelling, tobacco smoke, and VOCs from the construction/furnishing materials (plastics and fabrics) used in the vehicle – as well as from air fresheners deliberately placed inside the vehicle. The impact of in-vehicle pollution on children's health has recently been recognised by legislation banning smoking in cars if children are present. Aircraft have an additional, if minor, problem of fumes from aviation fuel and products of lubricating oil combustion that may infiltrate the cabin at certain stages of flight.

Until recently, pollution in public places was dominated by second-hand smoke, but this problem has been all but eradicated by the introduction of smoking legislation (see Chapter 1). Other causes of pollution in public places are largely dependent upon the type of space, and will include some or all of the domestic indoor air pollutants listed above. Many public places are also workplaces, as recognised in section 2.3 below.

2.2.6 *How can indoor air pollution be controlled – and what happens if it isn't?*

The practicalities of setting guidelines and establishing control policies for indoor air pollutants have previously been explored.¹⁸ There are a number of difficult issues, including the complexity of pollution sources and the multitude of parties potentially responsible for causing, monitoring and/or regulating indoor air pollution. As already noted, the quality of indoor air in any particular building or location is dependent on a number of factors that are themselves governed by a range of different influences, including the quality of the outdoor air, the design and condition of the building, ventilation exchange rates, the furnishings present, and the occupiers' lifestyle, habits and behaviours, including their management of the building and use of products.

Although acknowledgement of the importance of indoor air quality has often led to calls for legislation, specific legislation for this purpose is often not available, has gaps, or is perceived as too 'intrusive' on

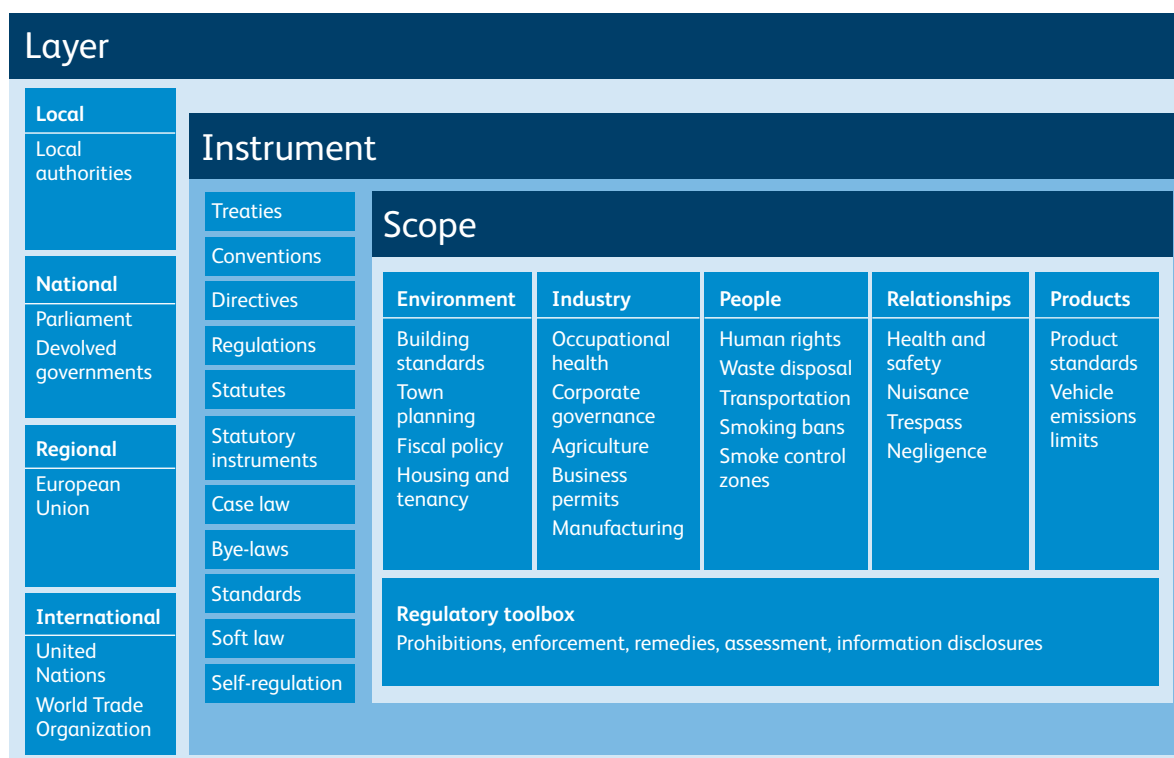


Fig 4. The regulatory framework for the control of air pollution. Courtesy Nikhil Gokani, School of Law and Social Justice, University of Liverpool.

individuals' lifestyles; moreover, very often no single authority or profession has overall responsibility for indoor air quality (see Fig 4). Positive examples of legislative instruments that apply to indoor air quality include the provisions of the building regulations, which do exert some control over ventilation requirements, radon ingress etc, and the highly successful policy to ban smoking in public places, which undoubtedly has led to reduced smoking indoors – although, apart from smoking in vehicles carrying children, legislation does not cover smoking inside private spaces.

Despite this complexity, in 2010 the WHO published quantitative guidelines¹⁹ for the protection of public health from risks due to indoor benzene, CO, formaldehyde, naphthalene, NO₂, PAHs (especially benzo(a)pyrene), radon, trichloroethylene and tetrachloroethylene. These guidelines are targeted at public health professionals involved in preventing health risks of environmental exposures, as well as at specialists and authorities involved in the design and use of buildings, indoor materials and products. In 2014, the WHO report was followed by indoor air quality guidelines for household fuel combustion,²⁰ targeting predominantly low- and middle-income countries where poorly vented and inefficient stoves using highly polluting biomass fuels are common, and responsible for a significant health burden. A recent commission report on household air pollution²¹ established that nearly 3 billion people worldwide are exposed to the threat of household air pollution every day from the use of solid fuel for cooking, heating and lighting. The authors concluded that household air pollution is a major contributor to global figures for morbidity and mortality, with major effects on respiratory symptoms and disease, including non-smoking COPD.

A WHO global burden of disease analysis²² identified household air pollution as an extremely important risk factor accounting for an estimated 4.3 million deaths worldwide in 2012, mostly in low- and

middle-income countries and including some 99,000 in Europe. Around 60% of these deaths are due to stroke (34%) and ischaemic heart disease (26%), with the remainder accounted for by COPD (22%), acute lower respiratory disease (12%) and lung cancer (6%). According to WHO, the guidelines that it has produced provide a scientific basis for legally enforceable standards. They may also be useful as the basis for emissions standards/limits to achieve appropriate source control indoors, which is the specific aim of various existing emission limit-setting and product-labelling initiatives in Europe.²³ Other building- and housing-related policy options to improve indoor air quality and associated quantified impacts on occupant health are presented in the EnVIE report.²⁴

*2.2.7 Possible future impacts of climate change and associated factors**

There are many direct and indirect consequences of climate change, and society's adaptations to it, that may significantly affect the indoor environment and, in turn, its influence on occupant health and wellbeing. At present, initiatives to conserve energy in homes (in an effort to reduce carbon emissions) have generally led to reductions in ventilation (air change rates) and hence the potential build-up of pollutants from indoor sources. Unless properly addressed in a coordinated fashion through planning, design and construction policies, the pressure to save energy will continue to degrade the quality of the indoor environment. The possibility of colder winters will add to this pressure while, if summers become hotter, the use of air conditioning in domestic buildings may supplant window opening, which will similarly lead to a more polluted, and less healthy, indoor environment.

2.2.8 Quick summary

There are many different sources of indoor air pollution, with numerous substances emitted. Pressures to conserve energy have often led to reduced ventilation and hence an increased propensity for the build-up of hazardous substances inside buildings. Drivers associated with climate change may add increasing burdens to maintaining clean, healthy indoor air. Maintenance of good ventilation is key, together with appropriate source control, which might include emission standards/limits linked to quantitative indoor air guideline values. Although the ban on smoking in public places has been highly effective, there remains a need to reduce exposure to second-hand smoke inside the home.

2.3 Workplace air

2.3.1 Some special concerns

Some of the general indoor issues discussed above also apply to workplaces, but there are in addition a number of special concerns.

Workplaces constitute a unique form of environment where exposures to harmful inhaled agents may occur; they may be predominantly indoor (typical factory settings, offices, salons and commercial environments), outdoor (including agricultural- and environmental-based jobs), underground (including mining and hyperbaric tunnelling) or in hostile environments (including offshore, altitude and deep-sea work). Health risks arising from air pollutants in the workplace can be appreciably higher than those of the same pollutants occurring in the domestic situation, as exposure concentrations can be substantially higher. While the focus of this section is on air quality in interior spaces, it is clear that

*See also Chapter 7 of this report.

outdoor air quality issues still apply to many workers, particularly those who work near sources of outdoor air pollution including, for example, urban-based traffic police and street cleaners. Also, some workplaces can be local ‘hotspots’ of air pollution, giving rise to increased exposure and possible elevated risk of respiratory conditions among neighbourhood inhabitants.

Workers in these diverse types of workplace may find it difficult to reduce or materially influence their exposure to harmful inhaled agents. Because of the reliance on the employer to control the nature of the air breathed by workers, workplace air quality is normally regulated, although the nature and type of the regulation will vary from country to country. In the UK, the Health and Safety at Work Act and subsequent Control of Substances Hazardous to Health (COSHH) regulations contain much of the legislation used to control harmful inhaled exposures at work. These underpin a risk-based approach, where those responsible for the quality of air in workplaces assess likely exposure, estimate the likely risk, and develop a set of interventions in the so-called ‘hierarchy of control’ to reduce the risks to health.

The lung is vulnerable to exposure to a broad range of harmful substances in the workplace, including allergens, asthmagens, organic dusts, mineral dusts and fibres, solvents and VOCs, gases and chemical carcinogens. Newer types of exposure include engineered hypoxic environments to control ignition risks. This serves to illustrate how complex workplace air quality considerations can be, and shows that these considerations need to evolve with changes in workplace design.

Four examples – asthmagens, organic dusts, mineral dusts and fibres, and hypoxic environments – are detailed in Box 9 to illustrate some current and future risks among the UK workforce.

In addition to the more traditional work-related respiratory diseases, perhaps more difficult to quantify are illnesses that arise from a particular workplace environment where a single, particular responsible exposure has not yet been identified. These include building-related illness (otherwise known as ‘sick building syndrome’) – a constellation of non-specific upper airway, eye and nasal symptoms often associated with newer building occupancy and also with perceived lack of control by the occupants over their environment. The roles of various exposures, including VOCs, O₃ and PM, have been assessed, and work continues to identify the best preventative strategies. Other issues relevant to workplaces/offices include the use of large numbers of printers and photocopiers that may emit O₃. More generally, poor air quality is known to have measurable impacts on worker productivity.

It is also important to recognise that certain workplaces are public areas, and here air quality will influence, and be influenced by, both public and worker occupancy. While exposure to environmental tobacco smoke may be less of a risk nowadays given recent UK-based legislation, the use of cleaning and personal care products, sprays and vaporisers, and also emissions from large quantities of stored items and products (eg inside shops and warehouses), may pose particular air quality issues.

The structure/fabric of workplaces can also give rise to low-level inhalation exposures known to cause respiratory ill health, including exposure to asbestos fibres in older buildings, and to formaldehyde and VOCs in newer builds. Builders, carpenters and electricians, for example, have been at particular risk from exposure in their day-to-day work to asbestos in old domestic and commercial buildings (see Box 9). Exposure of teachers to asbestos, used in the construction of many schools during the 1940s to 1980s,¹³ is also a matter of ongoing concern. Work continues in an effort to understand the risks posed by these vicarious exposures in both public and privately owned buildings.

Box 9: Examples of workplace risks

Asthmagens

Bakers, flour confectioners, metal machiners, welders, assemblers, vehicle paint sprayers, chemical process workers, cleaners and nurses are particularly at risk of asthma. The responsible agents include di-isocyanates (typically in 'twin-pack' paints), flours, cutting oils, animal proteins, wood dusts, metals, welding fumes and cleaning agents. A particular ongoing concern relates to 'novel' chemicals designed as high-performance adhesives, resin or polymer precursors, sealants or coatings.

Organic dusts

Exposures are highly varied and include exposure to bioaerosols, including endotoxins, bacteria, viruses and fungi. These have a variety of potential health consequences, including infections and inflammatory lung diseases such as hypersensitivity pneumonitis. Healthcare workers in particular are exposed to bioaerosols, and the consequences of such exposures can be serious for certain infectious agents. Also, exposure to wood dust is an acknowledged risk for potentially serious respiratory diseases, including nasal cancer.

Mineral dusts and fibres

Large numbers of workers are exposed, in a variety of workplace sectors, to respirable crystalline silica. While the dangers of these exposures have long been recognised, silica exposure still causes new cases of lung disease both in the UK and further afield. Exposure to asbestos fibres (notably the amphibole forms crocidolite and amosite) constitutes a serious and long-recognised health issue. The legacy of past extensive use of asbestos in buildings as heat and sound insulation and as fire protection is still causing exposure issues for tradesmen such as demolition workers, plumbers, electricians and carpenters, who may encounter asbestos-containing materials on a day-to-day basis.²⁵ Elevated exposure can cause asbestosis (pulmonary fibrosis), lung cancer and mesothelioma. Because of these risks, there is a duty on building owners to identify and record the presence of asbestos materials and to take appropriate action to isolate or, if necessary, remove them.

Engineered hypoxic environments

These constitute a relatively new workplace risk. Workers may be required to work in spaces where the partial pressure of oxygen has been reduced, or engineered, in order to reduce ignition risk. This is important for certain electrical considerations, or where spaces contain materials that may be irretrievably lost by fire damage. These workplaces represent interesting health and workplace challenges, particularly for those workers with established lung diseases.

Looking to the future, newer 'green' workplaces will be constructed, and newer technologies will be developed for use within them. The latter include significant developments in, for example, the use of advanced materials and three-dimensional printing. The construction, occupancy and exposure profiles of newer workplaces will lead to the potential for novel inhaled hazards and risks, and vigilance will be required in order to identify the occupational lung problems attributed to the workplaces of tomorrow.

2.3.2 Main points

The workplace can be the location of significant exposures to airborne substances that can pose real and measurable risks to health. That is why stringent workplace regulations are in place – to protect workers

who might otherwise suffer as a consequence of their occupation. However, a number of concerns remain, particularly for some workers who are engaged in occupations or in environments that are less adequately or thoroughly controlled. It is also important to consider that some workplaces are public places, and vice versa.

2.4 A word on the regulatory control of air pollution

Fig 4 illustrates that regulatory strategies in the control of air pollution can be made at local, national, regional and international levels. Although there is not necessarily a hierarchy between these levels, with the notable exception of EU law having supremacy over UK law, the constraints imposed by law on policymakers must be considered in order to create rules that are both effective and binding.

Control policies can be implemented using a variety of instruments across different areas relevant to the control of air pollution in particular situations and environments. These instruments range from binding legislation to voluntary self-regulatory schemes. In developing air pollution control laws, it is necessary for policymakers to make evidence-based decisions in order to determine who or what is sought to be regulated, together with the desired outcomes, and thus choose the most effective regulatory tools.

2.5 Conclusions

Our exposure to air pollutants depends on age and occupation, and varies throughout the day according to the different micro-environments that we encounter. Fig 5 clearly shows how a person's exposure to 'black carbon' (representing airborne carbon particles) varies according to the individual and their activities; who they are, what they do, and where they are during the day. Some case studies about our everyday exposure to air pollutants are presented in Box 10, illustrating the diverse range of pollutants to which we are all exposed. In addition, in Box 11 we offer some tips on how to improve the air that we breathe indoors, based upon two UK government leaflets.^{26,27}

It is clear that pollutants with potentially significant impacts on health are encountered outdoors, in the home, in transport and public places, at work and at school – and even *in utero*. Pressures to conserve energy to reduce the carbon footprint of buildings and hence reduce climate change (see Chapter 7) can have potentially detrimental effects on ventilation provision within buildings, leading to potential increases in exposure to pollutants with indoor sources. This complexity mandates a holistic approach to the control of exposure to substances in the breathed environment and the need to consider the full range of available regulatory tools.

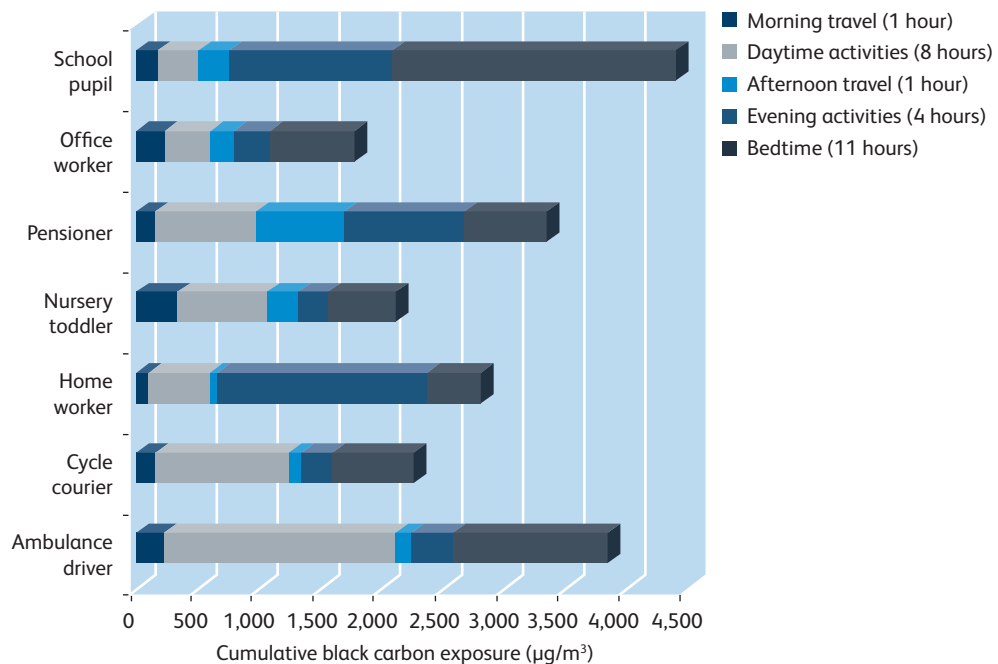


Fig 5. How a person’s daily exposure to ‘black carbon’ (representing airborne carbon particles) varies according to the individual – what they do and where they are over 24 hours. Numerous factors will influence actual measured levels for any one individual, including, for example, where they live and work (eg near a busy road), how and for how long they travel, and whether they are exposed to indoor sources (eg open fires). Courtesy Benjamin Barratt, MRC PHE Centre for Environment and Health.

Box 10: Exposure to air pollution – a ‘day in the life’ of a schoolchild and an office worker

Schoolchild

- Breakfast in kitchen with gas cooker – exposed to NO₂, CO₂ and other gas combustion products.
- Travels to school in parent’s car – exposed to vehicle exhaust and VOCs from car air freshener.
- Studies in crowded classroom – elevated exposure to CO₂, plus some exposure to dusts and fibres.
- Outdoor playtime – exposed to range of outdoor air pollutants.
- Art lesson – exposed to VOCs from paints, resins and adhesives.
- Returns home, watches television in the lounge – exposed to parent’s second-hand tobacco smoke.
- Supper in dining room – exposed to lead in dust from old paintwork through recent redecoration.
- Asleep overnight in downstairs bedroom – exposed to radon ingress from bedrock.

Office worker

- Bathroom ablutions – exposed to VOCs from personal care products.
- Breakfast in kitchen – exposed to PAHs from burned bacon and PM from burned toast!
- Travels to work on the London underground – exposure to PM (metallic particles) in the tunnels.
- Office duties – exposed to O₃ from printers and photocopiers.
- Returns home and rests in lounge with signs of damp – exposed to mould spores.
- Supper in dining room – exposed to formaldehyde from ‘flat-pack’ furniture and PM from candles.
- Decorates spare room – exposed to VOCs from gloss paint and asbestos through drilling the ceiling.
- Bedtime – exposed overnight to CO from faulty gas boiler and to house-dust mite allergens in pillow.

Box 11: How to reduce exposure to air pollutants in the home

Although some indoor pollutants are unavoidable, there are things that you can do to reduce exposures and lessen possible adverse health effects:

- do not smoke at home
- avoid using appliances without flues (eg freestanding gas and paraffin heaters)
- properly maintain all boilers, stoves, fires and other appliances that burn fuel of any kind
- ensure adequate fresh air ventilation at all times
- use cooker extractor hoods and kitchen/bathroom extractor fans where supplied
- treat condensation, and kill and remove mould. Avoid condensation by keeping boiling pans covered and not drying your wet washing inside the home. Properly insulate your home and do not block existing permanent ventilation provision (air blocks, trickle vents etc)
- heat the home sufficiently to prevent damp, while maintaining adequate ventilation – try to achieve the necessary balance between energy efficiency and pollution control
- consider reducing the use of products and materials that release high levels of VOCs.

In addition, depending on the pollutant in question, you may need to seek professional help – for example, to prevent entry of radon from the ground or to remove, or seal, asbestos-containing materials.

2.6 References

- 1 Committee on the Medical Effects of Air Pollutants. *Long term exposure to air pollution: effect on mortality*. London: Health Protection Agency, 2009. www.gov.uk/government/uploads/system/uploads/attachment_data/file/304667/COMEAP_long_term_exposure_to_air_pollution.pdf [Accessed 2 December 2015].
- 2 World Health Organization. *Review of evidence on health aspects of air pollution – REVIHAAP Project*. Bonn: World Health Organization, 2013. www.euro.who.int/__data/assets/pdf_file/0004/193108/REVIHAAP-Final-technical-report-final-version.pdf [Accessed 2 December 2015].
- 3 Newby DE, Mannucci PM, Tell GS *et al*. Expert position paper on air pollution and cardiovascular disease. *Eur Heart J* 2015;36:83–93.
- 4 Committee on the Medical Effects of Air Pollutants. *The mortality effects of long-term exposure to particulate air pollution in the United Kingdom*. London: Health Protection Agency, 2010. www.gov.uk/government/publications/comeap-mortality-effects-of-long-term-exposure-to-particulate-air-pollution-in-the-uk [Accessed 30 November 2015].
- 5 Department for Environment Food and Rural Affairs. *Valuing impacts on air quality: Updates in valuing changes in emissions of oxides of nitrogen (NO_x) and concentrations of nitrogen dioxide (NO₂)*. London: Defra, 2015. www.gov.uk/government/uploads/system/uploads/attachment_data/file/460401/air-quality-econanalysis-nitrogen-interim-guidance.pdf [Accessed 15 December 2015].
- 6 Committee on the Medical Effects of Air Pollutants. *Nitrogen dioxide: interim view on long-term average concentrations and mortality*. London: Health Protection Agency, 2015. www.gov.uk/government/publications/nitrogen-dioxide-interim-view-on-long-term-average-concentrations-and-mortality [Accessed 17 December 2015].
- 7 Brunekreef B, Annesi-Maesano I, Ayres JG *et al*. Ten principles for clean air. *Eur Respir J* 2012;39:525–8.
- 8 Tam CW, Bevan RJ, Harrison PTC, Youngs LC, Crump D. Public health impacts of exposure to carbon monoxide from gas appliances in UK homes – are we missing something? *Indoor Built Environ* 2012;21:229–40.
- 9 Institute for Environment and Health. *Indoor air quality in the home*. Leicester: IEH, 1995.
- 10 Harrison PTC. Health effects of indoor air pollutants. In: Harrison RM, Hester RE (eds), *Air pollution and health (Issues in environmental science and technology, vol 10)*. Cambridge: Royal Society of Chemistry, 1998:101–25.

- 11 European Commission. *INDEX: Critical appraisal of the setting and implementation of indoor exposure limits in the EU*. Ispra, Italy: EC, 2004. http://ec.europa.eu/health/ph_projects/2002/pollution/fp_pollution_2002_exs_02.pdf [Accessed 4 December 2015].
- 12 European Commission. *SINPHONIE: schools indoor pollution and health observatory network in Europe, final report*. EC, 2014. http://ec.europa.eu/health/healthy_environments/docs/2015_sinphonie_frep_en.pdf [Accessed 4 December 2015].
- 13 Institute for Environment and Health. *Fibrous materials in the environment*. Leicester: IEH, 1997.
- 14 Committee on Carcinogenicity of Chemicals in Food, Consumer Products and the Environment. *Statement on the relative vulnerability of children to asbestos compared to adults*. London: Public Health England, 2013. www.gov.uk/government/publications/relative-vulnerability-of-children-to-asbestos-compared-to-adults/ [Accessed 4 December 2015].
- 15 Centers for Disease Control and Prevention. *Health effects of secondhand smoke*. Atlanta, GA: CDC, 2014. www.cdc.gov/tobacco/data_statistics/fact_sheets/secondhand_smoke/health_effects/ [Accessed 4 December 2015].
- 16 De Brouwere KD, Cornelis C, Arvanitis A *et al*. Application of the maximum cumulative ratio (MCR) as a screening tool for the evaluation of mixtures in residential indoor air. *Sci Total Environ* 2014;479–80:267–76.
- 17 European Commission. *State of the art report on mixture toxicity*. London: EC, 2009. http://ec.europa.eu/environment/chemicals/effects/pdf/report_mixture_toxicity.pdf [Accessed 4 December 2015].
- 18 Harrison PTC. Editorial: Indoor air quality guidelines. *Occup Environ Med* 2002;59:73–4.
- 19 World Health Organization. *WHO guidelines for indoor air quality: selected pollutants*. Copenhagen: WHO, 2010. www.euro.who.int/__data/assets/pdf_file/0009/128169/e94535.pdf [Accessed 4 December 2015].
- 20 World Health Organization. *WHO guidelines for indoor air quality: household fuel combustion*. Geneva: WHO, 2014. http://apps.who.int/iris/bitstream/10665/141496/1/9789241548885_eng.pdf [Accessed 4 December 2015].
- 21 Gordon SB, Bruce NG, Grigg J *et al*. Respiratory risks from household air pollution in low and middle income countries. *Lancet Respir Med* 2014;2:823–60.
- 22 World Health Organization. *Burden of disease from household air pollution for 2012*. Geneva: WHO, 2014. www.who.int/phe/health_topics/outdoorair/databases/FINAL_HAP_AAP_BoD_24March2014.pdf [Accessed 4 December 2015].
- 23 Brown VM, Crump DR, Harrison PTC. Assessing and controlling risks from the emission of organic chemicals from construction products into indoor environments. *Environ Sci Process Impacts* 2013;15:2164–77.
- 24 EnVIE. *Co-ordination action on indoor air quality and health effects*. Porto, Portugal: EnVIE, 2009. <http://paginas.fe.up.pt/envie/documents/finalreports/Final%20Reports%20Publishable/Publishable%20final%20activity%20report.pdf> [Accessed 4 December 2015].
- 25 Harrison PTC, Llewellyn JW. Exposure to asbestos and man-made mineral fibres in buildings and the consequences for health. In: Moschandreas DJ (ed), *Design, construction, and operation of healthy buildings: solutions to global and regional concerns*. Atlanta, GA: ASHRAE, 1998:83–93.
- 26 UK Government. *Keep your home free from damp and mould*. London: Crown copyright, 1992.
- 27 UK Government. *Good air quality in your home*. London: Crown copyright, 1993.

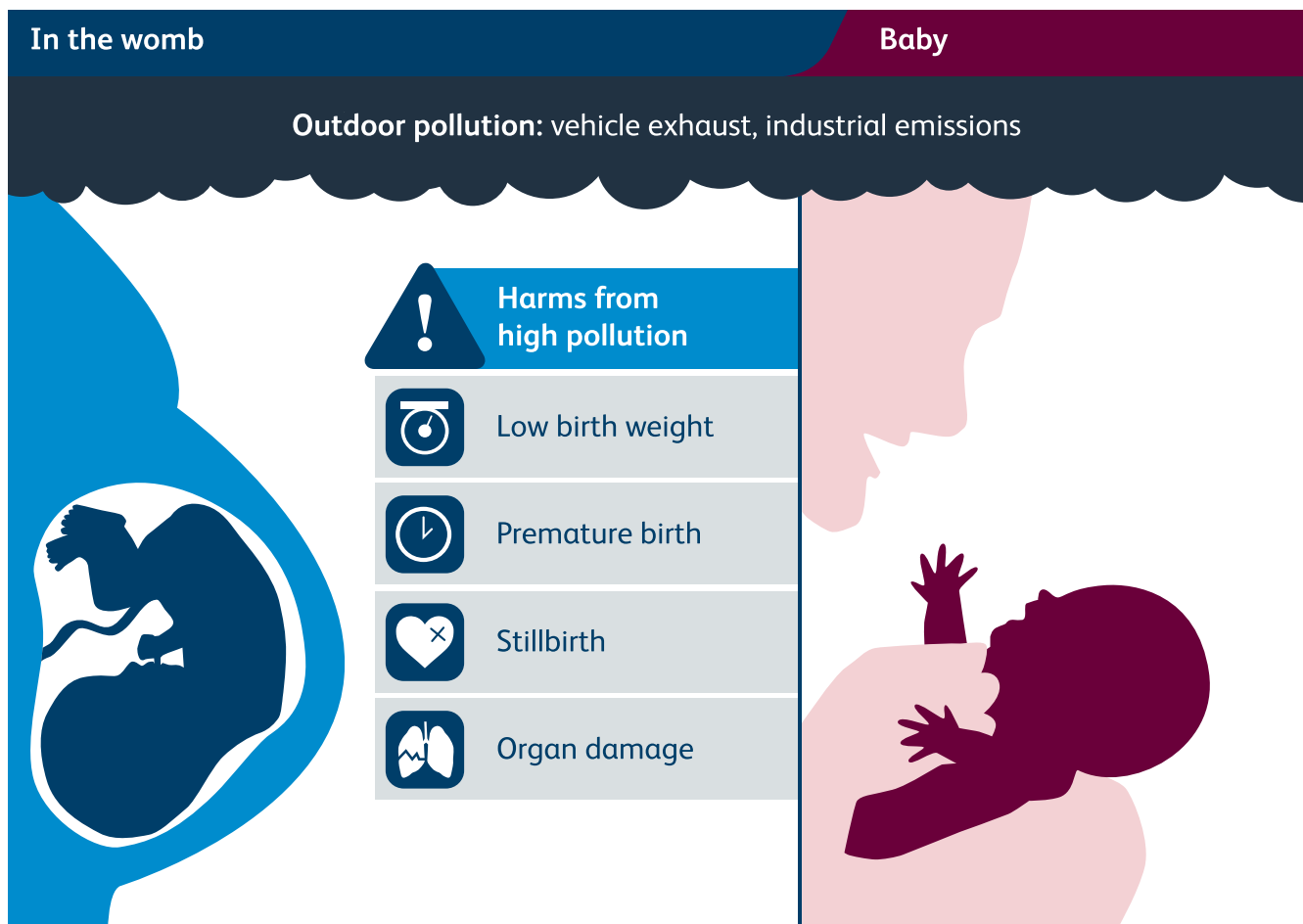
Chapter 3: Summary

Pregnancy, infancy and early childhood are critical times when all the body's systems are formed, and start maturing. This process happens at a lightning pace. It is controlled by genes, which must switch on and off at just the right time, in just the right order.

