



# Personalising the Health Impacts of Air Pollution – Summary for Decision Makers



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## Executive Summary

Air pollution is an important public health issue in the UK.

It is now well established that air pollution constitutes a significant public health problem in the UK, in Europe and in many other countries across the globe. However, summary statements on air pollution and health are typically on a whole population basis and on rarer but more severe endpoints such as mortality. While important for overall public health terms, it may be harder for the public to identify with these summary statements, compared with summary statements based on specific susceptible groups and/or more commonly observed health outcomes. Breathing polluted air can affect your physical wellbeing at every stage of life, from the womb to old age, and can lead to a lifetime of symptoms of ill health in some people. Yet, most people are unaware of the full effects of polluted air on their health and that of their family.

This report provides a series of statements about the potential risks to the public in the UK and Poland from exposure to air pollutants. The statements focus on the impact of pollution on the specific population group e.g. asthmatics and is at the local level, in contrast to the majority of previous work which focusses on a national population basis and on rarer but more severe endpoints such as mortality. The statements provide accessible, easily understandable and scientifically credible information about how pollution has a widespread and personal impact on individual health.

### Cities where the statements can be applied

This work has chosen to focus in cities in the UK and Poland. Both suffer widespread impacts from pollution and have sufficient data to calculate the statements but differ in the sources of pollution. The report focusses on nine UK and 4 Polish cities:

UK cities	Polish cities
London	Warsaw
Birmingham	Wroclaw
Bristol	Poznan
Oxford	Bielsko-Biala
Southampton	
Liverpool	
Manchester	
Derby	
Nottingham	

### Health risks considered in this report

Detailed meta-analysis of studies connecting air pollution and human health have been undertaken by other people, and these results, large studies across Europe and opinions from expert Committees have been assessed to see if the evidence is reliable enough to use for quantification. From this, 31 health outcomes have been identified where this is good evidence to link exposure and an impact on health. These are as follows:

1. *Reduced Lung function (FEV1)<sup>1</sup> in children from long term exposures*
2. Numbers of children age 6-8 with FEV1 less than 85% predicted: 'low lung function'
3. *Lung function (FVC)<sup>2</sup> at age 15.*
4. Lung function (FVC) growth in children from long term exposures (% change in FVC in children from age 11-15)
5. Lung cancer long-term
6. *Myocardial Infarction (MI), short term*
7. Coronary heart disease, long-term
8. Out of hospital cardiac arrest short-term
9. Respiratory admissions short-term all ages
10. *Respiratory admissions short-term elderly*
11. Stroke admissions, short term
12. First occurrence of stroke long term
13. *Heart failure short term*
14. *Heart failure long-term*
15. Asthma admissions in children
16. Asthma admissions in adults
17. Asthmatic symptoms in asthmatic children, short-term
18. Term low birth weight
19. Cardiovascular Disease (CVD) admissions all ages short-term
20. *Cardiovascular Disease (CVD) admissions elderly*
21. Pneumonia admissions in children short term
22. COPD admissions short term all ages
23. *COPD admissions short-term elderly*
24. *Hypertension short-term*
25. *Hypertension long-term*
26. *Diastolic Blood Pressure (DBP) short-term*
27. *DBP long-term*
28. *Cardiac arrhythmia short-term*
29. *Atrial fibrillation short-term*
30. Prevalence bronchitic symptoms in asthmatic children long-term
31. Bronchitis prevalence in children (long-term exposure)

For a variety of reasons, statements were not produced for those outcomes in italics e.g. overlap for all ages and elderly, not prioritised in focus group testing, difficulties finding baseline rates, ambiguity over exact definition. Further outcomes were considered and ruled out due to lack of evidence or too much uncertainty, as described in section 3.3 of the report.

## Method

Each statement is calculated from several basic components in a flow diagram:



<sup>1</sup> FEV<sub>1</sub> is forced expiratory volume in 1 second, a measure of how fast someone can breathe out. It is often low in asthmatics.

<sup>2</sup> FVC is forced vital capacity, a measure of the volume of the lung used for breathing.

In summary, the methodology is as follows:

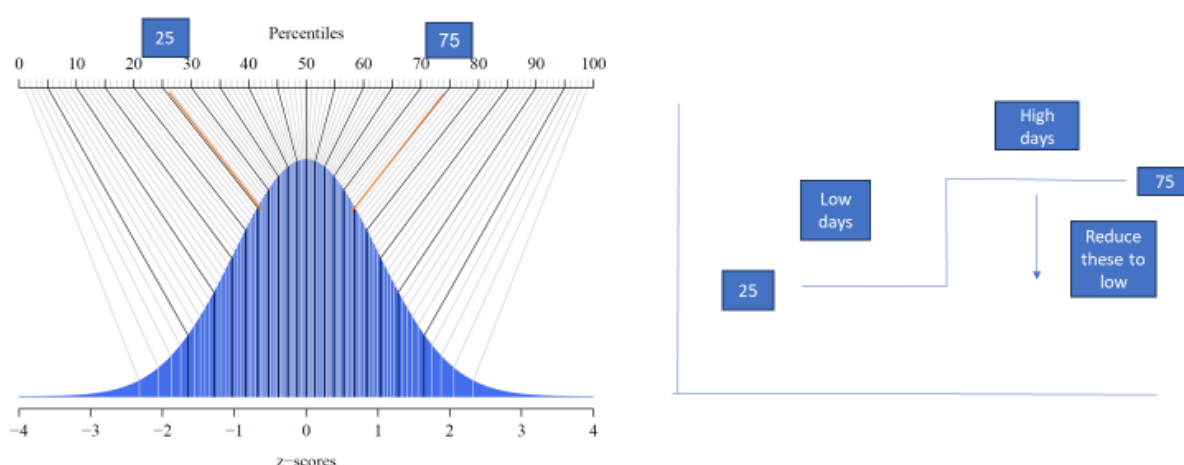
1. Air quality monitoring data for each city is used as a surrogate for exposure.
2. A numerical relationship established between exposure of air pollution and health outcomes, termed 'concentration response function' or CRF in previous studies is selected.
3. A baseline rate is calculated which is the numbers of a health outcome over the population in the absence of an air pollution exposure change.
4. Some of the statements focus on health outcomes for which only a subset of the total population can be regarded at risk, such as children. To approximate these subsets of people by age, we used yearly data from the Office for National Statistics (ONS). Other sources have been used for e.g. numbers of asthmatics.
5. Health impact assessments are used to quantify effects in particular places or for particular scenarios.
6. From the full set of evidence, a summary statement is provided for each health outcome and scenario in each city.

## Scenarios

Many of the statements in this report use statements such as 'high' and 'low' pollution days and 'busy' and 'non-busy' roads.

### *Higher pollution days vs lower pollution days:*

We defined this by assuming that typical higher air pollution days were at the middle of the top half of the annual range of pollutant levels and typical lower air pollution days were at the middle of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations. We simplified the distribution to assume that the top half of the days were all at the 75<sup>th</sup> percentile level and the bottom half at the 25<sup>th</sup> percentile. We then did calculations for a hypothetical scenario where the days at the 75<sup>th</sup> percentile were reduced to the 25<sup>th</sup> percentile.



*Schematic frequency distribution<sup>3</sup> illustrating the procedure for defining 'high' and 'low' pollution days and the associated scenario.*

<sup>3</sup> Note this frequency distribution is purely illustrative; real air pollutant concentration frequency distributions are virtually all log-normal rather than normal.



### *Living near busy roads:*

This scenario used the difference between concentrations at monitoring sites close to busy roads compared with concentrations away from busy roads from London background monitoring sites. Only London data was used for statements using numbers of people due to availability of numbers of people living within 50m of a major road from a previous project. A full explanation of the assumptions made here are available in section 5.1 Scenarios.

### **Some example statements**

There are a large number of statements given in section 5 Personalised health statements of the report. In fact, the wide range of health outcomes and cities covered is a key feature of the report. It does mean, however, that only a few examples of the results can be given here. These examples span different cities, short and long-term exposure, types of scenarios, types of statements (% change in risk or numbers affected)), severe outcomes affecting fewer people and more minor outcomes affecting more people.

*Roadside air pollution in Oxford stunts lung growth in children by 14.1%<sup>4</sup>.*

*Living near a busy road in London may contribute to 306 strokes each year<sup>5</sup>.*

*In London on high air pollution days, 142 more children with asthma experience asthma symptoms than on lower pollution days<sup>6</sup>.*

*Cutting air pollution in Warsaw by one fifth may contribute to 542 fewer cases of coronary heart disease each year (long-term)<sup>7</sup>.*

*The risk of emergency hospitalisations for pneumonia in children in Manchester is 2.3% higher on high air pollution days than on lower air pollution days<sup>8</sup> (short-term).*

### **Use of the statements**

This report provides the detailed scientific method and approach to calculating the numbers in the statements. It provides statements in simple language that are accompanied by footnotes explaining

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<sup>4</sup> Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the Oxford background).

<sup>5</sup> Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background).

<sup>6</sup> Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.

<sup>7</sup> Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g. (fatty diet) but air pollution may contribute too.

<sup>8</sup> Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average ozone concentrations.

the scenario from which the calculations come. The statements were developed in consultation with Purpose Europe (see below) to ensure simplicity of language, while retaining scientific accuracy. We see the statements and footnote as essential pieces of information that should always be used together. Together with links to this report, they provide a clear link between the statement and the simpler statement and their scientific basis.

For London, we also prepared extended 2 page 'statements and justification' that provide further details of the science behind the calculations in one convenient place. To support their use and uptake, we have worked with Purpose Europe to summarise the outputs in a user-friendly toolkit. The toolkit offers support for disseminating the statements with the public, highlights the health conditions that most people care about and recommends the most effective language and imagery choices to communicate messages effectively.

The guidance has been developed to help translate the complex and detailed scientific explanations provided in this report. It used focus groups, online message testing, and consultation with key organisations working on air pollution in order to uncover the best ways to engage and drive public action on dirty air.

The toolkits will be downloadable from the Clean Air Fund website from 27 November 2019: [www.cleanairfund.org](http://www.cleanairfund.org).

# 1 Introduction

## 1.1 The importance of the problem

It is now well established that air pollution constitutes a significant public health problem in many countries across the globe. However, summary statements on air pollution and health are typically on a whole population basis and on rarer but more severe endpoints such as mortality. While important for overall public health terms, it may be harder for the public to identify with these summary statements, compared with summary statements based on specific susceptible groups and/or more commonly observed health outcomes. The aim of this work is to produce statements about the potential risks to the public in the UK and Poland from exposure to air pollutants. We have chosen to work with the UK and Poland as these two countries represent extremes of pollution sources in their cities. The UK has for many decades reduced the use of solid fuel in small scale residential, commercial and industrial uses to the extent that road transport is the dominant national source of air pollutant exposures in UK cities. The transboundary contribution to PM<sub>2.5</sub>, PM<sub>10</sub> and ozone (O<sub>3</sub>) is also of importance of course. In Poland there is still a significant amount of solid fuel use for heating in the major urban areas and hence the UK and Poland provide a helpful example of two extremes of air pollutant exposures in Europe.

The statements we have produced deal with health outcomes other than mortality since it was considered that these outcomes would resonate more with the general public than broad statements on mortality or the loss of life-years and also because a considerable amount of work has already been done on the associations between air pollutants and mortality. The work has involved discussions with experts in communications (Purpose London) who have trialled the statements with the public in face-to-face sessions and via digital testing. The report below discusses more concentration response functions (CRFs) and more health outcomes than were finally used in producing personalised statements. This arose as a result of the outcomes of the focus groups and digital testing carried out by Purpose London. Nonetheless we have included the remaining CRFs for completeness and as a source for further work should that prove of interest. The final statements represent the combination of detailed scientific assessments of the air pollution and health effects literature, and feedback from the wider public community. This combination of robust scientific assessment coupled with a translation into statements which are relevant and clear to the public represents an important milestone in communicating the risks of exposure to air pollution across a range of health outcomes.

## 1.2 Background

The public are understandably interested in the size of the effect of air pollution on health and, in particular, the risks to them as individuals, or at least to individuals like them. Typical statements are in terms of numbers of premature deaths or life years lost. This is mainly because (i) it is assumed that people are most concerned about the most severe endpoints and these usually have a dominant influence on cost-benefit analysis (ii) most places collect mortality statistics routinely, there are a lot more studies for this endpoint (iii) there are also more studies on all-cause mortality because it is a clearly defined endpoint without confusion as to whether a disease has been diagnosed correctly (iv) overall impact on the population as a whole is the output of interest for public health practitioners.

As a measure of population impact, an input into cost-benefit analysis and a general headline for the media, the above types of statements remain influential. However, the acknowledgement of the risks of air pollution and the motivation to change behaviour may be increased by summary statements with which individuals can identify to a greater extent. Life years can seem a rather

abstract concept and deaths may seem too distant in time for many in the population. So, there is a role for summary statements on more common adverse health effects of air pollution and, in particular, statements directed at groups which may be susceptible to specific health problems or may also live in regions of higher pollution levels e.g. near busy roads.

There are many scientific studies on the effects of air pollution on a wide variety of disease outcomes but their conclusions are written for scientists rather than the public, and it could be difficult for a member of the public to judge its quality or put a particular study into context. There are, however, documents that pull together consensus positions on the evidence including Committee reports (e.g. COMEAP<sup>9</sup>, 2010; WHO, 2013a and b; US EPA *various dates*) and, also, systematic reviews/meta-analyses (e.g. Brook et al 2010; Mills et al, 2015; Hoek et al 2012). Meta-analyses pool quantitative information across studies so are useful to give a sound basis for estimating the size of the air pollution effect. Finally, there are health impact assessments which have quantified effects in particular places or for particular policy scenarios. Some of these only cover mortality (e.g. EEA, 2017) and some are becoming outdated (COMEAP, 1998) but other more recent publications do cover disease outcomes (Holland 2014; APHEKOM 2011; Walton *et al* 2015). Their methods vary substantially, and they are not necessarily written in language easily accessible by less specialist readers.

Our appraisal of the literature and the conclusions we draw in formulating the final statements will clearly remain valid only until such time as new research adds to the evidence base on the impacts of air pollutants on health. In addition, we did not consider toxicological evidence in this report that can affect whether links between air pollutants and a health outcome are regarded as causal. We relied instead on past considerations of this aspect. This could change with new expert Committee considerations. Research on all aspects of air pollution and health is continually evolving and detailed reviews are periodically carried out, for example at the time of writing the WHO are engaged in producing systematic reviews of the literature in the process of revising the air quality guidelines. Nonetheless, the statements presented here should prove useful to stakeholders, local and central governments, NGOs, foundations and the general public in assessing the risks to a range of health outcomes from exposure to air pollutants.

Although the main product of this work is the set of health statements, phrased in such a way as to be readily accessible by a lay audience, the statements are based on relatively sophisticated epidemiological studies. Readers may wish to delve more deeply into this background and various texts could be helpful (Nieuwenhuijsen, 2015, Landon and Wilkinson, 2006).

### 1.3 Methods: how these air pollution impact statements were calculated.

The statements are calculated from several basic components in an ‘impact pathway’ approach as illustrated in Figure 1.



Figure 1. The flow diagram illustrating the methodological step in producing the statements.

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<sup>9</sup> Committee on the Medical Effects of Air Pollutants, an expert advisory committee to the UK Department of Health and Social Care; World Health Organisation a United Nations body concerned with all aspects of global health.

The first of these is some measure of exposure, and where we have used air quality monitoring data from regulatory based monitoring networks in the UK and Poland. It is recognised that fixed point monitoring data are at best a surrogate for the actual exposures of people as they go about their daily lives. However, the overwhelming majority<sup>10</sup> of epidemiological studies are based on this measure of exposure, hence its use here. We have produced statements which relate to populations across cities as a whole and here we have used concentration data from urban background monitoring locations. We have also produced statements of risks relating to populations living near busy roads and here we have used air quality data from roadside/traffic locations. However, we should stress here that the statements on risk near busy roads were derived from averages over all roadside monitors in a given city and so should NOT be applied to any specific local road.

The next step is to obtain a numerical relationship between the air pollutant concentration ('exposure') and the change in the health outcome in question. This numerical relationship is termed the 'concentration response function' or CRF. It usually takes the form of a single numerical coefficient in a form equivalent to a percentage increase over the baseline. The change in the health outcome due to pollutant exposure in the population considered to be at risk (i.e. those living in the areas noted above) then has to be added to the baseline rate of the outcome or disease. Here the 'baseline rate' is the incidence of the outcome in the absence of air pollution exposure. The result then allows us to calculate quantitative statements giving the effect of a given exposure to an air pollutant on a particular health outcome or disease.

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<sup>10</sup> Time-series studies which relate daily variations in monitored concentrations of air pollution with daily health outcomes are relatively straightforward to do and therefore many hundreds of them exist. Studies using modelled air pollution concentrations to study effects of long-term exposure are increasing in number but are much more labour intensive.

## 2 Air Pollution Exposures

In the strict sense of the word, the ‘exposure’ of individuals to pollutants is a time series of successive concentrations as an individual moves through a variety of microenvironments during daily activities. Such data are as yet not feasible to collect for whole populations of cities and so some surrogate measure of exposure has to be used. As noted in the introduction we used air quality data from regulatory monitoring networks to represent the exposure of populations in the cities we investigated. Urban background monitoring stations – that is locations not directly influenced by a single nearby source such as a busy road – are arguably the most appropriate surrogate for a person’s total exposure. However, bearing in mind that many schools, hospitals and habitation are along busy roads we have also used data from roadside locations.

In the UK we used the Automatic Urban and Rural Network (AURN) with data published by Defra (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>). In London, specifically, data were also used from the London Air Quality Network (LAQN) run by King’s College London. Some monitoring stations were included in both networks, so in order to avoid double counting, we identified these stations and kept only the LAQN measurements (concentrations were almost identical with the AURN). All the UK air pollution exposure data were downloaded from the *openair* package in R (<http://www.openair-project.org/>). Information about the type of each monitor within the UK cities was also collected. Thus, for those cities that have both urban background and roadside monitors we report descriptive statistics by monitoring type, apart from the overall data (**Table A2** and **Table A3**).

For Poland, we used the data submitted under the requirements of the Air Quality Directive of 2008 and available on the AIRBASE database run by the European Environment Agency (<https://www.eea.europa.eu/themes/air/links/data-sources/airbase-public-air-quality-database>). More specifically, data are available for every monitoring station within each Polish city with an hourly resolution. For each city we used both urban background and roadside data. Although arguably the urban background stations are a better surrogate for the exposure of the whole population, there are nonetheless situations where houses, schools, hospitals and other sensitive locations may be situated at the roadside.

Air pollutant concentrations can vary from one year to the next depending on meteorological conditions. In order to average out potentially ‘bad’ or ‘good’ years, we used three years of data from 2015 to 2017 inclusive. More specifically, we downloaded hourly air pollution data for UK and Polish cities and created daily time series of the daily mean values for all pollutants except ozone for which we calculated 8-hour maximum, as this is the exposure that has mainly been associated with various health endpoints in epidemiological literature. Finally, we created a one-year daily time series dataset by averaging the measurements across the same calendar days of the years 2015–2017 (e.g. 01/01/2015, 01/01/2016 and 01/01/2017) to account for potentially ‘bad’ or ‘good air quality’ years and get a better representative dataset of the true long-term ambient air pollution in the cities included in this project. We checked the distribution of each pollutant and because of some skewness in the data, we decided to use the median and the interquartile range, i.e. 25<sup>th</sup> and 75<sup>th</sup> percentile, as typical values for a ‘typical’, ‘low’ and ‘high’ air pollution day respectively.

Descriptive statistics for six pollutants in 13 UK and Polish cities based on the data collected from all the monitors within each city are shown in tables A1 and A2 in Annex A.

In some UK cities, there are both urban background and roadside monitors that measure air quality. For those cities, we report in the tables the descriptive statistics by type of monitor. The data downloaded from the AIRBASE database for Poland did not provide this information. This means

that for the later statements, scenarios based on comparing living near a busy roads with quieter streets cannot be done for Derby, Manchester or the Polish cities.

## 3 Concentration response functions

### 3.1 Our approach

In our preliminary work we considered CRFs based on WHO (2013b) and COMEAP Reports and/or more recent meta-analyses. We also covered potentially interesting health outcomes for which there was no sufficient or consistent evidence.

After extensive discussion, we have chosen the health outcomes for which we considered the evidence to be persuasive and adequately quantified and, in this chapter and in Annex B, we have included these health outcomes and CRFs that associate them with the concentrations of specific pollutants. We should stress that we have chosen CRFs on the basis of robust science involving statistical significance and well-characterised studies. We have made no attempt to assess causality in this assessment of the literature and consequently the phrasing of our statements reflects this. We recognise that conclusions on representative CRFs often result from a consensus among experts, as was the case in the WHO HRAPIE exercise (WHO, 2013b) or in the reports of the UK advisory group COMEAP. Where we felt there was no better evidence than that included in HRAPIE, COMEAP or other consensus assessments we have used them.

Where these sources may have been superseded by more recent evidence and where this evidence was persuasive, we have made our own judgements and chosen appropriate CRFs. In these cases, what we have done is to set out clearly our reasoning for choosing the CRFs that we have used in formulating the statements and to cite the source of the studies we have used. We also recognise that as the science develops and as more studies emerge, and with further reports from expert groups, the conclusions we reach and the quantitative statements that we have produced would need updating in the light of emerging research.