Therefore, it is clearly a vulnerable phase of life. The developing heart, lung, brain, hormone systems and immunity can all be harmed by pollution. Environmental effects on the embryo, fetus, baby and toddler may last a lifetime, but may take years or even decades to become apparent.

Smoking in pregnancy is probably the most serious source of harm, causing slow fetal growth, prematurity and stillbirth.

There is also clear evidence that early exposure to air pollution can damage the lungs, and increase the risk of lung infections that may be fatal. It is known to have an effect on heart health in adult life. Research is beginning to point towards effects on growth, intelligence, asthma, and development of the brain and coordination.



*Includes exhaust gases from cooking, heating and burning solid fuels, use of household cleaners and other chemicals, VOCs, etc



Key facts

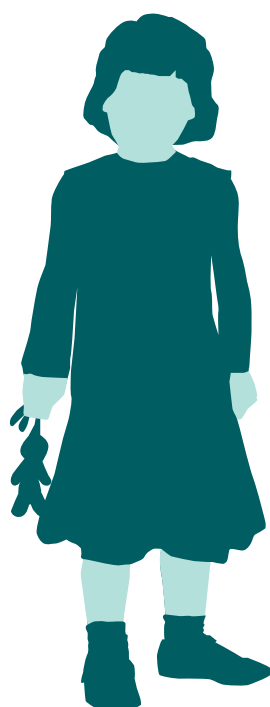
- Before birth, the health of the baby is tied closely to the health of the mother.
 - Smoking in pregnancy is linked to slow fetal growth, premature birth and stillbirth. It can also cause the placenta to break away (abruption), which is very dangerous to both mother and baby. The mother's risk of high blood pressure is increased.
 - Some pollutants, when breathed by the mother, can cross through the placenta to the developing baby. Particulates and heavy metals are two examples.
 - Air pollution can affect growth of the unborn baby and may be linked to premature birth.
- Development in the womb is rapid.
 - By 3–4 weeks of pregnancy, the heart is beating.
 - The major organs are formed by 12 weeks, and the endocrine (hormone) system is functioning.
 - At 16 weeks, the main tube system of the lungs (the bronchial tree) is formed.
 - By 6 months, most of the brain, spinal cord and nerves are in place.
 - Around 7 months, the lungs' air sacs (alveoli) begin to form. Half of them are completed by the end of a full-term pregnancy.
 - These are all critical points where air pollution or exposure to smoking could cause harm.

Toddler




Indoor pollution: tobacco smoke, household fumes*

Harms from high pollution

-  More lung infections
-  Increased infant mortality



Harms from high pollution

-  More coughs and wheezing
-  More A&E visits
-  Decreased lung function

Chapter 3: In the beginning: protecting our future generations

3.1 Early human development

To understand why fetuses, babies and children are more susceptible to external insults, including the effects of air pollution, it is useful to understand, at least in outline, aspects of early human development and growth.

3.1.1 *Development and growth: a short background to early development*

Development implies more than just growth; it includes changes in the nature and structure of tissues. In biological terms, it requires differentiation of cells into specialised types with varying functions, eg heart cells, which contract rhythmically, or cells in the gut, which secrete liquid and absorb nutrients.

Growth is increase in size; in the body, this means an increase in the number of cells in a particular tissue, rather than the cells themselves enlarging.

The amazing transformation of the fertilised human egg – a single cell – into a complex organism in a very short time requires the integrated and precisely controlled coordination of differentiation and growth of cells. These processes are controlled by the switching on and off of genes in a specific sequence in a timely manner. It is believed that over half the genes in the human genome are used only for early development.

The majority of organs are formed by about 10 weeks after fertilisation (12 weeks of pregnancy).

The heart

The heart is one of the first tissues in the body to develop into a functional organ; it starts to beat and to pump blood from around 3–4 weeks' gestation.

The lungs

The lungs will be the first point of contact for air pollution throughout life and, therefore, are particularly vulnerable to its adverse effects. The bronchial tree (air passages in the lungs) is completely formed by 16 weeks of pregnancy (Fig 6). The air sacs (alveoli) start developing at 28 weeks and about half the final adult number of alveoli are present at birth (40 weeks). Most of the remainder develop by about 2 years of age, but it is likely that they continue to develop through adolescence until body growth ceases. The peak of lung function is not achieved until the early to mid-twenties, making the lung vulnerable for many years, but perhaps maintaining an ability to recover some lost function.

The brain and central nervous system

The primitive brain and spinal cord are present from very early pregnancy, but development of the nervous system is an ongoing process that continues throughout fetal life and after birth to adolescence. Most of the nerve cells have developed by the middle of pregnancy and afterwards they go on to develop

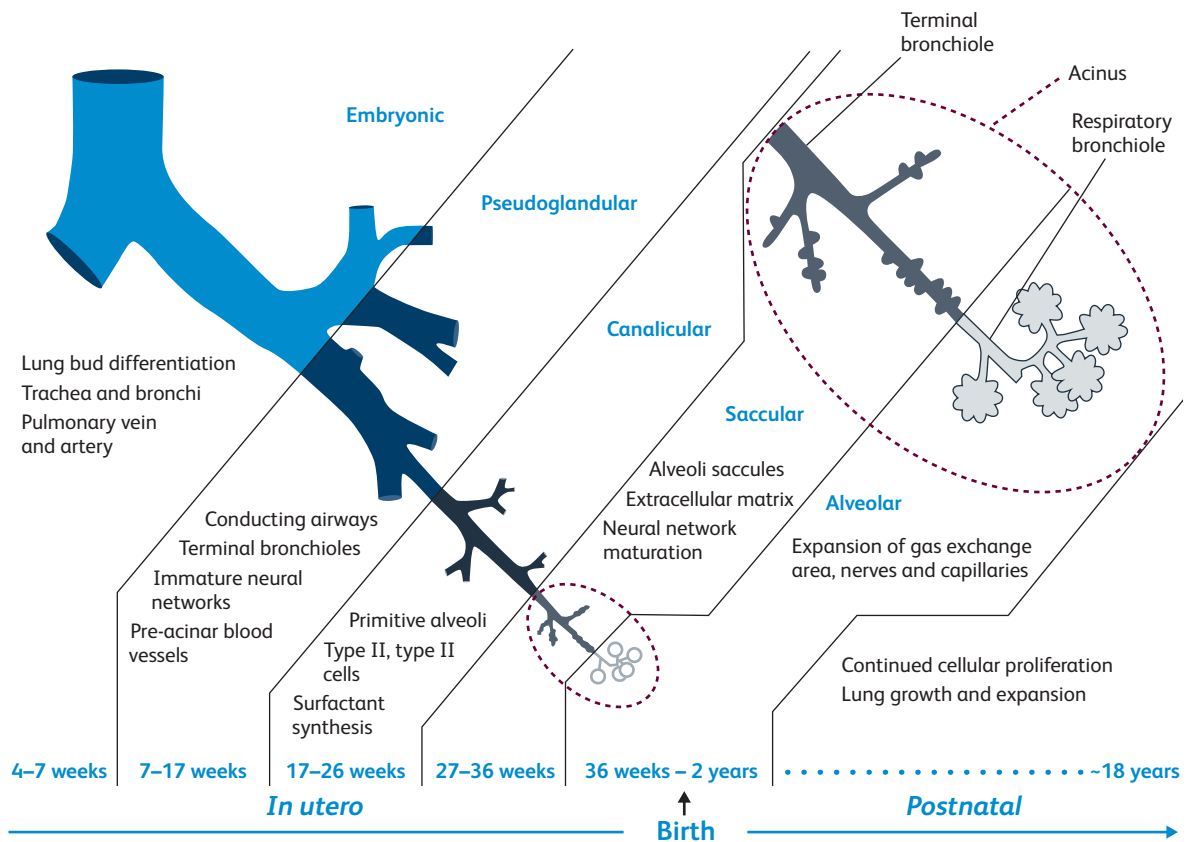


Fig 6. Principal stages of lung development in humans. Adapted with permission from: Kajekar R. Environmental factors and developmental outcomes in the lung. *Pharmacol Ther* 2007;114:129-45.

their specialised functions in the nervous system. Therefore, they remain vulnerable to injury throughout childhood.

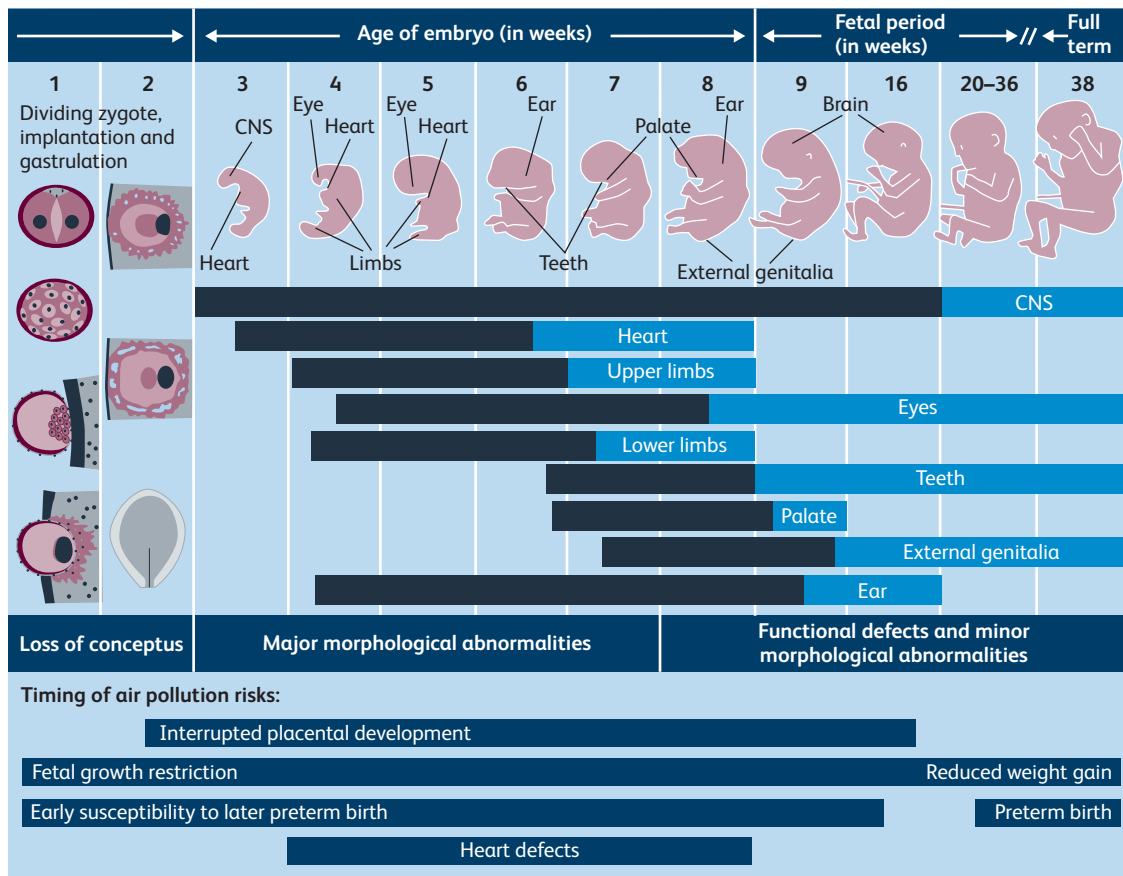
Glands and hormones

The fetus is able to produce hormones from early gestation. These are important for maintenance of pregnancy, and the fetus responds to stress by producing stress hormones. This can result in long-term resetting of hormone systems so that they do not function normally after birth. Because hormones produced by the endocrine system are vital for many functions of the body, including growth and development, such resetting can have adverse consequences for future health.

Immunity, infections and allergies

Babies' immune systems are not fully developed at birth and they are vulnerable to infections. Although the fetus is capable of producing antibodies and can develop allergic responses, most of the antibodies present at birth come from the mother, so the baby benefits from the mother's own immunity to infections. After birth, the immune system matures through exposure to infections and micro-organisms in the gut.

In view of the very intricate and rapid changes in this early period of development, the fetus is very vulnerable to external insults, be they chemical, physical or types of radiation. Small changes or disturbances in the process can have permanent, lifelong consequences (Fig 7). A good example of this can be demonstrated by German measles (rubella) infection of a pregnant mother. The effect on the



Note: Grey bars indicate time periods when major morphological abnormalities can occur, while light-blue bars correspond to periods at risk from minor abnormalities and functional defects.

Fig 7. Critical periods of risk from air pollution during fetal development. CNS = central nervous system. *Figure reprinted with permission.* Ritz B, Wilhelm M. Air pollution impacts on infants and children. UCLA Institute of the Environment: Southern California Environmental Report Card – Fall 2008, Los Angeles, CA: 2008.

developing fetus is crucially dependent on the timing in pregnancy of the infection and on which organs are developing most rapidly at that time. Infection in very early pregnancy affects the heart, but only a few days later produces permanent damage in the brain, eyes and ears. In contrast, infection near the end of pregnancy has few damaging effects. When children and adults contract German measles, it is considered to be a trivial disease, demonstrating that the sensitivity to and consequences of rubella infection are highly dependent on the stage of development.

3.2 Why are infants and young children vulnerable to the effects of air pollution?

Pregnancy and early childhood are critical times for the formation and maturation of all the important body systems; there is no other time in life during which such rapid changes take place. This means that factors that exert an adverse influence on human development, including air pollution, can have a far greater influence during this period than at other times; the rapidity of change magnifies their effects, and important organ systems, once their physical development is harmed, may not have the capacity to recover. This means that organ damage that occurs as a result of harm in early life, including before birth, will be present for the rest of that individual's life. This does not always mean that such changes

will show themselves during the early years of life. There is often a threshold of organ function below which symptoms occur and it may not be until the influences of natural physical decline in adulthood and older age are added to early injury that disease will become obvious.

There are three major periods of vulnerability to the adverse effects of air pollution during this critical period of early childhood development.

Pregnant women are susceptible to the effects of air pollution. The health of the developing fetus is intricately dependent on the health and wellbeing of its mother and on the function of the placenta. Illnesses or exposure of the mother to harmful substances, such as tobacco smoke, can result in slowing of fetal growth. This can affect the growth of different organs and, if growth is hampered at a critical time of development, can lead to permanent damage. This is an example of *indirect* harm due to air pollution. However, *direct* harm to the fetus may also occur owing to transfer of toxic substances from the mother's blood across the placenta. Although the placenta has a barrier function to filter out substances harmful to the fetus, several pollutants, such as heavy metals and fine particles (PM_{2.5} and smaller), are able to cross the placental barrier¹ and have the potential to cause injury to the fetus and its developing organs.

The developing fetus is susceptible to the effects of air pollution. From the earliest stages of development, the building block of life, DNA, is susceptible to changes arising from exposure to air pollution. This can be in the form of physical changes to the DNA structure or influences on how genes function. Even modifications of mothers' DNA before conception could influence fetal development. The fetus is also undergoing a remarkable and coordinated process of organ development, which follows a time-dependent course. As discussed above, even seemingly trivial interference during critical periods can irrevocably harm organs and tissues (the fetal brain and nervous system are exquisitely sensitive to these effects) or change their developmental trajectory so that their function is permanently impaired.

Infants are susceptible to the effects of air pollution. Even after birth, there is considerable development and maturation of organs that makes them vulnerable to harmful effects of pollution. Infants are relatively immobile and dependent on their parents to protect them or move them from sources of pollution, yet their main mode of outdoor transport seems designed to put them at precisely the level of motor vehicle exhaust emissions (Fig 8). Infants have a relatively high metabolic rate, so they breathe a greater volume of air per minute than an adult relative to their size. This is a double jeopardy: they get exposed to relatively higher doses of toxic pollutants, as well as being more vulnerable to their harmful effects.

3.3 How does air pollution affect human development?

3.3.1 What does air pollution do to pregnancy outcomes?

The placenta contains a myriad of blood vessels from both the fetal and the maternal circulations, which grow very rapidly in early pregnancy. There are several possible mechanisms through which air pollution can harm placental and hence fetal development, although it is unclear which of these is the most important. It is known that air pollution harms blood vessels in later life, giving rise to heart attacks and strokes. It would not be unreasonable, then, to expect some effect of air pollution on the placental blood vessels, but whether the time course of 9 months is sufficient for any effects to



Fig 8. Young children are at risk of high levels of air pollution exposure.

become apparent is unknown.² Maternal exposure to air pollution can cause inflammation of the placenta, but the evidence for this comes from animal rather than human studies. The placenta produces hormones that are important for fetal growth and wellbeing, and which help to regulate the timing of labour.

The pollutant known to cause the greatest avoidable harm during pregnancy is tobacco smoke. Maternal smoking during pregnancy is associated with stillbirth, placental separation, pregnancy-associated hypertension, premature delivery, and slowing of fetal growth (intrauterine growth retardation) resulting in low birth weight. The associations of other air pollutants with adverse pregnancy outcomes are not as strong as they are for tobacco smoke, and there is more uncertainty about the specific pollutants causing adverse outcomes; the most consistent evidence is for PM. Most studies in humans are based on modelled estimates rather than direct measurements of exposure. Because levels of different pollutants are highly correlated in the estimation models, separation of their individual effects is more difficult.

Air pollution exposure is associated with premature birth.³ Depending on the maturity of the fetus at birth, effects can range from minor inconvenience to severe disability and early death. Modern neonatology has improved the outcomes for premature babies enormously over the past 50 years, and babies born after 28 weeks' gestation have generally favourable outcomes. More premature infants, between 24 and 28 weeks' gestation, are at higher risk and their outcomes are closely dependent on their gestational age; owing to their rapid development, even a few days makes a difference to their maturity. A small shift in gestation at birth can have a huge effect on their survival and subsequent complications, particularly of the brain and respiratory system. Epidemiological studies suggest a link between air pollution exposure and premature birth, with the strongest evidence for gaseous pollutants (O₃ and SO₂) and weaker evidence for particulates (PM_{2.5} and PM₁₀).

The strongest evidence from epidemiological studies of pregnancy outcomes is that air pollution affects fetal growth and birth weight.⁴ It is estimated that traffic-related air pollution exposure of pregnant women accounts for more than one-fifth of all cases of low birth weight at term. There is consistent evidence that exposure to particulates during pregnancy increases the risk of low birth weight.⁵ The timing of exposure is critical, with the greatest harm during early pregnancy, and there is some evidence that girls and boys have different susceptibility to these adverse effects. There is also evidence that naturally occurring variations in some genes that reduce the activity of enzymes involved in detoxifying air pollutants increase the risk of harm associated with air pollution exposure during pregnancy, thus providing supporting evidence that this is a true biological effect. The evidence is less consistent for gaseous pollutants, but O₃, NO₂ and possibly CO have been linked with low birth weight (REVIHAAP Project: Technical Report; question B1),⁶ as has the combustion of indoor solid fuels.⁷ It is important to note that high exposure to specific air pollutants is socially patterned and linked with other lifestyle factors, including smoking and diet. The effects described appear to be independent of these confounding influences, but there is some suggestion that socio-economic deprivation increases the adverse effects of air pollution on low birth weight.

Although the average size of the effects on birth weight is modest (such effects are generally of the order of 100–200 g), the shift of the exposed population to the lighter end of the spectrum of birth weights means that babies in the lower tail of the population distribution are at increased risk of serious problems at birth and thereafter. Similarly, a shift in the average gestational age at delivery by only a small amount can have a substantial influence on the numbers of babies born very preterm (<28 weeks' gestation), with consequent implications for their health outcomes and also the need for sufficient neonatal intensive care facilities.

3.3.2 What does air pollution do to young children?

Air pollution exposure during pregnancy

The effects of air pollution on fetal outcomes, principally premature birth and fetal growth retardation/low birth weight,⁸ are themselves associated with impact on the developing organ systems. Interference with normal placenta development, evidenced by variations of the size and structure of the placenta at birth, has been associated with several chronic diseases, including heart disease, obesity and type 2 diabetes. Low birth weight for gestation is associated with low lung function, COPD, cardiovascular disease and early death in adulthood.⁹ Poor fetal growth is linked to abnormal development of the kidneys, and to hypertension and kidney disease in later life.¹⁰ Low birth weight for gestation is associated with rapid postnatal growth, which is linked in turn to the development of

obesity, asthma and low lung function. Additional to these consequences of poor fetal growth, there may be direct toxic effects of air pollution on specific organ development.

The best evidence for prenatal pollution exposure affecting subsequent development is for the brain and nervous system. Heavy metals, particularly lead (Pb) and mercury (Hg), which can cross the placenta and accumulate in the fetus, have been associated with neurodevelopmental harm, leading to reduced cognitive function, lower IQ, attention deficit hyperactivity disorder (ADHD), and possibly autism spectrum disorder during childhood.¹¹ It is suggested that similar effects could be associated with exposure to ultra-fine particles, which freely enter the bloodstream. Exposure to airborne PAHs in pregnancy has also been linked to adverse developmental abnormalities in children, including low IQ.¹² These compounds bind to DNA and have been associated with epigenetic remodelling (see section 3.4.1 on epigenetic effects) and DNA mutations, indicating the potential for increased cancer risk, although the evidence for this is currently limited.¹³

Intrauterine lung development is almost certainly hampered by tobacco smoke, either active smoking on the part of the mother or second-hand exposure of the mother to others' smoking. Low birth weight for gestation is associated with low lung function in childhood and later life, and babies of normal weight who were exposed to maternal smoking in pregnancy have low airway function shortly after birth.¹⁴ The evidence that exposure to airborne substances other than tobacco smoke alters lung development in the same way is not so well developed, but there is evidence that PM₁₀ exposure alters lung function in the postnatal period,¹⁵ and that prenatal PM_{2.5} exposure is associated with low forced expiratory volume in 1 second (FEV₁) and forced vital capacity (FVC) in preschool children.¹⁶

There is compelling evidence in children and adults that air pollution exposure is associated with new-onset asthma, but it is more difficult to be certain about the effects of exposure during pregnancy. This is partly due to the problem of trying to separate highly correlated exposures in the prenatal and early postnatal periods in observational studies of long-term outcomes. There is certainly evidence that tobacco smoke exposure, as well as affecting lung function, can influence the development of asthma and possibly other allergic diseases in children. This could be mediated through effects on the developing immune system. Maternal exposure to PM₁₀ and NO₂ has been linked to altered proportions of immune cells in cord blood, and PAH and PM_{2.5} exposure is associated with high cord blood immunoglobulin E (IgE) levels, but only in children of mothers without any reported allergies. Although IgE is raised in allergic diseases, high IgE by itself is not proof of allergic sensitisation in infants. However, current evidence is insufficient to determine whether similar effects can be attributed to air pollution exposure during pregnancy and developmental changes predisposing to asthma in children, although prenatal tobacco smoke exposure may increase vulnerability to postnatal exposure to air pollution.¹⁷

For other organ systems with long latencies of disease manifestation, such as the cardiovascular and metabolic systems, epidemiological studies are not well suited to separating organ-specific effects of prenatal exposures from cumulative exposure to pollutants through the remainder of the lifecourse. The strongest evidence comes from links between low birth weight and disease outcomes in adulthood.

Air pollution exposure after birth

Infants living in areas with high levels of particulate air pollution are at increased risk of death during the first year of life, particularly from respiratory illnesses. There is also evidence that air pollution, like tobacco smoke, may exacerbate the effects of respiratory infections in young children.¹⁸ There is less certain evidence of a link between pollution exposure in the postneonatal period and SIDS.¹⁹

There is little doubt that acute exposure to high levels of air pollution results in increased respiratory symptoms in children, including parent- and self-reported cough and wheeze, and increased visits to emergency departments with respiratory illnesses. Whether air pollution causes asthma in previously healthy children is a more contentious issue.^{20,21}

Epidemiological studies have suggested associations of air pollution with asthma onset, but results have been inconsistent and no specific pollutant has been identified. There are also difficulties in separating the effects of pollution on respiratory symptoms (wheeze) from true asthma, particularly in young children. Additionally, these studies are prone to confounding from a large number of social and lifestyle factors, including housing, diet, obesity, exercise and exposure to indoor and outdoor allergens, which all changed rapidly in society as asthma prevalence increased towards the end of the 20th century. There is biological evidence that diesel exhaust particles (DEPs) can enhance responses to inhaled allergens; this is supported by an epidemiological link between estimated traffic-related air pollution exposure and allergic diseases, although the evidence is still relatively weak.²² Exposure to VOCs, particularly formaldehyde in indoor air, has been suggested as a possible cause of asthma onset, but the current evidence remains inconsistent. Exposure of young children to second-hand tobacco smoke remains one of the most important sources of indoor air pollution and is associated with asthma prevalence, although the effects of exposure to maternal smoking in pregnancy are probably stronger than for childhood exposure. There seems little doubt that air pollution adversely affects the normal growth of lung function during childhood,^{23,24} right up to the late teens (see next chapter). Interestingly, this reduction in lung function is reversible to some extent, but it is not yet known how long the potential for recovery persists into later life.

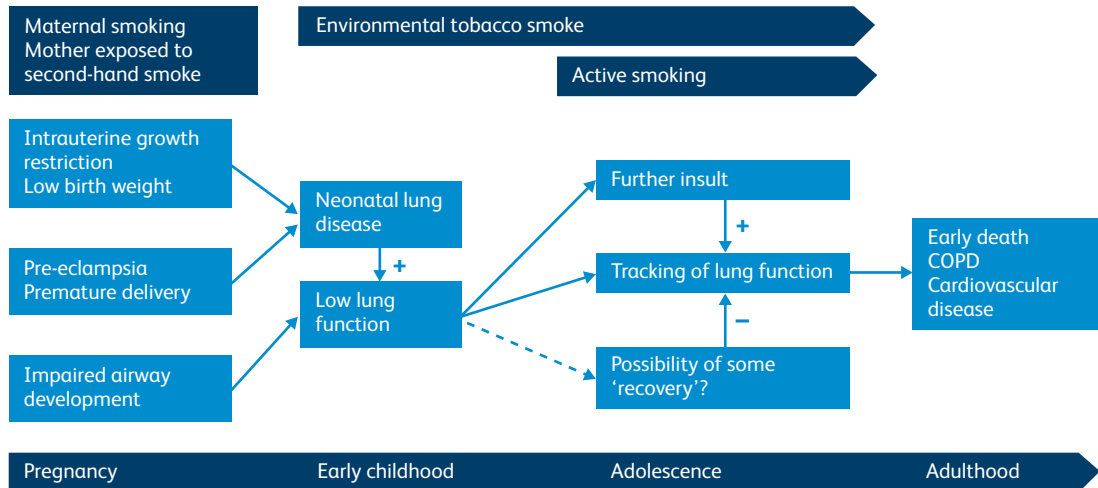
Because the central nervous system is still developing rapidly after birth, children remain susceptible to harmful effects of air pollution on their neurodevelopment and long-term cognitive health.²⁵ Several types of air pollution have been associated with harmful effects on neurocognitive development. As with prenatal effects, exposure of young children to heavy metals, even at very low levels, impairs cognitive development and lowers IQ. There is evidence that high levels of NO₂ impair all domains of neurodevelopment, including sensory, motor and psychomotor function. Children exposed to high indoor NO₂ levels from cooking and heating sources have been shown to have poorer cognitive function and seem to be at increased risk of ADHD.

There is compelling evidence that air pollution is a major risk factor for cardiovascular diseases (see next chapter) and a link has been suggested between air pollution and the development of obesity in children. In addition, there are concerns that air pollution can cause changes in human DNA that are biological markers of increased cancer risk. However, there is currently limited evidence of an increased risk of childhood cancers or of longer-term effects on adult cancers associated with high levels of exposure to air pollutants in childhood, with the possible exception of second-hand tobacco smoke.

3.4 How do early developmental changes manifest themselves throughout life?

Although tobacco smoke and air pollution are not equivalent exposures, the mechanism by which tobacco smoke exposure in early life influences future health is used in Box 12 as an illustration to show how exposure at different periods through life can affect lung growth and development. Although tobacco smoke is used here as an example because there is a great deal of research evidence about its harmful effects, air pollution exposure can have similar consequences for the lungs.

Box 12: The lifecourse influences of air pollution (eg tobacco smoke) exposure on lung function and disease



A pregnant woman can be exposed to many types of potentially harmful environmental poisons including alcohol, heavy metals, drugs and air pollution (including tobacco smoke). Some of these can be avoided by lifestyle choices, but for others, such as air pollution, this may not be possible. We use tobacco smoke exposure as an example because it is a common exposure and one for which the best evidence exists. However, the same pathways may operate for unavoidable risks, such as outdoor air pollution. A baby born of a mother who smokes during her pregnancy is likely to be born early and to have low birth weight, both of which are associated with low lung function at birth. Frequent respiratory illnesses in the newborn period may further hamper lung development. Continued tobacco smoke exposure during early childhood leads to increased respiratory illnesses and slows lung growth, exacerbating the prenatal effects.

Smoking is socially patterned. Therefore, children of smoking mothers are more likely to live in deprived urban areas and to be exposed to other harmful influences, including air pollution, which also slows lung function growth. Lung function established in early life 'tracks' to low peak lung function in adulthood. Even without further harm, the natural decline in lung function that occurs during adult life leads to earlier onset of diseases associated with low lung function, eg COPD. However, children of smoking parents are themselves more likely to smoke, and lack of social mobility leads to a greater risk of continued exposure to harmful air pollutants. These accelerate lung function decline, resulting in earlier onset of respiratory diseases and early death.

Even relatively small disturbances to normal development of organs can have marked changes on the number of individuals in a population that develop diseases as a consequence. In the example of lung function given above, a small change in lung function in an individual may not cause that person to have symptoms of lung disease. However, as shown in Fig 9, lung function values follow a normal distribution around the average. If a toxic exposure such as air pollution shifts the population average downwards, then a greater number of individuals in the lower tail of the population will have low enough lung function to cause disease.

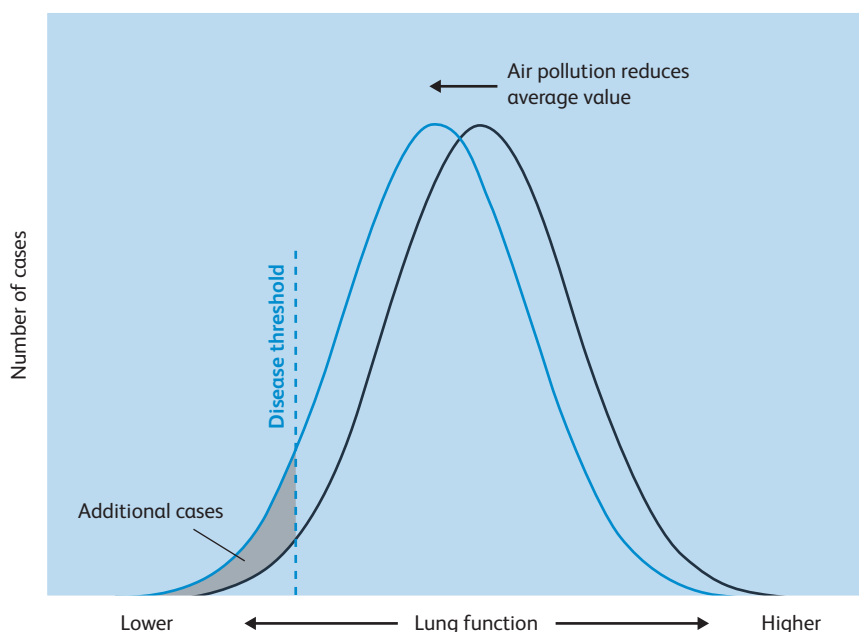


Fig 9. A small change in the average value of lung function leads to a far greater number of people falling below the disease threshold (shaded).

3.4.1 Epigenetic effects

Another possible mechanism for the long-term effects of early-life exposure to air pollution on health is by changes in DNA, called epigenetic modification.²⁶ There are several ways in which DNA can be changed without altering its underlying genetic code; the most studied of these is DNA methylation, in which methyl (-CH₃) chemical groups are bound to cytosine residues that comprise one of the four building blocks of DNA, usually resulting in gene silencing (see Fig 10). Both acute and chronic exposures to indoor and outdoor air pollutants, such as indoor solid fuel use, particulates, Pb, arsenic (As), nickel (Ni) and PAHs, have been shown to be associated with changes in DNA methylation.^{27,28} This can alter how genes work and is thought to be a way for humans to adapt to their environment without having to wait for evolutionary changes over many generations.²⁹ Fig 10 shows an example of how epigenetic modification of DNA by intrauterine exposures could affect lung function at birth.

Fetal life is a particularly sensitive period when maternal environmental exposures can result in epigenetic changes that can persist into adult life. For example, DNA methylation patterns associated with intrauterine tobacco smoke exposure have been shown to persist into adulthood and to occur in genes that could influence the biological response to personal smoking in later life. Studies currently underway will establish whether comparable intrauterine air pollution exposure has similar long-term epigenetic effects.

One fascinating aspect of epigenetic changes is their potential to be inherited from parent to child and to subsequent generations. This has been observed in animals and, although the evidence in humans is less clear, several studies have now reported that grandmaternal smoking while pregnant, irrespective of whether the mother herself smokes, is associated with increased risk of asthma in grandchildren. These findings indicate how early-life air pollution exposure could have consequences for future generations, even if current exposure is reduced.

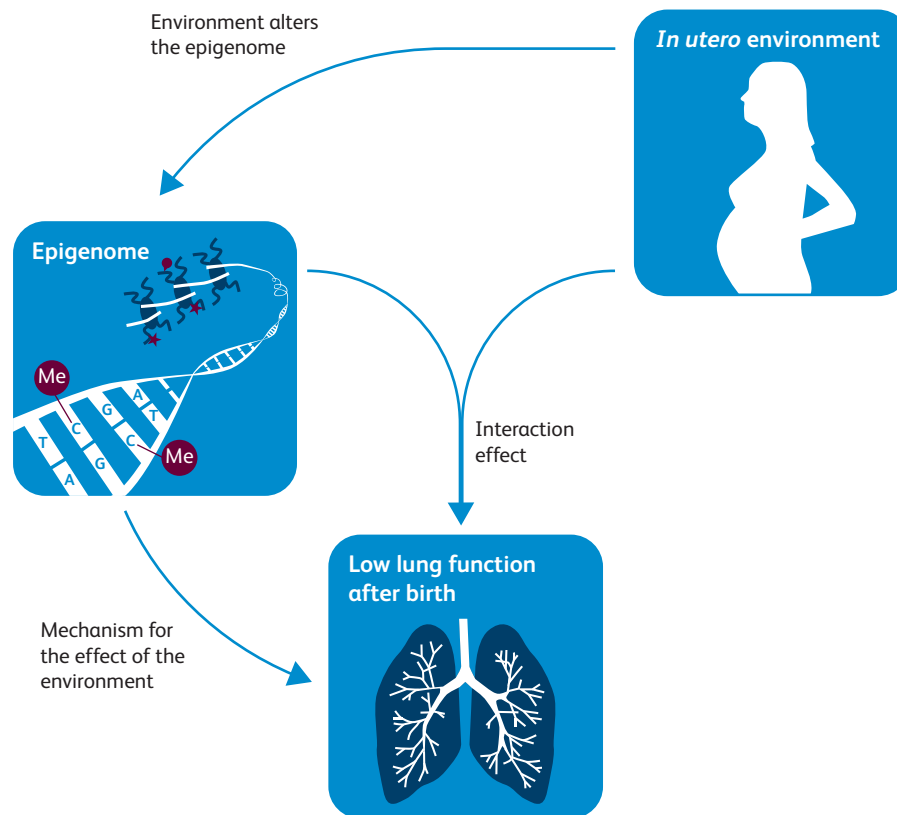


Fig 10. Harmful environmental exposures during intrauterine development can modify DNA through epigenetic effects, resulting in long-lasting alterations to gene regulation ('Me' indicates methylation). This could not only alter development of organs (such as the lungs), leading to physiological differences at birth, but may also lead to altered responses to further exposure later in life. Thus, epigenetic programming provides a pathway through which early life environmental exposures can influence health through the lifecourse. Figure modified with permission from: Lockett GA, Huoman J, Holloway JW. Does allergy begin *in utero*? *Pediatr Allergy Immunol* 2015;26:394–402.

3.5 Conclusions

There is no doubt that air pollution can affect the fetus, either indirectly through the health of the mother, or directly by affecting developing fetal organs and systems. These effects can have a permanent influence on growth and health throughout life. Exposure of the young child to air pollution can produce definite harm and even increase the risk of death from lung infections. There is evidence that it may produce deleterious effects on growth, intelligence and neurological development. Increasingly, it is being discovered that certain genetic polymorphisms make some individuals more susceptible than others to the effects of particular pollutants. In studies that have examined 'dose' effects of air pollution, harmful effects have been detected at current regulatory levels and there appears to be no lower limit below which harmful effects do not occur.

The evidence of harm due to air pollution to the fetus and the young child is not as strong as it is for adults, because the topic is relatively new and has not been so heavily researched. In addition, the effects on the baby and child may be more subtle and take many decades to appear, so that a causal relationship

is not observed unless it is specifically looked for over a long period of time. It is likely that maternal air pollution exposure interacts with other stressors in pregnancy such as poor diet, tobacco smoking and exposure to certain drugs. Moreover, the evidence for the effects of air pollution on cardiovascular disease and death in later life is very strong, so it is logical to conclude that reducing exposure to air pollution from as early an age as possible will be beneficial in order to reduce morbidity and early death.

3.6 References

- 1 Wick P, Malek A, Manser P *et al.* Barrier capacity of human placenta for nanosized materials. *Environ Health Perspect* 2010;118:432–6.
- 2 van den Hooven EH, Pierik FH, de Kluienaar Y *et al.* Air pollution exposure and markers of placental growth and function: the generation R study. *Environ Health Perspect* 2012;120:1753–9.
- 3 Ha S, Hu H, Roussos-Ross D *et al.* The effects of air pollution on adverse birth outcomes. *Environ Res* 2014;134:198–204.
- 4 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.
- 5 Glinianaia SV, Rankin J, Bell R *et al.* Particulate air pollution and fetal health: a systematic review of the epidemiologic evidence. *Epidemiology* 2004;15:36–45.
- 6 World Health Organization. *Review of evidence on health aspects of air pollution – REVIHAAP Project*. Bonn: WHO, 2013. www.euro.who.int/__data/assets/pdf_file/0004/193108/REVIHAAP-Final-technical-report-final-version.pdf [Accessed 2 December 2015].
- 7 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:e113920.
- 8 Pedersen M, Giorgis-Allemand L, Bernard C *et al.* Ambient air pollution and low birthweight: a European cohort study (ESCAPE). *Lancet Respir Med* 2013;1:695–704.
- 9 Risnes KR, Vatten LJ, Baker JL *et al.* Birthweight and mortality in adulthood: a systematic review and meta-analysis. *Int J Epidemiol* 2011;40:647–61.
- 10 Luyckx VA, Bertram JF, Brenner BM *et al.* Effect of fetal and child health on kidney development and long-term risk of hypertension and kidney disease. *Lancet* 2013;382:273–83.
- 11 Canfield RL, Henderson CR Jr, Cory-Slechta DA *et al.* Intellectual impairment in children with blood lead concentrations below 10 microg per deciliter. *New Engl J Med* 2003;348:1517–26.
- 12 Perera F, Weiland K, Neidell M, Wang S. Prenatal exposure to airborne polycyclic aromatic hydrocarbons and IQ: estimated benefit of pollution reduction. *J Public Health Policy* 2014;35:327–36.
- 13 Perera F, Hemminki K, Jedrychowski W *et al.* In utero DNA damage from environmental pollution is associated with somatic gene mutation in newborns. *Cancer Epidemiol Biomarkers Prev* 2002;11:1134–7.
- 14 Stick SM, Burton PR, Gurrin L, Sly PD, LeSouëf PN. Effects of maternal smoking during pregnancy and a family history of asthma on respiratory function in newborn infants. *Lancet* 1996;348:1060–4.
- 15 Latzin P, Röösl M, Huss A, Kuehni CE, Frey U. Air pollution during pregnancy and lung function in newborns: a birth cohort study. *Eur Respir J* 2009;33:594–603.
- 16 Jedrychowski WA, Perera FP, Maugeri U *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;24:492–501.
- 17 Sonnenschein-van der Voort AM, de Kluienaar Y, Jaddoe VW *et al.* Air pollution, fetal and infant tobacco smoke exposure, and wheezing in preschool children: a population-based prospective birth cohort. *Environ Health Perspect* 2012;11:91.
- 18 MacIntyre EA, Gehring U, Mölter A *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;122:107–13.
- 19 Woodruff TJ, Darrow LA, Parker JD. Air pollution and postneonatal infant mortality in the United States, 1999–2002. *Environ Health Perspect* 2008;116:110–15.
- 20 Dick S, Friend A, Dynes K *et al.* A systematic review of associations between environmental exposures and development of asthma in children aged up to 9 years. *BMJ Open* 2014;4:e006554.

- 21 Mölter A, Simpson A, Berdel D *et al.* A multicentre study of air pollution exposure and childhood asthma prevalence: the ESCAPE project. *Eur Respir J* 2015; 45:610–24.
- 22 Gruzieva O, Bellander T, Eneroth K *et al.* Traffic-related air pollution and development of allergic sensitization in children during the first 8 years of life. *J Allergy Clin Immunol* 2012;129:240–6.
- 23 Chen Z, Salam MT, Eckel SP, Breton CV, Gilliland FD. Chronic effects of air pollution on respiratory health in Southern California children: findings from the Southern California Children's Health Study. *J Thor Dis* 2015;7:46–58.
- 24 Gehring U, Gruzieva O, Agius RM *et al.* Air pollution exposure and lung function in children: the ESCAPE project. *Environ Health Perspect* 2013;121:1357–64.
- 25 Liu J, Lewis G. Environmental toxicity and poor cognitive outcomes in children and adults. *J Environ Health* 2014;76:130–8.
- 26 Fernandez-Twinn DS, Constância M, Ozanne SE. Intergenerational epigenetic inheritance in models of developmental programming of adult disease. *Semin Cell Dev Biol* 2015;43:85–95.
- 27 Jiang R, Jones MJ, Sava F, Kobor MS, Carlsten C. Short-term diesel exhaust inhalation in a controlled human crossover study is associated with changes in DNA methylation of circulating mononuclear cells in asthmatics. *Part Fibre Toxicol* 2014;11:71.
- 28 Janssen BG, Godderis L, Pieters N *et al.* Placental DNA hypomethylation in association with particulate air pollution in early life. *Part Fibre Toxicol* 2013;10:22.
- 29 Hou L, Zhang X, Wang D, Baccarelli A. Environmental chemical exposures and human epigenetics. *Int J Epidemiol* 2012;41:79–105.

Chapter 4: Summary

Exposure to air pollution has health effects at every stage of life, from before birth into old age. The damage is sometimes gradual, and may not be apparent for many years.

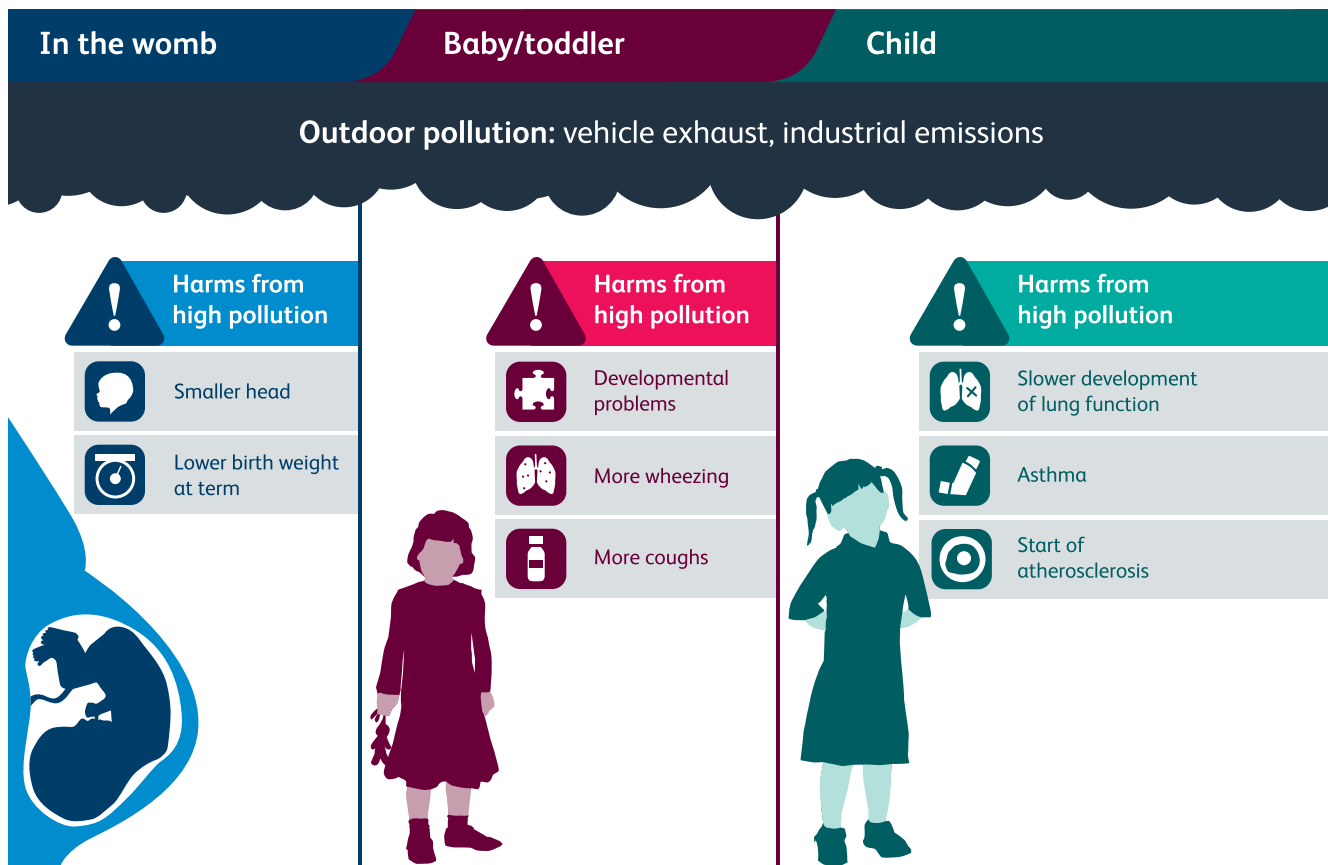
Lung function naturally develops throughout childhood, and there is clear evidence that long-term exposure to outdoor air pollution suppresses this process. In addition, it may speed up the decline of lung function through adulthood and into older age.

There is also good evidence that outdoor air pollution causes lung cancer.

It is likely that long-term exposure to air pollution is linked to the development of asthma. For people who already have asthma, there's strong evidence that air pollution can make it worse.

We still need more research, but it's possible that exposure to air pollution could be associated with the appearance of diabetes, and may also damage the brain's thinking abilities (cognition) in subtle ways that build up over time.

Large studies have shown a strong link between air pollution and cardiovascular disease (heart disease and strokes).



*Includes exhaust gases from cooking, heating and burning solid fuels, use of household cleaners and other chemicals, VOCs, etc

Key facts

- As the levels of air pollution increase, so does the harmful effect on lung function.
 - Children living in highly polluted areas are four times more likely to have reduced lung function in adulthood. Improving air quality for children has been shown to halt and reverse this effect.
 - For older people, living near a busy road speeds up the rate of lung function decline that is associated with ageing.
- Young children who live in polluted areas have more coughs and wheezes.
- The evidence is so convincing that the International Agency for Research on Cancer has classified air pollution as a known cause of lung cancer. This condition is thought to take many years to develop. Therefore, exposure in childhood could be linked to lung cancer in adults.
- Exposure to air pollution may affect mental and physical development in children, and thinking skills (cognition) in older people.
- Over the long term, breathing air pollution is linked to the development of cardiovascular disease in adults, including atherosclerosis (furring of the arteries). Once people have a heart condition, spikes in air pollution can make their symptoms worse, leading to more hospital admissions and deaths.



Chapter 4: Health effects of air pollution over our lifetime

In the previous chapter, we reviewed the reasons why the fetus and the child are especially vulnerable to air pollution, and why this vulnerability has implications for health across the lifecourse. This chapter reviews the evidence that long-term exposure to air pollution has adverse effects on health in infants, children, young people and adults. In reviewing the evidence, we focus mainly on new-onset (incident) disease and, therefore, greater weight is placed on evidence from studies where large groups of individuals have been followed over long periods of time.

4.1 The growing and the ageing lung

Maximising lung growth during childhood and minimising lung function decline during ageing are important because the development of low lung function (measured by spirometry as FEV₁ and FVC) means that there is less reserve if lung disease develops. For example, in asthmatic children, low lung function detected by spirometry predisposes to more severe asthma symptoms and decreased quality of life. In healthy young adults in the Coronary Artery Risk Development in Young Adults (CARDIA) study, accelerated decline in lung function over a 10-year period was associated with lower output of blood from the heart,¹ and recent analyses of data from the Framingham Offspring Cohort, the Copenhagen City Heart Study and the Lovelace Smokers Cohort found that low FEV₁ in early adulthood predisposes to the development of COPD in later life.² A lung function of <80% of that expected is normally used as a cut-off to indicate an increased risk of respiratory symptoms.

4.1.1 Infants

As discussed earlier, adverse effects of air pollution on the developing fetus may have long-term effects on lung development during extrauterine life. Separating the effects of exposure during gestation from the effects of exposure during infancy is difficult, because new mothers with infants usually live at the same address as when they were pregnant. However, a recent study reported an effect of fetal exposure *per se* on lung function in later life. In this study, increased exposure to NO₂ during the second trimester of gestation was independently associated with reduced FEV₁ measured at 4.5 years of age.³

4.1.2 Schoolchildren

In schoolchildren, the effects of air pollution over time on the increase of FEV₁ and FVC (as indices of lung function growth) have been examined in the Children's Health Study. This study recruited more than 11,000 schoolchildren selected from classrooms in 16 communities in California, USA.⁴ Children were chosen from different areas to reflect the widest range of background regional pollution, which is the component of air pollution that all children are exposed to within a community. Lung function was measured every year and long-term background levels of air pollution were measured using monitoring stations in each community. After adjusting for a wide range of confounding variables, suppression of lung function growth was found in children living in communities with the highest concentrations of PM₁₀, PM_{2.5}, elemental carbon and NO₂ (Fig 11).

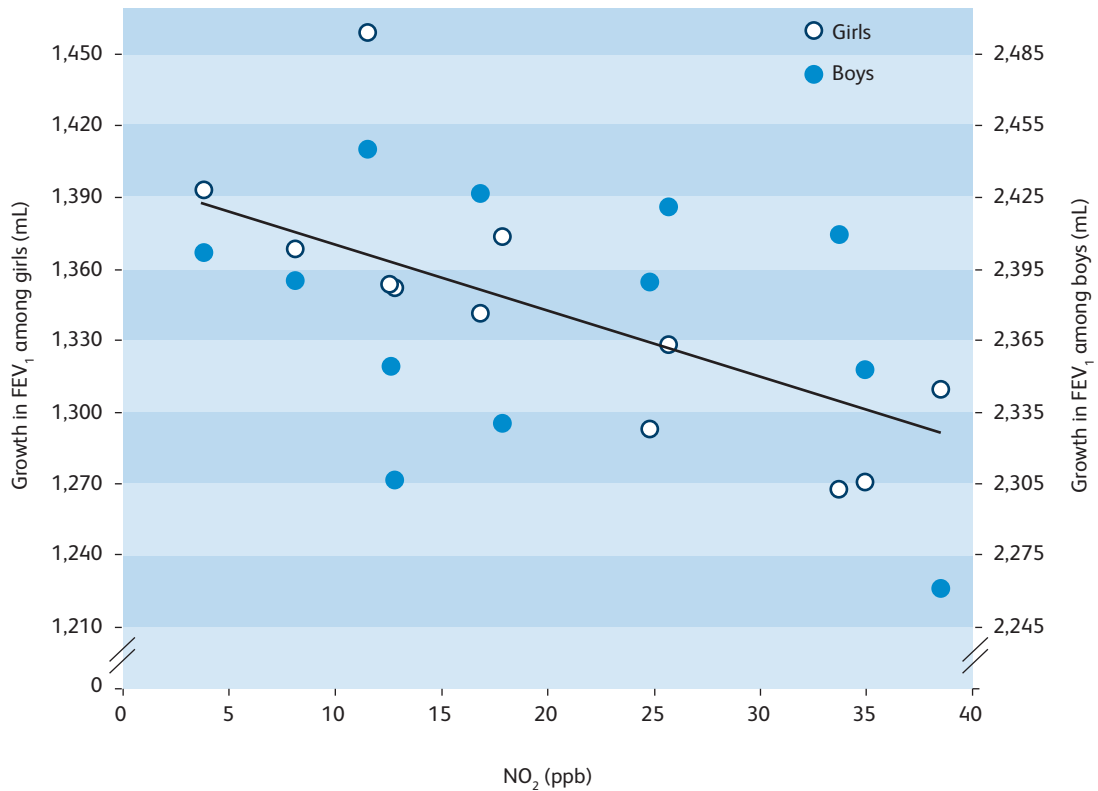
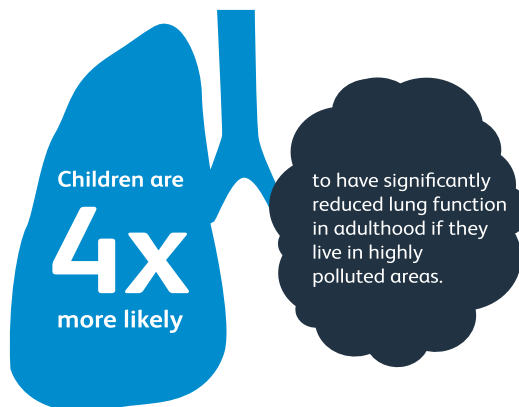


Fig 11. Average growth in FEV₁ in school-age girls (open circles) and boys (closed circles) during an 8-year period, plotted against average NO₂. Each dot represents a separate community. As background NO₂ increases in communities, the rate of growth in FEV₁ decreases. A similar effect was observed for background PM. Adapted with permission from Gauderman *et al.*,⁵ © 2004 Massachusetts Medical Society.

In the California study, suppression of lung function growth by air pollution was clinically significant. For example, the proportion of young adults with an FEV₁ <80% of that expected was four times higher in the community exposed to the highest background level of air pollution than in the least pollution-exposed community.



The Children's Health Study also addressed the independent effect of exposure to locally generated air pollution from traffic.⁴ Researchers found that children living within 500 m of a heavily used road had

suppression of growth of lung function (measured as FEV₁) over an 8-year period, compared with children living >1,500 m from a heavily used road.

The association between air pollution and impaired lung function growth has also been observed in other populations. For example, in a study of Taiwanese children followed over a 2-year period, reduced lung function growth (as FEV₁) was strongly associated with increased exposure to PM_{2.5}.⁶

More direct evidence that air pollution causes suppression of lung function growth is provided by a cross-sectional study of healthy schoolchildren in Leicester, UK.⁷ This study used the capacity of macrophages resident on the mucosal surface of the lower airways to take up inhaled material, including pollution particles (PM) (Fig 12).

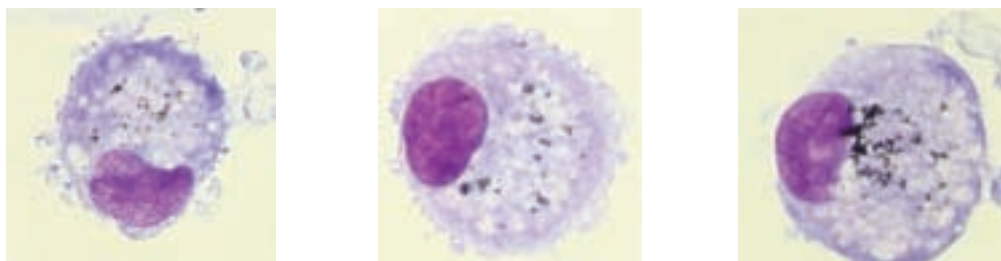


Fig 12. Examples of macrophages recovered from the lower airways of healthy children living in Leicester. The black spots in some of the cells are inhaled fossil fuel-derived particles.

In this study, each 1 μm^2 increase in the area of macrophage black carbon was associated with a 17% reduction in the expected FEV₁ (Fig 13).

The question of whether reducing the levels of air pollution improves lung function growth was recently addressed. In its most recent analysis, the Children's Health Study found that declining levels of NO₂ and PM were associated with improvements in lung function growth.⁸ Improved lung function growth, as a consequence of improved air quality, reduced the proportion of young people with an FEV₁ <80% of normal from 7.9% to 3.6%. In the UK, 7.9% of the population of 18-year-olds translates to approximately 58,500 individuals.

Whether exposure to O₃ (a predominantly summer pollutant produced in the atmosphere by photochemical oxidation of primary pollutants on sunny days) reduces lung function growth is unclear. However, an effect of O₃ on 2-year lung growth was found in the Taiwanese study referred to above,⁶ and a study of students who were lifelong residents of the Los Angeles and San Francisco Bay areas found that those with the highest exposure to O₃ had reduced measures of small airways lung function.⁹

4.1.3 Adults

As pointed out earlier, lung function in adulthood slowly declines with age. A recent analysis linked the long-term lung function of the US adults recruited into the Framingham Offspring or Third Generation studies with exposure in the home to air pollution, expressed as either distance of home from a major road or as modelled exposure to PM_{2.5}.¹⁰ This study found that adults living <100 m from a major road had a greater decline in FEV₁ than those living >400 m from a major road. In a study of older US men

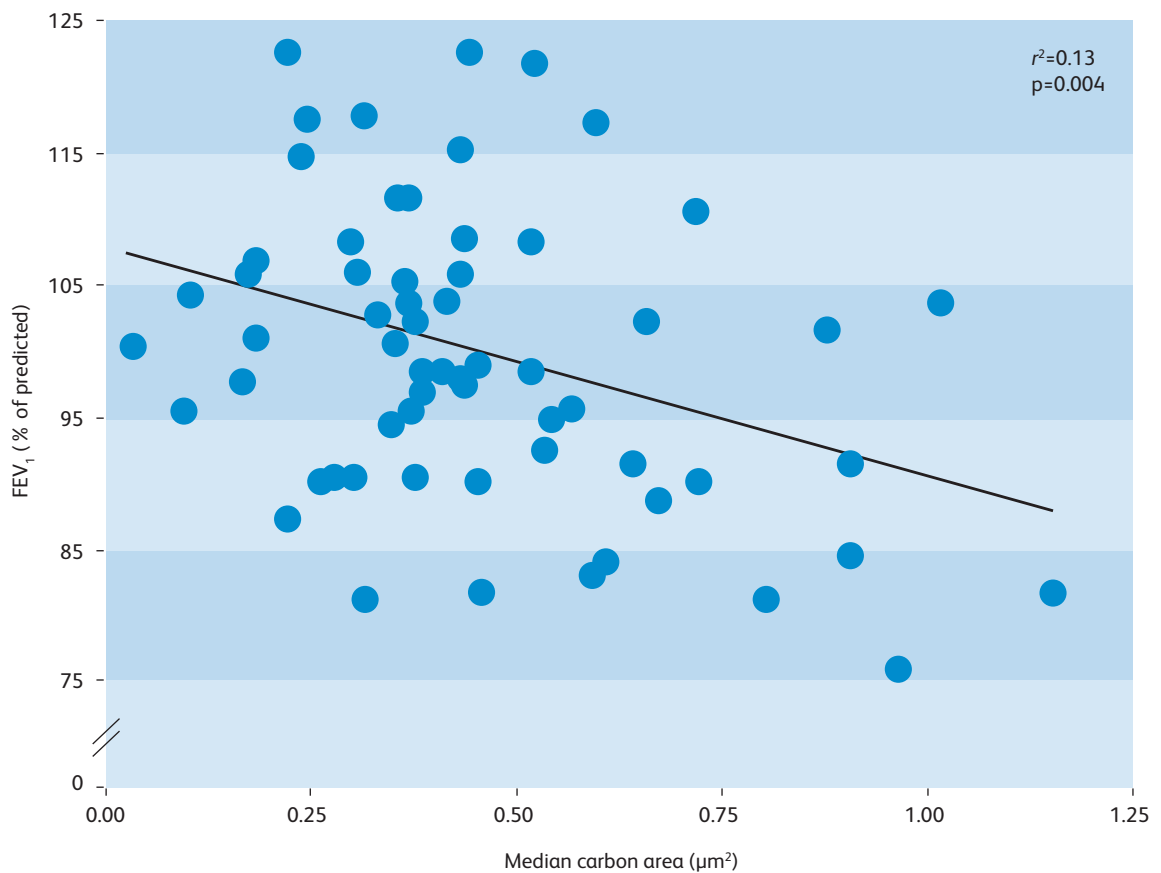


Fig 13. Association between FEV₁ (expressed as a percentage of the predicted value based on height and sex) and area of fossil fuel-derived particles in healthy children living in Leicester, UK. As the number of particles in macrophages increases, lung function decreases. Adapted with permission from Kulkarni *et al*,⁷ © 2006 Massachusetts Medical Society.