The CRFs we have used, depending on the study design and the question under investigation, may reflect either the effects of short-term exposures, i.e. effects taking place on the same day or a few days after the occurrence of higher pollutant concentrations, or the effects of long-term exposures, i.e. those occurring after many years or life-long exposures. The reference time period is specified in the corresponding Tables and text and leads to different types of statements. Moreover, for consistency purposes we decided to report the metric of association that was reported in the original studies, be it a relative risk, an odds ratio or a percentage increase in the risk of experiencing a health outcome. To convert the relative risk per unit or per any other fixed increase in air pollution concentration to the corresponding percentage risk change, one can use the following formula:

$$\% \text{ change} = (RR - 1) * 100 \%$$

where RR is the relative risk. The same formula applies for the conversion of an odds ratio (OR) to the percentage change in the odds of experiencing an outcome.

We preferred to use CRFs that were based on European studies. However, when there was not enough evidence coming from European studies and there was no reason to think that the effects in Europe would be largely different, we used CRFs based on global estimates. We placed emphasis in using CRFs either included in established reports, such as those from WHO or COMEAP, which are based on the collective opinion of many prominent experts, or in good quality meta-analyses for more recent findings.

### 3.2 The CRFs



Details of the CRFs, their sources in the literature and our comments and justification for choosing them are given in Annex B. Table 1 below shows the outcomes for which we have considered CRFs for at least one pollutant. The pollutant concentrations described in Annex B are 24-hour averages unless otherwise noted.

Although we have tabulated a list of CRFs for a range of health outcomes and pollutants, we have not used all of them in formulating our final statements. The reason for this is twofold. Firstly, in trialling the potential list of statements with focus groups and in digital testing, we concentrated on those statements which appeared to resonate most strongly with the public. Secondly, the production of the final statements is constrained by the availability of data in each of the process steps shown in Figure 1, and in some cases baseline rates for the relevant health outcome were not available.

*Table 1 - Health outcomes, exposure time reference, age or sensitive group and pollutants for the considered Concentration Response Functions (CRFs)<sup>11</sup>*

Health outcome	Exposure reference period	Age group	Pollutants
Lung function FEV <sub>1</sub>	Long-term	Children	PM <sub>2.5</sub> , NO <sub>2</sub>
Low (<80% of predicted value) lung function	Long-term	Children	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub>
Lung function FVC California statements	Long-term	Children	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub>
Lung function FEV <sub>1</sub> California statements	Long-term	Children	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub>
CVD admissions	Short-term	All ages	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub>
CVD admissions	Short-term	Elderly	PM <sub>10</sub> , NO <sub>2</sub> , Ozone
Lung cancer	Long-term	All ages	PM <sub>2.5</sub> , PM <sub>10</sub>
COPD admissions	Short-term	All ages	CO, PM <sub>10</sub> , NO <sub>2</sub> , PM <sub>2.5</sub> , Ozone
COPD admissions	Short-term	Elderly	CO, PM <sub>10</sub> , NO <sub>2</sub> , PM <sub>2.5</sub> , Ozone
Myocardial Infarction (MI)	Short-term	All ages	CO, PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub>
MI	Long-term	All ages	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub>
Pneumonia admissions	Short-term	Children	PM <sub>10</sub> , NO <sub>2</sub> , PM <sub>2.5</sub> , Ozone
Respiratory admissions	Short-term	All ages	NO <sub>2</sub> , PM <sub>2.5</sub> , Ozone
Respiratory admissions	Short-term	Elderly	NO <sub>2</sub> , PM <sub>2.5</sub> , Ozone
Cerebrovascular disease (stroke)	Short-term	All ages	PM <sub>2.5</sub> PM <sub>10</sub> CO Ozone NO <sub>2</sub>
Cerebrovascular disease (stroke)	Long-term	All ages	PM <sub>2.5</sub> PM <sub>10</sub>
Heart failure	Short-term	All ages	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub> , CO, Ozone, SO <sub>2</sub>
Heart failure	Long-term	All ages	PM <sub>10</sub> , NO <sub>2</sub> , SO <sub>2</sub>
Hypertension	Short-term	All ages	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub>

<sup>11</sup> The use of these CRFs is discussed in more detail in Annex B and in section 5 of this report where statements on specific health outcomes are discussed.

Hypertension	Long-term	All ages	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub>
Diastolic Blood Pressure (DBP)	Short-term	All ages	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub> , Ozone
DBP	Long-term	All ages	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub> , Ozone
Cardiac arrest	Short-term	All ages	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub> , CO, Ozone
Cardiac arrhythmia	Short-term	All ages	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub> , CO
Atrial fibrillation	Short-term		PM <sub>2.5</sub> , NO <sub>2</sub> , CO, Ozone, SO <sub>2</sub>
Asthma admissions	Short-term	Children	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub> , Ozone, (SO <sub>2</sub> )
Asthma admissions	Short-term	Adults	PM <sub>10</sub> , NO <sub>2</sub> , Ozone
Asthmatic symptoms	Short-term	Asthmatic children	PM <sub>10</sub>
Bronchitic symptoms	Long-term	Asthmatic children 5-14yrs	NO <sub>2</sub>
Asthma prevalence & incidence	Long-term	Children, adults	PM <sub>2.5</sub> , PM <sub>10</sub> , NO <sub>2</sub> , O <sub>3</sub> , SO <sub>2</sub> , CO
Chronic bronchitis	Long-term	Adults	PM <sub>10</sub>
Bronchitis prevalence	Long-term	Children	PM <sub>10</sub>
Term low birth weight	Full pregnancy	Mothers	PM <sub>2.5</sub> , PM <sub>10</sub> , CO, NO <sub>2</sub> , SO <sub>2</sub>
Lung function in COPD patients	Short term	All ages	PM <sub>10</sub>
Incidence of chronic bronchitis in adults	Long term	Adults	PM <sub>10</sub>

### 3.3 Outcomes which are not used.

As noted in the introduction, we exercised critical judgement in assessing the literature. If we did not feel the underlying science behind any potential associations of a health outcome with air pollutants was of sufficient quality, we decided not to incorporate that outcome in our statements. The outcomes relating to pre-term low birthweight have been discussed in section B20. (Term) Low birthweight. The other outcomes are listed below:

- (i) FEV<sub>1</sub> in children – results in terms of millilitres per second FEV<sub>1</sub> would not be well understood. Numbers of children with low lung function was used instead.
- (ii) Lung function decrements and symptoms in COPD patients – unclear definition of population at risk (mixture of asthma and COPD patients).
- (iii) Asthma incidence and asthma prevalence – suggestive but contradictory evidence
- (iv) Pre-term – while there were some studies which showed statistical significance, these were of lower quality than better quality studies which showed non-statistically significant associations with air pollutants.
- (v) Stillbirth – insufficient evidence
- (vi) Chronic bronchitis in adults – overall evidence unconvincing, although some studies show effects on chronic bronchitis symptoms

From the authors' prior experience several outcomes were ruled out before writing up or investigating concentration-response functions. These were:

- (i) Restricted activity days, all ages and Minor restricted activity days: we decided against using these as there was only 1 old US study on each in the literature (Ostro 1987; Ostro and Rothschild 1989)
- (ii) Work days lost in the working population aged between 20 and 65 years, again only one old US study was available (Ostro 1987)
- (iii) School absences - we partially investigated this but concluded that there were not enough studies.
- (iv) Dementia incidence - we were aware that COMEAP was considering air pollution and dementia and a report that had considered this issue was due out relatively soon (2020 on [www.comeap.org.uk](http://www.comeap.org.uk)). The minutes of COMEAP 5th November 2018 and later meetings indicate that evidence was considered suggestive but there were too few studies once considered across specific outcome definitions and pollutants for a stronger conclusion.

A further set of outcomes were not pursued further beyond this stage as other outcomes were given higher priority. This was informed by focus group testing of qualitative statements and other factors such as partial overlap with other outcomes (e.g. heart failure, arrhythmia admissions and atrial fibrillation can be a consequence of coronary heart disease) and likely difficulty in obtaining baseline rates in the time available. These could be pursued further at a later date. These were:

- Heart failure
- Hypertension
- Diastolic blood pressure
- Hospital admissions for arrhythmia
- Atrial fibrillation

### **3.3 Summary**

Concentration-response functions for a wide range of minor to severe health outcomes from different causes (respiratory symptoms to low birth weight to coronary heart disease) have been identified. This represents a significant part of our work. These can be used as the basis for both qualitative statements about links between a pollutant/pollution and a health outcome and statements based on a percentage change in risk for a change in pollution. For statements based on numbers of health outcomes further steps are needed. The following chapter considers whether baseline rates of the health outcomes (the total numbers occurring in a population) are available, and, where necessary, whether information on the population at risk is available e.g. numbers of asthmatic children.

## 4 Baseline rates and population at risk

### 4.1 United Kingdom

The concentration-response functions in the previous chapter give a change in the health outcome for a particular standard change in air pollution. This is then adjusted to match the actual difference in air pollution described in the scenario section in Chapter 5, using data from Chapter 2 and its associated annex. This may be expressed in different ways but is most easily explained as a new percentage change for the particular increase or decrease in pollution for the chosen scenario. Where statements of numbers affected are needed, this percentage change is then applied to the baseline rate for the health outcome i.e. the usual numbers of the health outcome per unit population without a change in air pollution. These baseline rates are sometimes routinely available, and if not directly available at the local level can be inferred if it is assumed the rate is the same as for a wider geographical region. Baseline rates for hospital admissions data as described below is an example of this. Other baseline rates may not be available routinely and come from a variety of sources as explained in Annex C.

We describe a further example in detail because we investigated but were not successful in finding an appropriate baseline rate. Myocardial infarction (heart attack) after short-term exposure did not therefore proceed to the next stage of preparing statements and it is useful to be clear why.

#### 4.1.1 Hospital Admissions

Disease-specific emergency hospital admissions for all ages in England were available online for the years 2014/15-2017/18 from the National Health Service (NHS Digital, [link](#)). To calculate annual figures that match the air pollution data, we used the average across these years was calculated. If the calculations were for a specific age group, we calculated age-specific emergency admissions by applying a scaling factor of age-specific Finished Consultant Episodes which were the only available online in the same link.

Moreover, only all-cause, city-specific emergency hospital admissions were available online ([link](#)) up to 2014/15. We calculated an average based on the 2012/13, 2013/14 and 2014/15 admissions. Then, to calculate cause-specific figures for all the cities, we assumed that the proportion of the cause-specific admissions over the total admissions is the same for all the UK cities (e.g. all respiratory emergency hospital admissions for those aged 65+ were estimated to contribute 7.62% to all emergency admissions in England, so we applied the same percentage to all the English cities).

There were some differences between the two datasets used for these approximations, probably due to the different time periods covered, so we decided to apply a correction factor in the estimated numbers. More specifically, number of admissions in England are 5,020,649 in the dataset with the region-specific estimates (2012-2015 average), while the corresponding number from the dataset with the disease- and age-specific (but no region) estimates is 5,841,004 (2014-2018 average). This is a 16.34% increase, so we applied a correction of 16.34% increase in the estimated city-specific values.

Finally, for London we used data from hospital episode statistics accessed via PHE for emergency hospital admissions, London residents, finished consultant episodes for the equivalent years. When we compared our estimates with these values the differences were relatively small (approximately 10% or less, except for asthma emergency admissions in children 0-14 years of age for which the real data were almost double the estimated ones).

Region-specific values were downloaded from this link: <https://digital.nhs.uk/data-and-information/publications/clinical-indicators/compendium-of-population-health-indicators/compendium-hospital-care/current/emergency-admissions/emergency-hospital-admissions-all-conditions-indirectly-standardised-rate-all-ages-annual-trend-f-m-p>

Disease- and age-specific values were downloaded from this link: <https://digital.nhs.uk/data-and-information/publications/statistical/hospital-admitted-patient-care-activity/>

#### **4.1.2 Myocardial infarction (short-term)**

Lanki et al (2006) used hospital admissions for first myocardial infarction. This is not routinely available – the standard statistics are for all myocardial infarction admissions. Initial investigation did not reveal data on the proportion of total myocardial infarction admissions that are for first MI. An alternative would be to use the baseline numbers of admissions for first MI from the study itself, convert these to a rate per unit population and assume these applied in the relevant cities. This would need population data for a specific age range (e.g. over 35 or 35-74 for some) in the study cities. This might be available with more investigation but not within the time constraints of this project.

Myocardial infarction (long-term). Cesaroni *et al* (2014) used both hospital admissions and mortality data sources to identify incidence of acute coronary events. Acute myocardial infarction and ‘other acute and sub-acute forms of ischaemic heart disease (ICD 10 I20.0, I21, I23 and I24) as an outcome for the hospital admissions data and deaths from ischaemic heart disease (I20-I25) as an outcome for the mortality data. The data could be linked to avoid double counting e.g. deaths where there was a hospital admission for an MI within 28 days of the death were excluded. Ideally, the baseline rates used would match these definitions. The study was analysed using Cox Proportional Hazards modelling. Follow-up varied by cohort from 3 years upwards. For incidence data care usually needs to be taken that the new cases are being calculated over the same time period for the health impacts as in the original study. For Cox proportional hazards modelling however, it is assumed in the analysis that the hazard ratio does not vary with age (increasing time). Thus, the same hazard ratio would apply to a 1-year period as to a longer period. Therefore, this can be applied to baseline rates for annual incidence.

## **4.2 Poland**

### **4.2.1 Data sources in Poland**

Data on baseline rates and populations were also available for Poland and as for the UK, we use hospital admissions as an example, with the remaining baseline rates for Poland described in Annex D.

Population data for Poland and Polish cities were downloaded from:  
<https://stat.gov.pl/en/topics/population/population/structure-of-the-population-by-2016,7,1.html>  
and from:  
<http://worldpopulationreview.com/world-cities/warsaw-population/>  
or from  
<http://population.city/poland/poznan/>

### **4.2.2 Emergency hospital admissions**

Data on the annual emergency hospital admissions for all the health outcomes included in the analysis except stroke in the four Polish cities were provided by the Polish Department of Population Health Monitoring and Analysis for 2015, 2016 and 2017. We used the average across the three years. For stroke admissions, we used data from London and calculated the number of admissions in the Polish cities by assuming that the percentage of stroke admissions over the total CVD emergency HA is the same as in London.

#### **4.2.3 Summary**

We were successful at obtaining baseline rates for most of the outcomes, although direct data was not always easily found in the time available. In these cases, baseline rates had to be assumed to be the same as that from another location or a wider geographical region. This could be improved upon in further work. One outcome was dropped – there was a lack of information on baseline rates for first myocardial infarction, although this might be resolvable with further time. Population at risk was available in many cases although not always using local data. The population living beside roads was only available for London but could be obtained for some other locations with separate research.

## 5 Personalised health statements

As will be clear from the earlier chapters, a lot of technical details about the inputs to health impact calculations need to be checked and need to match up e.g. the definition of the health outcome for the baseline rate needs to be the same as that in the original study for statements in terms of numbers of people affected. In addition, the statistical method of analysis in the original study needs to be understood to choose the correct method for calculating health impacts. Exact interpretation is also needed e.g. a study on new cases of disease cannot be assumed to mean annual new cases; it may be across the length of the study. This chapter only covers those health outcomes for which these steps have been checked and direct data, or reasonable approximations, found for the context of this project.

### 5.1 Scenarios and calculations

Before introducing the statements themselves, we need to describe the processes we used to define concepts like 'high' and 'low' pollution days and 'busy' and 'non-busy' roads. Quantitative statements need to be related to a specific concentration difference in pollutant levels. To give statements of interest, this concentration difference needs to come from a scenario that can be visualised. The following scenarios were used:

#### *Higher pollution days vs lower pollution days:*

We defined this by assuming that typical higher air pollution days were at the middle of the top half of the annual range of pollutant levels and typical lower air pollution days were at the middle of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations. We simplified the distribution to assume that the top half of the days were all at the 75<sup>th</sup> percentile level and the bottom half at the 25<sup>th</sup> percentile. We then did calculations for a hypothetical scenario where the days at the 75<sup>th</sup> percentile were reduced to the 25<sup>th</sup> percentile.

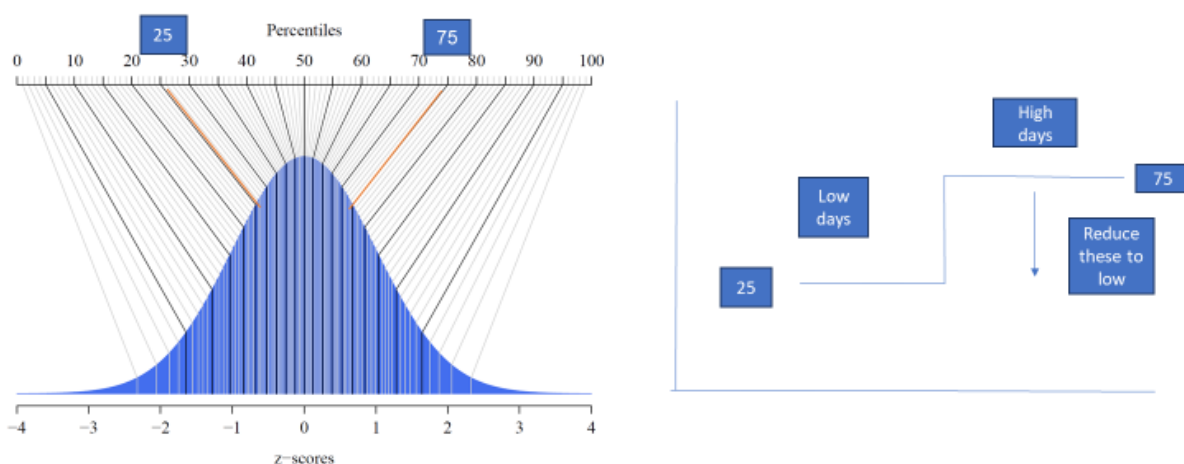


Figure 1. Schematic frequency distribution<sup>12</sup> illustrating the procedure for defining 'high' and 'low' pollution days and the associated scenario.

<sup>12</sup> Note this frequency distribution is purely illustrative; real air pollutant concentration frequency distributions are virtually all log-normal rather than normal.

### *Living near busy roads:*

This scenario used the difference between concentrations at monitoring sites close to busy roads compared with concentrations away from busy roads from London background monitoring sites. Even though exposure data from roadside and background monitoring stations were available for other cities as well (e.g. Birmingham, Bristol, etc. – see table **Table A3**), we could not collect any information for the addresses of the people living in these cities. Thus, we were able to produce statements only for the difference in risk for people living near busy roads compared with those living near quieter streets. This difference in risk was not able to be converted in attributable cases because we did not have an estimation of the population at risk.

For London, from an analysis in a previous project we had information from the Ordnance Survey Mastermap system regarding the numbers of people living near busy roads in the Greater London Authority (GLA) Area. For the numbers of health outcomes, we assumed that the relevant population at risk was those living within 50 meters of a road. More specifically, we created a buffer of 50m around major roads in London and calculated the number of all addresses within these buffers in the GLA area. This was estimated by an approximate method as 33% of the London population. Where the calculations of health outcomes was for children, we assumed the proportion of children of the specified age was the same as the proportion for the London population as a whole.

We have also used an arbitrary 20% reduction in concentrations. For Poland, 20% happened to be roughly equivalent to the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places.

### *Calculations:*

In order to produce these statements, we performed similar health impact calculations as the analysis done for the CAFE project and more specifically as described in the methodological report from Hurley et al (2005) for the Cost-Benefit analysis for CAFE. We had two main types of CRFs, i.e. relative risks mainly from Poisson regression, where the underlying population is very large, and the probability of the occurrence of a health outcome in one particular individual is very small, and odds ratios from logistic regression, where the probability of occurrence is approximated by the relative frequency of occurrences in the long run.

For relative risks of each health outcome, we transformed them to percentage changes for the adjusted changes in air pollution (following the calculations described in 3.1) and applied this percentage change to the baseline number of cases of the outcome. For odds ratios, we first transformed the odds (o) into probability (p) based on the following formula:

$$p = \frac{o}{1 + o}$$

or equivalently,  $o = p/(1-p)$ . Then, calculated the odds of the corresponding background rates for a health outcome using the formulas above. For example, we assumed the prevalence of bronchitic symptoms among asthmatic children to be 21.1% (McConnell et al 2003) which implies an odds of  $0.211/(1-0.211) = 0.2674$ . Using the odds ratios collected from the literature for each health endpoint, we calculated the new odds for the adjusted change in air pollution and subsequently, the new probability. Finally, we used this probability as the new baseline rate and calculated the change in the probability of the occurrence of a health outcome as the difference between new and original baseline rate.

## **5.2 Personalised health statements.**



In formulating the statements, we need to recognise that there are different types of studies of the health effects of air pollution. Not all forms of statements are appropriate for all types of studies e.g. statements relating to daily variations in pollutants are not suitable for studies based on spatial comparison of long-term averages. On the other hand, a particular form of statement may be applicable to several health outcomes and several pollutants.

For any one type of study, a variety of comparisons are possible e.g. high vs low air pollution day, comparison across cities, % increase in risk, numbers of people with the outcome.