(average age 70 years) whose lung function was measured up to six times from 1995 to 2011, exposure to traffic-derived air pollution, determined for the home address, revealed that increased long-term exposure to black carbon correlated with an accelerated decline in both FEV₁ and FVC.¹¹ The European Study of Cohorts for Air Pollution Effects (ESCAPE) also studied the effect of outdoor air pollution on adult lung function decline.¹² Subjects in this study had to be at least 20 years old, with lung function data from two different time points approximately 10 years apart. While no association was found between either background or locally generated air pollution and lung function decline, cross-sectional analysis found that reduced FEV₁ was associated with increased long-term exposure to NO₂ and PM₁₀. The researchers concluded that their findings were consistent with accelerated decline in lung function caused by air pollution.

4.1.4 Summary

Long-term exposure to either background or locally generated air pollution impairs lung function growth in children. Reducing exposure to air pollution reverses this effect, thereby allowing more young people to achieve their maximum lung function growth potential. In adults, there is emerging evidence that air pollution accelerates the decline in lung function during ageing.

4.2 Wheeze and asthma

Asthma is a long-term inflammatory condition of the conducting airways of the lungs. It causes the airways to contract too much and too easily (airways hyper-responsiveness), leading to cough, wheeze, chest tightness and shortness of breath. It is a disease whose onset often begins in early childhood and is associated with acquisition of atopy – the inherited predisposition to become allergic to common environmental allergens such as dust mites, animal dander, fungi and pollens. Superimposed upon day-to-day asthma symptoms are exacerbations lasting from days to weeks, which are caused by a variety of stressors such as air pollution episodes, respiratory viral infection and allergen exposure. Asthma is potentially a life-threatening condition, with a high burden on health services and quality of life. There are over 5 million people in the UK who are currently receiving treatment for asthma, 1 million of these being children.¹³ The UK still has some of the highest rates of asthma in Europe and, on average, three people a day still die from asthma.¹³

4.2.1 Preschool children

In preschool children, wheeze occurs in episodes triggered by viral colds. Unlike asthma in school-age children and adults, in preschool children wheeze is not associated with allergy and is not usually associated with wheeze between colds. Clinicians therefore label wheeze in this age group as ‘preschool wheeze’ – although the label ‘preschool asthma’ is also used. Preschool wheeze is common, with up to 25% of all preschool children experiencing at least one episode of wheeze.¹⁴ Analysis of children aged 1–5 years in the Leicester Birth Cohort found that exposure to locally generated PM₁₀ at the home address was associated with new-onset preschool wheeze.¹⁵ A study in British Columbia, Canada, which identified all new cases of preschool asthma diagnosed up to 3–4 years of age, found an increased risk of preschool asthma with increased modelled exposure to either NO₂ or PM₁₀ both during gestation and during the first year of life.¹⁶ A 3-year longitudinal study that recruited both preschool and early school-age children in different communities in southern California, USA, found that increased risk of developing early school-age asthma was associated both with markers of traffic-associated outdoor air pollution near the home and with exposure to background NO₂.¹⁷

4.2.2 Schoolchildren

In the Californian Children’s Health Study, exposure to higher local concentrations of NO₂ was associated with new-onset asthma.⁴ Furthermore, in this study the risk of lifetime asthma was higher in children living closer to a freeway. Children living within 75 m of a major road had a 29% increased risk of lifetime asthma, and traffic-related air pollutants near the home and school were associated with a 1.5-fold-increased risk of new-onset asthma. A meta-analysis by Gasana *et al*¹⁸ of the effect of traffic-generated air pollution and asthma in children, which included 19 studies, concluded that:

- 1 increased exposure to NO₂ is associated with new-onset asthma
- 2 increased exposure to PM is associated with new-onset wheeze.

NO₂ is also generated indoors during gas cooking. In a meta-analysis of 19 cross-sectional studies on the effect of either indoor NO₂ or gas cooking and asthma, Lin *et al*¹⁹ found that gas cooking was also associated with increased risk of both current and lifetime asthma. The risk estimates were similar for preschool wheeze, asthma in 6–10-year-olds, and asthma in children >10 years of age.

4.2.3 Adults

The Swiss study on Air Pollution and Lung Disease in adults (SAPALDIA) cohort found that new-onset asthma was associated with exposure to traffic-related PM₁₀ at the home address.²⁰ A study of women in the USA reported an association between exposure to PM_{2.5} and new-onset asthma.²¹ A recent meta-analysis of the effect of long-term exposure to air pollution and new-onset asthma in both schoolchildren and adults by Anderson *et al*,²² using data from 17 cohorts, found that a 7% and a 16% increase in new-onset asthma was associated with a 10 µg/m³ increase in either NO₂ or PM_{2.5}, respectively.

4.2.4 Summary

There is now consistent evidence that outdoor air pollution is associated with new-onset asthma across the lifecourse. There is also accumulating evidence that indoor NO₂ may also be associated with new-onset asthma.

4.3 Diabetes

Type 2 diabetes is due to a combination of the body not producing sufficient insulin to function properly, and/or the body's cells losing their response to insulin (insulin resistance). Type 2 diabetes is more common than type 1 diabetes (when the body doesn't produce any insulin at all), and is associated with obesity and metabolic syndrome with increased risk of cardiovascular disease. Most adults with diabetes have type 2 that is linked to diet and obesity. In the UK, 6% of adults (approximately 2.7 million individuals) have a diagnosis of diabetes.²³

4.3.1 Children

A study of healthy 10-year-old children reported an association between increased insulin resistance and exposure of the home address to outdoor air NO₂ and PM₁₀.²⁴ However, it remains unclear whether an abnormal insulin resistance index during childhood is associated with an increased risk of developing type 2 diabetes.

4.3.2 Adults

A prospective cohort study of women followed up for 10 years assessed the association between new-onset type 2 diabetes and modelled outdoor air pollution (both locally generated and background) at the home address.²⁵ New-onset type 2 diabetes was associated with exposure to both PM_{2.5} and oxides of nitrogen (including NO₂). An analysis of 23-year follow-up data from the Nurses' Health Study and the Health Professionals Follow-Up Study found that living within 50 m of a major road, compared with those living >200 m away, was associated with new-onset type 2 diabetes.²⁶ A meta-analysis of the effect of long-term exposure to air pollution on type 2 diabetes by Wang *et al*,²⁷ which included data from these studies along with data from eight other cohorts, concluded that increased risk of developing type 2 diabetes is associated with long-term exposure to PM_{2.5}, PM₁₀ and NO₂. In addition, a recent meta-analysis by Eze *et al*,²⁸ which included three studies on PM_{2.5} and four studies on NO₂, showed an 8–10% increased risk of type 2 diabetes per 10 µg/m³ increase in exposure to both pollutants, with stronger associations being observed in women.

4.3.3 Summary

There is emerging evidence that exposure to air pollution is associated with new-onset type 2 diabetes in adults.

4.4 The brain

4.4.1 Fetus

As discussed in the previous chapter, there is clear evidence that exposure of pregnant women to air pollution affects fetal growth. An analysis of pooled data from 14 population-based mother–child cohort studies from 12 European countries found an inverse association between head circumference at term and outdoor air pollution, assessed using the ESCAPE methodology.²⁹ As head circumference is associated with brain volume, these results suggest that exposure to air pollution affects fetal brain growth. Evidence that effects of air pollution on the developing brain may have long-term consequences is provided by data from children recruited into six European population-based birth cohorts, which found that exposure of the fetus to increased NO₂ at the home address was associated with reduced psychomotor development at 1–6 years of age.³⁰ However, no association was found between air pollution and measures of cognitive development.

Using prospective cohort data from the Nurses' Health Study II Cohort, researchers compared the exposure to air pollution during pregnancy of participants' children with autism spectrum disorder (cases) with that of controls without autism spectrum disorder, and found that fetal exposure to PM_{2.5} at the home address was associated with increased risk of autism spectrum disorder.³¹

4.4.2 Preschool children

In a study of mother–child pairs in the US Project Viva, children living <50 m from a heavily used road had lower non-verbal IQ,³² but disentangling this association from socio-economic factors is difficult owing to the small number of children living near a heavily used road. A single case–control study found that children with autism spectrum disorder were more likely to live at residences with the highest exposure to traffic-related air pollution prenatally and during the first year of life.³³

4.4.3 Adults

A cross-sectional analysis of data from the US Health and Retirement Study found reduced cognitive function in older adults living in areas with higher PM_{2.5} concentrations.³⁴ Analysis of older men in the US Department of Veterans Affairs Normative Aging Study found that increased exposure to fossil fuel-derived black carbon was associated with increased risk of having a Mini-Mental State Examination score ≤ 25 , and that living in more polluted areas was associated with a decrease in cognitive function that is similar to a 1.7–2.8-year difference in age.³⁵ Evidence that exposure to air pollution affects brain structure was found by magnetic resonance imaging (MRI) of participants in the Framingham Offspring Study, indicating that higher exposure to PM_{2.5} is associated with a reduction in total brain volume.³⁶

4.4.4 Summary

There is emerging evidence that air pollution adversely affects both the developing and the ageing brain. The associations need to be validated in more studies, and studies need to be sufficiently robust to control for socio-economic factors.

4.5 The heart and blood vessels

There are 7 million people in the UK living with disease of the heart and blood vessels (cardiovascular disease) and 2.3 million with coronary heart disease, which is the leading cause of death in the UK.³⁷

4.5.1 Children

Cardiovascular disease in children is generally caused by genetic factors; children may be born with an abnormality of the heart or the blood vessels of the heart. In some cases, childhood disease can be acquired owing to infections, such as from rheumatic fever. In children, biomarkers of cardiovascular function show changes after exposure to air pollution. For example, studies have shown effects on blood pressure after exposure to different pollutants. A study in Mexican children found that long-term exposure to increased PM_{2.5} is associated with increased mean pulmonary arterial pressure.³⁸ Similarly, in a study of 12-year-old European children, long-term exposure to outdoor NO₂ and PM₁₀ at the home address was found to be associated with increased diastolic blood pressure.³⁹ The relevance of such changes in children to the risk of developing adult cardiovascular disease is unclear.

4.5.2 Adults

Epidemiological studies have shown significant associations between air pollution and a range of cardiovascular effects in adults. Both short- and long-term exposure to air pollution can increase the risk of myocardial infarction (heart attacks), heart failure, arrhythmias (abnormal rhythms of the heart) and stroke in susceptible individuals, such as older people or those with pre-existing medical conditions including cardiovascular disease, high blood pressure and diabetes.^{40–42} For example, analysis of data in the UK Myocardial Infarction Audit demonstrated an increase in myocardial infarction in the hours following exposure to traffic-related air pollution.⁴³ A recent large review and meta-analysis of short-term exposures to gaseous and particulate pollutants and admissions to hospital for stroke or mortality from stroke found strong associations with the day of exposure, with more persistent effects observed for fine PM.⁴⁴ Long-term exposure to fine PM is associated with cardiovascular morbidity and a reduction in life expectancy, which is due to increased deaths from cardiovascular disease (as well as respiratory disease and lung cancer).^{40–42} For example, the ESCAPE study, which collected data from across Europe, found associations between long-term exposures to fine PM and an increased risk of stroke in older people,⁴⁵ as well as an increased risk of first coronary events such as myocardial infarctions.⁴⁶

Coronary heart disease and other circulatory diseases are caused by atherosclerosis; this is when arteries become clogged with collections of fatty substances referred to as plaques or atheroma. Plaques can cause arteries to narrow and harden, restricting blood flow. Rupture of a plaque can cause a blood clot and could lead to a heart attack or stroke. It has been proposed that exposure to PM increases the likelihood of these plaques to rupture, by generating an inflammatory response in the lung and release of inflammatory mediators into the circulation, adversely affecting the cardiovascular system – for example,

by increasing circulating platelet stickiness. Short-term exposures to air pollution have demonstrated an increase in the acute-phase inflammatory markers C-reactive protein and fibrinogen.⁴⁷ Other proposed mechanisms, which could occur simultaneously, include the stimulation of receptors in the lung by PM, which then affect the autonomic nervous system and alter the control of the heart. Small particles may also affect the blood vessels directly.⁴²

Atherosclerosis develops over a number of years and begins in childhood, but effects of the disease do not become clinically significant until adulthood. Long-term exposure to air pollution has been associated with markers of atherosclerosis, such as intima-media thickness (IMT; a measure of the thickness of the walls of the artery). For example, the Multi-Ethnic Study of Atherosclerosis (MESA) in the USA was designed to investigate associations between long-term PM_{2.5} exposures and the progression of atherosclerosis over a 10-year follow-up period, using information from a large cohort of people who were without pre-existing cardiovascular disease at recruitment. Early analysis of results shows that higher long-term PM_{2.5} concentrations are associated with increased IMT of carotid arteries, and greater reductions in PM_{2.5} are related to a slower increase in IMT.⁴⁸

4.5.3 Summary

The evidence for the effects of both short- and long-term exposures to air pollution on cardiovascular disease in adults is strong. Exposure to air pollution can exacerbate existing heart conditions and contribute to the development of cardiovascular disease, resulting in increased hospital admissions and deaths from cardiovascular disease. Strongest associations have been observed for PM. It is unclear whether exposure to air pollution during childhood affects the development of cardiovascular disease in later life.

Box 13: Reductions in air pollution levels during the Beijing Olympics

In Beijing and other cities in China, high levels of air pollution are frequently experienced by the population; these are due to increases in coal-fired power stations, traffic and factories, as a result of China's increasing economy and demand for energy. During the Beijing Olympics, measures were taken to reduce levels of air pollution. This gave researchers an opportunity to look at the effect of short-term changes in exposure on markers of inflammation and of cardiovascular physiology in healthy young adults. The studies revealed changes in these markers that, although not clinically significant on their own, provide support to the proposed mechanisms for how air pollution can cause cardiovascular effects.⁴⁹

4.6 Cancer

4.6.1 Children

Cancer is rare in children, and the WHO reported in 2005 that there was 'insufficient evidence for ambient air pollution and childhood cancer'.⁵⁰ Since 2005, studies on traffic-related air pollution and childhood cancers have produced equivocal results. One Californian study in 2013 found a greater risk of acute lymphoblastic leukaemia and retinoblastoma in children after mothers were exposed to high levels

of traffic pollution during pregnancy.⁵¹ However, a meta-analysis in 2014 looked at nine studies of residential traffic exposure and childhood cancer from January 1980 to July 2011. This analysis reported an association of childhood leukaemia with residential traffic exposure in the postnatal, but not the prenatal, period.⁵² Another meta-analysis in 2014 of 11 studies that investigated traffic density and risk of childhood leukaemia from January 1979 to December 2013 found no significant associations.⁵³

Air pollution exposures have been linked to lung cancer, but this is not a type of cancer generally seen in children. It is believed that the development of lung cancer after exposure has a long latency period; this could be 15, 20, even 30 years later. Therefore, exposure of children to high levels of air pollution could contribute to the development of cancers in later life.

4.6.2 Adults

Two American studies (the American Cancer Society Study⁵⁴ and the Six Cities study⁵⁵) in the early 1990s were the first large studies to show associations between long-term exposure to air pollution (specifically PM) and deaths from lung cancer. Since then, other air pollution studies have reported associations with, deaths from, and incidence of lung cancer. The ESCAPE study used data from 17 cohort studies in nine European countries and found that PM contributes to the incidence of lung cancer in Europe.⁵⁶ Based on such studies, the International Agency for Research on Cancer (IARC) classifies outdoor air pollution and PM from outdoor air pollution as carcinogenic to humans (group 1).⁵⁷ They concluded that there is strong evidence that exposures to outdoor air pollution are associated with changes in gene expression and genetic damage, which are linked to increased cancer risk in humans. They further state that there is 'sufficient evidence that exposure to outdoor air pollution causes lung cancer'. The IARC has also classified diesel engine exhaust as carcinogenic,⁵⁸ although a recent report by the Health Effects Institute suggests that new-technology diesel exhaust may not be carcinogenic.⁵⁹

4.6.3 Summary

There is insufficient evidence of cancers due to air pollution in childhood. There is strong evidence that outdoor air pollution exposure is linked to lung cancer.

4.7 Conclusions

4.7.1 Lung function

There is strong evidence that long-term exposure to outdoor air pollution suppresses normal lung function growth in children, and there is emerging evidence that air pollution accelerates lung function decline in adults.

4.7.2 Asthma

There is highly suggestive evidence that long-term exposure to outdoor air pollution causes new-onset asthma in both children and adults. There may be an additional effect of indoor-generated NO₂. This evidence base is increasingly being strengthened. For example, in a population-based birth cohort study published online in November 2015, Gehring *et al* reported that increased exposure to NO₂ at the birth address is associated with increased risk of new-onset (incident) asthma up to age 14–16 years.⁶⁰ It is

therefore likely that, for individuals living in areas of high pollution throughout their lifecourse, reduced lung growth – and accelerated lung function decline – significantly increase their risk of developing respiratory symptoms.

4.7.3 Diabetes

The emerging association between air pollution and new-onset type 2 diabetes in adults is concerning.

4.7.4 Neurodevelopment and cognition

Small cognitive deficits during childhood, when combined with accelerated cognitive decline during adulthood, may result in clinically significant cognitive effects if high exposure continues over the lifecourse. The link between air pollution and autism is unclear. No such link was found in a recent analysis of data from the ESCAPE project published online in June 2015.⁶¹

4.7.5 Cardiovascular

Exposure to air pollution exacerbates existing cardiovascular disease, causes deaths from cardiovascular disease and is associated with a range of cardiovascular effects including myocardial infarction, heart failure and stroke.

4.7.6 Cancer

There is strong evidence that outdoor air pollution causes cancer.

4.8 References

- 1 Cuttica MJ, Colangelo LA, Shah SJ *et al.* Loss of lung health from young adulthood and cardiac phenotypes in middle age. *Am J Respir Crit Care Med* 2015;192:76–85.
- 2 Lange P, Celli B, Agustí A *et al.* Lung-function trajectories leading to chronic obstructive pulmonary disease. *N Engl J Med* 2015;373:111–22.
- 3 Morales E, Garcia-Esteban R, de la Cruz OA *et al.* Intrauterine and early postnatal exposure to outdoor air pollution and lung function at preschool age. *Thorax* 2015;70:64–73.
- 4 Chen Z, Salam MT, Eckel SP, Breton CV, Gilliland FD. Chronic effects of air pollution on respiratory health in Southern California children: findings from the Southern California Children’s Health Study. *J Thorac Dis* 2015;7:46–58.
- 5 Gauderman WJ, Avol E, Gilliland F *et al.* The effect of air pollution on lung development from 10 to 18 years of age. *N Engl J Med* 2004;351:1057–67.
- 6 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.
- 7 Kulkarni N, Pierser N, Rushton L, Grigg J. Carbon in airway macrophages and lung function in children. *N Engl J Med* 2006;355:21–30.
- 8 Gauderman WJ, Urman R, Avol E *et al.* Association of improved air quality with lung development in children. *N Engl J Med* 2015;372:905–13.
- 9 Tager IB, Balmes J, Lurmann F *et al.* Chronic exposure to ambient ozone and lung function in young adults. *Epidemiology* 2005;16:751–9.
- 10 Rice MB, Ljungman PL, Wilker EH *et al.* Long-term exposure to traffic emissions and fine particulate matter and lung function decline in the Framingham heart study. *Am J Respir Crit Care Med* 2015;191:656–64.

- 11 Lepeule J, Litonjua AA, Coull B *et al.* Long-term effects of traffic particles on lung function decline in the elderly. *Am J Respir Crit Care Med* 2014;190:542–8.
- 12 Adam M, Schikowski T, Carsin AE *et al.* Adult lung function and long-term air pollution exposure. ESCAPE: a multicentre cohort study and meta-analysis. *Eur Respir J* 2015;45:38–50.
- 13 Asthma UK. *Asthma facts and FAQs*. www.asthma.org.uk/asthma-facts-and-statistics [Accessed 31 March 2015].
- 14 Kuehni CE, Davis A, Brooke AM, Silverman M. Are all wheezing disorders in very young (preschool) children increasing in prevalence? *Lancet* 2001;357:1821–5.
- 15 Piere N, Rushton L, Harris RS *et al.* Locally generated particulate pollution and respiratory symptoms in young children. *Thorax* 2006;61:216–20.
- 16 Clark NA, Demers PA, Karr CJ *et al.* Effect of early life exposure to air pollution on development of childhood asthma. *Environ Health Perspect* 2010;118:284–90.
- 17 McConnell R, Islam T, Shankardass K *et al.* Childhood incident asthma and traffic-related air pollution at home and school. *Environ Health Perspect* 2010;118:1021–6.
- 18 Gasana J, Dillikar D, Mendy A *et al.* Motor vehicle air pollution and asthma in children: a meta-analysis. *Environ Res* 2012;117:36–45.
- 19 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;42:1724–37.
- 20 Künzli N, Bridevaux PO, Liu LJ *et al.* Traffic-related air pollution correlates with adult-onset asthma among never-smokers. *Thorax* 2009;64:664–70.
- 21 Young MT, Sandler DP, DeRoo LA *et al.* Ambient air pollution exposure and incident adult asthma in a nationwide cohort of U.S. women. *Am J Respir Crit Care Med* 2014;190:914–21.
- 22 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.
- 23 NHS Choices. *Type 2 diabetes*. www.nhs.uk/Conditions/Diabetes-type2/Pages/Introduction.aspx [Accessed 31 March 2015].
- 24 Thiering E, Cyrys J, Kratzsch J *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;56:1696–704.
- 25 Coogan PF, White LF, Jerrett M *et al.* Air pollution and incidence of hypertension and diabetes mellitus in black women living in Los Angeles. *Circulation* 2012;125:767–72.
- 26 Puett RC, Hart JE, Schwartz J *et al.* Are particulate matter exposures associated with risk of type 2 diabetes? *Environ Health Perspect* 2011;119:384–9.
- 27 Wang B, Xu D, Jing Z *et al.* Effect of long-term exposure to air pollution on type 2 diabetes mellitus risk: a systematic review and meta-analysis of cohort studies. *Eur J Endocrinol* 2014;171:R173–82.
- 28 Eze IC, Hemkens LG, Bucher HC *et al.* Association between ambient air pollution and diabetes mellitus in Europe and North America: systematic review and meta-analysis. *Environ Health Perspect* 2015;123:381–9.
- 29 Pedersen M, Giorgis-Allemand L, Bernard C *et al.* Ambient air pollution and low birthweight: a European cohort study (ESCAPE). *Lancet Respir Med* 2013;1:695–704.
- 30 Guxens M, Garcia-Esteban R, Giorgis-Allemand L *et al.* Air pollution during pregnancy and childhood cognitive and psychomotor development: six European birth cohorts. *Epidemiology* 2014;25:636–47.
- 31 Raz R, Roberts AL, Lyall K *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;123:264–70.
- 32 Harris MH, Gold DR, Rifas-Shiman SL *et al.* Prenatal and childhood traffic-related pollution exposure and childhood cognition in the Project Viva cohort (Massachusetts, USA). *Environ Health Perspect* 2015;123:1072–8.
- 33 Volk HE, Lurmann F, Penfold B, Hertz-Picciotto I, McConnell R. Traffic-related air pollution, particulate matter, and autism. *JAMA Psychiatry* 2013;70:71–7.
- 34 Ailshire JA, Crimmins EM. Fine particulate matter air pollution and cognitive function among older US adults. *Am J Epidemiol* 2014;180:359–66.
- 35 Power MC, Weisskopf MG, Alexeeff SE *et al.* Traffic-related air pollution and cognitive function in a cohort of older men. *Environ Health Perspect* 2011;119:682–7.

- 36 Wilker EH, Preis SR, Beiser AS *et al.* Long-term exposure to fine particulate matter, residential proximity to major roads and measures of brain structure. *Stroke* 2015;46:1161–6.
- 37 British Heart Foundation. *BHF cardiovascular disease statistics UK factsheet*. London: BHF, 2015. www.bhf.org.uk/research/heart-statistics [Accessed 19 June 2015].
- 38 Calderón-Garcidueñas L, Vincent R, Mora-Tiscareño A *et al.* Elevated plasma endothelin-1 and pulmonary arterial pressure in children exposed to air pollution. *Environ Health Perspect* 2007;115:1248–53.
- 39 Bilenko N, van Rossem L, Brunekreef B *et al.* Traffic-related air pollution and noise and children's blood pressure: results from the PIAMA birth cohort study. *Eur J Prev Cardiol* 2015;22:4–12.
- 40 Brook RD, Rajagopalan S, Pope CA 3rd *et al.* Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation* 2010;121:2331–78.
- 41 World Health Organization. *Review of evidence on health aspects of air pollution – REVIHAAP Project*. Bonn: WHO, 2013. www.euro.who.int/__data/assets/pdf_file/0004/193108/REVIHAAP-Final-technical-report-final-version.pdf [Accessed 2 December 2015].
- 42 Newby DE, Mannucci PM, Tell GS *et al.* Expert position paper on air pollution and cardiovascular disease. *Eur Heart J* 2015;36:83–93b.
- 43 Bhaskaran K, Hajat S, Armstrong B *et al.* The effects of hourly differences in air pollution on the risk of myocardial infarction: case crossover analysis of the MINAP database. *BMJ* 2011;343:d5531.
- 44 Shah AS, Lee KK, McAllister DA *et al.* Short term exposure to air pollution and stroke: systematic review and meta-analysis. *BMJ* 2015;350:h1295.
- 45 Stafoggia M, Cesaroni G, Peters A *et al.* Long-term exposure to ambient air pollution and incidence of cerebrovascular events: results from 11 European cohorts within the ESCAPE project. *Environ Health Perspect* 2014;122:919–25.
- 46 Cesaroni G, Forastiere F, Stafoggia M *et al.* Long term exposure to ambient air pollution and incidence of acute coronary events: prospective cohort study and meta-analysis in 11 European cohorts from the ESCAPE Project. *BMJ* 2014;348:f7412.
- 47 Langrish JP, Bosson J, Unosson J *et al.* Cardiovascular effects of particulate air pollution exposure: time course and underlying mechanisms. *J Intern Med* 2012;272:224–39.
- 48 Adar SD, Sheppard L, Vedal S *et al.* Fine particulate air pollution and the progression of carotid intima-medial thickness: a prospective cohort study from the multi-ethnic study of atherosclerosis and air pollution. *PLoS Med* 2013;10:e1001430.
- 49 Zhang J, Zhu T, Kipen H *et al.* Cardiorespiratory biomarker responses in healthy young adults to drastic air quality changes surrounding the 2008 Beijing Olympics. *Res Rep Health Eff Inst* 2013;5–174.
- 50 World Health Organization. *Effects of air pollution on children's health and development: a review of the evidence*. Bonn: WHO, 2005. www.euro.who.int/__data/assets/pdf_file/0010/74728/E86575.pdf [Accessed 10 December 2015].
- 51 Heck JE, Wu J, Lombardi C *et al.* Childhood cancer and traffic-related air pollution exposure in pregnancy and early life. *Environ Health Perspect* 2013;121:1385–91.
- 52 Ghosh JK, Heck JE, Cockburn M *et al.* Prenatal exposure to traffic-related air pollution and risk of early childhood cancers. *Am J Epidemiol* 2013;178:1233–9.
- 53 Sun XX, Zhang SS, Ma XL. No association between traffic density and risk of childhood leukemia: a meta-analysis. *Asian Pac J Cancer Prev* 2014;15:5229–32.
- 54 Pope CA 3rd, Thun MJ, Namboodiri MM *et al.* Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am J Respir Crit Care Med* 1995;151:669–74.
- 55 Dockery DW, Pope CA 3rd, Xu X *et al.* An association between air pollution and mortality in six U.S. cities. *N Engl J Med* 1993;329:1753–9.
- 56 Raaschou-Nielsen O, Andersen ZJ, Beelen R *et al.* Air pollution and lung cancer incidence in 17 European cohorts: prospective analyses from the European Study of Cohorts for Air Pollution Effects (ESCAPE). *Lancet Oncol* 2013;14:813–22.
- 57 International Agency for Research on Cancer. *Outdoor air pollution a leading environmental cause of cancer deaths*. Press release no 221. Lyon: IARC, 2013. www.iarc.fr/en/media-centre/iarcnews/pdf/pr221_E.pdf [Accessed 19 June 2015].
- 58 International Agency for Research on Cancer. *Diesel and gasoline engine exhausts and some nitroarenes (Monographs on the evaluation of carcinogenic risks to humans 2013, volume 105)*. <http://monographs.iarc.fr/ENG/Monographs/vol105/mono105.pdf> [Accessed 19 June 2015].

- 59 Health Effects Institute. *Advanced Collaborative Emissions Study (ACES): lifetime cancer and non-cancer assessment in rats exposed to new-technology diesel exhaust* (Health Effects Institute research report no 184). Boston, MA: Health Effects Institute, 2015. <http://pubs.healtheffects.org/getfile.php?u=1039> [Accessed 10 December 2015].
- 60 Gehring U, Wijga AH, Hoek G *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;doi:10.1016/S2213-2600(15)00426-9.
- 61 Guxens M, Ghassabian A, Gong T *et al.* Air pollution exposure during pregnancy and childhood autistic traits in four European population-based cohort studies: the ESCAPE project. *Environ Health Perspect* 2015; doi:10.1289/ehp.1408483.

Chapter 5: Summary

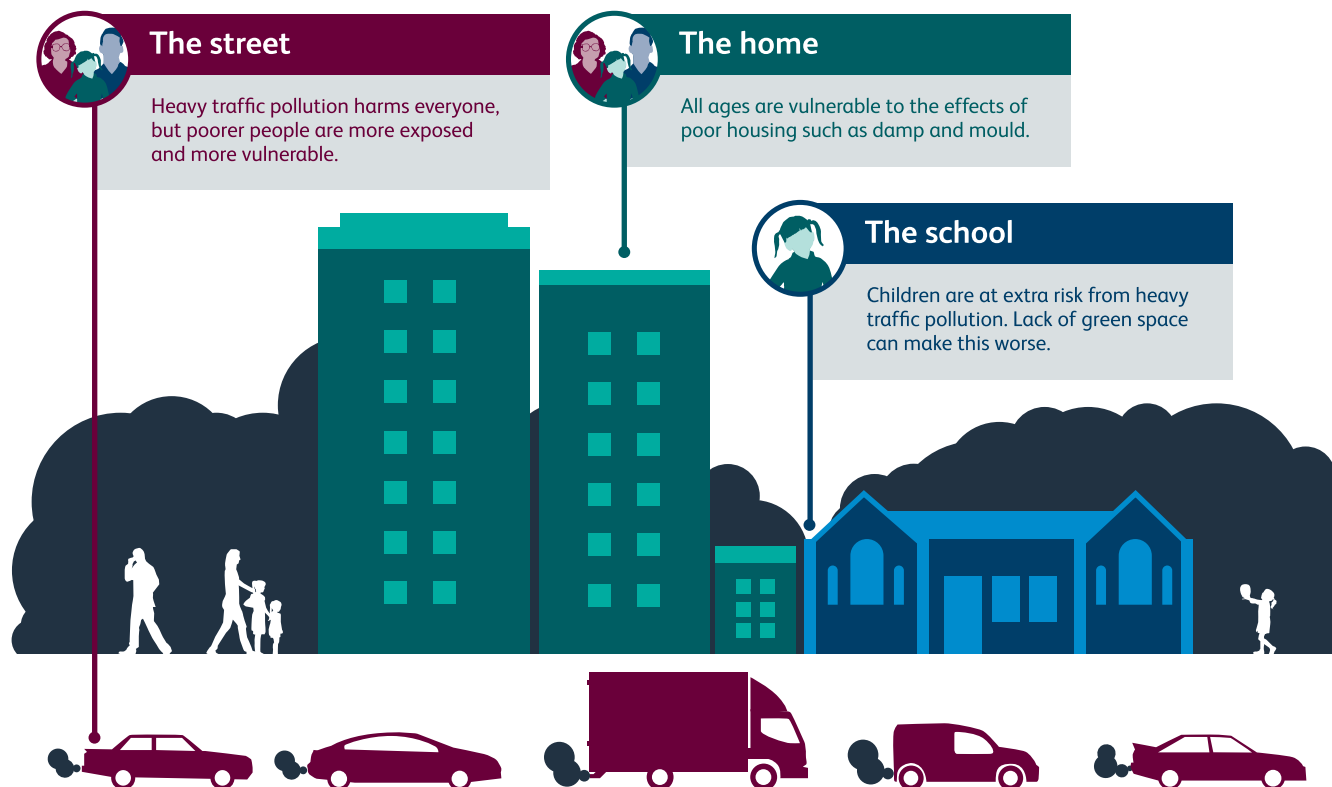
Air pollution is harmful to everyone. However, some people suffer more because they are:

- more likely to live in polluted areas
- exposed to higher levels of air pollution
- more vulnerable to health problems caused by air pollution.

Some people face all of these disadvantages.

Low income is one factor that can have such multiple effects. Poorer people are more likely to have existing medical conditions, and tend to live in areas where the outdoor and indoor environments – including the quality of the air – are not as healthy. Less access to decent housing, green spaces, jobs and healthy food all contribute to poor health. These stressful conditions may also affect the body's response to air pollution.

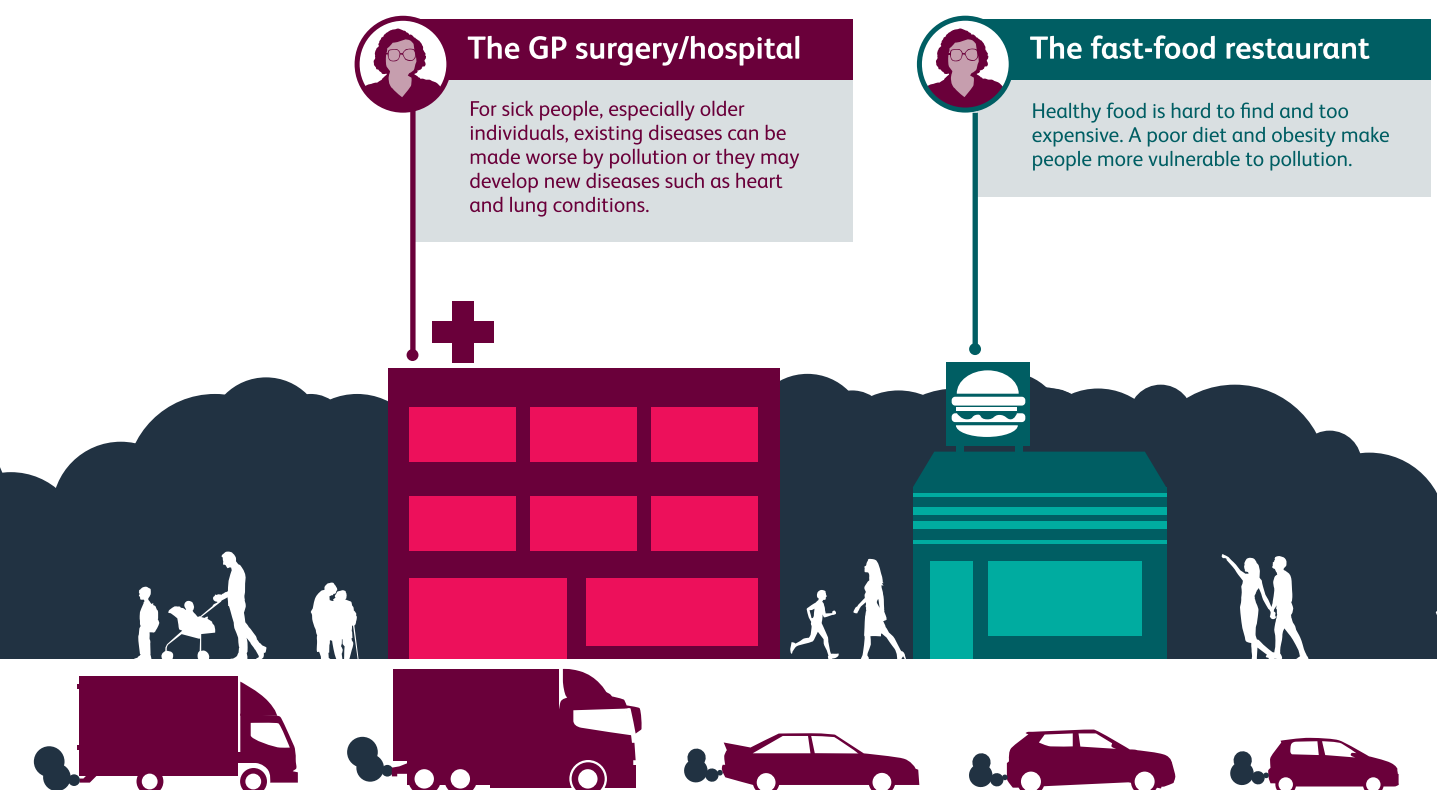
In some ways, it is a vicious circle. For example, research suggests that some chemicals in air pollution may be implicated in the development of obesity – and we also know that obese people are more sensitive to air pollution.



To make the injustice worse, poorer people often can't afford to move away and leave the problem to someone else – and they may not want to. People in low-income areas need more resources and opportunities to create a healthy local environment.

Key facts

- The body's defences against hazards like air pollution are partly controlled by our genes.
- Older people are more vulnerable to the harmful effects of air pollution.
- There is evidence that air pollution can also harm the health of children, starting from the time they are in the womb.
- Some health problems, such as heart and lung conditions, can make a person more vulnerable to harm from air pollution.
- Being overweight can also make people more vulnerable to the harmful effects of air pollution, while a diet that is rich in antioxidant nutrients (such as many vitamins), or which includes vitamin and mineral supplements, may give some protection.
- Poorer people can often find it easier to buy cheap, unhealthy foods than a healthy diet, which puts them at a significant disadvantage.
- Poorer people also tend to live in environments where they are more exposed to air pollution, for example from busy roads or in unhealthy housing.
- All of these disadvantages add up, so poorer people are at a greater risk from air pollution and its damaging health consequences.



Chapter 5: Our vulnerable groups

5.1 Concepts

As with all toxic substances, the adverse effects of air pollution vary between individuals. This imbalance reflects a complex set of intrinsic and external factors and is variously described as ‘vulnerability’, ‘susceptibility’ or ‘sensitivity’, terms which are used differently and often interchangeably.¹

Here, we use the term ‘vulnerability’ to express the broad range of determinants whereby the health impacts of pollution are unequal. These determinants include those innate to an individual, such as their age and their genetically regulated capacity to metabolise inhaled pollutants, and those that are acquired, such as the presence of respiratory or heart disease. Together, these influences are reflections of an individual’s biological ‘susceptibility’. In addition, vulnerability includes environmental, social and/or behavioural factors that determine an individual’s level of exposure and their capacity to manage it; these factors include, for example, the proximity of one’s home to an external source of pollution, co-exposures in the workplace, and access to preventive measures or healthcare. These concepts are depicted in Fig 14.

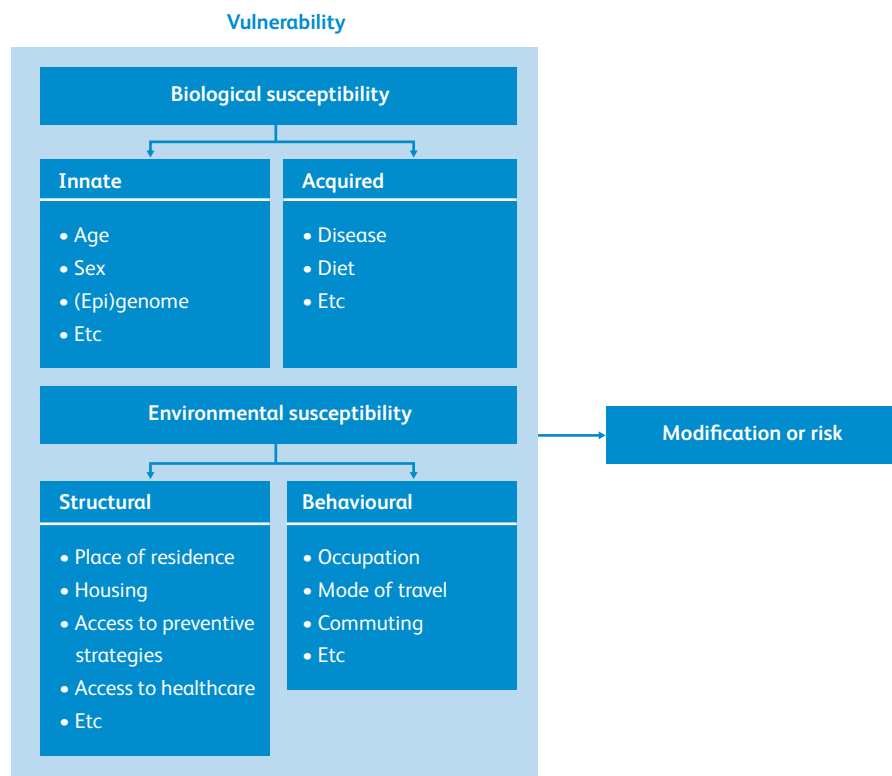


Fig 14. Concepts of vulnerability to the effects of air pollution.

The distinction between biological and environmental vulnerability, or innate and acquired susceptibility, is conceptually useful, but in truth it is somewhat artificial. For example, older individuals become more vulnerable because they have an age-related loss of antioxidant defence mechanisms in the

lung and elsewhere, because they are more likely to have developed chronic cardiorespiratory diseases, or because their place of residence and modes of travel expose them to higher levels of urban pollution.

5.2 Impacts of vulnerability

The concept of vulnerability indicates that increases in exposure to pollution may have substantial effects on a vulnerable portion of the population, even if the change in risk for the whole population is small; and, conversely, that reductions in pollution levels may lead to pronounced health benefits in population groups with the highest vulnerability.

Individuals, or groups of similar individuals, who are especially vulnerable to the effects of air pollution may:

- for any given exposure to pollution, have a higher risk of an adverse health outcome (Fig 15a)
- for any given exposure to pollution, experience a more severe adverse health outcome (Fig 15b)
- experience an adverse health outcome at a lower level ('threshold') of exposure to pollution (Fig 15c)
- be more likely to experience an above-threshold exposure to pollution (Fig 15d).

The adverse effects of air pollution measured in epidemiological studies are depicted as an average of effects across a whole population of individuals, within which there will exist varying individual states of

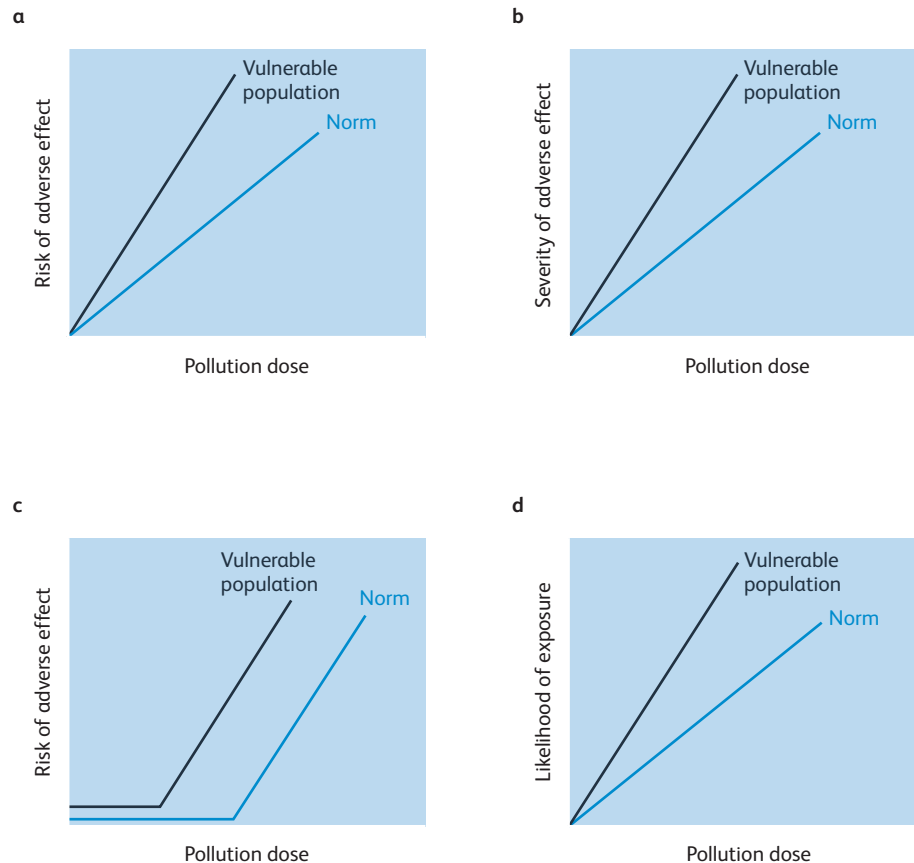


Fig 15. Different impacts of air pollution vulnerability.

vulnerability. Generally, the effect sizes (risk estimates) are small, but important because they have an impact on a large number of people. To a statistician, factors that determine vulnerability modify the risk estimate; in other words, the size of the estimate varies according to the presence or absence of a vulnerability factor. Studies that set out to examine vulnerability need to be especially large and, in general, have used only crude measures of vulnerability such as age or sex.

To complicate matters further, different factors that increase vulnerability often interact. For example, relatively socio-economically deprived communities will include a high proportion of individuals with diseases or poor diets that render them vulnerable to the effects of air pollution, and may be exposed to higher levels of pollution: a double injustice. These issues are discussed more fully below, but they beg several questions:

- Are these differences inequalities or injustices?
- Is it fair that those populations most exposed to air pollution are also more likely to have other stressors, such as poverty, poor housing, low educational attainment, obesity, long-term illnesses and higher levels of smoking?
- How much do these factors affect individual and population health?
- Which are the most important?
- Are they inevitable?
- Should we be reducing them and, if so, how do we do it?
- What options, if any, are available to deprived communities to avoid or reduce exposure and its effects?
- How much will intervention cost and who is responsible?

5.3 Biological (innate/acquired) susceptibility

5.3.1 Genetic and epigenetic influences

Human defences against external hazards are determined in part by factors that are under genetic control. For example, the capacity to neutralise inhaled toxins is regulated by enzymes in the airway, whose nature and production are governed by inherited genes. Variation in these genes explains part of the difference in how individuals respond to air pollution.

Several such genetically controlled factors have been identified (eg in asthma, polymorphism in the *TNF* (tumour necrosis factor) gene promoter and *GSTM1* (glutathione S-transferase μ 1) gene affects responsiveness to O_3 exposure), but even collectively they explain only a small part of individual variation in susceptibility. More recently, attention has moved to the 'epigenome', a term used to describe inheritable material other than the sequence of DNA (see Chapter 3). Epigenetic mechanisms include the switching on or off of genes, a process that may be fixed through life, but may also be prompted by environmental exposures including air pollutants such as PM.² These effects may vary by age. There is thus the intriguing possibility that past exposures to air pollution determine future responses, favourable or unfavourable, to the same pollutants (see Chapter 3).

5.3.2 Age

There is consistent evidence that older people are particularly vulnerable to the adverse effects of air pollution. A systematic review of studies that examined deaths in association with exposure to PM,³

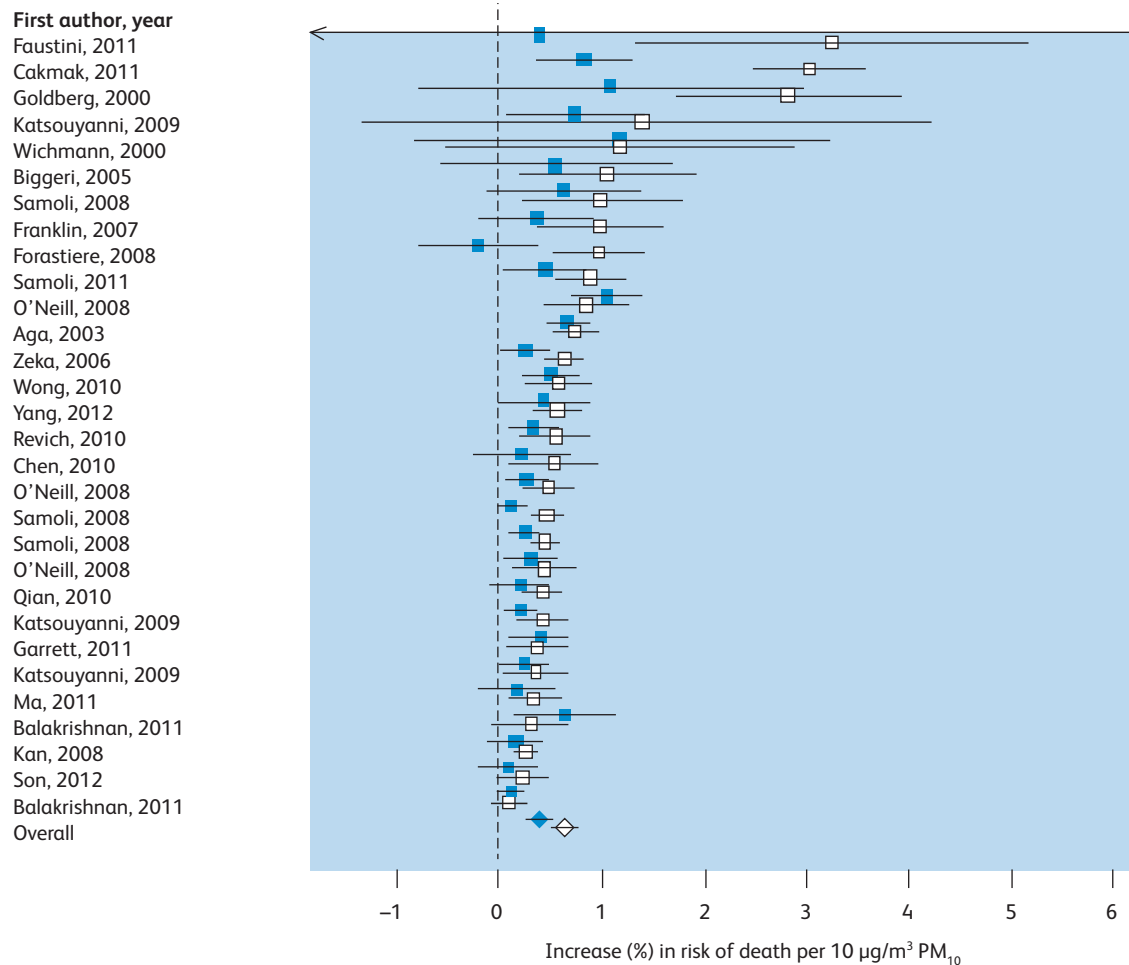


Fig 16. Meta-analysis of the association with age of increased risk of death from exposure to PM_{10} . Solid squares represent results from younger populations; open squares represent those for older populations. Each square represents a central risk estimate, and each horizontal line its 95% confidence interval. Diamonds represent overall results from the meta-analysis. Adapted from Bell *et al*,³ by permission of Oxford University Press.

for example, indicated a risk in older people of about twice that observed in younger persons (see Fig 16); similar findings are reported for hospitalisations. These comparisons are relatively crude ('older', variously categorised, versus 'not older') and probably obscure a stronger effect. The explanation(s) are likely to be several and have not been examined fully, but they will include the higher frequency of pollution-sensitive pulmonary, cardiovascular and metabolic diseases in older populations.

Few studies have explicitly compared risks in children with those in adults, but there is evidence of an adverse effect of air pollution on the health of children who spend a higher proportion of their time outdoors and are therefore at greater risk of exposure to ambient pollutants.⁴ Moreover, in 2015 nearly 25% of all schoolchildren in London were exposed to levels of air pollution that exceeded legal limits.⁵

5.3.3 Sex

The issue of sex differences in vulnerability to air pollution is complex.⁶ The evidence is inconsistent in studies of adults, although research in older adults and studies that have used estimates of exposure based on place of residence suggest that the effects of air pollution are more pronounced in women. Any differences are likely to have several explanations that include biological differences between men and women (such as in the absorption of gases through the lung or hormonal differences in chemical regulation), the higher proportion of older and vulnerable women than men, sex-related behavioural differences in factors such as smoking and occupational exposures (that may mask, in men, the effects of air pollution), or simply that estimates of exposure may be more accurate in adult women.

In infants and young children, the effects of pollution appear stronger in boys than girls, whereas in older children (>5 years old) the opposite is the case. The reasons for these differences are probably complex, reflecting sex- and age-related differences in lung function and growth, and behaviour.

5.3.4 Disease

Studies of air pollution frequently include comparisons between individuals who do or do not have pre-existing disease. The conditions that have been most often examined in this way are respiratory and cardiovascular diseases and, more recently, diabetes. These are very common in the general population, and understanding whether their presence confers a particular vulnerability is an important issue. The picture is complicated by the facts that older people often have more than one such condition (multiple comorbidities), and that air pollution itself may give rise to new disease (such as asthma) that then renders an individual more vulnerable to future pollution exposures. While it might reasonably be assumed that individuals with, say, heart disease would experience more serious effects from air pollution or effects at lower doses, the evidence that this is so is not entirely consistent.⁷ This may reflect the fact that the concomitant use of medication may offset some or all of any vulnerability conferred by disease.

Nonetheless, some conclusions can be drawn:

- Asthma is a condition of chronic airway inflammation and, consequently, of airways that are hypersensitive to inhaled irritants such as many constituents of ambient air pollution. Therefore, it is unsurprising that individuals with asthma report more symptoms from pollution than those without asthma, and may require more controller anti-inflammatory and preventer bronchodilator treatments or are more likely to have an ‘attack’ of asthma (exacerbation) when ambient outdoor air pollution levels are high than when they are not. As might be expected, those individuals with more severe asthma and those whose asthma is undertreated are especially vulnerable.
- COPD is common in older populations, causally linked to many years of tobacco smoking and/or biomass fuel burning in homes in developing countries. Patients with COPD have a diminished capacity to clear inhaled material from their lungs and may, as a result, incur a higher-than-normal ‘dose’ at any level of air pollution. In response to elevated levels of pollution, individuals with COPD experience a greater fall in lung function and a higher risk of admission to hospital than healthy persons of the same age.

Inhaled gaseous and particulate pollutants (PM) are able to enter the circulation from the lungs, thus becoming systemically bioavailable to cause adverse effects on the heart, brain and other organs. Patients who already have cardiovascular or other disease of the internal organs may be especially vulnerable to the adverse effects of pollution.

The presence of coronary artery disease increases the risk of adverse events from traffic-related pollutants and, in particular, those emitted from diesel engines. These events include myocardial infarctions (heart attacks), but also more subtle effects such as autonomic nervous system abnormalities in heart rhythm or rate, hypertension, accelerated heart failure and type 2 diabetes.

5.3.5 Obesity

English children from deprived areas are almost twice as likely to be obese than those in affluent areas.⁸ UK adult obesity is also associated with deprivation, particularly in women.⁹ There is evidence that obese people are more vulnerable than others to the effects of exposure to air pollutants, which, given the number of people involved, is of particular concern. For example, the attenuation of age-related decline in lung function associated with improved air quality is not observed in overweight or obese people, and there are reports that obesity may also exacerbate the impact of particulate exposure, including several studies suggesting that obese individuals (and/or those with metabolic syndrome) may be at greater risk of cardiovascular events due to PM exposure.¹⁰ Obesity can modify the associations between air pollution and markers of systemic inflammation, and between childhood asthma and respiratory symptoms. Recent research has suggested that obese young children may be more likely than non-obese children to develop asthma in association with exposure to PAHs.¹¹ Maternal obesity can exacerbate the risk associated with exposure to airborne PAHs on reproductive outcomes. Research has also suggested that some components of air pollution may be implicated in the development of obesity; prenatal exposure to endocrine-disrupting chemicals, for example, has been reported to increase children's risk of obesity.¹²

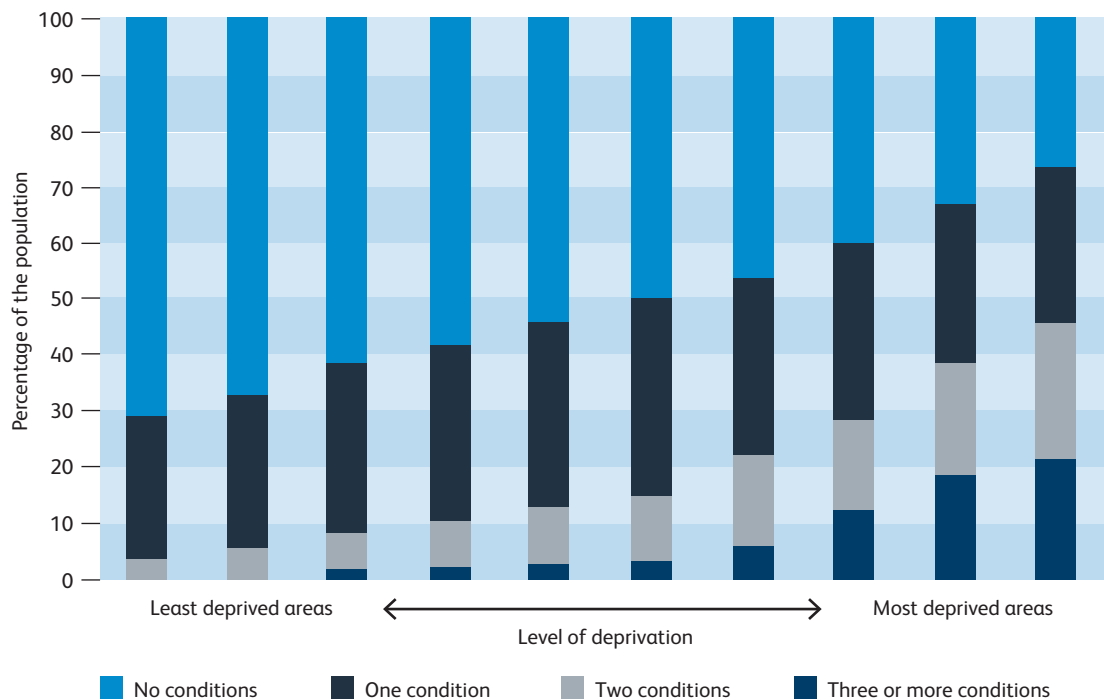
There is as yet little research on obesity and indoor air quality, although long-term occupational exposure to airborne pollutants has been reported as increasing the risk of obesity, and being overweight or obese has been reported to increase vulnerability to indoor PM_{2.5} and NO₂ in children with asthma living in urban areas.¹³

5.4 Environmental vulnerability

5.4.1 Deprivation

There are well-documented inequalities in the distribution of pollutants in the UK, although the relationship with deprivation is not straightforward. Deprived communities live in poorer-quality environments that experience higher levels of air pollution¹⁴ (Fig 17), a relationship reported in other developed nations and in the former communist states of Eastern Europe. UK populations living in air quality management areas (AQMA) by definition will encounter high air pollution – although large AQMA may include some areas that meet national air quality objectives – and are disproportionately deprived. There are exceptions where affluent populations tend to live in more trafficked areas, although the impact of congestion charging in central London on reducing levels of NO₂ and PM has been greatest in the most deprived areas.¹⁰ Levels of O₃ are generally lower in urban areas, given the chemistry of its formation as a photochemical-oxidant secondary pollutant and its removal by reacting with NO₂, which is higher in such areas.

Poor indoor air quality, including high levels of VOCs, PM and NO₂, is associated with unfit or inadequate housing standards, conditions that overwhelmingly affect those who are deprived.



Environmental conditions: river water quality, air quality, green space, habitat favourable to biodiversity, flood risk, litter, detritus, housing conditions, road accidents, regulated sites (eg landfill)

Fig 17. Proportion of the UK population living with adverse environmental conditions associated with deprivation, 2001–2006.¹⁴ Data originally from Department for Environment Food and Rural Affairs (2007): www.ltnetwork.org/SITE/UPLOAD/DOCUMENT/defra_sustain.pdf.