Often there are concentration-response functions for several pollutants for the same health outcome. However, it may not be appropriate to do calculations for each pollutant and add them up. This is because the effects of the different pollutants are difficult to disentangle in the original studies. Because of their common sources, pollutants are often closely correlated. Thus, for example, in still weather both PM<sub>2.5</sub> and NO<sub>2</sub> might be high and in windy weather they may both be low. So, a study appearing to relate a health effect to NO<sub>2</sub> may actually also be reflecting the effects of PM<sub>2.5</sub> and *vice versa*. Adding the results derived from the two concentration-response functions would then involve double counting. Thus, we chose only one pollutant for each statement, usually the one that gave the largest answer after taking into account the concentration of the pollutant and the size of the concentration-response function. In the section with detailed justifications, the pollutant used is specified. There may be a small underestimate involved in doing this but to add the impacts from several pollutants which may be correlated could lead to a very much larger overestimate.

The statements are given as stand-alone statements for each health outcome. For summarising overall information, users might wish to add up numbers across different health outcomes. This is not always appropriate. Firstly, some of the health outcomes are sub-sets of other outcomes e.g. asthma admissions are included within all respiratory admissions, so adding them would be double counting. Secondly, outcomes are of different types. Adding up types of hospital admissions that are not subsets of each other is fine. Adding across out-of-hospital cardiac arrests and hospital admissions is less useful. Also, it is likely that for a given outcome, different populations might be affected, for example one couldn't add an effect in the elderly to one involving all ages, again there would be possible double counting. While the totals could be referred to as 'health events', if only the total is given, it becomes much more difficult for expert readers to work out the derivation of the numbers.

Another point to bear in mind when reading the statements, particularly for the effects of long-term exposure to pollutants on incidence of disease, is that multiple risk factors are involved in the development or worsening of the disease. If the results of a calculation indicate that air pollution increases numbers of people with disease, it does not necessarily mean that air pollution is the sole cause of that disease. Many steps may be required to lead to disease and air pollution may just provide one step. People may even actually already have had the disease but because air pollution makes it worse, it may be diagnosed when it had not been before.

Each of the statements comes together with a note. THIS IS AN ESSENTIAL PART OF THE STATEMENT. It allows readers to understand where the numbers in the statement come from. Communication materials should be able to incorporate a footnote to include this information.

### 5.3 Statements for London

We give below the details of the background information relevant to each statement. We will present a full description of the methods used in deriving the statements using London as an

example. These consist of two types of short statement followed by a technical justification. In the following sections 5.4 Statements for UK Cities and 5.5 Statements for Poland Cities , we present just the simplest statement with its accompanying note for the other UK cities and the Polish cities. We have used similar methods to derive these statements but have not at this stage provided the detailed technical justification in the same place. The information is still available in the annexes.

### 5.3.1 Out-of-hospital cardiac arrests

Out-of-hospital cardiac arrests refer to people whose hearts have stopped when they are not in hospital but are either at home or out in the street. Some but not all survive and are admitted to hospital. Estimates of baseline numbers come from ambulance call data.

#### High vs low pollution days, change in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Living in London, your risk of out-of-hospital cardiac arrest would be reduced by 2.2%, if higher pollution days were reduced to lower pollution days instead.**

*Note: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**Living in London, your risk of experiencing an out-of-hospital cardiac arrest would be reduced by 2.2%, if air pollution (PM<sub>2.5</sub>) was reduced by 37.2% on half the days of the year. This air pollution reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of out-of-hospital cardiac arrests than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in out-of-hospital cardiac arrests with a particular change in air pollution level. In this case, the concentration-response functions come from: Zhao et al (2017) (relative risk of 1.04 per 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> or equivalently a 4% increase in the risk of out-of-hospital cardiac arrest per 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>). These studies pooled together several other studies so are quite robust. The upper 95% confidence intervals for the relative risk was 1.07.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

PM<sub>2.5</sub> 25<sup>th</sup> percentile 9.1 µg/m<sup>3</sup>  
PM<sub>2.5</sub> 75<sup>th</sup> percentile 14.5 µg/m<sup>3</sup>

Change 5.4 µg/m<sup>3</sup> (37.2%)

Out-of-hospital cardiac arrests have been linked with other pollutants as well, such as PM<sub>10</sub>, NO<sub>2</sub>, O<sub>3</sub> and CO (see section B9. Out of hospital cardiac arrest). Calculations are only quoted for PM<sub>2.5</sub> and note that the concentrations at these percentiles will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Zhao et al. The impact of short-term exposure to air pollutants on the onset of out-of-hospital cardiac arrest: A systematic review and meta-analysis. International journal of cardiology 2017; Jan 1;226:110-117. doi: 10.1016/j.ijcard.2016.10.053

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### High vs low pollution days, change in numbers of admissions

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**If higher air pollution days in London were lower instead, we could avoid 87 cardiac arrests each year.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**There would be 87 fewer people experiencing out-of-hospital cardiac arrest if air pollution (PM<sub>2.5</sub>) was reduced by 37.2% on half the days of the year. This reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of out-of-hospital cardiac arrests than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in cardiac arrests with a particular change in air pollution level. In this case, the concentration-response functions come from: Zhao et al (2017) (relative risk of 1.04 per 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub> or equivalently a 4% increase in the risk of out-of-hospital cardiac arrest per 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>). These studies pooled together several other studies so are quite robust. The upper 95% confidence intervals for the relative risk was 1.07.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

PM<sub>2.5</sub> 25<sup>th</sup> percentile 9.1 µg/m<sup>3</sup>  
PM<sub>2.5</sub> 75<sup>th</sup> percentile 14.5 µg/m<sup>3</sup>

Change 5.4 µg/m<sup>3</sup> (37.2%)

The new percentage change was then applied to the baseline numbers of out-of-hospital cardiac arrests in London (British Heart Foundation 2015). In this case it was the annual number of 8,069 cases (averaged over the years 2014/15-2017/18; UK wide data scaled by population size) divided by 2 because the scenario changed concentration on half the days of the year. Out-of-hospital cardiac arrests have been linked with other pollutants as well, such as PM<sub>10</sub>, NO<sub>2</sub>, O<sub>3</sub> and CO (see section B9. Out of hospital cardiac arrest). Calculations were done separately for all pollutants but only quoted for PM<sub>2.5</sub> because the stronger health effects were found for this pollutant. Given the overlap in effects between pollutants we just used this number rather than adding the results.

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**Sources:** Zhao et al. The impact of short-term exposure to air pollutants on the onset of out-of-hospital cardiac arrest: A systematic review and meta-analysis. International journal of cardiology 2017; Jan 1;226:110-117. doi: 10.1016/j.ijcard.2016.10.053  
British Heart Foundation, Resuscitation Council UK. "Consensus Paper on Out-of-Hospital Cardiac Arrest in England." (2015)

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#### **Concentration-change roadside vs background, difference in risk**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Living near a busy road in London increases your risk of out-of-hospital cardiac arrest by 3.0%.**

*Note: Based on the difference between the middle of the range of air pollution levels at roadsides and the middle of the range of air pollution levels away from roads.*

*For text in articles; body of press releases etc*

**Living in London, your risk of out-of-hospital cardiac arrest would be reduced by 3.0%, if air pollution (NO<sub>2</sub>) was reduced by 49.1%. This air pollution reduction over time is the same as the difference between typical air pollution levels at a busy roadside location compared with at an urban background location<sup>1</sup>.**

<sup>1</sup> A typical air pollution day was defined as the middle of the range of air pollution levels in a year as measured at roadside or urban background monitoring stations within London.

**Justification:** This calculation is based on studies which show that higher air pollution concentrations can result in larger numbers of out-of-hospital cardiac arrests. These studies give us a concentration-response function – an equation relating a percentage increase in cardiac arrests with a particular change in air pollution level. In this case, the concentration-response functions come from: Zhao et al (2017) (relative risk of 1.02 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub> or equivalently a 2% increase in the risk of out-of-hospital cardiac arrest per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>). These studies pooled together several other studies so are quite robust. The upper 95% confidence intervals for the relative risk was 1.03.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the median value of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>) for the years 2015-2017 at roadside and urban background stations. We assumed that a typical air pollution day near busy roads in

London is the median (roughly the average of the range of concentrations) at roadside monitoring stations and all days were reduced to the median value at background stations.

NO<sub>2</sub> Median at roadside stations 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median at background stations 29.7 µg/m<sup>3</sup> Change 28.4 µg/m<sup>3</sup> (49.1%)

Out-of-hospital cardiac arrests have been linked with other pollutants as well, such as PM<sub>10</sub>, PM<sub>2.5</sub>, O<sub>3</sub> and CO (see section B9. Out of hospital cardiac arrest). Calculations are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

**Sources:** Zhao et al. The impact of short-term exposure to air pollutants on the onset of out-of-hospital cardiac arrest: A systematic review and meta-analysis. International journal of cardiology 2017; Jan 1;226:110-117. doi: 10.1016/j.ijcard.2016.10.053

### Concentration-change roadside vs background, difference in admission numbers

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Living near a busy road in London can contribute to 81 more out-of-hospital cardiac arrests each year.**

*Note: Based on the difference between the middle of the range of air pollution levels at roadsides and the middle of the range of air pollution levels away from roads over one year.*

*For text in articles; body of press releases etc*

**There would be 81 fewer people experiencing cardiac arrest if air pollution (NO<sub>2</sub>) was reduced by 49.1%. This reduction is the difference between typical air pollution days near roadside and background monitors<sup>1</sup>.**

<sup>1</sup> A typical air pollution day was defined as the middle of the range of air pollution levels in a year.

**Justification:** This calculation is based on studies which show that higher air pollution concentrations can result in larger numbers of out-of-hospital cardiac arrests. These studies give us a concentration-response function – an equation relating a percentage increase in cardiac arrests with a particular change in air pollution level. In this case, the concentration-response functions come from: Zhao et al (2017) (relative risk of 1.02 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub> or equivalently a 2% increase in the risk of out-of-hospital cardiac arrest per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>). These studies pooled together several other studies so are quite robust. The upper 95% confidence intervals for the relative risk was 1.03.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>).

NO<sub>2</sub> Median at roadside stations 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median at background stations 29.7 µg/m<sup>3</sup> Change 28.4 µg/m<sup>3</sup> (49.1%)

The new percentage change was then applied to the baseline numbers of out-of-hospital cardiac arrests in London (British Heart Foundation 2015). In this case it was the annual number of 8,069 cases (averaged over the years 2014/15-2017/18; UK wide data scaled by population size) divided by 2 because the scenario changed concentration on half the days of the year.

Out-of-hospital cardiac arrests have been linked with other pollutants as well, such as PM<sub>10</sub>, PM<sub>2.5</sub>, O<sub>3</sub> and CO (see section B9. Out of hospital cardiac arrest). Calculations are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Zhao et al. The impact of short-term exposure to air pollutants on the onset of out-of-hospital cardiac arrest: A systematic review and meta-analysis. International journal of cardiology 2017; Jan 1;226:110-117. doi: 10.1016/j.ijcard.2016.10.053  
British Heart Foundation, Resuscitation Council UK. "Consensus Paper on Out-of-Hospital Cardiac Arrest in England." (2015)

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### 5.3.2 Stroke

#### Stroke admissions

#### High vs low pollution days, change in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Living in London, your risk of emergency hospitalisation for stroke would be reduced by 2.7%, if higher pollution days were reduced to lower pollution days instead.**

*Note: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**Living in London, your risk of emergency hospitalisation for stroke would be reduced by 2.7%, if air pollution (NO<sub>2</sub>) was reduced by 22% on half the days of the year. This air pollution reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of emergency hospital admissions for stroke than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in stroke admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Shah et al (2015) (relative risk of 1.023 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub> or equivalently a 2.3% increase in the risk of out-of-hospital cardiac arrest per 10 µg/m<sup>3</sup> increase in

PM<sub>2.5</sub>). These studies pooled together several other studies so are quite robust. The upper 95% confidence intervals for the relative risk was 3.5%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

NO<sub>2</sub> 25<sup>th</sup> percentile 41.8 µg/m<sup>3</sup>

NO<sub>2</sub> 75<sup>th</sup> percentile 53.6 µg/m<sup>3</sup> Change 11.8 µg/m<sup>3</sup> (22.0%)

Stroke admissions have been linked with other pollutants as well, such as PM<sub>10</sub>, NO<sub>2</sub>, O<sub>3</sub> and CO (see section B6. Cerebrovascular Disease (Stroke)). Calculations are only quoted for NO<sub>2</sub> and note that the concentrations at these percentiles will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Shah *et al* (2015) Short term exposure to air pollution and stroke: systematic review and meta-analysis. BMJ. 2015 Mar 24;350:h1295. doi: 10.1136/bmj.h1295.

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## Stroke admissions

### High vs low pollution days, change in numbers of admissions

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**If higher air pollution days in London were lower instead, we could avoid 144 emergency hospital admissions for stroke each year.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**There would be 144 fewer people sent to hospital for stroke if air pollution (NO<sub>2</sub>) was reduced by 22% on half the days of the year. This reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of emergency hospital admissions for stroke than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in stroke admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Shah *et al* (2015) (relative risk of 1.023 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub> or equivalently a 2.3% increase in the risk of out-of-hospital cardiac arrest per 10 µg/m<sup>3</sup> increase in

PM<sub>2.5</sub>). These studies pooled together several other studies so are quite robust. The upper 95% confidence intervals for the relative risk was 3.5%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

NO<sub>2</sub> 25<sup>th</sup> percentile 41.8 µg/m<sup>3</sup>

NO<sub>2</sub> 75<sup>th</sup> percentile 53.6 µg/m<sup>3</sup> Change 11.8 µg/m<sup>3</sup> (22.0%)

The new percentage change was then applied to the baseline numbers of emergency hospital admissions for stroke in London. In this case it was the annual number of 10,610 cases (averaged over the years 2014/15-2017/18; PHE personal communication via NHS Digital) divided by 2 because the scenario changed concentration on half the days of the year. Stroke admissions have been linked with other pollutants as well, such as PM<sub>10</sub>, NO<sub>2</sub>, O<sub>3</sub> and CO (see section B6. Cerebrovascular Disease (Stroke)). Calculations were done separately for all pollutants are only quoted for NO<sub>2</sub> and note that the concentrations at these percentiles will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Shah *et al* (2015) Short term exposure to air pollution and stroke: systematic review and meta-analysis. BMJ. 2015 Mar 24;350:h1295. doi: 10.1136/bmj.h1295.

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## Stroke admissions

### Concentration-change roadside vs background, difference in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

### **Living near a busy road in London increases your risk of hospitalisation for stroke by 6.6%.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background).*

*For text in articles; body of press releases etc*

### **In London, air pollution may contribute to a 6.6% greater chance of hospitalisation for stroke if you live beside a polluted road compared with living on a quieter street. Stroke occurs through many steps and smoking is the major cause, but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background. Note that a 6.6% greater chance is not the same as a 6.6% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of emergency hospital admissions for stroke than days with lower pollution. These studies



give us a concentration-response function – an equation relating a percentage increase in stroke admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Shah et al (2015) (relative risk of 1.023 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub> or equivalently a 2.3% increase in the risk of out-of-hospital cardiac arrest per 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>). These studies pooled together several other studies so are quite robust. The upper 95% confidence intervals for the relative risk was 3.5%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>).

NO<sub>2</sub> Median at roadside stations 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median at background stations 29.7 µg/m<sup>3</sup> Change 28.6 µg/m<sup>3</sup> (49.1%)

Stroke admissions have been linked with other pollutants as well, such as PM<sub>10</sub>, NO<sub>2</sub>, O<sub>3</sub> and CO (see section B6. Cerebrovascular Disease (Stroke)). Calculations are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Shah *et al* (2015) Short term exposure to air pollution and stroke: systematic review and meta-analysis. BMJ. 2015 Mar 24;350:h1295. doi: 10.1136/bmj.h1295.

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## Stroke admissions

### Concentration-change roadside vs background, change in numbers of admissions

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

### **Living near a busy road in London may contribute to 230 hospital admissions for stroke each year.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background).*

*For text in articles; body of press releases etc*

### **In London, air pollution may contribute to 230 more emergency hospital admissions for stroke if you live beside a polluted road compared with living on a quieter street. Stroke occurs through many steps and smoking is the major cause, but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup>*Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background.*

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of emergency hospital admissions for stroke than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in stroke admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Shah et al (2015) (relative risk of 1.023 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub> or equivalently a 2.3% increase in the risk of out-of-hospital cardiac arrest per 10 µg/m<sup>3</sup> increase in

PM<sub>2.5</sub>). These studies pooled together several other studies so are quite robust. The upper 95% confidence intervals for the relative risk was 3.5%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>).

NO<sub>2</sub> Median at roadside stations 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median at background stations 29.7 µg/m<sup>3</sup> Change 28.6 µg/m<sup>3</sup> (49.1%)

The new percentage change was then applied to the baseline numbers of emergency hospital admissions for stroke in people living near busy roads in London, i.e. approximately 33% of the population. In this case it was the annual number of 10,610 cases (averaged over the years 2014/15-2017/18; PHE personal communication via NHS Digital) multiplied by 33%. Stroke admissions have been linked with other pollutants as well, such as PM<sub>10</sub>, NO<sub>2</sub>, O<sub>3</sub> and CO (see section B6. Cerebrovascular Disease (Stroke)). Calculations were done separately for all pollutants but only quoted for PM<sub>2.5</sub> because the stronger health effects were found for this pollutant. Given the overlap in effects between pollutants we just used this number rather than adding the results.

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**Sources:** Shah *et al* (2015) Short term exposure to air pollution and stroke: systematic review and meta-analysis. BMJ. 2015 Mar 24;350:h1295. doi: 10.1136/bmj.h1295.

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### **Stroke (first occurrence, all ages)**

#### **Concentration-change roadside vs background, difference in risk**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Living near busy roads in London may contribute to a 10.2% greater chance of stroke.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background).*

*For text in articles; body of press releases etc*

#### **In London, air pollution may contribute to a 10.2% greater chance of stroke if you live beside a polluted road compared with living on a quieter street. Stroke occurs through many steps and smoking is the major cause, but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup>*Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background. Note that a 10.2% greater chance is not the same as a 10.2% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that people from places with higher particulate (PM<sub>2.5</sub>) pollution have a higher risk of a first stroke. These studies give us a concentration-response function – an equation relating a change in risk of stroke with a particular change in air pollution level. In this case, the concentration-response function comes from: Stafoggia *et al* (2014) (19 % change in cases of cerebrovascular disease (stroke), all ages, per 5 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>). This study used pooled raw data from several

European cohorts so is quite robust, although the association for PM<sub>2.5</sub> is not quite statistically significant and the confidence intervals are wide (indicates uncertainty). The upper 95% confidence intervals for this study was 62% per 5 µg/m<sup>3</sup>.

The relative risk per 5 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>).

PM<sub>2.5</sub> Median at roadside stations 12.5 µg/m<sup>3</sup>

PM<sub>2.5</sub> Median at background stations 9.7 µg/m<sup>3</sup> Change 2.8 µg/m<sup>3</sup> (22.4%)

The incidence of cerebrovascular disease has been linked with other pollutants as well, such as PM<sub>10</sub> (see section B6. Cerebrovascular Disease (Stroke)). Calculations are only quoted for PM<sub>2.5</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Source:** Stafoggia *et al* (2014) 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 Sep;122(9):919-25. doi: 10.1289/ehp.1307301.

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### **Stroke first occurrence (all ages)**

#### **Concentration-change roadside vs background, change in numbers with first occurrence of stroke**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

### **Living near a busy road in London may contribute to 306 strokes each year.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background).*

*For text in articles; body of press releases etc*

### **In London, air pollution may contribute to 306 strokes if you live beside a polluted road compared with living on a quieter street. Stroke occurs through many steps and smoking is the major cause, but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup>*Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that people from places with higher particulate (PM<sub>2.5</sub>) pollution have a higher risk of stroke. These studies give us a concentration-response function – an equation relating a change in risk of stroke with a particular change in air pollution level. In this case, the concentration-response function comes from: Stafoggia *et al* (2014) (19 % change in cases of cerebrovascular disease (stroke), all ages, per 5 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>). This study used pooled raw data from several European cohorts so is quite robust, although the association for PM<sub>2.5</sub> is not quite statistically

significant and the confidence intervals are wide (indicates uncertainty). The upper 95% confidence intervals for this study was 62% per 5  $\mu\text{g}/\text{m}^3$ .

The relative risk per 5  $\mu\text{g}/\text{m}^3$  was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>).

PM<sub>2.5</sub> Median at roadside stations 12.5  $\mu\text{g}/\text{m}^3$

PM<sub>2.5</sub> Median at background stations 9.7  $\mu\text{g}/\text{m}^3$  Change 2.8  $\mu\text{g}/\text{m}^3$  (22.4%)

The new percentage change was then applied to the baseline numbers of incident stroke (first occurrence) in people living near busy roads in London, i.e. approximately 33% of the population. In this case it was the annual number of 9,063 cases (averaged over the years 2014/15-2017/18; UK wide data scaled by population size) multiplied by 33%. The incidence of cerebrovascular disease has been linked with other pollutants as well, such as PM<sub>10</sub> (see section B6. Cerebrovascular Disease (Stroke)). Calculations are only quoted for PM<sub>2.5</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

**Source:** Stafoggia *et al* (2014) 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 Sep;122(9):919-25. doi: 10.1289/ehp.1307301.