Stress, at both the individual and the community levels, can weaken the body’s defences against external insult and influence the internal dose of toxicants.¹⁵ While the health experience of the relatively affluent is effectively independent of geographical region of residence, the Marmot Review¹⁴ clearly shows that geography has a major impact on the health effects of deprivation in England (Fig 18). It is entirely plausible that environmental stressors such as air quality contribute to this inequity.

Deprivation has been identified as increasing susceptibility to PM in a number of separate studies. A European review reported that poorer communities were more vulnerable to the effects of PM₁₀ exposure, including morbidity and mortality.¹⁶ Other factors closely associated with deprivation, such as obesity and pre-existing cardiovascular and respiratory diseases, also increase vulnerability. Deprivation modifies the impacts of both PM on preterm birth and black smoke exposure on respiratory mortality. Effects on cardiorespiratory disease and gestational hypertension continue to be reported.

5.4.2 Diet

One of the main mechanisms through which air pollutants affect lung and heart health is by the activation of ‘oxidative stress’ in the airways and circulation. Diets that are rich in antioxidant nutrients (such as many vitamins), or which include micronutrient supplements, may help to protect against the harmful effects of air pollution, although the evidence here is based only on short-term studies.¹⁷ Poorer people have more limited access to a healthy diet,¹⁸ which may reduce the body’s defences against air

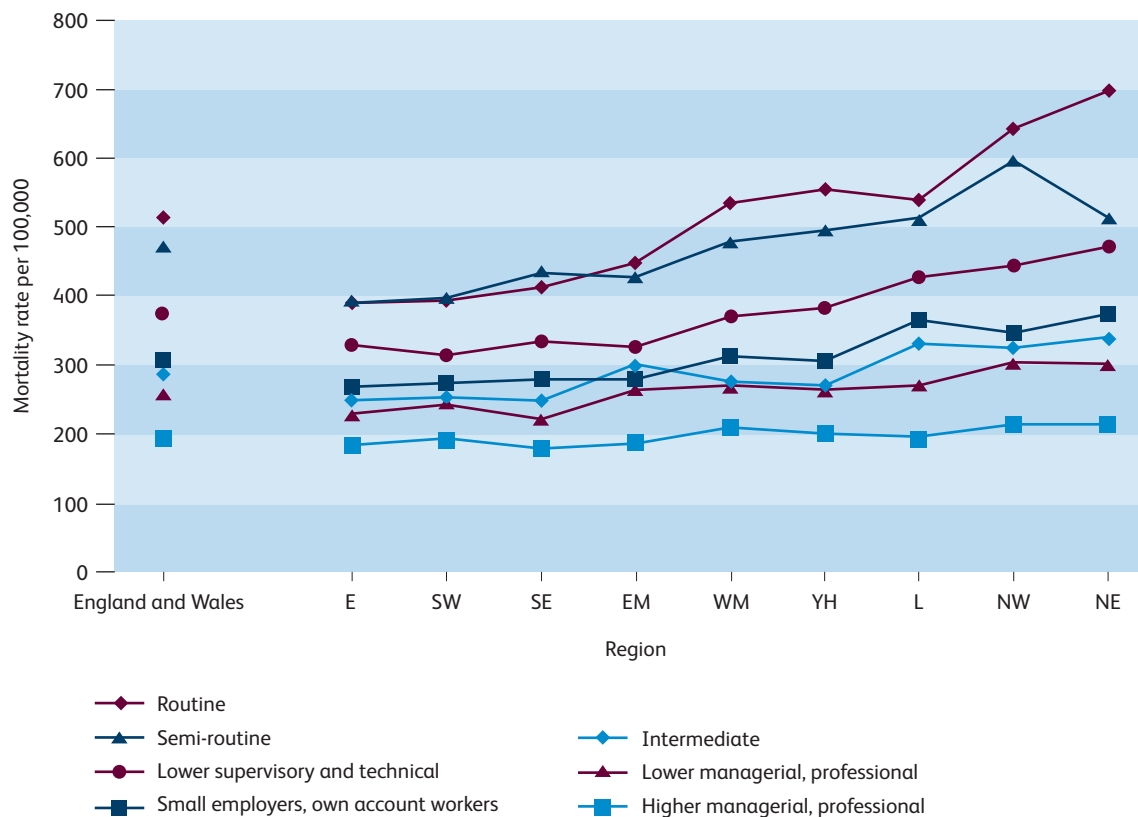


Fig 18. Age-standardised mortality rates by socio-economic classification and region, for men aged 25–64 years, 2001–2003.¹⁴ Data originally from Office for National Statistics (2008): www.ons.gov.uk/ons/rel/hsq/health-statistics-quarterly/no-40-winter-2008/regional-differences-in-male-mortality-inequalities-using-the-national-statistics-socio-economic-classification-england-and-wales-2001-03.pdf.

pollutants, as nutrition can have a buffering or synergistic effect on PM-induced cardiovascular responses and effects on the fetus.

5.5 Conclusions

There is clear evidence that, with a few exceptions, poorer people tend to live in lower-quality environments and are more exposed to air pollution. This inequality is not necessarily an injustice, provided that the levels of exposure are not hazardous and that deprivation does not increase vulnerability; however, neither of these conditions seems to apply.

While many aspects of air quality have improved over the past few decades, there are still breaches of air quality standards, particularly in urban areas, and some important air pollutants are ‘non-threshold toxins’, meaning that any level of exposure will have an impact at a population level. Deprivation seems particularly linked with increasing susceptibility to PM exposure and cardiorespiratory effects, although the precise mechanisms remain unclear. There is a general pattern that deprived populations, although not always more exposed, experience greater harmful effects of air pollution because of vulnerability factors. There is also some research suggesting that obesity may negatively modify risk and that exposure to some chemicals may be implicated in the development of metabolic syndrome. It is also clear that poorer people have other health, social and environmental stressors, such as poor-quality housing, higher

unemployment, less access to environmental resources such as green spaces, poor diet and higher levels of pre-existing disease. These stresses may modify the effect of exposure to pollutants.

These inequities affect people across the lifecourse from the prenatal stage through to old age. They are compounded by the limited opportunities available for deprived communities to improve or escape their environments.

5.6 References

- 1 Parkin RT, Balbus JM. Variations in concepts of 'susceptibility' in risk assessment. *Risk Anal* 2000;20:603–11.
- 2 Janssen BG, Byun HM, Gyselaers W *et al*. Placental mitochondrial methylation and exposure to airborne particulate matter in the early life environment: An ENVIRONAGE birth cohort study. *Epigenetics* 2015;10:536–44.
- 3 Bell ML, Zanobetti A, Dominici F. Evidence on vulnerability and susceptibility to health risks associated with short-term exposure to particulate matter: a systematic review and meta-analysis. *Am J Epidemiol* 2013;178:865–76.
- 4 Bateson TF, Schwartz JJ. Children's response to air pollutants. *Toxicol Environ Health A* 2008;71:238–43.
- 5 Howard R. *Up in the air: how to solve London's air quality crisis: part 1*. London: Policy Exchange, 2015. www.policyexchange.org.uk/publications/category/item/up-in-the-air-how-to-solve-london-s-air-quality-crisis-part-1 [Accessed 11 December 2015].
- 6 Clougherty JE. A growing role for gender analysis in air pollution epidemiology. *Cien Saude Colet* 2011;16:2221–38.
- 7 Sacks JD, Stanek LW, Luben TJ *et al*. Particulate matter-induced health effects: who is susceptible? *Environ Health Perspect* 2011;119:446–54.
- 8 National Obesity Observatory. *National Child Measurement Programme: Changes in children's body mass index between 2006/07 and 2011/12*. Oxford: National Obesity Observatory, 2013. www.noo.org.uk/uploads/doc/vid_17929_NCMP_Changes_children.pdf [Accessed 15 December 2015].
- 9 Public Health England. *Health inequalities*. National Obesity Observatory. www.noo.org.uk/NOO_about_obesity/inequalities [Accessed 28 October 2014].
- 10 World Health Organization. *Review of evidence on health aspects of air pollution – REVIHAAP Project*. Bonn: WHO, 2013. www.euro.who.int/__data/assets/pdf_file/0004/193108/REVIHAAP-Final-technical-report-final-version.pdf [Accessed 2 December 2015].
- 11 Jung KH, Perzanowski M, Rundle A *et al*. Polycyclic aromatic hydrocarbon exposure, obesity and childhood asthma in an urban cohort. *Environ Res* 2014;128:35–41.
- 12 Rundle A, Hoepner L, Hassoun A *et al*. Association of childhood obesity with maternal exposure to ambient air polycyclic aromatic hydrocarbons during pregnancy. *Am J Epidemiol* 2012;175:1163–72.
- 13 Lu KD, Breyse PN, Diette GB *et al*. Being overweight increases susceptibility to indoor pollutants among urban children with asthma. *J Allergy Clin Immunol* 2013;131:1017–23.
- 14 The Marmot Review. *Fair society, healthy lives: strategic review of health inequalities in England post-2010*. London: Marmot Review, 2010. www.instituteofhealthequity.org/projects/fair-society-healthy-lives-the-marmot-review [Accessed 11 December 2015].
- 15 Gee GC, Payne-Sturges DC. Environmental health disparities: a framework integrating psychosocial and environmental concepts. *Environ Health Perspect* 2004;112:1645–53.
- 16 Deguen S, Zmirou-Navier D. Social inequalities resulting from health risks related to ambient air quality – a European review. *Eur J Public Health* 2010;20:27–35.
- 17 Romieu I, Castro-Giner F, Kunzli N, Sunyer J. Air pollution, oxidative stress and dietary supplementation: a review. *Eur Respir J* 2008;31:179–97.
- 18 Saunders P, Saunders A, Middleton J. Living in a 'fat swamp': exposure to multiple sources of accessible, cheap, energy-dense fast foods in a deprived community. *Br J Nutr* 2015;113:1828–34.

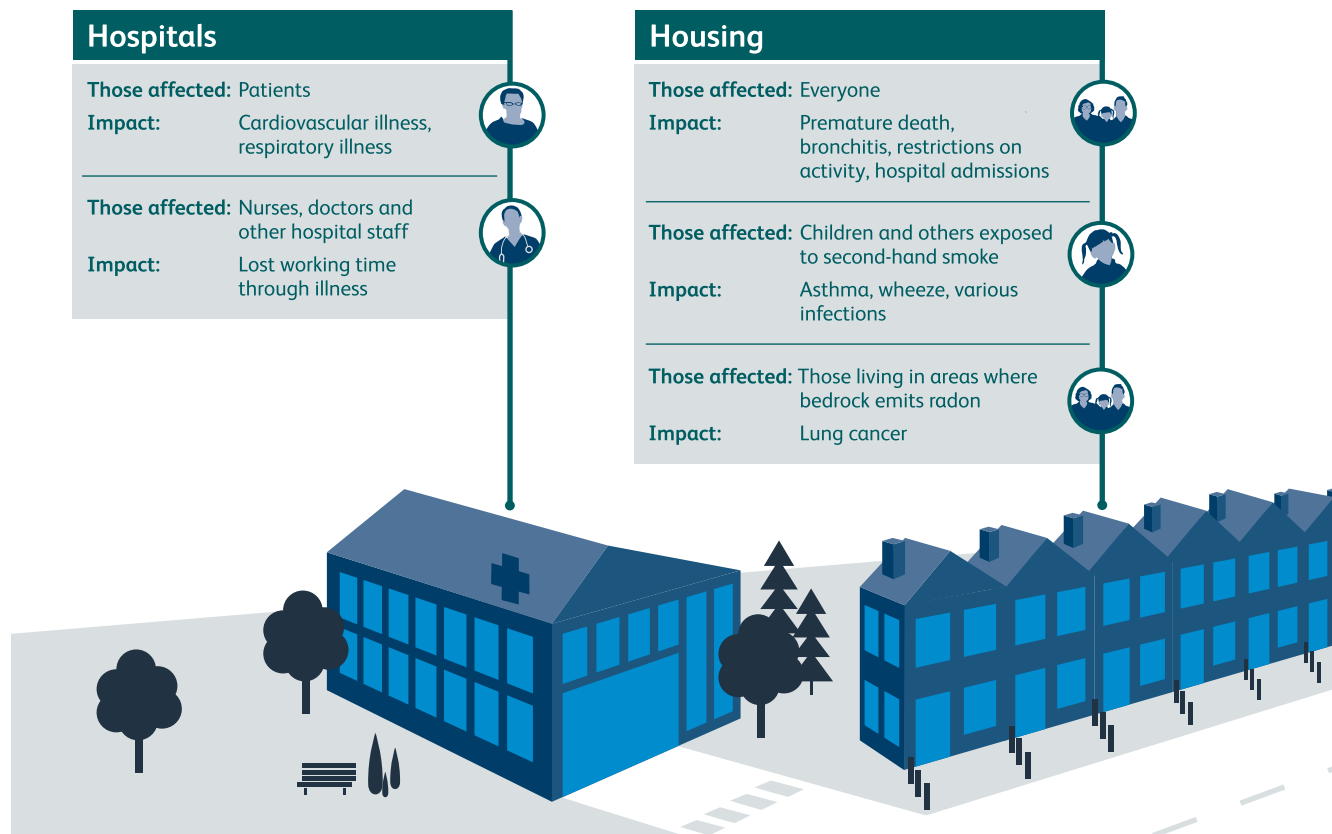
Chapter 6: Summary

Putting numbers on the harmful effects of air pollution allows policymakers to compare the costs of action with the benefits that will follow. This helps them to develop cost-effective plans, ensuring that we get the greatest benefit from investments in cleaning up air pollution.

Calculating the impact of air pollution also highlights areas where the evidence base is weak, and where further research would be most useful. For example, we have a lot of information about the impact of outdoor air pollution, but much less knowledge about indoor air pollution. We also know very little about the long-term health and economic effects of childhood illness caused by air pollution.

However, we already have clear evidence that air pollution is costing society dearly. It is forcing people to miss work and school, and to change their lifestyles to avoid exposure on high-pollution days. Exposure to fine particles, nitrogen dioxide and other pollutants in the air we breathe is causing pain and suffering – and additional healthcare costs – through increased illness. It has been estimated to cause 44,750–52,500 early deaths every year. Emerging evidence suggests a slightly lower figure, and therefore we have opted for a best estimate of around 40,000 attributable deaths per year with a range of $\pm 25\%$.

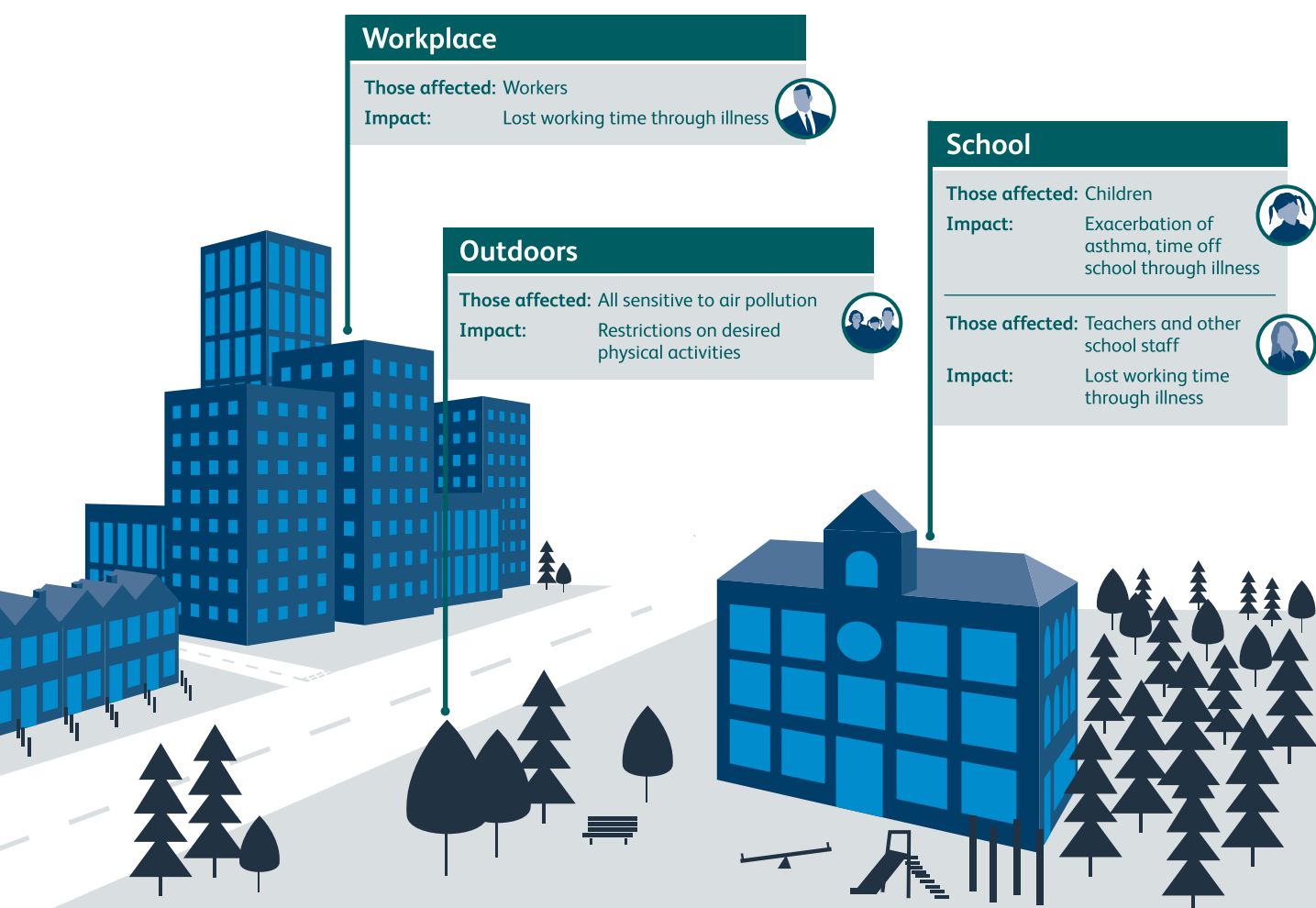
The European Commission, the US Environmental Protection Agency and various other bodies have concluded that further measures to control air pollution are economically justified. The costs of cutting emissions are outweighed by the benefits of action, because it would reduce pain and suffering, lower healthcare costs and get people back to work.



Countries across Europe, including the UK, have already paid a high price for failing to act more strongly earlier. The longer action is delayed, the more this damage will mount up.

Key facts

- Exposure to particulates and nitrogen dioxide is linked to around 40,000 early deaths in the UK each year.
- Air pollution is also linked to illness. For an individual, this can range from a minor illness that requires some medication, to a very serious situation – such as admission to hospital caused by a stroke, heart attack, lung condition or a range of other diseases.
- The health problems caused by air pollution impose many costs on society, through reduced productivity and an added burden on the health service.
- Overall, the estimated cost to individuals and society is more than £20 billion annually for the UK.
- According to 2010 estimates, the economic impact of exposure to air pollution across the European Union is more than €240 billion each year.
- We have less evidence about the costs of indoor air pollution. However, second-hand tobacco smoke and radon gas also cause deaths, and impose a significant burden on the NHS.



Chapter 6: The heavy cost of air pollution

6.1 Introduction

The development of policy for reducing the risks of air pollution is informed through the quantification and monetisation of effects. Quantification highlights both the magnitude and the variety of impacts. The monetisation process typically accounts for healthcare costs, lost productivity, and ‘welfare’ or ‘utility’, placing a value on good health *per se*. Monetisation permits the costs of action to reduce pollutant exposure to be compared with the benefits, to ensure that there is a sound economic basis for action.

In this chapter, particular attention is given to the effects of outdoor (ambient) air pollution, as this has been the focus of a large number of health impact and economic assessments. Reference is also made to some work on indoor air quality, but this focuses on specific examples (second-hand smoke and radon) and is significantly less complete.

Quantification is limited to effects for which there is considered to be sufficient evidence available, not only for acceptance of effect, but also for quantification. Important elements of the emerging literature on childhood illness, in particular, have yet to be taken through to quantification.

6.2 Outdoor air pollution

6.2.1 Methods

All of the major air pollution health impact assessments follow the same broad method, referred to as the impact pathway approach, which was first elaborated in detail in the EC–US Fuel Cycles Study of the early 1990s, a collaborative exercise between the European Commission and the US Department of Energy.¹ This approach tracks air pollution from its source in a logical, sequential manner, to describe the exposure of the population and then the impacts on the population either as a whole or for specific groups (eg those over the age of 65 years, or children with asthma).

The development of methods by various bodies, including the UK’s Committee on the Medical Effects of Air Pollutants (COMEAP),^{2,3} the US Environmental Protection Agency (USEPA)^{4,5} and the WHO,⁶ has required a number of decisions to be made, of which the following are particularly important:

- Analysis is based on the use of data from epidemiological studies in preference to toxicology, in order to capture effects on the whole population, and a full range of effects from those that range from mild to severe for individuals.
- Various reviews of evidence from the western hemisphere have concluded that analysis should focus on exposure to fine particles (PM_{2.5}) and O₃. There is growing evidence for inclusion of additional impacts of exposure to NO₂ and these effects are now being integrated with damage assessments.
- PM_{2.5} is a complex mix of primary and secondary particles, varying in their physical and chemical characteristics. However, available evidence does not support a systematic distinction between different particle types within the PM_{2.5} size range.
- Epidemiological research of fine particles has found no evidence for a threshold at the level of the whole population.⁷ For O₃, expert opinion⁸ has concluded that, while there is no firm evidence

for a threshold for health effects, there is more convincing evidence for effects above a certain level (35 ppb) than below, and this should be adopted as a cut-off point for analysis. The question of a threshold for NO₂ assessment is currently still under review, although a preliminary opinion of COMEAP in the UK is that there is unlikely to be a threshold at the level of the whole population (bearing in mind that this includes people in a variety of health states).⁹

- The most-studied effects, on mortality and hospital admissions linked to short-term exposure to fine particles, capture only a small part of the range of the total health effects reported for air pollution. For policy evaluation, this makes it desirable to include other endpoints, but the question arises of how far one should proceed, given increasing uncertainty as one pursues endpoints for which the evidence base is limited. Variable conclusions on this point have been reached by different bodies, noting here that analysis for the European Commission and USEPA has tended to quantify a large number of endpoints, while analysis for the UK government has so far considered fewer.

It is notable that the major quantification studies in the UK, European Union and USA have all come to the same conclusions on several of the most important of these questions, specifically that:

- 1 PM has significant effects on health
- 2 the mortality effects of long-term exposure to PM should be quantified without threshold and without distinguishing between different types of particle
- 3 these mortality impacts should be quantified using a response function in the order of a 6% change in impact per 10 µg/m³ PM_{2.5}.

6.2.2 Analysis of impacts in the UK

COMEAP² provides a detailed account of the quantification of the mortality burden of exposure to PM_{2.5} in the UK. Results are shown in Table 1, expressed in terms of deaths and the impact on longevity.

The COMEAP report considers the meaning of the estimate of the number of deaths shown in Table 1, noting that effects are principally from cardiovascular disease, which has multiple established and likely causes at the population level, and almost certainly has a complex mixture of factors affecting initiation and progression at the individual level. On this basis, the reported number of deaths should be considered as more of a statistical construct, with air pollution playing some role in bringing forward the deaths of a larger number of people than the 28,861 shown in Table 1.

Table 1. COMEAP results for effects of outdoor PM_{2.5} exposure on mortality for the UK²

Measure of mortality	Impact
Number of attributable deaths	28,861
Attributable deaths per 100,000 aged over 30 years	75
Burden on total survival (life-years lost)	340,000
Difference in life expectancy for the 2008 cohort (days)	
Females	194
Males	182

For some time, COMEAP has also made recommendations on quantification of the effects of O₃ on mortality, and of NO₂ and SO₂ in addition to PM and O₃ on hospital admission rates.² These recommendations have been applied by the Interdepartmental Group on Costs and Benefits in evaluating policies that influence emissions of air pollution in the UK. However, the ensuing results are dominated by the mortality burden of fine particles. COMEAP is currently undertaking a number of activities to investigate extension of the range of effects for which quantification may be recommended for UK policy analysis. In related work, the function for PM-related mortality has been applied to generate a list of attributable deaths and life-years lost for each local authority in the UK.¹⁰

Preliminary estimates of NO₂ impacts on mortality were published by the UK's Department for Environment Food and Rural Affairs (Defra) in September 2015.¹¹ These indicate that NO₂ is linked to 23,500 deaths annually in the UK (with a range of 9,500–38,000), based on pollutant levels in 2013. It is noted that this figure will include some overlap with the impact quantified against exposure to PM_{2.5}.

When quantifying the total impact associated with exposure to both NO₂ and PM_{2.5}, it is therefore necessary to account for this overlap in the response functions. Defra estimates that the annual equivalent number of attributable deaths associated with the two pollutants combined is 44,750–52,500, with an associated annual social cost of £25.3 billion – £29.7 billion. However, a subsequent paper issued by COMEAP in December 2015 indicates that the level of overlap in estimates between pollutants may be greater than originally thought.¹² On this basis, while recognising that COMEAP's research on this issue is continuing, this report adopts a combined estimate of effect of around 40,000 deaths annually with an associated annual social cost of £22.6 billion (both with a range for a central estimate of ±25%).

6.2.3 Global burden of disease

With respect to air pollution, the Global Burden of Disease (GBD) 2010 study¹³ focused on quantification of mortality impacts from exposure to PM_{2.5} and O₃. Overall, results indicate that a total of 7 million deaths in 2010 across the world were attributable to indoor and outdoor air pollution, making air pollution one of the most important risk factors globally. Results for air pollutant impacts for the UK, expressed as both disability-adjusted life-years (DALYs) and deaths (GBD did not include morbidity), are shown in Table 2.

Table 2. GBD results for outdoor air pollution for the UK¹³

	DALYs	Deaths	%
Ambient PM pollution			
Cerebrovascular disease	73,061	5,448	20 %
COPD	27,558	1,681	8 %
Ischaemic heart disease	220,643	13,907	61 %
Lower respiratory infections	815	9	0 %
Trachea, bronchus and lung cancers	38,623	2,328	11 %
Total (all causes)	360,700	23,373	100 %
Ozone			
Total (all COPD)	8,865	772	100 %

It will be noted that the estimate for PM_{2.5} by COMEAP, presented above, is higher than the GBD estimate by about 20%. The COMEAP estimate is preferred here, because it is based on much finer-scale modelling. The two sets of results are, however, sufficiently close to provide a good level of corroboration.

The GBD analysis addresses household air pollution in relation to emissions generated from solid fuel use, and particularly from crude, poorly ventilated stoves used in the developing world. Results for the UK for this source are negligible. There are further indoor air quality issues considered in the GBD work, some of which are highlighted towards the end of this chapter.

6.2.4 USEPA analyses

USEPA has quantified health benefits of the US Clean Air Act of 1970 and subsequent legislation, including quantification of the costs and benefits of the 1990 Clean Air Act Amendments.⁴ Estimated health impacts and associated economic benefits for the USA are shown in Table 3.

Like most other analysis, the US work is focused on impacts related to PM and O₃ exposure, of which the PM effects are predominant. While the economic assessment is driven largely by mortality, the results showing change in incidence demonstrate a substantial burden on a wide range of morbidity endpoints.

The costs of the US Clean Air Act Amendments are large, with an estimated net present value of \$380 billion from 1990 to 2020. However, the overall benefits to society are considerably greater, estimated

Table 3. USEPA results showing the estimated benefits of the US Clean Air Act Amendments for the USA in 2020.⁴ Values are expressed in \$million, price year 2006

Health effect	Pollutant	Incidence	Value (\$million)
Mortality, attributed deaths*	PM, O ₃	230,000	1,800,000
Chronic bronchitis	PM	75,000	36,000
Non-fatal myocardial infarction	PM	200,000	21,000
Respiratory hospital admissions	PM, O ₃	66,000	1,100
Cardiovascular hospital admissions	PM	69,000	2,000
Respiratory emergency room visits	PM, O ₃	120,000	44
Acute bronchitis	PM	180,000	94
Lower respiratory symptom days	PM	2,300,000	42
Upper respiratory symptom days	PM	3,300,000	60
Asthma exacerbation days	PM	6,700,000	130
Minor restricted activity days	PM, O ₃	140,000,000	6,700
Work loss days	PM	19,000,000	2,700
School loss days	O ₃	8,600,000	480
Outdoor worker productivity	O ₃	Not applicable	170

*The mortality estimate shown includes effects on infants as well as adults.
Source: USEPA,⁴ Tables 5–7.

between a factor of 4 and 92 higher (the range accounting for variability in the response functions and valuations).

6.2.5 European Commission analyses

The European Commission has used cost–benefit analysis in the development of policy on air pollution since the mid-1990s. Results from the latest analysis^{14,15} are shown in Table 4 as health impact assessment and Table 5 as economic assessment, including results for the European Union as a whole, and separately for the UK. Again, the estimate of mortality linked to PM_{2.5} exposure is broadly similar to that generated by COMEAP. Like the approach used by USEPA, the EC analyses consider a broad range of effects on morbidity, the demonstration of which provides support for there being a significant burden of air pollution on mortality. The economic assessment considers several dimensions of value: healthcare costs, lost productivity, pain and suffering, and aversion to risk. It is notable that the economic analysis indicates that the most-studied morbidity endpoint from the epidemiology literature, hospital admissions, represents only a small part of the overall morbidity impact.

Table 4. Estimated impacts in 2010 in the European Union and the UK¹⁵

O₃ effects	Units	EU	UK
Mortality	Premature deaths	23,507	1,371
Respiratory hospital admissions	Cases	19,117	1,368
Cardiovascular hospital admissions	Cases	86,279	2,678
Minor restricted activity days	Days	108,845,140	6,662,683
PM_{2.5} effects			
Mortality	Life-years lost	4,030,653*	327,769*
Mortality	Premature deaths	379,420*	30,018*
Infant mortality	Premature deaths	777	70
Chronic bronchitis	Cases	316,685	25,582
Bronchitis in children aged 6–12	Cases	1,068,990	102,386
Respiratory hospital admissions	Cases	142,243	11,652
Cardiac hospital admissions	Cases	108,989	4,523
Restricted activity days	Days	436,351,761	40,809,466
Asthma symptom days in children	Days	11,290,673	1,171,559
Lost working days	Days	121,378,612	6,097,215
NO₂ effects			
Chronic mortality	Life-years lost	NQ [†]	NQ [†]
Bronchitis in children	Cases	NQ [†]	NQ [†]
Respiratory hospital admissions	Cases	NQ [†]	NQ [†]

*Alternative estimates of the same impact (effect of long-term exposure to particles on mortality).

[†]NQ: recommended for quantification by the WHO, but not quantified in the European analysis.

Table 5. Monetised estimates of impacts in 2010 in the European Union and UK, €million/year¹⁵

Damage, € million/year	EU	UK
Ozone effects		
Mortality (valued as loss of life expectancy)	1,043–2,785	61–146
Hospital admissions	180	7
Minor restricted activity days	3,516	215
PM_{2.5} effects		
Mortality (valued against life expectancy)	179,000–430,000*	14,600–34,600*
Mortality (valued against deaths)	318,000–648,000*	25,400–51,500*
Infant mortality	977–1,990	88–227
Chronic bronchitis	13,057	1,055
Bronchitis in children aged 6–12	484	46
Hospital admissions	429	28
Restricted activity days	30,880	2,888
Asthma symptom days in children	365	38
Lost working days	12,138	610
Totals	243,000–712,000	20,000–56,200

*Alternative estimates of the same impact (effect of long-term exposure to particles on mortality).

The results of this analysis have been used by the European Commission to identify the socially optimal target for its Clean Air Policy Package (CAPP) released in December 2013. Technical measures for reducing emissions were identified and brought together in an abatement cost curve, showing how the costs of abatement per unit of emission increase as emissions are reduced further. Health benefits were overlaid onto this graph and indicated that a target of around 75% of the abatement potential is economically justifiable. Results reveal a substantial surplus of benefit to society over cost for the CAPP, although the policy package is only the latest in a long series of actions to improve air quality going back 60 years or more. This shows that the failure to act earlier has generated substantial costs of inaction for public health, much of which could have been avoided.

A report for the European Environment Agency¹⁶ applies the approaches developed for the European Commission at the level of all the industrial facilities that are required to report emissions to the European Pollutant Release and Transfer Register (E-PRTR). A total in excess of 14,000 industrial plants have been considered, generating estimates of total burden and economic equivalent, and highlighting those plants considered to cause the highest levels of damage. It is notable that 50% of the damage costs were linked to emissions from only 147 facilities, 1% of the total number of industrial plants assessed (Fig 19). With its focus on the impacts of industry, this analysis excludes assessment of other sources such as traffic, but does highlight the type of plant most likely to generate harm and variation in applied emission standards, enabling emission controls to be targeted on the most damaging facilities.

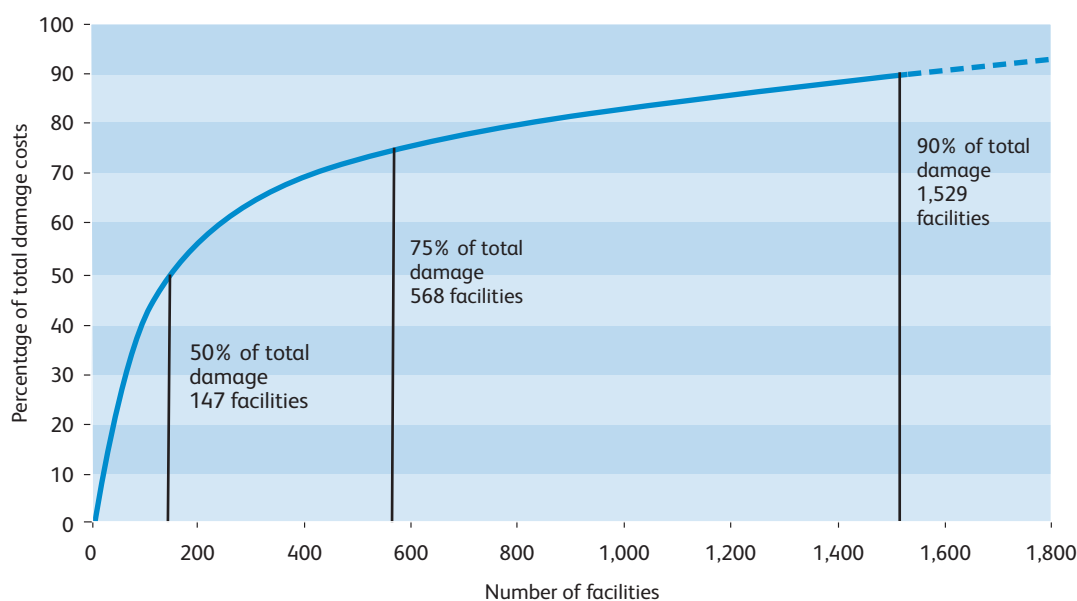


Fig 19. Cumulative distribution of the estimated damage costs associated with emissions of selected pollutants from facilities reporting emissions to the E-PRTR.¹⁶

6.2.6 Overview of estimates of impacts of outdoor air pollutants

A number of bodies and groups around the world have reviewed the evidence for health impacts of outdoor air pollutants. While there are differences in the extent to which effects are quantified by each group, and in the precise response functions selected, there is also much common ground. Overall:

- each group has recognised that the effects of air pollutants on health are real
- no group considers there to be evidence to support adoption of a threshold for impacts of PM
- all groups indicate a substantial mortality burden associated with PM in particular, for the UK equivalent to around 29,000 attributable deaths.

The figure for attributable early deaths in the UK increases to up to 50,000 and probably around 40,000 when the impacts of NO₂ exposure are brought into the analysis.

Analyses of morbidity impacts for USEPA and the European Commission indicate a variety of effects, from hospital admissions to heart failure and chronic bronchitis, down to effects that are minor at the level of the individual (eg days of restricted activity). When taken together, this information on morbidity provides support for the large mortality burden. Analysis for the UK government has taken a more conservative approach to morbidity quantification, reflecting the limited evidence base from which some morbidity endpoints can be quantified and indicating the need for further research on the less-studied effects.

Results of these analyses have been used to inform the development of policies to reduce pollutant emissions. That cited above for the European Commission and USEPA demonstrates major societal benefits for continued action on air pollution, well in excess of cost.

6.3 Quantitative estimates of impacts of indoor air pollution for the UK

The quantification of impacts of indoor air pollution has received far less attention than that for outdoor air. The following examples, for radon and second-hand smoke, provide national estimates demonstrating that the effects of indoor air pollution are far from trivial. However, they do not paint a complete picture of the problem and further quantification work is urgently needed to highlight the most important indoor sources and associated risk factors.

6.3.1 Radon

The Health Protection Agency (now part of Public Health England) has provided a review on radon in relation to public health.¹⁷ The burden of disease from radon in the UK is quantified at about 1,100 deaths from lung cancer annually, equivalent to just over 1 in 500 from all causes. There is a close link between the risks of radon exposure and smoking, although over 40% of deaths caused jointly by radon and smoking occur in people who have already given up smoking.

Analysis under the GBD Project suggests a higher figure of 2,320 deaths per year from radon exposure in the UK,¹⁸ along with an estimated 38,500 DALYs (Fig 20). The results generated by the Health Protection Agency are likely to be more reliable, as this analysis should take better account of conditions within the UK. However, the GBD results suggest that the estimate of 1,100 deaths provided by the Health Protection Agency may be an underestimate, and provides a useful insight into the development of disease with age, with effects starting in some people as young as their mid-30s.

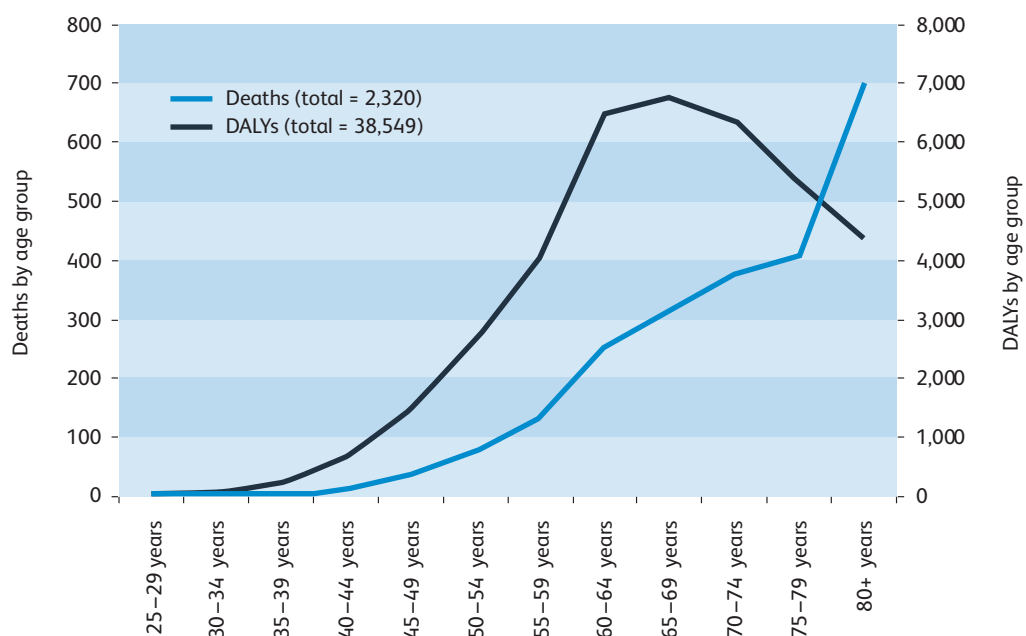


Fig 20. Estimate of deaths and DALYs associated with exposure to radon in the UK in 2010, from the GBD Project.¹⁸

6.3.2 Second-hand smoke

It has been estimated that second-hand smoke, or 'passive smoking', accounted for 600 deaths in the UK

in 2003 for those exposed in the workplace, and for 10,700 for those exposed at home (2,700 of whom were aged under 65 years, and 8,000 over 65 years).¹⁹

Analysis by the RCP²⁰ focuses on the effects of second-hand smoke on children, highlighting links to SIDS, lower respiratory infection, wheeze and asthma, middle ear infection and meningitis. Results are summarised in Table 6, with estimated health costs in Table 7, totalling £22 million.

The healthcare costs presented in Table 7 are only a partial estimate of economic cost, as they omit welfare costs (pain and suffering), impacts on productivity through parents needing to take time off work, etc. By focusing on the impacts on children they also, of course, omit effects of passive smoking on the adult population.

As for radon, the GBD Project¹⁸ provides an estimate of deaths and DALYs associated with exposure to second-hand smoke (Fig 21), demonstrating the progress of disease with age. Effects are noted in the very young and for adults from their mid-20s. Total results are broadly similar to the GBD estimates for

Table 6. Disease in UK children associated with exposure to second-hand smoke²⁰

Effect	Age group in years	Cases	Consultations	Hospital admissions
Lower respiratory infections	≤2	20,500	26,000	3,361
Middle ear infections	0–16*	121,400	160,200	2,517*
Wheeze	≤2	7,200	10,300	938
Asthma	3–4	1,700	7,600	236
Asthma	5–16*	13,700	99,000	1,211*
Meningitis	0–16*	600	800	231*
Total		165,100	303,900	8,494

*Results for hospital admissions go up to age 14, not 16.

Table 7. Healthcare costs for children in the UK associated with exposure to second-hand smoke (2007 prices, £million)²⁰

Effect	Age group in years	Consultations	Hospital admissions
Lower respiratory infections	≤2	0.78	5.08
Middle ear infections	0–16*	4.81	5.06*
Wheeze	≤2	0.31	1.12
Asthma	3–4	0.23	0.28
Asthma	5–16*	2.97	1.46*
Meningitis	0–16*	0.02	0.60*
Total		9.12	13.60

*Results for hospital admissions go up to age 14, not 16.

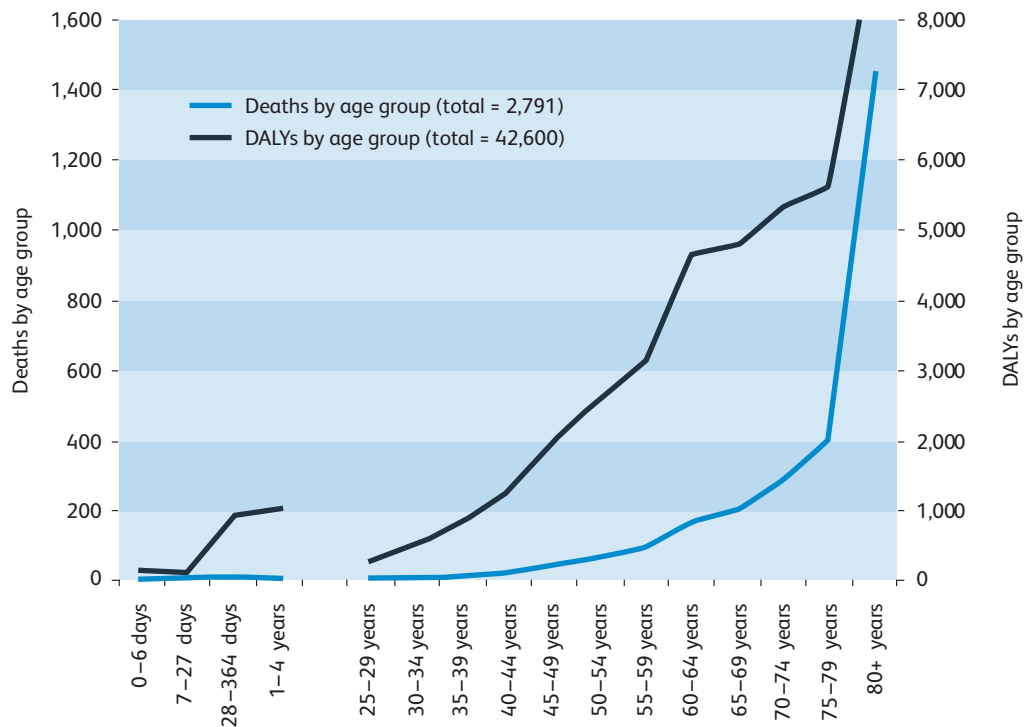


Fig 21. Estimate of deaths and DALYs associated with exposure to second-hand smoke in the UK in 2010, from the GBD Project.¹⁸

radon, although there are differences in the progression of effects with age. GBD also provides information on the illnesses linked to exposure to second-hand smoke (Table 8).

The 2,800 deaths estimated by the GBD study for the UK in 2010¹⁸ are clearly much lower than the estimate of around 11,000 deaths made previously for 2003.¹⁹ Reduced prevalence of smoking and legislation against smoking indoors will have had some effect over this period, although the difference is likely to be linked more to the methods used than to trends over the period. Further detailed analysis for the UK specifically would clearly be beneficial in targeting future policies.

Table 8. Breakdown of DALYs and deaths from second-hand smoke exposure in the UK in 2010 by type of disease¹⁸

	DALYs	Deaths
Haemorrhagic and other non-ischaemic stroke	4,493	264
Ischaemic heart disease	27,938	1,844
Ischaemic stroke	5,402	513
Lower respiratory infections	1,687	18
Middle ear infection	510	0
Trachea, bronchus and lung cancers	2,569	152
Total (all causes)	42,599	2,791

6.4 Conclusions

Despite actions undertaken over many decades, outdoor air pollution remains a major risk to health in the UK and other countries. These risks generate a burden to both mortality and morbidity, with a general consensus around the COMEAP estimate of 29,000 attributable deaths brought forward because of exposure to ambient PM_{2.5} air pollution each year in the UK. Reported morbidity effects vary greatly in severity, from impacts that are seriously debilitating, such as COPD or cardiac events, to those that are less serious individually (eg 'restricted activity days' or 'symptom days'), but which affect a large number of people. The costs of these impacts, for welfare, healthcare and productivity, are truly substantial. Quantification of effects associated with exposure to NO₂ remains under discussion, although current indications are that PM_{2.5} and NO₂ combined will bring forward around 40,000 attributable deaths annually.

Over time, it is to be expected that the costs of controlling air pollution will rise, as the cheapest measures, including efficiency improvements that save money by reducing energy costs, are fully implemented. At some point, then, it is to be expected that the costs of reducing emissions will exceed the benefits gained. However, analyses for the European Commission, USEPA and others suggest that sufficient inexpensive measures remain available to justify continued action. In part, this reflects new understanding of the sources of pollution, with sources that have in the past attracted little attention now being considered important enough to be taken into account. A good example concerns emissions of ammonia from agriculture, which are linked to the formation of secondary sulphate and nitrate particles, an important part of the mixture of pollutants that together form PM.

Most measures for reducing emissions of greenhouse gases (eg CO₂) also have benefits for reducing emissions of local and regional air pollutants. The 'co-benefits' so generated are substantial and, in some cases, sufficient on their own to justify the abatement measures, irrespective of resulting climate benefits. This results from the two types of pollutant sharing a major common source, the combustion of fossil fuels. In contrast, some measures for control of greenhouse gases may have a negative impact on air quality. Combustion of biomass fuel, for example, which is commonplace in middle- and lower-income countries, has the potential to increase emissions of fine particles, especially where the biomass is burned indoors for domestic heating and cooking.

From this limited review of the health effects of indoor pollutants, it has been shown that they cause, at a minimum, several thousands of deaths per year in the UK, and are associated with healthcare costs in the order of tens of millions of pounds. A more systematic approach to the quantification of the effects of indoor air pollution would be beneficial, not least as this is where people in the UK spend most of their time.

6.5 References

- 1 ExternE (1995) *Volume 2: methodology. Method for estimation of physical impacts and monetary valuation for priority impact pathways*. ExternE: Externalities of Energy. www.externe.info/externe_d7?q=node/37 [Accessed 15 December 2015].
- 2 Committee on the Medical Effects of Air Pollutants. *The mortality effects of long-term exposure to particulate air pollution in the United Kingdom*. London: Health Protection Agency, 2010. www.gov.uk/government/publications/comeap-mortality-effects-of-long-term-exposure-to-particulate-air-pollution-in-the-uk [Accessed 30 November 2015].
- 3 Committee on the Medical Effects of Air Pollutants. *Statement on the evidence for differential health effects of particulate matter according to source or components*. London: Health Protection Agency, 2015. www.gov.uk/government/publications/particulate-air-pollution-health-effects-of-exposure/ [Accessed 2 April 2015].

- 4 USEPA. *Prospective study: the benefits and costs of the Clean Air Act, 1990 to 2020*. United States Environmental Protection Agency, 2011. www.epa.gov/clean-air-act-overview/benefits-and-costs-clean-air-act-1990-2020-second-prospective-study [Accessed 3 April 2015].
- 5 USEPA. *Manual and appendices for BenMAP 4.0*. United States Environmental Protection Agency, 2012.
- 6 World Health Organization. *HRAPIE: Health risks of air pollution in Europe – HRAPIE project. Recommendations for concentration–response functions for cost–benefit analysis of particulate matter, ozone and nitrogen dioxide*. Bonn: WHO, 2013. www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications/2013/health-risks-of-air-pollution-in-europe-hrapie-project-recommendations-for-concentrationresponse-functions-for-costbenefit-analysis-of-particulate-matter,-ozone-and-nitrogen-dioxide/ [Accessed 15 December 2015].
- 7 Crouse DL, Peters PA, van Donkelaar A *et al*. Risk of nonaccidental and cardiovascular mortality in relation to long-term exposure to low concentrations of fine particulate matter: a Canadian national-level cohort study. *Environ Health Perspect* 2012;120:708–14.
- 8 World Health Organization. *Review of evidence on health aspects of air pollution – REVIHAAP Project*. Bonn: WHO, 2013. www.euro.who.int/__data/assets/pdf_file/0004/193108/REVIHAAP-Final-technical-report-final-version.pdf/ [Accessed 2 December 2015].
- 9 Committee on the Medical Effects of Air Pollutants. *Working group on the quantification of mortality associated with long-term average concentrations of nitrogen dioxide (NO₂): Note of the meeting held on Monday 6 July 2015*. London: Committee on the Medical Effects of Air Pollutants, 2015. www.gov.uk/government/publications/nitrogen-dioxide-interim-view-on-long-term-average-concentrations-and-mortality [Accessed 15 December 2015].
- 10 Gowers AM, Miller BG, Stedman JR. *Estimating local mortality burdens associated with particulate air pollution* (report no PHE-CRCE-010). London: Public Health England, 2014. www.gov.uk/government/uploads/system/uploads/attachment_data/file/332854/PHE_CRCE_010.pdf [Accessed 15 December 2015].
- 11 Department for Environment Food and Rural Affairs. *Valuing impacts on air quality: Updates in valuing changes in emissions of oxides of nitrogen (NO_x) and concentrations of nitrogen dioxide (NO₂)*. London: Defra, 2015. www.gov.uk/government/uploads/system/uploads/attachment_data/file/460401/air-quality-econanalysis-nitrogen-interim-guidance.pdf [Accessed 15 December 2015].
- 12 Committee on the Medical Effects of Air Pollutants. *Nitrogen dioxide: interim view on long-term average concentrations and mortality*. London: Health Protection Agency, 2015. www.gov.uk/government/publications/nitrogen-dioxide-interim-view-on-long-term-average-concentrations-and-mortality [Accessed 17 December 2015].
- 13 Lim SS, Vos T, Flaxman AD *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;380:2224–60.
- 14 Holland M. *Implementation of the HRAPIE Recommendations for European Air Pollution CBA work*. Report to European Commission DG Environment, 2014. <http://ec.europa.eu/environment/air/pdf/CBA%20HRAPIE%20implement.pdf> [Accessed 18 December 2015].
- 15 Holland M. *Cost–benefit analysis of final policy scenarios for the EU Clean Air Package* (corresponding to IIASA TSAP report no 11). Report to European Commission DG Environment, 2014. <http://ec.europa.eu/environment/air/pdf/TSAP%20CBA.pdf> [Accessed 21 December 2015].
- 16 European Environment Agency. *Cost of air pollution from European industrial facilities 2008–12*. Luxembourg: European Environment Agency, 2014. www.eea.europa.eu/publications/costs-of-air-pollution-2008-2012/ [Accessed 2 April 2015].
- 17 Health Protection Agency. *Radon and public health: Report of the independent Advisory Group on Ionising Radiation*. Chilton: Health Protection Agency, 2009. http://webarchive.nationalarchives.gov.uk/20110109132023/http://hpa.org.uk/web/hpawebfile/hpaweb_c/1243838496865/ [Accessed 2 April 2015].
- 18 Global Burden of Disease Study 2010 (GBD 2010) data downloads. <http://ghdx.healthdata.org/global-burden-disease-study-2010-gbd-2010-data-downloads> [Accessed 27 May 2015].
- 19 Jamrozik K. Estimate of deaths attributable to passive smoking among UK adults: database analysis. *BMJ* 2005;330:812.
- 20 Royal College of Physicians. *Passive smoking and children*. A report by the Tobacco Advisory Group of the Royal College of Physicians. London: RCP, 2010. www.rcplondon.ac.uk/sites/default/files/documents/passive-smoking-and-children.pdf [Accessed 2 April 2015].

Chapter 7: Summary

In the first chapter of this report, we explained how our economy, industries and way of life have changed over recent generations, and how these trends have affected air pollution over that time.

We close the report by looking at the effect that these developments will have on our environment and the air we breathe in the years ahead.

We are using up natural resources at an unprecedented rate. Our pollution of the environment harms the delicate global ecosystems on which we rely. The air pollution we generate plays a key role in this damage, not least in the process of climate change, which places our food, air and water supplies at risk and threatens our health and wellbeing.

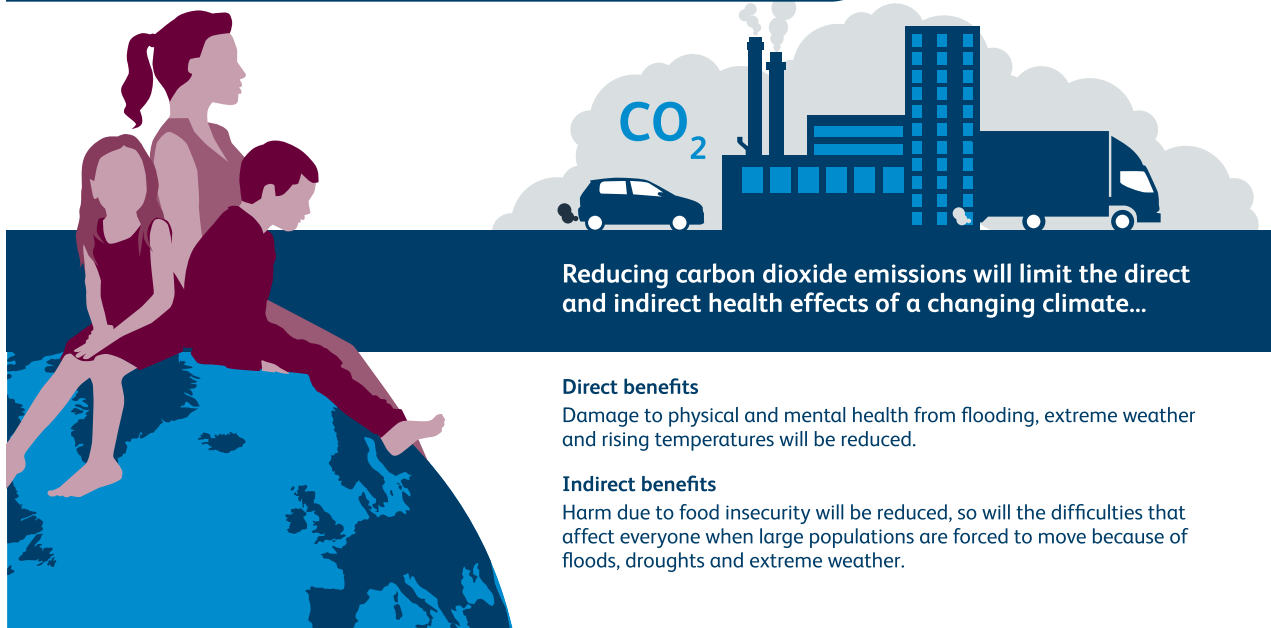
Many of the pollutants that cause this environmental damage are the same ones that are toxic to our bodies. These health problems will get worse if we continue on our current course.

There is hope, though, if we act quickly. Many of the changes that would decrease air pollution to protect our health – especially using energy more efficiently and burning less solid fuels and oil – would also help to slow down the overheating of our planet. If we take steps now to save lives by cleaning up our air, we may also protect the future of our home on Earth.

Key facts

- By 2013, the concentration of carbon dioxide in the atmosphere had increased by about 42% over the levels before the Industrial Revolution, and the concentration continues to rise. Carbon dioxide is one of the main gases causing the Earth to overheat.

Smart thinking on climate change can benefit our health



Reducing carbon dioxide emissions will limit the direct and indirect health effects of a changing climate...

Direct benefits

Damage to physical and mental health from flooding, extreme weather and rising temperatures will be reduced.

Indirect benefits

Harm due to food insecurity will be reduced, so will the difficulties that affect everyone when large populations are forced to move because of floods, droughts and extreme weather.

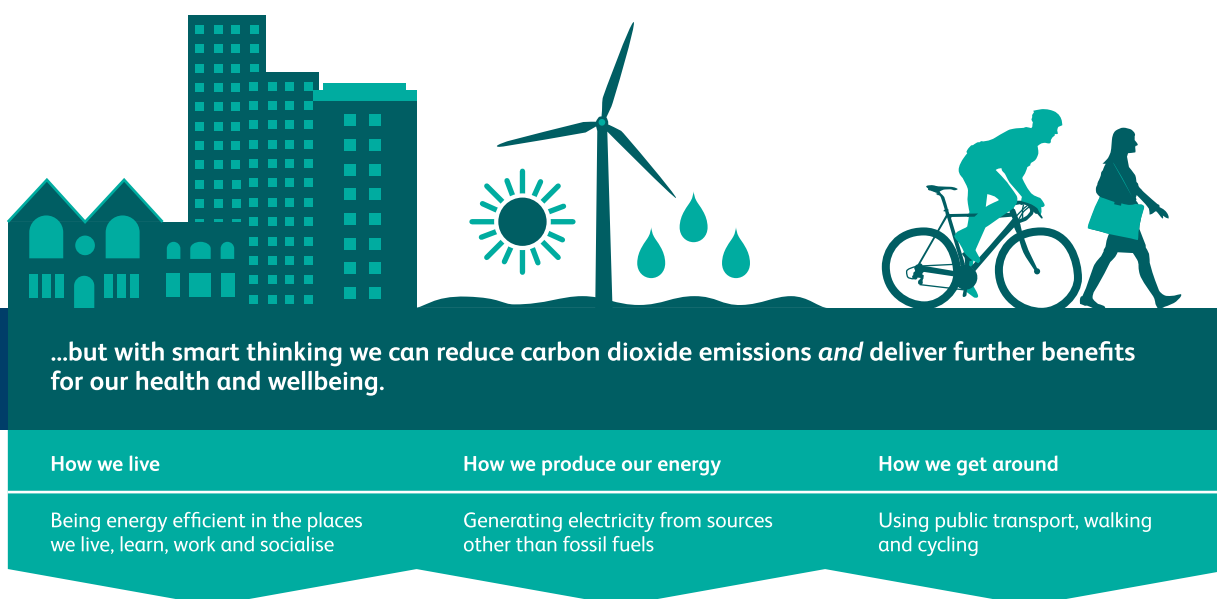
- Air pollution and climate change are intertwined. For example, the shifts in weather patterns due to climate change may cause more ozone to be produced at ground level, which harms our health. Increased ozone levels then contribute to more warming.
- Sometimes what is good for one of these problems is bad for the other. Diesel-fuelled vehicles cut down on carbon dioxide but they increase pollution from particulates, which damage health.
- On the other hand, many strategies to decrease air pollution are also ways to slow down climate change. We can make this happen by:
 - using less energy
 - using energy more efficiently
 - burning less oil, gas, coal and wood, while making more use of renewable energy sources
 - using hybrid and low emission vehicles
 - developing and using technology that captures carbon from power plants and factories, before it is released into the air.

If we act now to reduce greenhouse gas emissions to target levels by 2050, we can have a real impact. An analysis for the European Commission suggests that, each year in the UK, this would prevent the following impacts related to local and regional air pollutant exposure:

- 5,700 deaths
- 1,600 hospital admissions for lung and heart problems
- 2,400 new cases of bronchitis.

Reducing air pollution would also allow vulnerable people to be more active, take less medication, and live longer.

The economic value of these benefits would add up to €3.9 billion per year.



All of these steps will also reduce pollution, its health effects, and demands on health services. Energy efficiency will mean less illness and fewer deaths caused by energy poverty. Active transport will make us all fitter people, with huge gains for health.