### 5.3.3 Asthma admissions in children

#### High vs low pollution days, change in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Living in London, if you are aged below 14 years, your risk of being admitted to hospital for asthma would be reduced by 4.2%, if higher pollution days were reduced to lower pollution days instead.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**Living in London, if you are aged below 14 years, your risk of being admitted to hospital for asthma would be reduced by 4.2%, if air pollution (NO<sub>2</sub>) was reduced by 22% on half the days of the year. This air pollution reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for asthma than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions

with a particular change in air pollution level. In this case, the concentration-response functions come from: Walton et al (2019) (Relative risk of 1.036 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub> (24-hour average), or equivalently 3.6% change in asthma admissions in children per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>). This study pooled together several other studies, so it is quite robust. The upper 95% confidence intervals of the percentage change for this study was 5.4%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

NO<sub>2</sub> 25<sup>th</sup> percentile 41.8 µg/m<sup>3</sup>

NO<sub>2</sub> 75<sup>th</sup> percentile 53.6 µg/m<sup>3</sup> Change 11.8 µg/m<sup>3</sup> (22.0%)

Asthma admissions in children have been linked with other pollutants as well, such as PM<sub>2.5</sub>, PM<sub>10</sub>, O<sub>3</sub> and SO<sub>2</sub> (see section B16. Asthma admissions in children (short-term)). Calculations were done separately for all pollutants but are only quoted for NO<sub>2</sub> and note that the concentrations at these percentiles will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Walton, H., Dajnak, D., Evangelopoulos, D., Fehcht, D., (2019). "Health Impact Assessment of Air Pollution on Asthma in London" from <http://www.erg.kcl.ac.uk/Research/home/projects/air-pollution-and-asthma-admissions-in-London.html>

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#### High vs low pollution days, change in numbers of admissions

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**If higher air pollution days in London were lower instead, we could avoid 74 hospital admissions each year for asthma in people aged below 14.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**There would be 74 fewer people aged below 14 admitted to hospital for asthma if air pollution (NO<sub>2</sub>) was reduced by 22.0% on half the days of the year. This reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for asthma than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions

with a particular change in air pollution level. In this case, the concentration-response functions come from: Walton et al (2019) (Relative risk of 1.036 per 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{NO}_2$ , or equivalently 3.6% change in asthma admissions in children per 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{NO}_2$ ). This study pooled together several other studies, so it is quite robust. The upper 95% confidence intervals of the percentage change for this study was 5.4%.

The percentage increase per 10  $\mu\text{g}/\text{m}^3$  was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

$\text{NO}_2$  25<sup>th</sup> percentile 41.8  $\mu\text{g}/\text{m}^3$

$\text{NO}_2$  75<sup>th</sup> percentile 53.6  $\mu\text{g}/\text{m}^3$

Change 11.8  $\mu\text{g}/\text{m}^3$  (22.0%)

The new percentage change was then applied to the baseline numbers of emergency hospital admissions for asthma in adults in London. In this case it was the annual number of 3,472 cases (averaged over the years 2014/15-2017/18; PHE personal communication via NHS Digital) divided by 2 because the scenario changed concentration on half the days of the year. Asthma admissions in children have been linked with other pollutants as well, such as  $\text{PM}_{2.5}$ ,  $\text{PM}_{10}$ ,  $\text{O}_3$  and  $\text{SO}_2$  (see section B16. Asthma admissions in children (short-term)). Calculations were done separately for all pollutants but are only quoted for  $\text{NO}_2$  and note that the concentrations at these percentiles will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Walton, H., Dajnak, D., Evangelopoulos, D., Fehcht, D.,. (2019). "Health Impact Assessment of Air Pollution on Asthma in London" from <http://www.erg.kcl.ac.uk/Research/home/projects/air-pollution-and-asthma-admissions-in-London.html>

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### 5.3.4 Asthma admissions in adults

#### High vs low pollution days, change in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**In London, the risk of adults being admitted to hospital for asthma would be reduced by 1.4%, if higher pollution days were reduced to lower pollution days instead.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*



**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for asthma than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Walton et al (2019) (Relative risk of 1.012 per 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{NO}_2$ , or equivalently 1.2% change in asthma admissions in adults per 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{NO}_2$ ). This study pooled together several other studies, so it is quite robust. The upper 95% confidence intervals of the

**Living in London, the risk of adults being admitted to hospital for asthma would be reduced by 1.4%, if air pollution ( $\text{NO}_2$ ) was reduced by 22.0% on half the days of the year. This air pollution reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

percentage change for this study was 2.3%.

The percentage increase per 10  $\mu\text{g}/\text{m}^3$  was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

$\text{NO}_2$  25<sup>th</sup> percentile 41.8  $\mu\text{g}/\text{m}^3$

$\text{NO}_2$  75<sup>th</sup> percentile 53.6  $\mu\text{g}/\text{m}^3$

Change 11.8  $\mu\text{g}/\text{m}^3$  (22.0%)

Asthma admissions in adults have been linked with other pollutants as well, such as  $\text{PM}_{10}$  and  $\text{O}_3$  (see section B17. Asthma admissions in adults (short-term)). Calculations are only quoted for  $\text{NO}_2$  and note that the concentrations at these percentiles will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher and statistically significant estimates in the headline statement rather than adding the results.

**Sources:** Sources: Walton, H., Dajnak, D., Evangelopoulos, D., Fehst, D.,. (2019). "Health Impact Assessment of Air Pollution on Asthma in London" from <http://www.erg.kcl.ac.uk/Research/home/projects/air-pollution-and-asthma-admissions-in-London.html>.

## High vs low pollution days, change in numbers of admissions

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**If higher air pollution days in London were lower instead, we could avoid 33 hospital admissions each year for asthma in adults.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**There would be 33 fewer adults admitted to hospital for asthma if air pollution (NO<sub>2</sub>) was reduced by 22.0% on half the days of the year. This reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for asthma than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Walton et al (2019) (Relative risk of 1.012 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>, or equivalently 1.2% change in asthma admissions in adults per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>). This study pooled together several other studies, so it is quite robust. The upper 95% confidence intervals of the percentage change for this study was 2.3%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

NO<sub>2</sub> 25<sup>th</sup> percentile 41.8 µg/m<sup>3</sup>

NO<sub>2</sub> 75<sup>th</sup> percentile 53.6 µg/m<sup>3</sup>

Change 11.8 µg/m<sup>3</sup> (22.0%)

The new percentage change was then applied to the baseline numbers of emergency hospital admissions for asthma in adults in London. In this case it was the annual number of 4,611 cases (averaged over the years 2014/15-2017/18; PHE personal communication via NHS Digital) divided by 2 because the scenario changed concentration on half the days of the year. Asthma admissions in adults have been linked with other pollutants as well, such as PM<sub>10</sub> and O<sub>3</sub> (see section B17. Asthma admissions in adults (short-term)). Calculations were done separately for all pollutants but are only quoted for NO<sub>2</sub> and note that the concentrations at these percentiles will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

**Sources:** Walton, H., Dajnak, D., Evangelopoulos, D., Fecht, D., (2019). "Health Impact Assessment of Air Pollution on Asthma in London" from <http://www.erg.kcl.ac.uk/Research/home/projects/air-pollution-and-asthma-admissions-in-London.html>.



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### 5.3.5 Reduced lung growth and low lung function

#### Concentration-change roadside vs background, difference in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Living near busy roads in London may contribute to an 8.7% greater chance of low lung function in children.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background).*

*For text in articles; body of press releases etc*

**In London, air pollution may contribute to an 8.7% greater chance of low lung function for children live beside a polluted road compared those living on a quieter street.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background. Note that an 8.7% greater chance is not the same as a 8.7% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that children from places with higher air pollution (NO<sub>2</sub>) have a higher risk of low lung function. These studies give us a concentration-response function – an equation relating a change in risk of low lung function with a particular change in air pollution level. In this case, the concentration-response function comes from: Gehring et al (2013) (Odds Ratio equal to 1.35 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>). This study used pooled raw data from several European cohorts so is quite robust. The upper 95% confidence intervals for this OR was 1.73 per 10 µg/m<sup>3</sup>.

The relative risk per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>)

NO<sub>2</sub> Median at roadside stations 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median at background stations 29.7 µg/m<sup>3</sup> Change 28.6 µg/m<sup>3</sup> (49.1%)

Low lung function has been linked with other pollutants as well, such as PM<sub>10</sub> and PM<sub>2.5</sub> (see section B14. Lung function in children). Calculations are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Gehring et al. Air pollution exposure and lung function in children: the ESCAPE project. Environ Health Perspect. 2013 Nov-Dec;121(11-12):1357-64. doi: 10.1289/ehp

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#### 20% reduction, difference in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Cutting air pollution in London by one fifth may contribute to a 2.3% greater chance of better lung function in children.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario.*

*For text in articles; body of press releases etc*

**In London, current air pollution (NO<sub>2</sub>) levels may contribute to a 2.3% greater chance of low lung function for children compared to a hypothetical scenario of a 20% reduced air pollution levels.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels across London compared to the same concentrations reduced by one fifth. Note that a 2.3% greater chance is not the same as a 2.3% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that children from places with higher air pollution (NO<sub>2</sub>) have a higher risk of low lung function. These studies give us a concentration-response function – an equation relating a change in risk of low lung function with a particular change in air pollution level. In this case, the concentration-response function comes from: Gehring et al (2013) (Odds Ratio equal to 1.35 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>). This study used pooled raw data from several European cohorts so is quite robust. The upper 95% confidence intervals for this OR was 1.73 per 10 µg/m<sup>3</sup>.

The relative risk per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range.

The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels.

Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>)

NO<sub>2</sub> Median across London 47.2 µg/m<sup>3</sup>

NO<sub>2</sub> Median hypothetical reduction 37.8 µg/m<sup>3</sup> Change 9.4 µg/m<sup>3</sup> (20%)

Low lung function has been linked with other pollutants as well, such as PM<sub>10</sub> and PM<sub>2.5</sub> (see section B14. Lung function in children). Calculations are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

**Sources:** Gehring et al. Air pollution exposure and lung function in children: the ESCAPE project. Environ Health Perspect. 2013 Nov-Dec;121(11-12):1357-64. doi: 10.1289/ehp

**20% reduction, attributable cases**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Cutting air pollution in London by one fifth may result in 7,927 fewer children with low lung function each year.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario.*

*For text in articles; body of press releases etc*

**In London, current air pollution (NO<sub>2</sub>) levels may result in 7,927 more children with low lung function compared to a hypothetical scenario of a 20% reduced air pollution levels.<sup>1</sup>**

<sup>1</sup> Based on the difference between long term average air pollution levels across London compared to the same concentrations reduced by one fifth.

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that children from places with higher air pollution (NO<sub>2</sub>) have a higher risk of low lung function. These studies give us a concentration-response function – an equation relating a change in risk of low lung function with a particular change in air pollution level. In this case, the concentration-response function comes from: Gehring et al (2013) (Odds Ratio equal to 1.35 per 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>). This study used pooled raw data from several European cohorts so is quite robust. The upper 95% confidence intervals for this OR was 1.73 per 10 µg/m<sup>3</sup>.

The relative risk per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels.

Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>)

NO<sub>2</sub> Median across London 47.2 µg/m<sup>3</sup>

NO<sub>2</sub> Median hypothetical reduction 37.8 µg/m<sup>3</sup> Change 9.4 µg/m<sup>3</sup> (20%)

The new percentage change was then applied to the baseline numbers and population at risk of low lung function in children aged 6 to 8 years old living in London. In this case, the population at risk was the annual number of 349,143 children (averaged over the years 2015-2017; Estimates of the population for the UK, England and Wales, Scotland and Northern Ireland, Office for National Statistics (ONS)). The prevalence of low lung function of 7.7 % was taken from the MAAS cohort (based in Manchester) data with Gehring et al (2013). Low lung function has been linked with other pollutants as well, such as PM<sub>10</sub> and PM<sub>2.5</sub> (see section B14. Lung function in children). Calculations are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

**Sources:** Gehring et al. Air pollution exposure and lung function in children: the ESCAPE project. Environ Health Perspect. 2013 Nov-Dec;121(11-12):1357-64. doi: 10.1289/ehp

#### **Concentration-change roadside vs background, difference in risk**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Living near busy roads in London may stunt lung growth in children by 12.5%.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background).*

**In London, air pollution may contribute to a 12.5% greater chance to stunt lung growth in children live beside a polluted road compared those living on a quieter street.<sup>1</sup>**

<sup>1</sup> Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span. Note that a 12.5% greater chance is not the same as a 12.5% chance – the absolute chance for an individual depends on other factors not just air pollution.

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that children from places with higher air pollution (NO<sub>2</sub>) have a higher risk of low lung function. These studies give us a concentration-response function – an equation relating a change in lung function growth with a particular change in air pollution level. In this case, the concentration-response function comes from: Gauderman et al (2015) (predicted change in the Forced Vital Capacity (FVC) in children aged 11 to 15 years old equal to 168.9 ml on average for every 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>). This study used raw data from the California Children's study which is an extensive long-term investigation. The upper 95% confidence intervals for this change was 210.7 ml per 10 µg/m<sup>3</sup>.

For the ideal lung growth from age 11 to 15 we used data from Quanjer et al 2012 (worked examples for calculating lung function using tables from their supplementary material - Caucasian ethnic group assumed). We used the average (for boys and girls) predicted change in FVC and calculated the percentage change in this growth for for every 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>. The average change per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and-2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>)

NO<sub>2</sub> Median at roadside stations 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median at background stations 29.7 µg/m<sup>3</sup> Change 28.6 µg/m<sup>3</sup> (49.1%)

Lung function growth has been linked with other pollutants as well, such as PM<sub>10</sub> and PM<sub>2.5</sub> (see section B15. Lung function growth (associated with long-term decrease in pollutants from the California Children's Health Study)). Calculations are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Gauderman et al. Association of improved air quality with lung development in children. N Engl J Med. 2015 Mar 5;372(10):905-13. doi: 10.1056/NEJMoa1414123

Quanjer PH, Stanojevic S, Cole TJ, Baur X, Hall GL, Culver BH, et al. Multi-ethnic reference values for spirometry for the 3–95-yr age range: the global lung function 2012 equations. European Respiratory Journal. 2012;40(6):1324-43

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**20% reduction, difference in risk**

For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.

**Cutting air pollution in London by one fifth would increase children's lung capacity by around 4.1%.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario.*

For text in articles; body of press releases etc

**In London, current air pollution (NO<sub>2</sub>) levels may contribute to a 4.1% greater chance of reduced children's lung capacity compared to a hypothetical scenario of a 20% reduced air pollution levels.<sup>1</sup>**

<sup>1</sup> Based on the difference between long term average air pollution levels across London compared to the same concentrations reduced by one fifth. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span. Note that a 4.1% greater chance is not the same as a 4.1% chance – the absolute chance for an individual depends on other factors not just air pollution.

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that children from places with higher air pollution (NO<sub>2</sub>) have a higher risk of low lung function. These studies give us a concentration-response function – an equation relating a change in lung function growth with a particular change in air pollution level. In this case, the concentration-response function comes from: Gauderman et al (2015) (predicted change in the Forced Vital Capacity (FVC) in children aged 11 to 15 years old equal to 168.9 ml on average for every 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>). This study used raw data from the California Children's study which is an extensive long-term investigation. The upper 95% confidence intervals for this change was 210.7 ml per 10 µg/m<sup>3</sup>.

For the ideal lung growth from age 11 to 15 we used data from Quanjer et al 2012 (worked examples for calculating lung function using tables from their supplementary material - Caucasian ethnic group assumed). We used the average (for boys and girls) predicted change in FVC and calculated the percentage change in this growth for for every 10 µg/m<sup>3</sup> increase in NO<sub>2</sub>. The average change per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>)  
NO<sub>2</sub> Median across London 47.2 µg/m<sup>3</sup>  
NO<sub>2</sub> Median hypothetical reduction 37.8 µg/m<sup>3</sup> Change 9.4 µg/m<sup>3</sup> (20%)

Lung function growth has been linked with other pollutants as well, such as PM<sub>10</sub> and PM<sub>2.5</sub> (see section B15. Lung function growth (associated with long-term decrease in pollutants from the California Children's Health Study)). Calculations are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

**Sources:** Gauderman et al. Association of improved air quality with lung development in children. N Engl J Med. 2015 Mar 5;372(10):905-13. doi: 10.1056/NEJMoa1414123

### 5.3.6 Lung cancer

#### Concentration-change roadside vs background, difference in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Living near busy roads in London may contribute to a 9.7% greater chance of developing lung cancer.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background). Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.*

*For text in articles; body of press releases etc*

#### **In London, air pollution may contribute to a 9.7% greater chance of developing lung cancer if you live beside a polluted road compared with living on a quieter street. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background. Note that a 9.7% greater chance is not the same as a 9.7% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that people from places with higher particulate (PM<sub>2.5</sub>) pollution have a higher risk of lung cancer. These studies give us a concentration-response function – an equation relating a change in risk of lung cancer with a particular change in air pollution level. In this case, the concentration-response function comes from: Raaschou-Nielsen et al (2013) (18 % change in cases of lung cancer, all ages, per 5 µg/m<sup>3</sup> PM<sub>2.5</sub>). This study used pooled raw data from several European cohorts so is quite robust, although the association for PM<sub>2.5</sub> is not quite statistically significant and the confidence intervals are wide (indicates uncertainty). The upper 95% confidence intervals for this study was 46% per 5 µg/m<sup>3</sup>. This outcome has not been quantified before, although lung cancer mortality rather than incidence has been quantified for the Global Burden of Disease [https://www.who.int/gho/phe/outdoor air pollution/burden text/en/](https://www.who.int/gho/phe/outdoor_air_pollution/burden_text/en/).

The relative risk per 5 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and-2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>)

PM<sub>2.5</sub> Median at roadside stations 12.5 µg/m<sup>3</sup>

PM<sub>2.5</sub> Median at background stations 9.7 µg/m<sup>3</sup> Change 2.8 µg/m<sup>3</sup> (22.4%)

Calculations are only quoted for PM<sub>2.5</sub> and note that the median concentrations will be different in other cities. This is the most relevant pollutant for lung cancer as it is known to contain polycyclic



aromatic hydrocarbons which are known carcinogens. For the upper confidence intervals, the reduction in risk was 24% for PM<sub>2.5</sub>.

**Source:** Raaschou-Nielsen *et al* (2013) 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](#).14(9):813-22.

IARC (2010) IARC monographs on the evaluation of carcinogenic risks to humans Volume 92 Some Non-heterocyclic Polycyclic Aromatic Hydrocarbons and Some Related Exposures <https://monographs.iarc.fr/iarc-monographs-on-the-evaluation-of-carcinogenic-risks-to-humans-29/>

IARC (2014) IARC monographs on the evaluation of carcinogenic risks to humans Volume 105 Diesel and Gasoline Engine Exhausts and Some Nitroarenes <https://monographs.iarc.fr/iarc-monographs-on-the-evaluation-of-carcinogenic-risks-to-humans-11/>

Pope, C. A., 3rd, R. T. Burnett, M. J. Thun, E. E. Calle, D. Krewski, K. Ito and G. D. Thurston (2002). "Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution." *JAMA* 287(9): 1132-1141.

**Comment:** The carcinogenicity of polycyclic aromatic hydrocarbons is well established (IARC, 2010) More recently it has been concluded that diesel exhaust is carcinogenic to humans (IARC (2014) The PM<sub>2.5</sub> from diesel contains polycyclic aromatic hydrocarbons. Previous epidemiological findings have also shown links between PM<sub>2.5</sub> and lung cancer (Pope *et al* 2002). There are still uncertainties – as lung cancer is relatively rare very large studies are needed so confidence intervals are often wide. There have not been wide international discussions of quantifying lung cancer incidence. There may be more recently published individual studies but no new study pooling raw data from groups of European studies together.

It is particularly important not to give the impression that air pollution is the sole cause of these lung cancer cases. If the numbers were added to those caused by smoking (a dominant cause), the total could add up to more than the total number of lung cancer cases.

#### **Concentration-change roadside vs background, attributable cases**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Living near busy roads in London may contribute to 390 lung cancer cases.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background). Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.*

*For text in articles; body of press releases etc*

**In London, air pollution may contribute to 390 more lung cancer cases if you live beside a polluted road compared with living on a quieter street. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that people from places with higher particulate (PM<sub>2.5</sub>) pollution have a higher risk of lung cancer. These studies give us a concentration-response function – an equation relating a change in risk of lung cancer with a particular change in air pollution level. In this case, the concentration-response function comes from: Raaschou-Nielsen *et al* (2013) (18 % change in cases of lung cancer,

all ages, per 5  $\mu\text{g}/\text{m}^3$   $\text{PM}_{2.5}$ ). This study used pooled raw data from several European cohorts so is quite robust, although the association for  $\text{PM}_{2.5}$  is not quite statistically significant and the confidence intervals are wide (indicates uncertainty). The upper 95% confidence intervals for this study was 46% per 5  $\mu\text{g}/\text{m}^3$ . This outcome has not been quantified before, although lung cancer mortality rather than incidence has been quantified for the Global Burden of Disease [https://www.who.int/gho/phe/outdoor\\_air\\_pollution/burden\\_text/en/](https://www.who.int/gho/phe/outdoor_air_pollution/burden_text/en/).