Chapter 7: Changing our future

7.1 Introduction

Population growth, urbanisation, developments in the way that we travel, our pursuit and use of energy, new approaches to producing and sourcing food, and many other transitions have all delivered benefits for individuals and society. Yet, in combination, such changes have often created unintended new and complicated threats to health and wellbeing. Human beings are now using natural resources at an unprecedented rate and are damaging global systems and processes, on which we all rely for health and wellbeing.¹ Many of the global environmental changes – from depletion of the ozone layer, to ocean acidification, to climate change – can be linked to pollution of the atmosphere by human activity. This emphasises both the fragility and the interconnectivity of global systems and processes.^{2,3}

The pollutants primarily responsible for climate change often share common sources with the toxic pollutants that damage health in our towns and cities. When we burn fossil fuels in vehicles, in our homes or in industry, health-damaging chemicals (notably SO₂, oxides of nitrogen including NO₂, and PM) are released. At the same time, fossil fuel combustion produces gases such as CO₂ and NO₂, which contribute to warming of the planet. One consequence is that measures to reduce emissions of greenhouse gases through energy efficiency, and most of the options for switching from fossil to other fuels, also reduce local air pollution. Thus, policies and interventions that tackle local air pollution can address climate change, and vice versa. They are said to offer ‘co-benefits’. However, there are some policy options that generate ‘trade-offs’. For example, as discussed earlier, reducing reliance on fossil fuels by increased burning of biomass (typically biological material derived from recently living plants) may increase particle emissions. Similarly, ‘end-of-pipe’ options for cleaning flue gases reduce overall energy efficiency and increase the pollutants that they do not specifically target, potentially contributing to both climate change and local air quality problems.

Some policy options introduce unexpected complications and can have unanticipated negative consequences. For example, the shift away from petrol and towards diesel for the small engines that power our cars has reduced tailpipe CO₂ emissions. However, critically, the shift to diesel has also contributed to levels of health-damaging airborne particulates in the air of our towns and cities. Notably, too, the Air Quality Expert Group in its 2007 report *Air quality and climate change: a UK perspective*⁴ observed that, while the situation is complicated, the perceived climate benefits of reduced CO₂ in tailpipe emissions are, to an extent, offset by increased refinery emissions of CO₂ due to increased demand for diesel, and the climate-warming effects of black carbon particles that diesel engines emit. Fig 22 provides a simple illustration of how climate change and air pollution policies can interact.

7.2 Different pollutants – different behaviours and effects

The public health implications of many different emissions to the atmosphere are considered throughout this report. However, pollutants often differ markedly in their behaviour when introduced to the atmosphere. This is important for whether and how they impact on health and wellbeing and for the types of policy that are required to address the threats. When we think about air pollution in our towns and cities, we naturally focus on local pollutant concentrations and sources, such as busy roads or factories. These are important for people who live and work in the vicinity. However, the challenge from air pollutants assumes different characteristics when they are transported and transformed into secondary pollutants (such as O₃ and secondary particles) during dispersal in the atmosphere, perhaps

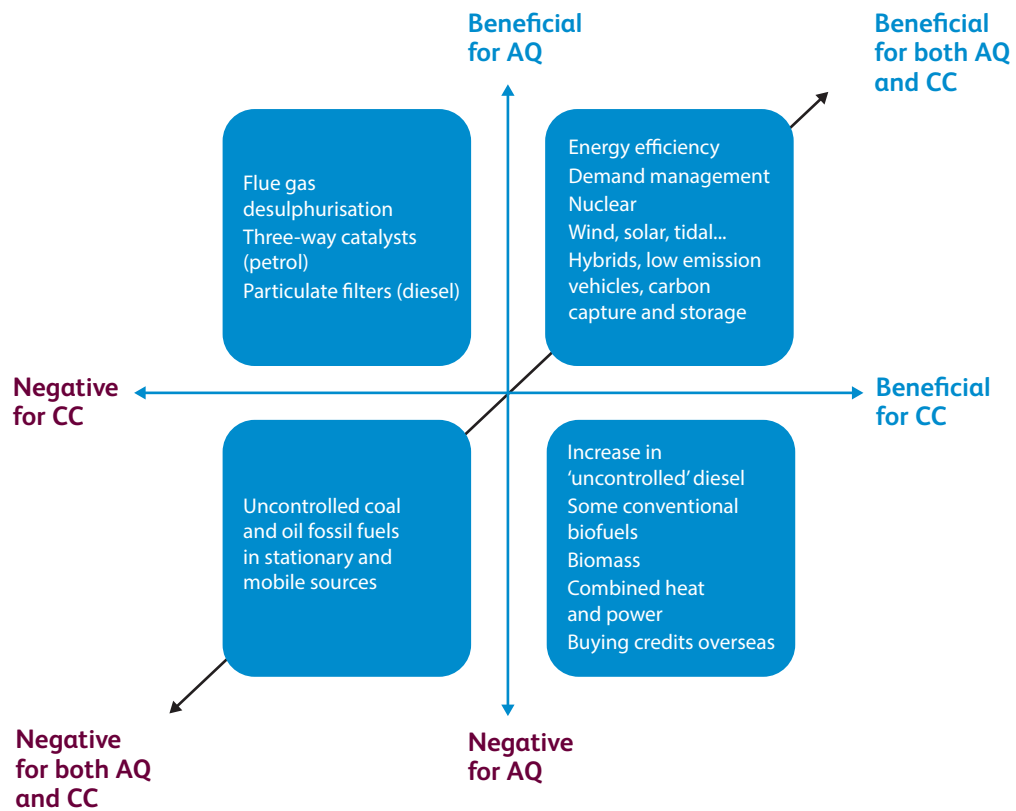


Fig 22. Policy map displaying air quality (AQ) / climate change (CC) interactions. Source: Defra (2010). *Air pollution: Action in a changing climate*. www.gov.uk/government/publications/air-pollution-action-in-a-changing-climate.

over several days. In these circumstances, the environment and the health of people living at some distance from the initial pollution source(s) is placed at risk. This has been recognised through the development of international action on air quality, as well as climate change, for example through a number of EU directives and the United Nations Economic Commission for Europe (UNECE) Convention on Long-range Transboundary Air Pollution.⁵

The global health threat from climate change has its origins in yet another type of pollutant behaviour. Because climate pollutants are less chemically reactive, they mix globally and respond much more slowly to emission reduction measures. They persist and penetrate throughout the atmosphere over timescales measured in decades and even centuries. This means that, although the need to address this dimension of air pollution and health is urgent and serious, the threat can appear very remote in space and time to residents in the locality or region where the emissions occur. People living in different locations will often experience the direct and indirect health impacts of climate change in different ways and over different timescales. Some communities will be affected much more severely than others, but invariably those most vulnerable to the impacts of climate change are, like those most vulnerable to the adverse health effects of air pollution, among the poorest in society. Thus, the release of pollutants into the atmosphere from human activity can be seen to initiate a number of quite different processes, which impact in different ways on human health and wellbeing. However, the public health response to air pollution should always be about protecting humans and the environment in ways that are socially inclusive and equitable, globally and across multiple generations.⁶

7.3 How local air pollution affects our climate

Average global temperatures are dictated by the balance between the incoming energy from the sun that is retained by, and warms, the Earth's surface, and the energy that is reflected back into space. When human activity pollutes the air, it upsets the Earth's energy balance, producing a warming effect. Both gases and particles are involved in this process. Greenhouse gases, such as CO₂ and CH₄, act directly, trapping radiation in the atmosphere. Others, such as oxides of nitrogen (NO_x), impact indirectly on the concentrations of CH₄ and O₃ (another powerful greenhouse gas). The role of aerosols in global warming is complex and can be positive and negative. By 2013, the concentration of CO₂ in the atmosphere had increased by around 42% over the levels present before the Industrial Revolution. In 2013, the Intergovernmental Panel on Climate Change (IPCC) concluded that, unless very stringent emission standards are achieved, by the end of the 21st century global surface temperatures will be more than 1.5°C above their 19th-century levels.⁷ Disturbingly, the IPCC expressed medium confidence that a 'business as usual' scenario, in which emissions remain high, carries a 50:50 chance that warming will exceed 4°C by 2100. Such a rise will have huge implications for health, wellbeing and the global community itself.^{8–11} A tragic but timely indication of the consequences of the extreme weather events likely to become more common in the world was provided by the devastation wrought by Cyclone Pam on Vanuatu in March 2015.

7.4 How climate change affects the air we breathe

Atmospheric chemistry is complex and, just as climate change is primarily caused by the release of greenhouse gases, the warming climate itself alters atmospheric chemistry in ways that can damage health and wellbeing. This can be illustrated by reference to O₃. Global warming increases levels of O₃ in the lower atmosphere, giving rise to airway damage, reduced lung function and increases in respiratory symptoms in exposed persons, as we have discussed throughout this report. Yet, the highest levels of atmospheric O₃ exist in the stratosphere (the second major layer of the Earth's atmosphere), where the 'O₃ layer' reduces the amount of ultraviolet radiation reaching the Earth's surface and, with it, damage to our DNA. In the 1970s, thinning of the O₃ layer prompted measures to tackle release of the chlorofluorocarbons (CFCs) present in aerosols that were primarily responsible. However, more recently it has been proposed that the 50–60-year timescale predicted for O₃ layer recovery may not in fact be achieved owing to climate change.^{12,13}

The Air Quality Expert Group report *Air quality and climate change: a UK perspective* provides an in-depth assessment of the interactions between the climate and aspects of the air we breathe.⁴

7.5 Health and wellbeing implications

Even in outline, the interconnections between air quality and climate change present a complex and sometimes confusing picture. However, the headline message for society, supported by an overwhelming scientific consensus, is that climate change is a product of human activity and, especially, emissions from the combustion of fossil fuels. By extension, any consideration of the health effects of human-induced air pollution is incomplete without including the many and diverse health and wellbeing impacts that are already taking place owing to the changing climate.

In a recent report, a working group of the IPCC observed that, through shifts in weather patterns and other consequences, climate change is both a direct and an indirect risk to health.¹⁴ Through its impacts on air quality, water and food, it is already affecting lives and livelihoods across the globe. In 2014 the

WHO estimated that, between 2030 and 2050, climate change will cause an additional 250,000 deaths worldwide per year from malaria, diarrhoea, heat exposure and undernutrition.¹⁵

For the UK, climate change will certainly impact negatively on air quality, but the most obvious changes will be in the seasonal, maximum, mean and minimum temperatures, in precipitation and in sea levels. Although the actual contribution of climate change to recent storms and floods in the UK cannot be accurately assessed, such events show the social and economic disruption that can be anticipated, should such events become more commonplace. They also illustrate the potential consequences of a changing climate for physical and mental health. The UK's Health Protection Agency (now part of Public Health England) recently considered impacts of climate-related environmental change specifically for the UK.¹⁶ Its report highlights the inherent difficulty in predicting the impacts of a process that is influenced by such a wide range of factors, not least the future level of man-made emissions and the effectiveness of mitigation and adaptation measures. However, the agency concluded that the medium-term prospect for the UK is more likely to be an exacerbation of existing health challenges, rather than the introduction of entirely new ones.¹⁶ The predictions make a valuable contribution, informing adaptation measures and emphasising the importance of climate change to a sometimes disengaged public. However, while it is essential for any country or community to consider the direct threats to health arising from changes close to home, it is also important to take account of a wider set of climate-related threats to health and wellbeing, including effects mediated through impacts on ecosystems.

The global nature of climate change means that, irrespective of the source of pollution, ecosystem-related changes can disrupt economies and societies anywhere in the world. Where these disruptions (eg floods and droughts) occur, they may damage material resources such as crops or the marine harvest, or affect non-material resources such as tourism and culture, which also underpin society. In turn, these impacts can rapidly undermine key determinants of health and wellbeing for the community, such as security, social relations, freedom of choice, and material resources. While such changes can appear remote to a country like the UK, the global connectivity of economic, social and ecological systems means that any sense of separation is often illusory.¹⁷ The migration of people, the availability of goods, and issues of food security can all impact societies that may not, themselves, be directly experiencing significant climate-related disruptions.

In summary, for any locality to appreciate fully the potential health-related impacts of climate change, it is necessary to consider the anticipated environmental changes close to home, but also those that, for a variety of reasons, may appear more remote and abstract.⁶ Fortunately, the co-benefits for climate change and local air quality that can flow from the right policies mean that actions to improve health through tackling local air pollution can be entirely consistent with securing the health of people in far-off lands at some point in the future.

7.6 Air pollution co-benefits from climate mitigation

Research for the European Commission under the ClimateCost Project¹⁸ indicates the magnitude of air quality benefits attributable to climate policies. Across the EU, a climate mitigation scenario designed to restrict warming to 2°C is estimated to reduce annual emissions of sulphur and nitrogen by 60% and 46% respectively in 2050 in Europe, with a 19% reduction for PM. As indicated in Table 9, these reductions have significant benefits for health, the monetary equivalent of total co-benefits being in the order of €40 billion per year.

Table 9. Health impacts and co-benefits for the EU of moving from the baseline to the mitigation scenario for 2050

Results for 2050	Pollutant(s)	Baseline	Mitigation	Co-benefit
Acute mortality (all ages), deaths	O ₃	24,000	21,000	3,400
Respiratory hospital admissions (≥65 years)	O ₃	28,000	24,000	3,800
Minor restricted activity days (15–64 years)	O ₃	36,200,000	31,200,000	4,940,000
Respiratory medication use (adults ≥20 years)	O ₃	16,700,000	14,400,000	2,280,000
Chronic mortality, life-years lost	PM	1,390,000	905,000	482,000
Infant mortality (1 month – 1 year), deaths	PM	200	130	70
Chronic bronchitis (≥27 years), new incidence	PM	77,000	50,000	27,000
Respiratory hospital admissions (all ages)	PM	29,000	19,000	10,000
Cardiac hospital admissions (all ages)	PM	18,000	12,000	6,100
Restricted activity days (15–64 years)	PM	124,000,000	81,400,000	42,700,000
Respiratory medication use, days	PM	14,990,000	9,847,000	5,184,000
Total annual monetary equivalent	O ₃ + PM	€125 billion	€82.5 billion	€42.8 billion

The analytical methods used for health impact assessment of air pollution in Europe have since been updated through the WHO-led health risks of air pollution in Europe (HRAPIE) study.^{19,20} While the detail of the recommended methods for European appraisal has changed, overall results, using the updated approach, will be broadly similar to those given above. These co-benefits equate to around €24 per tonne of CO₂ and are sufficient on their own to justify many actions to reduce greenhouse gas emissions. Similar findings have been reported elsewhere, for example by Balbus *et al.*,²¹ who reported air quality co-benefits from climate policies of between \$40 and \$93 per tonne of CO₂.

The conclusion from these studies is clear: that climate and air pollution policies are closely linked, and that there are substantial benefits from ensuring that the linkages present are fully exploited. The reverse also holds true, ie that a failure to exploit these links will incur unnecessary costs to health and ecosystems. The same applies to industry, for which inefficient policy will create additional demand for expensive emission controls.

7.7 Policy implications

The need to adapt to the now-unavoidable reality of climate change in the UK and elsewhere, and to mitigate the pace and extent of climate change through reducing emissions of greenhouse gases, is evident. It is clear that, while greenhouse gases are most active in the upper atmosphere and toxic pollutants are most active at ground level, they invariably share a source in the combustion of fossil fuels. This is important for policy, as improved energy conservation, measures to increase fuel efficiency and substitution of fossil fuels with alternative energy sources can have beneficial impacts on both air pollution and climate change. A number of policies targeting the way in which society lives, consumes and moves around have potential to produce co-benefits. For example, measures that discourage the use of private cars in urban areas deliver co-benefits to health and wellbeing through tackling climate change and air pollution. However, where such measures drive an increase in active travel (walking and cycling),

a much wider set of benefits to physical and mental health and wellbeing can result from increased physical activity levels.

The UK health sector is an important focus for climate change mitigation activities. It has been estimated that European health services have, in total, a carbon footprint equivalent to that of the whole of the Netherlands. With UK health services costing around 9.1% of gross domestic product (GDP),²² it follows that the sector uses a substantial amount of energy and is responsible for a substantial amount of greenhouse gas and local/regional air pollutant emissions.^{23–25} It follows, then, that improved energy efficiency within the health sector will have direct benefits to the health of the society that it serves through reduced burdens on climate and local air quality. For example, a reduction in hospital admissions and rates of other illnesses will save resources that can be reinvested to improve healthcare in other ways. An example of what NHS trusts can do to tackle air pollution is shown in Box 14.

Box 14: What can NHS trusts do?

- In collaboration with the charity Global Action Plan, Barts Health NHS Trust has worked with local boroughs to reduce staff and patients' exposure to air pollution and to reduce carbon and air pollution emissions.
- The Barts Health Cleaner Air Programme has focused on five core areas: clinical engagement (doctors and pharmacists), active travel, reducing community-based emissions through domestic housing, reducing hospitals' impact through fleet emissions, and protecting patients in high-risk groups (such as those with COPD).
- An example of the programme's outputs is a leaflet entitled *3 easy ways to reduce your contact with air pollution...* (co-designed with patients; Fig 23), which is freely available in clinics and local pharmacies.
- The seven partners in this programme are Barts Health NHS Trust, City of London Corporation, Waltham Forest Council, Newham Council, Tower Hamlets Council, the Greater London Authority, and delivery partner Global Action Plan. The programme is financed by the partners as well as by the Mayor's Air Quality Fund and Defra.

A big difference could be made if more trusts instigated activities like this, because the scale and activities of the NHS mean that it has a huge environmental impact.

In addition to local initiatives such as that described above, the NHS is very actively promoting sustainability in its operations through, for example, the outputs of its Sustainable Development Unit (www.sduhealth.org.uk/).



Fig 23. Information packs developed as part of the Barts Health Cleaner Air Programme. Maps provided by King's College London; photographs reproduced with permission from Global Action Plan.

7.8 References

- 1 Rayner G, Lang T. *Ecological public health: reshaping the conditions for good health*. Abingdon: Routledge, 2012.
- 2 Steffen W, Crutzen PJ, McNeill JR. The Anthropocene: are humans now overwhelming the great forces of nature? *Ambio* 2007;36:614–21.
- 3 Rockström J, Steffen W, Noone K *et al.* Planetary boundaries: exploring the safe operating space for humanity. *Ecol Soc* 2009;14:32.
- 4 Air Quality Expert Group. *Air quality and climate change: a UK perspective*. London: Defra, 2007. <http://uk-air.defra.gov.uk/assets/documents/reports/aqeg/fullreport.pdf> [Accessed 21 December 2015].
- 5 Reis S, Grennfelt P, Klimont Z *et al.* Policy Forum: From acid rain to climate change. *Science* 2012;338:1153–4.
- 6 Morris GP, Reis S, Beck S *et al.* *Climate change and health in the UK. Scoping and communicating the longer-term 'distal' dimensions*. Living with Environmental Change: Climate Change and Health Report Card, technical paper no 10. Swindon: Natural Environment Research Council, 2015. www.nerc.ac.uk/research/partnerships/lwec/products/report-cards/health/papers/source10/ [Accessed 22 December 2015].
- 7 Intergovernmental Panel on Climate Change. *Climate change 2013: the physical science basis*. Working Group I contribution to the fifth assessment report of the Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press, 2013. www.climatechange2013.org/report/ [Accessed 21 December 2015].
- 8 Anderson K, Bows A. Beyond 'dangerous' climate change: emission scenarios for a new world. *Phil Trans R Soc A* 2011;369:20–44.
- 9 New M. Four degrees and beyond: the potential for a global temperature increase of four degrees and its implications. *Phil Trans R Soc A* 2011;369:6–19.
- 10 Thornton PK, Jones PG, Ericksen PJ, Challinor AJ. Agriculture and food systems in sub-Saharan Africa in a 4°C+ world. *Phil Trans R Soc A* 2011;369:117–36.
- 11 Fung F, Lopez A, New M. Water availability in +2°C and +4°C worlds. *Phil Trans R Soc A* 2011;369:99–116.
- 12 Kelfkens G, Bregman A, de Gruijl FR *et al.* *Ozone layer–climate change interactions. Influence on UV levels and UV related effects*. Global Change NOP–NRP report 2002. Netherlands: RIVM, 2002. www.rivm.nl/bibliotheek/digitaaldepot/410200112_main_report.pdf [Accessed 21 May 2014].
- 13 Gao W, Schmoldt DL, Slusser JR (eds). *UV radiation in global climate change: measurements, modeling and effects on ecosystems*. Berlin: Springer-Verlag, 2010.
- 14 Intergovernmental Panel on Climate Change. *Climate change 2014: impacts, adaptation, and vulnerability*. Summary for policymakers. Working Group II contribution to the fifth assessment report climate change of the 2014 Intergovernmental Panel on Climate Change. Cambridge: Cambridge University Press, 2014. www.ipcc-wg2.gov/AR5/ [Accessed 2 February 2015].
- 15 Neira M. *Climate change: an opportunity for public health*. WHO Department of Public Health, Environmental and Social Determinants of Health, 2014. www.who.int/mediacentre/commentaries/climate-change/en/ [Accessed 14 May 2015].
- 16 Vardoulakis S, Heaviside C (eds). *Health effects of climate change in the UK 2012: current evidence, recommendations and research gaps*. London: Health Protection Agency, 2012. www.gov.uk/government/uploads/system/uploads/attachment_data/file/371103/Health_Effects_of_Climate_Change_in_the_UK_2012_V13_with_cover_accessible.pdf [Accessed 21 December 2015].
- 17 Adger WN, Eakin H, Winkels A. Nested and teleconnected vulnerabilities to environmental change. *Front Ecol Environ* 2009;7:150–7.
- 18 Holland M, Amann M, Heyes C *et al.* *Technical policy briefing 6: The reduction in air quality impacts and associated economic benefits of mitigation policy*. Summary of results from the EC RTD ClimateCost Project. In Watkiss P (ed), 2011. *The ClimateCost Project. Final Report. Volume 1: Europe*. Stockholm: Stockholm Environment Institute, 2011. www.climatecost.cc/images/Policy_Brief_master_REV_WEB_medium_.pdf [Accessed 21 December 2015].
- 19 World Health Organization. *HRAPIE: Health risks of air pollution in Europe – HRAPIE project. Recommendations for concentration–response functions for cost–benefit analysis of particulate matter, ozone and nitrogen dioxide*. Bonn: WHO, 2013. www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications/2013/health-risks-of-air-pollution-in-europe-hrapie-project-recommendations-for-concentrationresponse-functions-for-costbenefit-analysis-of-particulate-matter,-ozone-and-nitrogen-dioxide/ [Accessed 15 December 2015].
- 20 Holland M. *Cost–benefit analysis of final policy scenarios for the EU Clean Air Package* (corresponding to IIASA TSAP

- report no 11). Report to European Commission DG Environment, 2014. <http://ec.europa.eu/environment/air/pdf/TsAP%20CBA.pdf> [Accessed 21 December 2015].
- 21 Balbus JM, Greenblatt JB, Chari R, Millstein D, Ebi KL. A wedge-based approach to estimating health co-benefits of climate change mitigation activities in the United States. *Clim Change* 2014;127:199–210.
 - 22 The World Bank. *Health expenditure, total (% of GDP)*. Washington, DC: The World Bank, 2013. <http://data.worldbank.org/indicator/SH.XPD.TOTL.ZS> [Accessed 14 January 2016].
 - 23 National Institute for Health Research. *NIHR carbon reduction guidelines*. London: NIHR, 2014. www.nihr.ac.uk/policy-and-standards/nihr-carbon-reduction-guidelines.htm [Accessed 21 December 2015].
 - 24 Sustainable Development Unit. *Carbon footprint update for NHS in England 2012*. Cambridge: Sustainable Development Unit, 2013. www.sduhealth.org.uk/documents/Carbon_Footprint_summary_NHS_update_2013.pdf [Accessed 21 December 2015].
 - 25 Tomson C. Reducing the carbon footprint of hospital-based care. *Future Hosp J* 2015;2:57–62.

Methodology

Background

Sir Richard Thompson, immediate past president of the Royal College of Physicians (RCP), initially contacted Professor Stephen Holgate to discuss the health effects of climate change; this later developed into air pollution, with climate change as a sub-area of this, recognising new research that could inform the field. A working party, chaired by Professor Holgate, was set up to look at the topic of air pollution and its effect on health across the lifecourse, including looking at the effects *in utero*.

Administration

The working party was led by the RCP. It was agreed that the report should be jointly owned by the RCP and the Royal College of Paediatrics and Child Health (RCPCH), owing to the aspects of child health that are covered, as well as issues relating to later life.

- The working party was administered through the RCP Membership Support and Global Engagement Department.
- Production of the report was managed by the RCP Strategy, Communications and Policy Department.

Meetings

The working party met as follows.

- One planning meeting to set out the aims and objectives of the report. This allowed planning prior to the invitation of potential members of the working party, and was felt to be a good approach to ensure that the report's scope was available to share with potential members.
- Six meetings of the working party, which were each 3 hours long (plus an evidence-giving session of an additional 2 hours). These meetings were formally led by the chair of the working party, with an agenda that covered the minutes of the previous meeting, the breakdown of the report, and discussions around chapter development and recommendations. The project initiation document was assessed at each meeting and updated accordingly. This was considered to be a robust approach to ensure that all relevant areas were covered adequately and discussed. Members of RCP staff were invited to provide input on policy, recommendation setting and good publication practice.
- Sub-meetings were set up on an ad hoc basis, with attendance of the chair and vice chair for discussions around policy aspects, key recommendations and additional evidence. Additional meetings were held to ensure that policy aspects of the report could be discussed in detail with the RCP, and recommendations could be finalised.

The six meetings were well attended by working party members, with most members attending each meeting either in person or via teleconferencing. There was representation from both the RCP and the RCPCH at all meetings. Working party members / chapter authors were reminded at each meeting to disclose any potential new conflicts of interest. If members sent their apologies, written emails and updates were circulated.

Drafting the report

Subgroups of the working party with the appropriate expertise were charged with the responsibility to produce draft chapters, with additional working party members being co-opted as required to ensure that individual subject areas were covered in detail, and that the report had full ownership from those contributing to each section. However, while recognising that specific expertise was important in developing and analysing the evidence, the working party functioned as an expert committee rather than a committee of experts. Each chapter was discussed in depth at the meetings of the working party. The chapters were subsequently edited by the chair, Professor Stephen Holgate, to ensure coherence and connectivity between the different chapters. Where chapters overlapped in content, authors worked together to avoid duplication.

It was felt that chapter authors would be best placed to develop recommendations; therefore, each subgroup was responsible for developing key recommendations from each chapter to take forward. These were then assessed and edited by Ms Colleen Shannon, Professor Stephen Holgate, Mr Philip Insall and members of the RCP Strategy, Communications and Policy Department to ensure that the overarching key recommendations encapsulated the messages and views of the report.

RCP library staff conducted literature searches on the following topics: air pollution and development, air pollution and obesity, and long-term effects of air pollution. In each case, the Medline database was searched using MeSH and the title and abstract fields. The search strategies used in each case are available on request.

The addition of a professional healthcare writer was felt to be beneficial, to allow chapters to be summarised succinctly for a non-specialist audience. Ms Colleen Shannon was commissioned to develop accessible summaries of each chapter and the executive summary, and to work with authors in drafting recommendations. The summaries of each chapter feature in the main report and in a shorter, publicly accessible version of the report. It was felt this would help non-specialist readers to understand the key messages from each chapter.

Evidence

Evidence was gathered by selecting individuals/organisations that are key in the area of air pollution and related health issues. The RCP wrote to organisations and relevant individuals, requesting evidence. Most requests were for written evidence, as it was felt that this would give organisations the opportunity to submit evidence and position statements for incorporation into the report.

The relevance of the evidence to the report was assessed by the working party at its meetings. All evidence has been incorporated into the report in some format.

Most evidence was received as written submissions.¹ We had one oral evidence-giving session from Mr Philip Insall (formerly at Sustrans). Mr Insall was subsequently co-opted onto the working party.

Many of the submissions received contained both evidence-based and consensus views from organisations. The evidence referenced large studies in the field of air quality and health, and individual case studies.¹ Some submissions received were organisational statements in this area based on current evidence.

Evidence submitted to the report can be viewed on the RCP website.¹ This was seen to be a beneficial source to readers. Permissions were gained from evidence givers; if permission was not granted, the evidence is not provided online, but instead referenced in the report.

Consultation

Around 80 stakeholder organisations were identified. Many had already been involved with the report in the evidence-giving stage. The organisations were considered to be key in the area of air pollution and related health issues, and were from both the private and the public sectors.

The draft report, without recommendations, was sent to these stakeholders, giving them 4 weeks to comment and, in particular, to identify any omissions or inaccuracies. Stakeholders were also asked what they believed the main recommendations of the report should be.

Members of the working party considered the stakeholder feedback and made decisions about how the report should be amended, editing the report accordingly. Each amended section was approved by the working party chair.

The final text of the report was approved by the RCP Council and the RCPCH Executive Committee.

1 Royal College of Physicians. *Every breath we take: the lifelong impact of air pollution*. Report of a working party. London: RCP, 2016. www.rcplondon.ac.uk/projects/outputs/every-breath-we-take-lifelong-impact-air-pollution [Accessed 21 December 2015].

Acknowledgements

The RCP and RCPCH would like to acknowledge with thanks the following people and organisations for their contribution to the evidence that informs this report:

- Dr Abigail Whitehouse, clinical research fellow, Blizzard Institute, Barts and The London School of Medicine and Dentistry
- Amy Smullen, policy officer, British Heart Foundation
- Fiona Osgun, health information officer, Cancer Research UK
- Gavin Thomson, healthy air campaigner, ClientEarth
- Emma Ryland, geriatric registrar and medical leadership fellow in patient safety, Leeds Teaching Hospitals NHS Trust
- Caroline Watson, partner, Global Action Plan
- Annette Figueiredo, senior civil servant, Greater London Authority
- Anne Stauffer, deputy director, Health and Environment Alliance (HEAL)
- Abid Shah, clinical effectiveness manager, Royal College of Obstetrics and Gynaecology
- Professor Derek Bell, president, Royal College of Physicians of Edinburgh
- Robert Walker, senior technical manager – Environment, Public Policy and Vehicle Legislation Department, Society of Motor Manufacturers and Traders (SMMT)
- Philip Insall on behalf of Sustrans, now director of Insall and Coe active travel consulting
- Hannah Graff, senior policy researcher, UK Health Forum
- Dr Penny Woods, CEO, British Lung Foundation.

Every breath we take

The lifelong impact of air pollution

ISBN 978-1-86016-567-2
eISBN 978-1-86016-568-9

Royal College of Physicians
11 St Andrews Place
Regent's Park
London NW1 4LE

www.rcplondon.ac.uk



**Royal College
of Physicians**



Health & care
information
you can trust

The Information Standard

✓ Certified
Member