The relative risk per 5  $\mu\text{g}/\text{m}^3$  was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>)

$\text{PM}_{2.5}$  Median at roadside stations 12.5  $\mu\text{g}/\text{m}^3$

$\text{PM}_{2.5}$  Median at background stations 9.7  $\mu\text{g}/\text{m}^3$  Change 2.8  $\mu\text{g}/\text{m}^3$  (22.4%)

The new percentage change was then applied to the baseline numbers of lung cancer cases in London. In this case it was the annual number of 4,017 cases (averaged over the years 2015-2017; Cancer registration statistics, England, Office for National Statistics (ONS)). Calculations are only quoted for  $\text{PM}_{2.5}$  and note that the median concentrations will be different in other cities. This is the most relevant pollutant for lung cancer as it is known to contain polycyclic aromatic hydrocarbons which are known carcinogens. For the upper confidence intervals, the reduction in risk was 24% for  $\text{PM}_{2.5}$ .

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**Source:** Raaschou-Nielsen *et al* (2013) 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](https://doi.org/10.1016/j.lancet.2013.08.013). 14(9):813-22.

IARC (2010) IARC monographs on the evaluation of carcinogenic risks to humans Volume 92 Some Non-heterocyclic Polycyclic Aromatic Hydrocarbons and Some Related Exposures

<https://monographs.iarc.fr/iarc-monographs-on-the-evaluation-of-carcinogenic-risks-to-humans-29/>

IARC (2014) IARC monographs on the evaluation of carcinogenic risks to humans Volume 105 Diesel and Gasoline Engine Exhausts and Some Nitroarenes <https://monographs.iarc.fr/iarc-monographs-on-the-evaluation-of-carcinogenic-risks-to-humans-11/>

Pope, C. A., 3rd, R. T. Burnett, M. J. Thun, E. E. Calle, D. Krewski, K. Ito and G. D. Thurston (2002).

"Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution." *JAMA* 287(9): 1132-1141.

**Comment:** The carcinogenicity of polycyclic aromatic hydrocarbons is well established (IARC, 2010) More recently it has been concluded that diesel exhaust is carcinogenic to humans (IARC (2014) The  $\text{PM}_{2.5}$  from diesel contains polycyclic aromatic hydrocarbons. Previous epidemiological findings have also shown links between  $\text{PM}_{2.5}$  and lung cancer (Pope *et al* 2002). There are still uncertainties – as lung cancer is relatively rare very large studies are needed so confidence intervals are often wide. There have not been wide international discussions of quantifying lung cancer incidence. There may be more recently published individual studies but no new study pooling raw data from groups of European studies together.

It is particularly important not to give the impression that air pollution is the sole cause of these lung cancer cases. If the numbers were added to those caused by smoking (a dominant cause), the total could add up to more than the total number of lung cancer cases.

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## 20% reduction, difference in risk

For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.

### **Cutting air pollution in London by one fifth would decrease lung cancer cases by around 7.6%.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.*

For text in articles; body of press releases etc

### **In London, current levels of air pollution may contribute to a 7.6% greater chance of developing lung cancer compared with a hypothetical scenario of 20% reduction in pollution levels. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup> Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Note that a 7.6% greater chance is not the same as a 7.6% chance – the absolute chance for an individual depends on other factors not just air pollution.

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that people from places with higher particulate (PM<sub>2.5</sub>) pollution have a higher risk of lung cancer. These studies give us a concentration-response function – an equation relating a change in risk of lung cancer with a particular change in air pollution level. In this case, the concentration-response function comes from: Raaschou-Nielsen et al (2013) (18 % change in cases of lung cancer, all ages, per 5 µg/m<sup>3</sup> PM<sub>2.5</sub>). This study used pooled raw data from several European cohorts so is quite robust, although the association for PM<sub>2.5</sub> is not quite statistically significant and the confidence intervals are wide (indicates uncertainty). The upper 95% confidence intervals for this study was 46% per 5 µg/m<sup>3</sup>. This outcome has not been quantified before, although lung cancer mortality rather than incidence has been quantified for the Global Burden of Disease [https://www.who.int/gho/phe/outdoor air pollution/burden text/en/](https://www.who.int/gho/phe/outdoor%20air%20pollution/burden%20text/en/).

The relative risk per 5 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>)  
PM<sub>2.5</sub> Median across London 11.1 µg/m<sup>3</sup>  
PM<sub>2.5</sub> Median hypothetical reduction 8.9 µg/m<sup>3</sup> Change 2.2 µg/m<sup>3</sup> (20%)

Calculations are only quoted for PM<sub>2.5</sub> and note that the median concentrations will be different in other cities. This is the most relevant pollutant for lung cancer as it is known to contain polycyclic aromatic hydrocarbons which are known carcinogens. For the upper confidence intervals, the reduction in risk was 24% for PM<sub>2.5</sub>.

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**Source:** Raaschou-Nielsen et al (2013) 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](https://doi.org/10.1016/S1473-3099(13)70781-9).14(9):813-22.

IARC (2010) IARC monographs on the evaluation of carcinogenic risks to humans Volume 92 Some Non-heterocyclic Polycyclic Aromatic Hydrocarbons and Some Related Exposures <https://monographs.iarc.fr/iarc-monographs-on-the-evaluation-of-carcinogenic-risks-to-humans-29/>  
IARC (2014) IARC monographs on the evaluation of carcinogenic risks to humans Volume 105 Diesel and Gasoline Engine Exhausts and Some Nitroarenes <https://monographs.iarc.fr/iarc-monographs-on-the-evaluation-of-carcinogenic-risks-to-humans-11/>  
Pope, C. A., 3rd, R. T. Burnett, M. J. Thun, E. E. Calle, D. Krewski, K. Ito and G. D. Thurston (2002). "Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution." *JAMA* 287(9): 1132-1141.

**Comment:** The carcinogenicity of polycyclic aromatic hydrocarbons is well established (IARC, 2010) More recently it has been concluded that diesel exhaust is carcinogenic to humans (IARC (2014) The PM<sub>2.5</sub> from diesel contains polycyclic aromatic hydrocarbons. Previous epidemiological findings have also shown links between PM<sub>2.5</sub> and lung cancer (Pope et al 2002). There are still uncertainties – as lung cancer is relatively rare very large studies are needed so confidence intervals are often wide. There have not been wide international discussions of quantifying lung cancer incidence. There may be more recently published individual studies but no new study pooling raw data from groups of European studies together.

It is particularly important not to give the impression that air pollution is the sole cause of these lung cancer cases. If the numbers were added to those caused by smoking (a dominant cause), the total could add up to more than the total number of lung cancer cases.

#### **Concentration-change roadside vs background, attributable cases**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Cutting air pollution in London by one fifth would result in 306 fewer lung cancer cases each year.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.*

*For text in articles; body of press releases etc*

**In London, current levels of air pollution may contribute to 306 more lung cancer cases compared with a hypothetical scenario of 20% reduction in pollution levels. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that people from places with higher particulate (PM<sub>2.5</sub>) pollution have a higher risk of lung cancer. These studies give us a concentration-response function – an equation relating a change in risk of lung cancer with a particular change in air pollution level. In this case, the concentration-response function comes from: Raaschou-Nielsen et al (2013) (18 % change in cases of lung cancer, all ages, per 5 µg/m<sup>3</sup> PM<sub>2.5</sub>). This study used pooled raw data from several European cohorts so is quite robust, although the association for PM<sub>2.5</sub> is not quite statistically significant and the confidence intervals are wide (indicates uncertainty). The upper 95% confidence intervals for this

study was 46% per 5  $\mu\text{g}/\text{m}^3$ . This outcome has not been quantified before, although lung cancer mortality rather than incidence has been quantified for the Global Burden of Disease [https://www.who.int/gho/phe/outdoor\\_air\\_pollution/burden\\_text/en/](https://www.who.int/gho/phe/outdoor_air_pollution/burden_text/en/).

The relative risk per 5  $\mu\text{g}/\text{m}^3$  was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>)

PM<sub>2.5</sub> Median across London 11.1  $\mu\text{g}/\text{m}^3$

PM<sub>2.5</sub> Median hypothetical reduction 8.9  $\mu\text{g}/\text{m}^3$  Change 2.2  $\mu\text{g}/\text{m}^3$  (20%)

The new percentage change was then applied to the baseline numbers of lung cancer cases in London. In this case it was the annual number of 4,017 cases (averaged over the years 2015-2017; Cancer registration statistics, England, Office for National Statistics (ONS)). Calculations are only quoted for PM<sub>2.5</sub> and note that the median concentrations will be different in other cities. This is the most relevant pollutant for lung cancer as it is known to contain polycyclic aromatic hydrocarbons which are known carcinogens. For the upper confidence intervals, the reduction in risk was 24% for PM<sub>2.5</sub>.

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**Source:** Raaschou-Nielsen *et al* (2013) 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](https://doi.org/10.1016/j.lancet.2013.08.013).14(9):813-22.

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<https://monographs.iarc.fr/iarc-monographs-on-the-evaluation-of-carcinogenic-risks-to-humans-29/>

IARC (2014) IARC monographs on the evaluation of carcinogenic risks to humans Volume 105 Diesel and Gasoline Engine Exhausts and Some Nitroarenes <https://monographs.iarc.fr/iarc-monographs-on-the-evaluation-of-carcinogenic-risks-to-humans-11/>

Pope, C. A., 3rd, R. T. Burnett, M. J. Thun, E. E. Calle, D. Krewski, K. Ito and G. D. Thurston (2002).

"Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution." [JAMA](https://doi.org/10.1001/jama.287.9.1132) 287(9): 1132-1141.

**Comment:** The carcinogenicity of polycyclic aromatic hydrocarbons is well established (IARC, 2010) More recently it has been concluded that diesel exhaust is carcinogenic to humans (IARC (2014) The PM<sub>2.5</sub> from diesel contains polycyclic aromatic hydrocarbons. Previous epidemiological findings have also shown links between PM<sub>2.5</sub> and lung cancer (Pope et al 2002). There are still uncertainties – as lung cancer is relatively rare very large studies are needed so confidence intervals are often wide. There have not been wide international discussions of quantifying lung cancer incidence. There may be more recently published individual studies but no new study pooling raw data from groups of European studies together.

It is particularly important not to give the impression that air pollution is the sole cause of these lung cancer cases. If the numbers were added to those caused by smoking (a dominant cause), the total could add up to more than the total number of lung cancer cases.

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### 5.3.7 Asthma symptoms in children

#### High vs low pollution days, change in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**In London on high air pollution days, the risk of asthmatic children suffering asthma symptoms is 0.3% more than on lower pollution days.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.*

*For text in articles; body of press releases etc*

**Living in London, if you are aged below 14, your risk of suffering asthma symptoms would be reduced by 0.3%, if air pollution (PM<sub>10</sub>) was reduced by 27.6% on half the days of the year. This air pollution reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>. Asthmatic symptoms include cough, wheeze and breathlessness.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of asthmatic symptoms in asthmatic children than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in asthma symptoms with a particular change in air pollution level. In this case, the concentration-response functions come from: WHO (2013b) based on Weinmayr et al (2010) (Relative risk of 1.028 per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>, or equivalently 2.8% increased risk of experiencing asthma symptoms per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>). This CRF is recommended by the WHO HRAPIE report (2013b). The upper 95% confidence intervals for this estimate was 5.1%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

PM<sub>10</sub> 25<sup>th</sup> percentile 17.8 µg/m<sup>3</sup>

PM<sub>10</sub> 75<sup>th</sup> percentile 24.6 µg/m<sup>3</sup>

Change 6.8 µg/m<sup>3</sup> (27.6%)

Calculations were done for PM<sub>10</sub> and note that the concentrations at these percentiles will be different in other cities.

**Sources:** Weinmayr et al (2010) Short-term effects of PM<sub>10</sub> and NO<sub>2</sub> on respiratory health among children with asthma or asthma-like symptoms: a systematic review and meta-analysis. Environ Health Perspect. 2010 Apr;118(4):449-57. doi: 10.1289/ehp.0900844.

**Concentration-change roadside vs background, difference in risk**

For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.

**In London on high air pollution days, 142 more children with asthma experience asthma symptoms than on lower pollution days.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.*

For text in articles; body of press releases etc

**There would be 142 fewer children with asthma symptoms if air pollution (PM<sub>10</sub>) was reduced by 27.6% on half the days of the year. This reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of asthmatic symptoms in asthmatic children than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in asthma symptoms with a particular change in air pollution level. In this case, the concentration-response functions come from: WHO (2013b) based on Weinmayr et al (2010) (Relative risk of 1.028 per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>, or equivalently 2.8% increased risk of experiencing asthma symptoms per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>). This CRF is recommended by the WHO HRAPIE report (2013b). The upper 95% confidence intervals for this estimate was 5.1%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

PM<sub>10</sub> 25<sup>th</sup> percentile 17.8 µg/m<sup>3</sup>

PM<sub>10</sub> 75<sup>th</sup> percentile 24.6 µg/m<sup>3</sup>

Change 6.8 µg/m<sup>3</sup> (27.6%)

The new percentage change was then applied to the baseline numbers of children aged 5 to 14 years old in London. We assumed an asthma prevalence of 10% in this population (Lai et al 2009). In this case it was the annual number of 1,067,956 children in London (averaged over the years 2014/15-2017/18; Estimates of the population for the UK, England and Wales, Scotland and Northern Ireland, Office for National Statistics (ONS)) divided by 2 because the scenario changed concentration on half the days of the year. Calculations were done for PM<sub>10</sub> and note that the concentrations at these percentiles will be different in other cities.

**Sources:** Weinmayr et al (2010) Short-term effects of PM<sub>10</sub> and NO<sub>2</sub> on respiratory health among children with asthma or asthma-like symptoms: a systematic review and meta-analysis. Environ Health Perspect. 2010 Apr;118(4):449-57. doi: 10.1289/ehp.0900844.

### 5.3.8 Term low birthweight

#### Concentration-change roadside vs background, difference in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Living near busy roads in London may contribute to a 0.4% greater risk of babies being born underweight.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background). Babies born underweight refers to babies born at term with a birthweight less than 2,500g.*

*For text in articles; body of press releases etc*

#### **In London, air pollution may contribute to a 0.4% greater risk of babies being born underweight if mothers live beside a polluted road compared with living on a quieter street. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background. Note that a 0.4% greater chance is not the same as a 0.4% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that mothers from places with higher nitrogen dioxide (NO<sub>2</sub>) have a higher risk of giving birth to underweight babies. These studies give us a concentration-response function – an equation relating a change in risk of low birthweight with a particular change in air pollution level. In this case, the concentration-response function comes from: Pedersen et al (2013) (9 % change in cases of low birthweight per 10 ppb (NO<sub>2</sub>). This study used pooled raw data from several European cohorts within the ESCAPE study so is quite robust. The upper 95% confidence intervals for this study was 19% per 10 ppb.

The relative risk per 10 ppb was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>)

NO<sub>2</sub> Median at roadside stations 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median at background stations 29.7 µg/m<sup>3</sup> Change 28.6 µg/m<sup>3</sup> (49.1%)

Low birthweight has been linked with other pollutants as well, such as PM<sub>2.5</sub>, PM<sub>10</sub>, CO and SO<sub>2</sub> (see section B20. (Term) Low birthweight). Calculations were done separately for all pollutants but are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results (in fact, the estimates for PM<sub>2.5</sub> were the highest but because data for roadside and background stations were not available for all the cities we used the NO<sub>2</sub> CRF).

**Source:** Pedersen *et al* (2013) Ambient air pollution and low birthweight: a European cohort study (ESCAPE). *Lancet Respir Med.* 2013 Nov;1(9):695-704. doi: 10.1016/S2213-2600(13)70192-9.



### Concentration-change roadside vs background, attributable cases

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Living near busy roads in London may contribute to 144 babies born underweight each year.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background). Babies born underweight refers to babies born at term with a birthweight less than 2,500g.*

*For text in articles; body of press releases etc*

#### **In London, air pollution may contribute to 144 babies born underweight each year if mothers live beside a polluted road compared with living on a quieter street. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that mothers from places with higher nitrogen dioxide (NO<sub>2</sub>) have a higher risk of giving birth to underweight babies. These studies give us a concentration-response function – an equation relating a change in risk of low birthweight with a particular change in air pollution level. In this case, the concentration-response function comes from: Pedersen et al (2013) (9 % change in cases of low birthweight per 10 ppb (NO<sub>2</sub>). This study used pooled raw data from several European cohorts within the ESCAPE study so is quite robust. The upper 95% confidence intervals for this study was 19% per 10 ppb.

The relative risk per 10 ppb was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>)

NO<sub>2</sub> Median at roadside stations 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median at background stations 29.7 µg/m<sup>3</sup> Change 28.6 µg/m<sup>3</sup> (49.1%)

The new percentage change was then applied to the baseline numbers of total term births in London assuming a prevalence of 2.8% for term low birthweight (see section C1. Term low birthweight). In this case it was the annual number of 114,926 term births (averaged over the years 2015-2017; Birth characteristics, Office for National Statistics (ONS)). Low birthweight has been linked with other pollutants as well, such as PM<sub>2.5</sub>, PM<sub>10</sub>, CO and SO<sub>2</sub> (see section B20. (Term) Low birthweight). Calculations were done separately for all pollutants but are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results (in fact, the estimates for PM<sub>2.5</sub> were the highest but because data for roadside and background stations were not available for all the cities we used the NO<sub>2</sub> CRF).

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**Source:** Pedersen *et al* (2013) Ambient air pollution and low birthweight: a European cohort study (ESCAPE). *Lancet Respir Med.* 2013 Nov;1(9):695-704. doi: 10.1016/S2213-2600(13)70192-9.

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## 20% reduction, difference in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

### **Cutting air pollution in London by one fifth would decrease low birthweights by around 0.1%.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.*

*For text in articles; body of press releases etc*

### **In London, current levels of air pollution may contribute to a 0.1% greater chance of babies born underweight compared with a hypothetical scenario of 20% reduction in pollution levels. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Note that a 0.1% greater chance is not the same as a 0.1% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that mothers from places with higher nitrogen dioxide (NO<sub>2</sub>) have a higher risk of giving birth to underweight babies. These studies give us a concentration-response function – an equation relating a change in risk of low birthweight with a particular change in air pollution level. In this case, the concentration-response function comes from: Pedersen et al (2013) (9 % change in cases of low birthweight per 10 ppb (NO<sub>2</sub>). This study used pooled raw data from several European cohorts within the ESCAPE study so is quite robust. The upper 95% confidence intervals for this study was 19% per 10 ppb.

The relative risk per 10 ppb was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>)

NO<sub>2</sub> Median across London 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median hypothetical reduction 37.8 µg/m<sup>3</sup> Change 9.4 µg/m<sup>3</sup> (20%)

Low birthweight has been linked with other pollutants as well, such as PM<sub>2.5</sub>, PM<sub>10</sub>, CO and SO<sub>2</sub> (see section B20. (Term) Low birthweight). Calculations were done separately for all pollutants but are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results (in fact, the estimates for PM<sub>2.5</sub> were the highest but because data for roadside and background stations were not available for all the cities we used the NO<sub>2</sub> CRF).

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**Source:** Pedersen *et al* (2013) Ambient air pollution and low birthweight: a European cohort study (ESCAPE). *Lancet Respir Med.* 2013 Nov;1(9):695-704. doi: 10.1016/S2213-2600(13)70192-9.

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### Concentration-change roadside vs background, attributable cases

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Cutting air pollution in London by one fifth would result in 138 fewer babies born underweight each year.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.*

*For text in articles; body of press releases etc*

#### **In London, current levels of air pollution may contribute to 138 more babies born underweight compared with a hypothetical scenario of 20% reduction in pollution levels. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.<sup>1</sup>**

<sup>1</sup> Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario.

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that mothers from places with higher nitrogen dioxide (NO<sub>2</sub>) have a higher risk of giving birth to underweight babies. These studies give us a concentration-response function – an equation relating a change in risk of low birthweight with a particular change in air pollution level. In this case, the concentration-response function comes from: Pedersen et al (2013) (9 % change in cases of low birthweight per 10 ppb (NO<sub>2</sub>). This study used pooled raw data from several European cohorts within the ESCAPE study so is quite robust. The upper 95% confidence intervals for this study was 19% per 10 ppb.

The relative risk per 10 ppb was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>)

NO<sub>2</sub> Median across London 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median hypothetical reduction 37.8 µg/m<sup>3</sup> Change 9.4 µg/m<sup>3</sup> (20%)

The new percentage change was then applied to the baseline numbers of total term births in London assuming a prevalence of 2.8% for term low birthweight. In this case it was the annual number of 114,926 term births (averaged over the years 2015-2017; Birth characteristics, Office for National Statistics (ONS)). Low birthweight has been linked with other pollutants as well, such as PM<sub>2.5</sub>, PM<sub>10</sub>, CO and SO<sub>2</sub> (see section B20. (Term) Low birthweight). Calculations were done separately for all pollutants but are only quoted for NO<sub>2</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results (in fact, the estimates for PM<sub>2.5</sub> were the highest but because data for roadside and background stations were not available for all the cities we used the NO<sub>2</sub> CRF).

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**Source:** Pedersen *et al* (2013) Ambient air pollution and low birthweight: a European cohort study (ESCAPE). *Lancet Respir Med.* 2013 Nov;1(9):695-704. doi: 10.1016/S2213-2600(13)70192-9.

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### 5.3.9 Respiratory admissions all ages

#### High vs low pollution days, change in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Living in London, your risk of being admitted to hospital for respiratory disease would be reduced by 1.4%, if higher pollution days were reduced to lower pollution days instead.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**Living in London, your risk of being admitted to hospital for respiratory disease would be reduced by 1.4%, if air pollution (O<sub>3</sub>) was reduced by 34.0% on half the days of the year. This air pollution reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for respiratory disease than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: COMEAP (2015) (0.75% change in respiratory admissions, all ages, per 10 µg/m<sup>3</sup> increase in O<sub>3</sub> (daily 8-hour maximum)). This study pooled together several other studies so are quite robust and the CRF reported is recommended by the COMEAP. The upper 95% confidence intervals for this estimate was 1.2%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

O<sub>3</sub> 25<sup>th</sup> percentile 35.7 µg/m<sup>3</sup>

O<sub>3</sub> 75<sup>th</sup> percentile 54.1 µg/m<sup>3</sup>                      Change 18.4 µg/m<sup>3</sup> (34.0%)

Respiratory admissions have been linked with other pollutants as well, such as PM<sub>2.5</sub>, PM<sub>10</sub> and NO<sub>2</sub> (see section B2. Respiratory hospital admissions (short-term exposures, all ages and elderly)). Calculations were done separately for all pollutants but are only quoted for O<sub>3</sub> and note that the concentrations at these percentiles will be different in other cities. Also, note that ozone is higher in background compared to roadside stations. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Source:** Committee on the Medical Effects of Air Pollutants (COMEAP) 2015 Quantification of mortality and hospital admissions associated with ground-level ozone; ISBN 978-0-85951-776-8. Available at: <https://www.gov.uk/government/publications/comeap-quantification-of-mortality-and-hospital-admissions-associated-with-ground-level-ozone>

**Comment:** The PM<sub>2.5</sub> concentration-response function is the same as that recommended by WHO (2013b) [http://www.euro.who.int/\\_data/assets/pdf\\_file/0006/238956/Health\\_risks\\_air\\_pollution\\_HRAPIE\\_project.pdf?ua=1](http://www.euro.who.int/_data/assets/pdf_file/0006/238956/Health_risks_air_pollution_HRAPIE_project.pdf?ua=1). The concentration-response functions is based on papers published up to May 2011. There are more recently published individual studies but no new study pooling groups of studies together.

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#### **High vs low pollution days, change in numbers of admissions**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**If higher air pollution days in London were lower instead, we could avoid 654 hospital admissions each year for respiratory disease.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**There would be 654 fewer people admitted to hospital for respiratory disease if air pollution (O<sub>3</sub>) was reduced by 34.0% on half the days of the year. This reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for respiratory disease than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: COMEAP (2015) (0.75% change in respiratory admissions, all ages, per 10 µg/m<sup>3</sup> increase in O<sub>3</sub> (daily 8-hour maximum)). This study pooled together several other studies so are quite robust and the CRF reported is recommended by the COMEAP. The upper 95% confidence intervals for this estimate was 1.2%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

O<sub>3</sub> 25<sup>th</sup> percentile 35.7 µg/m<sup>3</sup>  
O<sub>3</sub> 75<sup>th</sup> percentile 54.1 µg/m<sup>3</sup>                      Change 18.4 µg/m<sup>3</sup> (34.0%)

The new percentage change was then applied to the baseline numbers of respiratory admissions in London. In this case it was the annual number of 94,841 admissions (averaged over the years 2014/15-2017/18; PHE personal communication via NHS Digital) divided by 2 because the scenario changed concentration on half the days of the year. Respiratory admissions have been linked with other pollutants as well, such as PM<sub>2.5</sub>, PM<sub>10</sub> and NO<sub>2</sub> (see section B2. Respiratory hospital admissions (short-term exposures, all ages and elderly)). Calculations were done separately for all pollutants but are only quoted for O<sub>3</sub> and note that the concentrations at these percentiles will be different in other cities. Also, note that ozone is higher in background compared to roadside stations. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Source:** Committee on the Medical Effects of Air Pollutants (COMEAP) 2015 Quantification of mortality and hospital admissions associated with ground-level ozone; ISBN 978-0-85951-776-8. Available at: <https://www.gov.uk/government/publications/comeap-quantification-of-mortality-and-hospital-admissions-associated-with-ground-level-ozone>

**Comment:** The PM<sub>2.5</sub> concentration-response function is the same as that recommended by WHO (2013b) [http://www.euro.who.int/\\_data/assets/pdf\\_file/0006/238956/Health\\_risks\\_air\\_pollution\\_HRAPIE\\_project.pdf?ua=1](http://www.euro.who.int/_data/assets/pdf_file/0006/238956/Health_risks_air_pollution_HRAPIE_project.pdf?ua=1). The concentration-response functions is based on papers published up to May 2011. There are more recently published individual studies but no new study pooling groups of studies together.

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### 5.3.10 Cardiovascular admissions all ages

#### High vs low pollution days, change in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Living in London, your risk of being admitted to hospital for cardiovascular disease would be reduced by 0.5%, if higher pollution days were reduced to lower pollution days instead.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**Living in London, your risk of being admitted to hospital for cardiovascular disease would be reduced by 0.5%, if air pollution (PM<sub>2.5</sub>) was reduced by 37.2% on half the days of the year. This air pollution reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for cardiovascular disease than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Atkinson et al (2014) (0.91% change in cardiovascular admissions, all ages, per 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{2.5}$ ). This study pooled together several other studies so are quite robust. We used the estimates from European studies. The upper 95% confidence intervals for this estimate was 1.66%.

The percentage increase per 10  $\mu\text{g}/\text{m}^3$  was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

$\text{PM}_{2.5}$  25<sup>th</sup> percentile 14.5  $\mu\text{g}/\text{m}^3$

$\text{PM}_{2.5}$  75<sup>th</sup> percentile 9.1  $\mu\text{g}/\text{m}^3$

Change 5.4  $\mu\text{g}/\text{m}^3$  (37.2%)

Cardiovascular admissions have been linked with other pollutants as well, such as  $\text{PM}_{10}$  and  $\text{NO}_2$  (see section B1. Cardiovascular hospital admissions (short-term exposures, all ages and elderly)). Calculations were done separately for all pollutants but are only quoted for  $\text{PM}_{2.5}$  and note that the concentrations at these percentiles will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Atkinson *et al* (2014) Epidemiological time series studies of  $\text{PM}_{2.5}$  and daily mortality and hospital admissions: a systematic review and meta-analysis. Thorax. 2014 Jul;69(7):660-5. doi: 10.1136/thoraxjnl-2013-204492

**Comment:** The  $\text{PM}_{2.5}$  concentration-response function is the same as that recommended by the HRAPIE project (WHO 2013b). There are more recently published individual studies but no new study pooling groups of studies together.

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#### High vs low pollution days, change in numbers of admissions

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**If higher air pollution days in London were lower instead, we could avoid 153 hospital admissions each year for cardiovascular disease.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

**There would be 153 fewer people admitted to hospital for cardiovascular disease if air pollution (PM<sub>2.5</sub>) was reduced by 37.2% on half the days of the year. This reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for cardiovascular disease than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Atkinson et al (2014) (0.91% change in cardiovascular admissions, all ages, per 10 µg/m<sup>3</sup> increase in PM<sub>2.5</sub>). This study pooled together several other studies so are quite robust. We used the estimates from European studies. The upper 95% confidence intervals for this estimate was 1.66%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

PM<sub>2.5</sub> 25<sup>th</sup> percentile 14.5 µg/m<sup>3</sup>

PM<sub>2.5</sub> 75<sup>th</sup> percentile 9.1 µg/m<sup>3</sup>

Change 5.4 µg/m<sup>3</sup> (37.2%)

The new percentage change was then applied to the baseline numbers of cardiovascular admissions in London. In this case it was the annual number of 62,180 admissions (averaged over the years 2014/15-2017/18; PHE personal communication via NHS Digital) divided by 2 because the scenario changed concentration on half the days of the year. Cardiovascular admissions have been linked with other pollutants as well, such as PM<sub>10</sub> and NO<sub>2</sub> (see section B1. Cardiovascular hospital admissions (short-term exposures, all ages and elderly)). Calculations were done separately for all pollutants but are only quoted for PM<sub>2.5</sub> and note that the concentrations at these percentiles will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

**Sources:** Atkinson *et al* (2014) Epidemiological time series studies of PM<sub>2.5</sub> and daily mortality and hospital admissions: a systematic review and meta-analysis. *Thorax*. 2014 Jul;69(7):660-5. doi: 10.1136/thoraxjnl-2013-204492

**Comment:** The PM<sub>2.5</sub> concentration-response function is the same as that recommended by the HRAPIE project (WHO 2013b). There are more recently published individual studies but no new study pooling groups of studies together.



### 5.3.11 Coronary Heart Disease (CHD) Incidence (all ages)

#### Concentration-change roadside vs background, difference in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Living near busy roads in London may contribute to a 6.3% greater chance of coronary heart disease.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background). Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g. (fatty diet) but air pollution may contribute too.*

*For text in articles; body of press releases etc*

#### **In London, air pollution may contribute to a 6.3% greater chance of coronary heart disease if you live beside a polluted road compared with living on a quieter street. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g. (fatty diet) but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background. Note that a 6.3% greater chance is not the same as a 6.3% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that people from places with higher particulate (PM<sub>10</sub>) pollution have a higher risk of CHD. These studies give us a concentration-response function – an equation relating a change in risk of CHD with a particular change in air pollution level. In this case, the concentration-response function comes from: Cesaroni et al (2014) (Relative risk of 1.12 per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> or equivalently 12 % increase in CHD occurrence, per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>). This study used pooled raw data from several European cohorts so is quite robust. The upper 95% confidence interval for this study was 25% change per 10 µg/m<sup>3</sup>.

The relative risk per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>)

PM<sub>10</sub> Median at roadside stations 21.8 µg/m<sup>3</sup>

PM<sub>10</sub> Median at background stations 16.4 µg/m<sup>3</sup> Change 5.4 µg/m<sup>3</sup> (24.8%)

Coronary heart disease has been linked with other pollutants as well, such as PM<sub>2.5</sub>, CO and NO<sub>2</sub> (see section B5. Myocardial Infarction (short- and long-term exposures, all ages)). Calculations were done separately for all pollutants but are only quoted for PM<sub>10</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher and statistically significant estimates in the headline statement rather than adding the results.

**Source:** Cesaroni *et al* (2014) 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 Jan 21;348:f7412. doi: 10.1136/bmj.f7412.

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### **Concentration-change roadside vs background, attributable cases**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Living near busy roads in London may contribute to 821 coronary heart disease cases.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background). Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g. (fatty diet) but air pollution may contribute too.*

*For text in articles; body of press releases etc*

#### **In London, air pollution may contribute to 821 coronary heart disease cases if you live beside a polluted road compared with living on a quieter street. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g. (fatty diet) but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that people from places with higher particulate (PM<sub>10</sub>) pollution have a higher risk of CHD. These studies give us a concentration-response function – an equation relating a change in risk of CHD with a particular change in air pollution level. In this case, the concentration-response function comes from: Cesaroni *et al* (2014) (Relative risk of 1.12 per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> or equivalently 12 % increase in CHD occurrence, per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>). This study used pooled raw data from several European cohorts so is quite robust. The upper 95% confidence interval for this study was 25% change per 10 µg/m<sup>3</sup>.

The relative risk per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and-2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>)

PM<sub>10</sub> Median at roadside stations 21.8 µg/m<sup>3</sup>  
PM<sub>10</sub> Median at background stations 16.4 µg/m<sup>3</sup> Change 5.4 µg/m<sup>3</sup> (24.8%)

The new percentage change was then applied to the baseline numbers of coronary heart disease incidence in the approximately 33% of the London population that live near busy roads . In this case it was the annual number of 39,432 cases (averaged over the years 2014/15-2017/18; UK wide data scaled by population size, Heart & Circulatory Disease Statistics 2019) multiplied by 33%. Coronary heart disease has been linked with other pollutants as well, such as PM<sub>2.5</sub>, CO and NO<sub>2</sub> (see section B5. Myocardial Infarction (short- and long-term exposures, all ages)). Calculations were done separately for all pollutants but are only quoted for PM<sub>10</sub> and note that the median concentrations



will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher and statistically significant estimates in the headline statement rather than adding the results.

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**Source:** Cesaroni *et al* (2014) 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 Jan 21;348:f7412. doi: 10.1136/bmj.f7412.

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### **20% reduction, difference in risk**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Cutting air pollution in London by one fifth would decrease the risk of coronary heart disease by around 4.8%.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g. (fatty diet) but air pollution may contribute too.*

*For text in articles; body of press releases etc*

**In London, current air pollution (NO<sub>2</sub>) levels may contribute to a 4.8% greater chance of coronary heart disease incidence compared to a hypothetical scenario of a 20% reduced air pollution levels. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g. (fatty diet) but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels across London compared to the same concentrations reduced by one fifth. Note that a 4.8% greater chance is not the same as a 4.8% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that people from places with higher particulate (PM<sub>10</sub>) pollution have a higher risk of CHD. These studies give us a concentration-response function – an equation relating a change in risk of CHD with a particular change in air pollution level. In this case, the concentration-response function comes from: Cesaroni *et al* (2014) (Relative risk of 1.12 per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> or equivalently 12 % increase in CHD occurrence, per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>). This study used pooled raw data from several European cohorts so is quite robust. The upper 95% confidence interval for this study was 25% change per 10 µg/m<sup>3</sup>.

The relative risk per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>)

PM<sub>10</sub> Median across London 20.6 µg/m<sup>3</sup>  
PM<sub>10</sub> Median hypothetical reduction 16.5 µg/m<sup>3</sup> Change 4.1 µg/m<sup>3</sup> (20%)

Coronary heart disease has been linked with other pollutants as well, such as PM<sub>2.5</sub>, CO and NO<sub>2</sub> (see section B5. Myocardial Infarction (short- and long-term exposures, all ages)). Calculations were done separately for all pollutants but are only quoted for PM<sub>10</sub> and note that the median concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the

pollutant that yielded the higher and statistically significant estimates in the headline statement rather than adding the results.

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**Source:** Cesaroni *et al* (2014) 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 Jan 21;348:f7412. doi: 10.1136/bmj.f7412.

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### **20% reduction, attributable cases**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

### **Cutting air pollution in London by one fifth may result in 1,885 fewer cases of coronary heart disease each year.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g. (fatty diet) but air pollution may contribute too.*

*For text in articles; body of press releases etc*

**In London, current air pollution (PM<sub>10</sub>) levels may result in 1,885 more cases of coronary heart disease each year compared to a hypothetical scenario of a 20% reduced air pollution levels. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g. (fatty diet) but air pollution may contribute too.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels across London compared to the same concentrations reduced by one fifth.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that people from places with higher particulate (PM<sub>10</sub>) pollution have a higher risk of CHD. These studies give us a concentration-response function – an equation relating a change in risk of CHD with a particular change in air pollution level. In this case, the concentration-response function comes from: Cesaroni *et al* (2014) (Relative risk of 1.12 per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> or equivalently 12 % increase in CHD occurrence, per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>). This study used pooled raw data from several European cohorts so is quite robust. The upper 95% confidence interval for this study was 25% change per 10 µg/m<sup>3</sup>.

The relative risk per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>)

PM<sub>10</sub> Median across London 20.6 µg/m<sup>3</sup>

PM<sub>10</sub> Median hypothetical reduction 16.5 µg/m<sup>3</sup> Change 4.1 µg/m<sup>3</sup> (20%)

The new percentage change was then applied to the baseline numbers of coronary heart disease incidence in London. In this case it was the annual number of 39,432 cases (averaged over the years 2014/15-2017/18; UK wide data scaled by population size, Heart & Circulatory Disease Statistics 2019). Coronary heart disease has been linked with other pollutants as well, such as PM<sub>2.5</sub>, CO and NO<sub>2</sub> (see section B5. Myocardial Infarction (short- and long-term exposures, all ages)). Calculations were done separately for all pollutants but are only quoted for PM<sub>10</sub> and note that the median

concentrations will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher and statistically significant estimates in the headline statement rather than adding the results.

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**Source:** Cesaroni *et al* (2014) 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 Jan 21;348:f7412. doi: 10.1136/bmj.f7412.

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### 5.3.12 Bronchitic symptoms (asthmatic children)

**LONDON (Concentration-change roadside vs background, difference in risk).**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Asthmatic children that live near busy roads in London may be subject to a 11.5% greater chance of experiencing bronchitic symptoms (cough and phlegm).**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background) Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.*

*For text in articles; body of press releases etc*

**In London, air pollution may be subject to a 11.5% greater chance of developing bronchitic symptoms for asthmatic children that live beside a polluted road compared with those living on a quieter street. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background. Note that a 11.5% greater chance is not the same as a 11.5% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that asthmatic children from places with higher air pollution (NO<sub>2</sub>) have a higher risk of bronchitic symptoms. These studies give us a concentration-response function – an equation relating a change in risk of bronchitic symptoms with a particular change in air pollution level. In this case, the concentration-response function comes from: WHO (2013b) based on McConnell *et al* (2003) (Odds Ratio of 1.021 per 1 µg/m<sup>3</sup> increase in NO<sub>2</sub>, or equivalently 2.1% increased odds of experiencing bronchitic symptoms per 1 µg/m<sup>3</sup> increase in NO<sub>2</sub>). This CRF is recommended by the WHO HRAPIE report (2013b). The upper 95% confidence interval for this study was 6.0% per 1 µg/m<sup>3</sup>.

The odds per 1 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from

the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>)

NO<sub>2</sub> Median at roadside stations 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median at background stations 29.7 µg/m<sup>3</sup> Change 28.6 µg/m<sup>3</sup> (49.1%)

Calculations were done for NO<sub>2</sub> and note that the median concentrations will be different in other cities.

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**Source:** McConnell *et al* (2003) Prospective study of air pollution and bronchitic symptoms in children with asthma. Am J Respir Crit Care Med. 2003 Oct 1;168(7):790-7.

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### **Concentration-change roadside vs background, difference in risk**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Air pollution may contribute to 4,067 more asthmatic children that live near busy roads in London experiencing bronchitic symptoms each year.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background) Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.*

*For text in articles; body of press releases etc*

**In London, air pollution may contribute to 4,067 more asthmatic children that live beside a polluted road experiencing bronchitic symptoms each year compared with those living on a quieter street. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that asthmatic children from places with higher air pollution (NO<sub>2</sub>) have a higher risk of bronchitic symptoms. These studies give us a concentration-response function – an equation relating a change in risk of bronchitic symptoms with a particular change in air pollution level. In this case, the concentration-response function comes from: WHO (2013b) based on McConnell *et al* (2003) (Odds Ratio of 1.021 per 1 µg/m<sup>3</sup> increase in NO<sub>2</sub>, or equivalently 2.1% increased odds of experiencing bronchitic symptoms per 1 µg/m<sup>3</sup> increase in NO<sub>2</sub>). This CRF is recommended by the WHO HRAPIE report (2013b). The upper 95% confidence interval for this study was 6.0% per 1 µg/m<sup>3</sup>.

The relative risk per 1 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>).  
NO<sub>2</sub> Median at roadside stations 58.3 µg/m<sup>3</sup>

NO<sub>2</sub> Median at background stations 29.7 µg/m<sup>3</sup> Change 28.6 µg/m<sup>3</sup> (49.1%)

The new percentage change was then applied to the baseline number of children aged 5 to 14 years old living near busy roads in the approximately 33% of the population living near busy roads in London. We assumed an asthma prevalence of 10% in this population (Lai et al, 2009), and also a prevalence of bronchitic symptoms among asthmatic children of 21.1% (WHO (2013b) HRAPIE Report). In this case it was the annual number of 1,067,956 children in London (averaged over the years 2014/15-2017/18; Estimates of the population for the UK, England and Wales, Scotland and Northern Ireland, Office for National Statistics (ONS)) multiplied by 33%. Calculations were done for NO<sub>2</sub> and note that the median concentrations will be different in other cities.

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**Source:** McConnell *et al* (2003) Prospective study of air pollution and bronchitic symptoms in children with asthma. Am J Respir Crit Care Med. 2003 Oct 1;168(7):790-7.

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### 20% reduction, difference in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Cutting air pollution in London by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 3.5%.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.*

*For text in articles; body of press releases etc*

**In London, current air pollution (NO<sub>2</sub>) levels may contribute to a 3.5% greater risk of bronchitic symptoms in asthmatic children compared to a hypothetical scenario of a 20% reduced air pollution levels. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels across London compared to the same concentrations reduced by one fifth. Note that a 3.5% greater chance is not the same as a 3.5% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that asthmatic children from places with higher air pollution (NO<sub>2</sub>) have a higher risk of bronchitic symptoms. These studies give us a concentration-response function – an equation relating a change in risk of bronchitic symptoms with a particular change in air pollution level. In this case, the concentration-response function comes from: WHO (2013b) based on McConnell et al (2003) (Odds Ratio of 1.021 per 1 µg/m<sup>3</sup> increase in NO<sub>2</sub>, or equivalently 2.1% increased odds of experiencing bronchitic symptoms per 1 µg/m<sup>3</sup> increase in NO<sub>2</sub>). This CRF is recommended by the WHO HRAPIE report (2013b). The upper 95% confidence interval for this study was 6.0% per 1 µg/m<sup>3</sup>.

The relative risk per 1 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and-2017 across London and the

equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>)

NO<sub>2</sub> Median across London 47.2 µg/m<sup>3</sup>

NO<sub>2</sub> Median hypothetical reduction 37.8 µg/m<sup>3</sup> Change 9.4 µg/m<sup>3</sup> (20%)

Calculations were done for NO<sub>2</sub> and note that the median concentrations will be different in other cities.

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**Source:** McConnell *et al* (2003) Prospective study of air pollution and bronchitic symptoms in children with asthma. Am J Respir Crit Care Med. 2003 Oct 1;168(7):790-7.

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### 20% reduction, attributable cases

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Cutting air pollution in London by one fifth could contribute to 3,685 fewer asthmatic children with bronchitic symptoms each year.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.*

*For text in articles; body of press releases etc*

**In London, current air pollution (PM<sub>10</sub>) levels may contribute to 1,885 more asthmatic children with bronchitic symptoms each year compared to a hypothetical scenario of a 20% reduced air pollution levels. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels across London compared to the same concentrations reduced by one fifth.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that asthmatic children from places with higher air pollution (NO<sub>2</sub>) have a higher risk of bronchitic symptoms. These studies give us a concentration-response function – an equation relating a change in risk of bronchitic symptoms with a particular change in air pollution level. In this case, the concentration-response function comes from: WHO (2013b) based on McConnell *et al* (2003) (Odds Ratio of 1.021 per 1 µg/m<sup>3</sup> increase in NO<sub>2</sub>, or equivalently 2.1% increased odds of experiencing bronchitic symptoms per 1 µg/m<sup>3</sup> increase in NO<sub>2</sub>). This CRF is recommended by the WHO HRAPIE report (2013b). The upper 95% confidence interval for this study was 6.0% per 1 µg/m<sup>3</sup>.

The relative risk per 1 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>).

NO<sub>2</sub> Median across London 47.2 µg/m<sup>3</sup>

NO<sub>2</sub> Median hypothetical reduction 37.8 µg/m<sup>3</sup> Change 9.4 µg/m<sup>3</sup> (20%)

The new percentage change was then applied to the baseline number of children aged 5 to 14 years old in London. We assumed an asthma prevalence of 10% in this population (Lai et al, 2009), and also a prevalence of bronchitic symptoms among asthmatic children of 21.1% (WHO (2013b) HRAPIE Report). In this case it was the annual number of 1,067,956 children in London (averaged over the years 2014/15-2017/18; Estimates of the population for the UK, England and Wales, Scotland and Northern Ireland, Office for National Statistics (ONS)). Calculations were done for NO<sub>2</sub> and note that the median concentrations will be different in other cities.

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**Source:** McConnell *et al* (2003) Prospective study of air pollution and bronchitic symptoms in children with asthma. *Am J Respir Crit Care Med*. 2003 Oct 1;168(7):790-7.

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### 5.3.13 Acute bronchitis in children

#### Concentration-change roadside vs background, difference in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Living near busy roads in London may contribute to a 0.6% greater risk of a chest infection (acute bronchitis) in children.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background). Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.*

*For text in articles; body of press releases etc*

**In London, air pollution may contribute to a 0.6% greater risk of a chest infection (acute bronchitis) for children living beside a polluted road compared with living on a quieter street. Acute bronchitis means short-lived inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background. Note that a 0.6% greater chance is not the same as a 0.6% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that children from places with higher particulate (PM<sub>10</sub>) pollution have a higher risk of acute bronchitis. These studies give us a concentration-response function – an equation relating a change in risk of acute bronchitis with a particular change in air pollution level. In this case, the concentration-response function comes from: WHO (2013b) based on Hoek et al (2012) (Relative risk of 1.08 per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> or equivalently 8% increase in acute bronchitis occurrence per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>). This study used pooled raw data from nine European countries so is quite robust. This CRF is recommended by the WHO HRAPIE report (2013b). The upper 95% confidence interval for this study was 19% change per 10 µg/m<sup>3</sup>.

The relative risk per 10  $\mu\text{g}/\text{m}^3$  was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>)

PM<sub>10</sub> Median at roadside stations 21.8  $\mu\text{g}/\text{m}^3$

PM<sub>10</sub> Median at background stations 16.4  $\mu\text{g}/\text{m}^3$  Change 5.4  $\mu\text{g}/\text{m}^3$  (24.8%)

Calculations were done for PM<sub>10</sub> and note that the median concentrations will be different in other cities.

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**Source:** Hoek *et al* (2012) PM<sub>10</sub>, and children's respiratory symptoms and lung function in the PATY study. Eur Respir J. 2012 Sep;40(3):538-47. doi: 10.1183/09031936.00002611.

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### **Concentration-change roadside vs background, attributable cases**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **Living near busy roads in London may contribute to 1,598 cases of a chest infection (acute bronchitis) in children.**

*Footnote: Based on the difference between long term average air pollution levels at roadsides compared to the long-term average at less polluted, quieter streets (the London background). Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.*

*For text in articles; body of press releases etc*

**In London, air pollution (PM<sub>10</sub>) may contribute to 1,598 cases of a chest infection (acute bronchitis) in children living beside a polluted road compared with living on a quieter street. Acute bronchitis means short-lived inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels at roadside monitoring sites compared to the long-term average at the London background.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that children from places with higher particulate (PM<sub>10</sub>) pollution have a higher risk of acute bronchitis. These studies give us a concentration-response function – an equation relating a change in risk of acute bronchitis with a particular change in air pollution level. In this case, the concentration-response function comes from: WHO (2013b) based on Hoek *et al* (2012) (Relative risk of 1.08 per 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> or equivalently 8% increase in acute bronchitis occurrence per 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub>). This study used pooled raw data from nine European countries so is quite robust. This CRF is recommended by the WHO HRAPIE report (2013b). The upper 95% confidence interval for this study was 19% change per 10  $\mu\text{g}/\text{m}^3$ .

The relative risk per 10  $\mu\text{g}/\text{m}^3$  was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each



day is the average of the same day of the year for 2015, 2016 and 2017 at roadside monitoring sites and the equivalent median over 2015-2017 at background monitoring sites. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>)

PM<sub>10</sub> Median at roadside stations 21.8 µg/m<sup>3</sup>

PM<sub>10</sub> Median at background stations 16.4 µg/m<sup>3</sup> Change 5.4 µg/m<sup>3</sup> (24.8%)

The new percentage change was then applied to the baseline numbers of acute bronchitis and population at risk of the 33% of children aged 6 to 12 years old living near busy roads in London. In this case it was the annual number of 759,572 children in London (averaged over the years 2014/15-2017/18; Estimates of the population for the UK, England and Wales, Scotland and Northern Ireland, Office for National Statistics (ONS)) multiplied by 33%. Calculations were done for PM<sub>10</sub> and note that the median concentrations will be different in other cities.

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**Source:** Hoek *et al* (2012) PM<sub>10</sub>, and children's respiratory symptoms and lung function in the PATY study. *Eur Respir J.* 2012 Sep;40(3):538-47. doi: 10.1183/09031936.00002611.

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### **20% reduction, difference in risk**

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

### **Cutting air pollution in London by one fifth would decrease the risk of a chest infection (acute bronchitis) in children by around 0.5%.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.*

*For text in articles; body of press releases etc*

**In London, current air pollution (PM<sub>10</sub>) levels may contribute to a 0.5% greater risk of a chest infection (acute bronchitis) in children compared to a hypothetical scenario of a 20% reduced air pollution levels. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels across London compared to the same concentrations reduced by one fifth. Note that a 0.5% greater chance is not the same as a 0.5% chance – the absolute chance for an individual depends on other factors not just air pollution.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that children from places with higher particulate (PM<sub>10</sub>) pollution have a higher risk of acute bronchitis. These studies give us a concentration-response function – an equation relating a change in risk of acute bronchitis with a particular change in air pollution level. In this case, the concentration-response function comes from: WHO (2013b) based on Hoek *et al* (2012) (Relative risk of 1.08 per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub> or equivalently 8% increase in acute bronchitis occurrence per 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>). This study used pooled raw data from nine European countries so is quite robust. This CRF is recommended by the WHO HRAPIE report (2013b). The upper 95% confidence interval for this study was 19% change per 10 µg/m<sup>3</sup>.

The relative risk per 10  $\mu\text{g}/\text{m}^3$  was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>)

PM<sub>10</sub> Median across London 20.6  $\mu\text{g}/\text{m}^3$

PM<sub>10</sub> Median hypothetical reduction 16.5  $\mu\text{g}/\text{m}^3$  Change 4.1  $\mu\text{g}/\text{m}^3$  (20%)

Calculations were done for PM<sub>10</sub> and note that the median concentrations will be different in other cities.

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**Source:** Hoek *et al* (2012) PM<sub>10</sub>, and children's respiratory symptoms and lung function in the PATY study. Eur Respir J. 2012 Sep;40(3):538-47. doi: 10.1183/09031936.00002611.

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### 20% reduction, attributable cases

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

### **Cutting air pollution in London by one fifth may result in 3,683 fewer children with a chest infection (acute bronchitis) each year.**

*Footnote: Based on the difference between long term average air pollution levels across London compared to a hypothetical 20% reduction scenario. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.*

*For text in articles; body of press releases etc*

**In London, current air pollution (PM<sub>10</sub>) levels may result in 3,683 more cases children with a chest infection (acute bronchitis) each year compared to a hypothetical scenario of a 20% reduced air pollution levels. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.<sup>1</sup>**

<sup>1</sup> *Based on the difference between long term average air pollution levels across London compared to the same concentrations reduced by one fifth.*

**Justification:** This calculation is based on studies which follow up groups of people over time, showing that children from places with higher particulate (PM<sub>10</sub>) pollution have a higher risk of acute bronchitis. These studies give us a concentration-response function – an equation relating a change in risk of acute bronchitis with a particular change in air pollution level. In this case, the concentration-response function comes from: WHO (2013b) based on Hoek *et al* (2012) (Relative risk of 1.08 per 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub> or equivalently 8% increase in acute bronchitis occurrence per 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>10</sub>). This study used pooled raw data from nine European countries so is quite robust. This CRF is recommended by the WHO HRAPIE report (2013b). The upper 95% confidence interval for this study was 19% change per 10  $\mu\text{g}/\text{m}^3$ .

The relative risk per 10  $\mu\text{g}/\text{m}^3$  was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the difference between the median of 365 days where each day is the average of the same day of the year for 2015, 2016 and 2017 across London and the

equivalent median over 2015-2017 under a hypothetical 20% reduction in air pollution levels. Concentrations were from the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>)  
PM<sub>10</sub> Median across London 20.6 µg/m<sup>3</sup>  
PM<sub>10</sub> Median hypothetical reduction 16.5 µg/m<sup>3</sup> Change 4.1 µg/m<sup>3</sup> (20%)

The new percentage change was then applied to the baseline numbers of children aged 6 to 12 years old in London. In this case it was the annual number of 759,572 children (averaged over the years 2014/15-2017/18; Estimates of the population for the UK, England and Wales, Scotland and Northern Ireland, Office for National Statistics (ONS)). Calculations were done for PM<sub>10</sub> and note that the median concentrations will be different in other cities.

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**Source:** Hoek *et al* (2012) PM<sub>10</sub>, and children's respiratory symptoms and lung function in the PATY study. Eur Respir J. 2012 Sep;40(3):538-47. doi: 10.1183/09031936.00002611.

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### 5.3.14 COPD admissions (all ages)

#### High vs low pollution days, change in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Living in London, your risk of being admitted to hospital for COPD would be reduced by 2.1%, if higher pollution days were reduced to lower pollution days instead.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**Living in London, your risk of being admitted to hospital for COPD would be reduced by 2.1%, if air pollution (O<sub>3</sub>) was reduced by 34% on half the days of the year. This air pollution reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for COPD than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Walton *et al* (2014) (1.12% change in COPD admissions, all ages, per 10 µg/m<sup>3</sup> increase in O<sub>3</sub>). This study pooled together several other studies so are quite robust. We used the estimates from European studies. The upper 95% confidence intervals for this estimate was 1.66%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half

of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

O<sub>3</sub> 25<sup>th</sup> percentile 35.7 µg/m<sup>3</sup>

O<sub>3</sub> 75<sup>th</sup> percentile 54.1 µg/m<sup>3</sup>                      Change 18.4 µg/m<sup>3</sup> (34.0%)

COPD admissions have been linked with other pollutants as well, such as PM<sub>2.5</sub>, PM<sub>10</sub>, CO and NO<sub>2</sub> (see section B3. COPD hospital admissions). Calculations were done separately for all pollutants but are only quoted for O<sub>3</sub> and note that the concentrations at these percentiles will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Walton *et al* (2014) Quantitative systematic review of the associations between short-term exposure to ambient ozone and mortality and hospital admissions.

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### High vs low pollution days, change in numbers of admissions

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**If higher air pollution days in London were lower instead, we could avoid 136 hospital admissions each year for COPD.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**There would be 136 fewer people admitted to hospital for COPD if air pollution (O<sub>3</sub>) was reduced by 34% on half the days of the year. This reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for COPD than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Walton *et al* (2014) (1.12% change in COPD admissions, all ages, per 10 µg/m<sup>3</sup> increase in O<sub>3</sub>). This study pooled together several other studies so are quite robust. We used the estimates from European studies. The upper 95% confidence intervals for this estimate was 1.66%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurn>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

O<sub>3</sub> 25<sup>th</sup> percentile 35.7 µg/m<sup>3</sup>

O<sub>3</sub> 75<sup>th</sup> percentile 54.1 µg/m<sup>3</sup>

Change 18.4 µg/m<sup>3</sup> (34.0%)

The new percentage change was then applied to the baseline numbers of COPD admissions in London. In this case it was the annual number of 13,224 admissions (averaged over the years 2014/15-2017/18; PHE personal communication via NHS Digital) divided by 2 because the scenario changed concentration on half the days of the year. COPD admissions have been linked with other pollutants as well, such as PM<sub>2.5</sub>, PM<sub>10</sub>, CO and NO<sub>2</sub> (see section B3. COPD hospital admissions). Calculations were done separately for all pollutants but are only quoted for O<sub>3</sub> and note that the concentrations at these percentiles will be different in other cities. Given the overlap in effects between pollutants we just used the pollutant that yielded the higher number in the headline statement rather than adding the results.

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**Sources:** Walton *et al* (2014) Quantitative systematic review of the associations between short-term exposure to ambient ozone and mortality and hospital admissions.

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### 5.3.15 Pneumonia admissions in children

#### High vs low pollution days, change in risk

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

**Living in London, the risk of children being admitted to hospital for pneumonia would be reduced by 2.3%, if higher pollution days were reduced to lower pollution days instead.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**Living in London, the risk of your child being admitted to hospital for pneumonia would be reduced by 2.3%, if air pollution (O<sub>3</sub>) was reduced by 34.0% on half the days of the year. This air pollution reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for pneumonia than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Nhung *et al* (2017) (2.40% change in pneumonia admissions in children, per 10 ppb increase in O<sub>3</sub> (daily 8-hour maximum)). This study pooled together several other studies so are quite robust. The upper 95% confidence intervals for this estimate was 3.80%.

The percentage increase per 10 ppb was then adjusted (on the log scale) for a new concentration range (in µg/m<sup>3</sup>). The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aur>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile,

roughly the average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

O<sub>3</sub> 25<sup>th</sup> percentile 35.7 µg/m<sup>3</sup>

O<sub>3</sub> 75<sup>th</sup> percentile 54.1 µg/m<sup>3</sup> Change 18.4 µg/m<sup>3</sup> (34.0%)

Pneumonia admissions in children have been linked with other pollutants as well, such as PM<sub>2.5</sub>, PM<sub>10</sub> and NO<sub>2</sub> (see section B13. Pneumonia admissions in children). Calculations were done separately for all pollutants but are only quoted for O<sub>3</sub> and note that the concentrations at these percentiles will be different in other cities. Also, note that ozone is higher in background compared to roadside stations. Given the overlap in effects between pollutants we just used the pollutant that yielded the highest and statistically significant estimates in the headline statement rather than adding the results.

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**Source:** Nhung *et al* (2017) Short-term association between ambient air pollution and pneumonia in children: A systematic review and meta-analysis of time-series and case-crossover studies. *Environ Pollut.* 2017 Nov;230:1000-1008. doi: 10.1016/j.envpol.2017.07.063.

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### High vs low pollution days, change in numbers of admissions

*For posters, article headlines etc NB Either footnote (for poster) or fuller statement later on (for articles) (see next box) needs to be included for statement to be scientifically complete.*

#### **If higher air pollution days in London were lower instead, we could avoid 9 hospital admissions each year for pneumonia in children.**

*Footnote: Assumes half the year was at the average of the top half of the annual range of particulate air pollution levels and these days were reduced to the average of the bottom half of the range of levels.*

*For text in articles; body of press releases etc*

**There would be 9 fewer children admitted to hospital for pneumonia if air pollution (O<sub>3</sub>) was reduced by 34.0% on half the days of the year. This reduction is the difference between a typical higher air pollution day and a typical lower pollution day<sup>1</sup>.**

<sup>1</sup> A typical higher air pollution day was defined as the middle of the top half of the range of particulate air pollution levels in a year and a typical low pollution day as the middle of the bottom half of the range.

**Justification:** This calculation is based on studies which show days with higher pollution have larger numbers of admissions to hospital for pneumonia than days with lower pollution. These studies give us a concentration-response function – an equation relating a percentage increase in hospital admissions with a particular change in air pollution level. In this case, the concentration-response functions come from: Nhung *et al* (2017) (2.40% change in pneumonia admissions in children, per 10 ppb increase in O<sub>3</sub> (daily 8-hour maximum)). This study pooled together several other studies so are quite robust. The upper 95% confidence intervals for this estimate was 3.80%.

The percentage increase per 10 µg/m<sup>3</sup> was then adjusted (on the log scale) for a new concentration range. The concentration range chosen was the 25<sup>th</sup> to 75<sup>th</sup> percentile of the range of concentrations in the London Air Quality Network ([www.londonair.org](http://www.londonair.org)) and the Automatic Urban and Rural Network (<https://uk-air.defra.gov.uk/networks/network-info?view=aurun>) for the years 2015-2017. We assumed half the days of the year were at the 75<sup>th</sup> percentile (roughly the average of the top half of the range of concentrations) to start with and were all reduced to the 25<sup>th</sup> percentile, roughly the

average of the lower half of the range of concentrations. (Air pollution distributions are often slightly skewed with more concentrations at the low end, but the above assumptions are still reasonable).

O<sub>3</sub> 25<sup>th</sup> percentile 35.7 µg/m<sup>3</sup>

O<sub>3</sub> 75<sup>th</sup> percentile 54.1 µg/m<sup>3</sup>                      Change 18.4 µg/m<sup>3</sup> (34.0%)

The new percentage change was then applied to the baseline numbers of pneumonia admissions in children in London. In this case it was the annual number of 763 admissions (averaged over the years 2014/15-2017/18; UK wide data scaled by population size, Hospital Admitted Patient Care Activity (NHS)) divided by 2 because the scenario changed concentration on half the days of the year. Pneumonia admissions in children have been linked with other pollutants as well, such as PM<sub>2.5</sub>, PM<sub>10</sub> and NO<sub>2</sub> (see section B13. Pneumonia admissions in children). Calculations were done separately for all pollutants but are only quoted for O<sub>3</sub> and note that the concentrations at these percentiles will be different in other cities. Also, note that ozone is higher in background compared to roadside stations. Given the overlap in effects between pollutants we just used the pollutant that yielded the highest and statistically significant estimates in the headline statement rather than adding the results.

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**Source:** Nhung *et al* (2017) Short-term association between ambient air pollution and pneumonia in children: A systematic review and meta-analysis of time-series and case-crossover studies. *Environ Pollut.* 2017 Nov;230:1000-1008. doi: 10.1016/j.envpol.2017.07.063.

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## 5.4 Statements for UK Cities

We give below the details of the background information relevant to each statement for UK cities. As explained previously in 5.3 Statements for London, we have not added detailed justification to these statements at this stage, although the information is still in the annexes. Where some UK cities do not feature in some statements it is because there were insufficient air quality data to allow a calculation to be made e.g. ozone data for Derby were not available.

### 5.4.1 Out-of-hospital cardiac arrests

#### Birmingham

*The risk of out of hospital cardiac arrest in Birmingham is 2.3% higher on high air pollution days than lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*Each year on average, higher air pollution days in Birmingham are responsible for 12 more cardiac arrests outside hospital than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate matter levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

#### Bristol



*The risk of out of hospital cardiac arrest in Bristol is 2.2% higher on high air pollution days than lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*Each year on average, higher air pollution days in Bristol are responsible for 4 more cardiac arrests outside hospital than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate matter levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

## **Derby**

*The risk of out of hospital cardiac arrest in Derby is 1.8% higher on high air pollution days than lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*Each year on average, higher air pollution days in Derby are responsible for 0 more cardiac arrests outside hospital than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate matter levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

## **Liverpool**

*The risk of out of hospital cardiac arrest in Liverpool is 2% higher on high air pollution days than lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*Each year on average, higher air pollution days in Liverpool are responsible for 4 more cardiac arrests outside hospital than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate matter levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

## **Manchester**



*The risk of out of hospital cardiac arrest in Manchester is 2.4% higher on high air pollution days than lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*Each year on average, higher air pollution days in Manchester are responsible for 6 more cardiac arrests outside hospital than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate matter levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

### **Nottingham**

*The risk of out of hospital cardiac arrest in Nottingham is 2.3% higher on high air pollution days than lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*Each year on average, higher air pollution days in Nottingham are responsible for 3 more cardiac arrests outside hospital than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate matter levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

### **Oxford**

*The risk of out of hospital cardiac arrest in Oxford is 1.9% higher on high air pollution days than lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*Each year on average, higher air pollution days in Oxford are responsible for 6 more cardiac arrests outside hospital than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate matter levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

### **Southampton**

*The risk of out of hospital cardiac arrest in Southampton is 1.9% higher on high air pollution days than lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*Each year on average, higher air pollution days in Southampton are responsible for 2 more cardiac arrests outside hospital than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate matter levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

## **5.4.2 Stroke**

### **Birmingham**

*The risk of emergency hospitalisations for stroke in Birmingham is 2.6% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Living near a busy road in Birmingham increases your risk of hospitalisation for stroke by 4.0% (short-term).*

Based on the difference between the middle of the range of daily average nitrogen dioxide levels at roadsides and the middle of the range of daily average nitrogen dioxide levels away from roads. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*On high air pollution days in Birmingham, there are on average 27 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Lowering air pollution by 32.1% on high air pollution days in Birmingham could save 27 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Each year on average, higher air pollution days in Birmingham can send up to 42 more people to hospital for stroke than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

## **Bristol**

*The risk of emergency hospitalisations for stroke in Bristol is 2.8% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Living near a busy road in Bristol increases your risk of hospitalisation for stroke by 2.8% (short-term).*

Based on the difference between the middle of the range of daily average nitrogen dioxide levels at roadsides and the middle of the range of daily average nitrogen dioxide levels away from roads. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*On high air pollution days in Bristol, there are on average 9 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Lowering air pollution by 35.9% on high air pollution days in Bristol could save 9 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Each year on average, higher air pollution days in Bristol can send up to 14 more people to hospital for stroke than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

## **Derby**

*The risk of emergency hospitalisations for stroke in Derby is 3.9% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*On high air pollution days in Derby, there are on average 8 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Lowering air pollution by 38.2% on high air pollution days in Derby could save 8 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Each year on average, higher air pollution days in Derby can send up to 13 more people to hospital for stroke than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

## **Liverpool**

*The risk of emergency hospitalisations for stroke in Liverpool is 2.6% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Living near a busy road in Liverpool increases your risk of hospitalisation for stroke by 2.4% (short-term).*

Based on the difference between the middle of the range of daily average nitrogen dioxide levels at roadsides and the middle of the range of daily average nitrogen dioxide levels away from roads. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*On high air pollution days in Liverpool, there are on average 12 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Lowering air pollution by 36.0% on high air pollution days in Liverpool could save 12 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Each year on average, higher air pollution days in Liverpool can send up to 19 more people to hospital for stroke than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

## **Manchester**

*The risk of emergency hospitalisations for stroke in Manchester is 2.8% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*On high air pollution days in Manchester, there are on average 14 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Lowering air pollution by 33.5% on high air pollution days in Manchester could save 14 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Each year on average, higher air pollution days in Manchester can send up to 22 more people to hospital for stroke than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

## **Nottingham**

*The risk of emergency hospitalisations for stroke in Nottingham is 3.3% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Living near a busy road in Nottingham increases your risk of hospitalisation for stroke by 1.5% (short-term).*

Based on the difference between the middle of the range of daily average nitrogen dioxide levels at roadsides and the middle of the range of daily average nitrogen dioxide levels away from roads. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*On high air pollution days in Nottingham, there are on average 8 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Lowering air pollution by 35.7% on high air pollution days in Nottingham could save 8 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Each year on average, higher air pollution days in Nottingham can send up to 13 more people to hospital for stroke than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

## **Oxford**

*The risk of emergency hospitalisations for stroke in Oxford is 2.2% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Living near a busy road in Oxford increases your risk of hospitalisation for stroke by 7.4% (short-term).*

Based on the difference between the middle of the range of daily average nitrogen dioxide levels at roadsides and the middle of the range of daily average nitrogen dioxide levels away from roads. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*On high air pollution days in Oxford, there are on average 2 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Lowering air pollution by 26.2% on high air pollution days in Oxford could save 2 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Each year on average, higher air pollution days in Oxford can send up to 4 more people to hospital for stroke than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

## **Southampton**

*The risk of emergency hospitalisations for stroke in Southampton is 3.0% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Living near a busy road in Southampton increases your risk of hospitalisation for stroke by 2.0% (short-term).*

Based on the difference between the middle of the range of daily average nitrogen dioxide levels at roadsides and the middle of the range of daily average nitrogen dioxide levels away from roads. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*On high air pollution days in Southampton, there are on average 7 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Lowering air pollution by 30.2% on high air pollution days in Southampton could save 7 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

*Each year on average, higher air pollution days in Southampton can send up to 10 more people to hospital for stroke than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

### **5.4.3 Asthma admissions in children**

#### **Birmingham**

*In Birmingham, your child is 4.1% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of nitrogen dioxide (NO<sub>2</sub>) levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Birmingham, an extra 15 children are hospitalised with asthma on days where air pollution is high compared to days where air pollution is low on average each year (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Calculation applies to children aged 0-14.

#### **Bristol**

*In Bristol, your child is 4.4% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of nitrogen dioxide (NO<sub>2</sub>) levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Bristol, an extra 5 children are hospitalised with asthma on days where air pollution is high compared to days where air pollution is low on average each year (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Calculation applies to children aged 0-14.



## Derby

*In Derby, your child is 6.2% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of nitrogen dioxide (NO<sub>2</sub>) levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Derby, an extra 5 children are hospitalised with asthma on days where air pollution is high compared to days where air pollution is low on average each year (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Calculation applies to children aged 0-14.

## Liverpool

*In Liverpool, your child is 4.0% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of nitrogen dioxide (NO<sub>2</sub>) levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Liverpool, an extra 7 children are hospitalised with asthma on days where air pollution is high compared to days where air pollution is low on average each year (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Calculation applies to children aged 0-14.

## Manchester

*In Manchester, your child is 4.4% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of nitrogen dioxide (NO<sub>2</sub>) levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Manchester, an extra 8 children are hospitalised with asthma on days where air pollution is high compared to days where air pollution is low on average each year (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Calculation applies to children aged 0-14.

## Nottingham

*In Nottingham, your child is 5.1% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of nitrogen dioxide (NO<sub>2</sub>) levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Nottingham, an extra 5 children are hospitalised with asthma on days where air pollution is high compared to days where air pollution is low on average each year (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Calculation applies to children aged 0-14.

## Oxford

*In Oxford, your child is 3.5% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of nitrogen dioxide (NO<sub>2</sub>) levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Oxford, an extra 1 child is hospitalised with asthma on days where air pollution is high compared to days where air pollution is low on average each year (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Calculation applies to children aged 0-14.

## Southampton

*In Southampton, your child is 4.7% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of nitrogen dioxide (NO<sub>2</sub>) levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Southampton, an extra 4 children are hospitalised with asthma on days where air pollution is high compared to days where air pollution is low on average each year (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Calculation applies to children aged 0-14.

## 5.4.4 Asthma admissions in adults

### Birmingham

*In Birmingham, adults are 1.4% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Birmingham, an extra 11 adults are taken to hospital with asthma on days of high air pollution compared to days with lower air pollution(short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of nitrogen dioxide concentrations. Calculation applies to adults age 15-64.

### Bristol

*In Bristol, adults are 1.5% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Bristol, an extra 4 adults are taken to hospital with asthma on days of high air pollution compared to days with lower air pollution(short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of nitrogen dioxide concentrations. Calculation applies to adults age 15-64.

### Derby

*In Derby, adults are 2.1% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Derby, an extra 3 adults are taken to hospital with asthma on days of high air pollution compared to days with lower air pollution(short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more

technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of nitrogen dioxide concentrations. Calculation applies to adults age 15-64.

### **Liverpool**

*In Liverpool, adults are 1.3% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Liverpool, an extra 5 adults are taken to hospital with asthma on days of high air pollution compared to days with lower air pollution(short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of nitrogen dioxide concentrations. Calculation applies to adults age 15-64.

### **Manchester**

*In Manchester, adults are 1.5% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Manchester, an extra 6 adults are taken to hospital with asthma on days of high air pollution compared to days with lower air pollution(short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of nitrogen dioxide concentrations. Calculation applies to adults age 15-64.

### **Nottingham**

*In Nottingham, adults are 1.7% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Nottingham, an extra 3 adults are taken to hospital with asthma on days of high air pollution compared to days with lower air pollution(short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of nitrogen dioxide concentrations. Calculation applies to adults age 15-64.

### **Oxford**

*In Oxford, adults are 1.2% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Oxford, an extra 1 adult are taken to hospital with asthma on days of high air pollution compared to days with lower air pollution(short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of nitrogen dioxide concentrations. Calculation applies to adults age 15-64.

### **Southampton**

*In Southampton, adults are 1.6% more likely to be hospitalised for asthma on days with high NO<sub>2</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations.

*In Southampton, an extra 3 adults are taken to hospital with asthma on days of high air pollution compared to days with lower air pollution(short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of nitrogen dioxide concentrations. Calculation applies to adults age 15-64.

## **5.4.5 Reduced lung growth and low lung function**

### **Birmingham**

*Roadside air pollution in Birmingham stunts lung growth in children by 7.7% (long-term).*

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Birmingham background). Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Cutting air pollution in Birmingham by one fifth would increase children's lung capacity by around 2.6% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Living near busy roads in Birmingham may contribute to an 4.7% greater chance of reduced lung function in children (long-term).*

Based on the difference between long term average air nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Birmingham background). Refers to children aged 6-8.

*Cutting air pollution in Birmingham by one fifth may contribute to a 1.3% greater chance of better lung function in children (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

*Cutting air pollution in Birmingham by one fifth would result in 659 fewer children with low lung function each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

## **Bristol**

*Roadside air pollution in Bristol stunts lung growth in children by 5.3% (long-term).*

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Bristol background). Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Cutting air pollution in Bristol by one fifth would increase children's lung capacity by around 2.3% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Living near busy roads in Bristol may contribute to an 3.0% greater chance of reduced lung function in children (long-term).*

Based on the difference between long term average air nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Bristol background). Refers to children aged 6-8.

*Cutting air pollution in Bristol by one fifth may contribute to a 1.2% greater chance of better lung function in children (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

*Cutting air pollution in Bristol by one fifth would result in 199 fewer children with low lung function each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

## **Derby**

*Cutting air pollution in Derby by one fifth would increase children's lung capacity by around 3.1% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Cutting air pollution in Derby by one fifth may contribute to a 1.7% greater chance of better lung function in children (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

*Cutting air pollution in Derby by one fifth would result in 179 fewer children with low lung function each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

## **Liverpool**

*Roadside air pollution in Liverpool stunts lung growth in children by 4.6% (long-term).*

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Liverpool background). Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Cutting air pollution in Liverpool by one fifth would increase children's lung capacity by around 2.1% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Living near busy roads in Liverpool may contribute to an 2.5% greater chance of reduced lung function in children (long-term).*

Based on the difference between long term average air nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Liverpool background). Refers to children aged 6-8.

*Cutting air pollution in Liverpool by one fifth may contribute to a 1.1% greater chance of better lung function in children (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

*Cutting air pollution in Liverpool by one fifth would result in 174 fewer children with low lung function each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

## **Manchester**

*Cutting air pollution in Manchester by one fifth would increase children's lung capacity by around 2.6% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Cutting air pollution in Manchester by one fifth may contribute to a 1.3% greater chance of better lung function in children (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

*Cutting air pollution in Manchester by one fifth would result in 284 fewer children with low lung function each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

## **Nottingham**



*Roadside air pollution in Nottingham stunts lung growth in children by 2.8% (long-term).*

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Nottingham background). Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Cutting air pollution in Nottingham by one fifth would increase children's lung capacity by around 2.8% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Living near busy roads in Nottingham may contribute to an 1.5% greater chance of reduced lung function in children (long-term).*

Based on the difference between long term average air nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Nottingham background). Refers to children aged 6-8.

*Cutting air pollution in Nottingham by one fifth may contribute to a 1.5% greater chance of better lung function in children (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

*Cutting air pollution in Nottingham by one fifth would result in 175 fewer children with low lung function each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

## **Oxford**

*Roadside air pollution in Oxford stunts lung growth in children by 14.1% (long-term).*

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Oxford background). Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Cutting air pollution in Oxford by one fifth would increase children's lung capacity by around 2.8% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Living near busy roads in Oxford may contribute to an 10.3% greater chance of reduced lung function in children (long-term).*

Based on the difference between long term average air nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Oxford background). Refers to children aged 6-8.

*Cutting air pollution in Oxford by one fifth may contribute to a 1.5% greater chance of better lung function in children (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

*Cutting air pollution in Oxford by one fifth would result in 77 fewer children with low lung function each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

## **Southampton**

*Roadside air pollution in Southampton stunts lung growth in children by 3.8% (long-term).*

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Southampton background). Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Cutting air pollution in Southampton by one fifth would increase children's lung capacity by around 3.2% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

*Living near busy roads in Southampton may contribute to an 2.0% greater chance of reduced lung function in children (long-term).*

Based on the difference between long term average air nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Southampton background). Refers to children aged 6-8.

*Cutting air pollution in Southampton by one fifth may contribute to a 1.7% greater chance of better lung function in children (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

*Cutting air pollution in Southampton by one fifth would result in 150 fewer children with low lung function each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Low lung function refers to children with FEV<sub>1</sub> (Forced expiratory volume in 1 second – a measure of how fast a child can breathe out) less than 85% of that predicted for healthy children of the same age and gender. It is typically low in asthmatics. Refers to children aged 6-8.

#### **5.4.6 Lung cancer**

##### **Birmingham**

*Cutting air pollution in Birmingham by one fifth would decrease lung cancer cases by around 6.4% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

*Cutting air pollution in Birmingham by one fifth would result in 50 fewer lung cancer cases each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

##### **Bristol**

*Cutting air pollution in Bristol by one fifth would decrease lung cancer cases by around 5.9% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

*Cutting air pollution in Bristol by one fifth would result in 18 fewer lung cancer cases each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

##### **Liverpool**

*Cutting air pollution in Liverpool by one fifth would decrease lung cancer cases by around 5.3% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

*Cutting air pollution in Liverpool by one fifth would result in 17 fewer lung cancer cases each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

## **Manchester**

*Cutting air pollution in Manchester by one fifth would decrease lung cancer cases by around 5.6% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

*Cutting air pollution in Manchester by one fifth would result in 20 fewer lung cancer cases each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

## **Nottingham**

*Cutting air pollution in Nottingham by one fifth would decrease lung cancer cases by around 6.7% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

*Cutting air pollution in Nottingham by one fifth would result in 15 fewer lung cancer cases each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

## **Oxford**

*Cutting air pollution in Oxford by one fifth would decrease lung cancer cases by around 6.0% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

*Cutting air pollution in Oxford by one fifth would result in 28 less lung cancer cases each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

## **Southampton**

*Cutting air pollution in Southampton by one fifth would decrease lung cancer cases by around 5.9% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

*Cutting air pollution in Southampton by one fifth would result in 10 fewer lung cancer cases each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>2.5</sub> concentrations. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

#### **5.4.7 Asthma symptoms in children**

##### **Birmingham**

*In Birmingham, children with asthma are 0.3% more likely to experience asthma symptoms on high air pollution days than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness.

*On high air pollution days, 42 more children with asthma in Birmingham experience asthma symptoms than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.

##### **Bristol**

*In Bristol, children with asthma are 0.2% more likely to experience asthma symptoms on high air pollution days than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness.

*On high air pollution days, 12 more children with asthma in Bristol experience asthma symptoms than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.

##### **Liverpool**

*In Liverpool, children with asthma are 0.2% more likely to experience asthma symptoms on high air pollution days than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness.

*On high air pollution days, 12 more children with asthma in Liverpool experience asthma symptoms than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.

### **Nottingham**

*In Nottingham, children with asthma are 0.3% more likely to experience asthma symptoms on high air pollution days than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness.

*On high air pollution days, 11 more children with asthma in Nottingham experience asthma symptoms than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.

### **Oxford**

*In Oxford, children with asthma are 0.2% more likely to experience asthma symptoms on high air pollution days than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness.

*On high air pollution days, 4 more children with asthma in Oxford experience asthma symptoms than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.

### **Southampton**

*In Southampton, children with asthma are 0.3% more likely to experience asthma symptoms on high air pollution days than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness.

*On high air pollution days, 9 more children with asthma in Southampton experience asthma symptoms than on lower pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range

of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.

#### **5.4.8 Term low birthweight**

##### **Birmingham**

*Living near busy roads in Birmingham may contribute to a 0.2% greater risk of babies being born underweight (long-term).*

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Birmingham background). Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Birmingham by one fifth would decrease the risk of babies being born underweight by around 0.1% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Birmingham by one fifth would result in 11 fewer babies born underweight each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

##### **Bristol**

*Living near busy roads in Bristol may contribute to a 0.2% greater risk of babies being born underweight (long-term).*

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Bristol background). Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Bristol by one fifth would decrease the risk of babies being born underweight by around 0.1% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Bristol by one fifth would result in 4 fewer babies born underweight each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

##### **Derby**

*Cutting air pollution in Derby by one fifth would decrease the risk of babies being born underweight by around 0.1% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Derby by one fifth would result in 3 fewer babies born underweight each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

## **Liverpool**

*Living near busy roads in Liverpool may contribute to a 0.1% greater risk of babies being born underweight (long-term).*

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Liverpool background). Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Liverpool by one fifth would decrease the risk of babies being born underweight by around 0.1% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Liverpool by one fifth would result in 3 fewer babies born underweight each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

## **Manchester**

*Cutting air pollution in Manchester by one fifth would decrease the risk of babies being born underweight by around 0.1% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Manchester by one fifth would result in 5 fewer babies born underweight each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

## **Nottingham**

*Living near busy roads in Nottingham may contribute to a 0.1% greater risk of babies being born underweight (long-term).*



Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Nottingham background). Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Nottingham by one fifth would decrease the risk of babies being born underweight by around 0.1% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Nottingham by one fifth would result in 3 fewer babies born underweight each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

## **Oxford**

*Living near busy roads in Oxford may contribute to a 0.4% greater risk of babies being born underweight (long-term).*

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Oxford background). Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Oxford by one fifth would decrease the risk of babies being born underweight by around 0.1% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Oxford by one fifth would result in 1 fewer baby born underweight each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

## **Southampton**

*Living near busy roads in Southampton may contribute to a 0.1% greater risk of babies being born underweight (long-term).*

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Southampton background). Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Southampton by one fifth would decrease the risk of babies being born underweight by around 0.1% (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

*Cutting air pollution in Southampton by one fifth would result in 3 fewer babies born underweight each year (long-term).*

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

#### **5.4.9 Respiratory admissions all ages**

##### **Birmingham**

*The risk of emergency hospitalisations for respiratory disease in Birmingham is 1.5% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum ozone concentrations.

*On high air pollution days in Birmingham, there are on average 149 more hospital admissions for respiratory disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 31.7% on high air pollution days in Birmingham could save 149 hospital admissions for respiratory disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 31.7%.

*Each year on average, higher air pollution days in Birmingham can send up to 238 more people to hospital for respiratory disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

##### **Bristol**

*The risk of emergency hospitalisations for respiratory disease in Bristol is 1.4% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum ozone concentrations.

*On high air pollution days in Bristol, there are on average 43 more hospital admissions for respiratory disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 27.7% on high air pollution days in Bristol could save 43 hospital admissions for respiratory disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 27.7%.

*Each year on average, higher air pollution days in Bristol can send up to 68 more people to hospital for respiratory disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Liverpool**

*The risk of emergency hospitalisations for respiratory disease in Liverpool is 1.4% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum ozone concentrations.

*On high air pollution days in Liverpool, there are on average 61 more hospital admissions for respiratory disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 26.7% on high air pollution days in Liverpool could save 61 hospital admissions for respiratory disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 26.7%.

*Each year on average, higher air pollution days in Liverpool can send up to 98 more people to hospital for respiratory disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Manchester**

*The risk of emergency hospitalisations for respiratory disease in Manchester is 1.4% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum ozone concentrations.

*On high air pollution days in Manchester, there are on average 68 more hospital admissions for respiratory disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 33.5% on high air pollution days in Manchester could save 68 hospital admissions for respiratory disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 33.5%.

*Each year on average, higher air pollution days in Manchester can send up to 109 more people to hospital for respiratory disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Nottingham**

*The risk of emergency hospitalisations for respiratory disease in Nottingham is 1.5% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum ozone concentrations.

*On high air pollution days in Nottingham, there are on average 36 more hospital admissions for respiratory disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 34.2% on high air pollution days in Nottingham could save 36 hospital admissions for respiratory disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 34.2%.

*Each year on average, higher air pollution days in Nottingham can send up to 57 more people to hospital for respiratory disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Southampton**

*The risk of emergency hospitalisations for respiratory disease in Southampton is 1.2% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum ozone concentrations.

*On high air pollution days in Southampton, there are on average 27 more hospital admissions for respiratory disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 27.7% on high air pollution days in Southampton could save 27 hospital admissions for respiratory disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 27.7%.

*Each year on average, higher air pollution days in Southampton can send up to 43 more people to hospital for respiratory disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

#### **5.4.10 Cardiovascular admissions all ages**

##### **Birmingham**

*The risk of emergency hospitalisations for cardiovascular disease in Birmingham is 0.5% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*On high air pollution days in Birmingham, there are on average 34 more hospital admissions for cardiovascular disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 42.9% on high air pollution days in Birmingham could save 34 hospital admissions for cardiovascular disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 42.9%.

*Each year on average, higher air pollution days in Birmingham can send up to 62 more people to hospital for cardiovascular disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Bristol**

*The risk of emergency hospitalisations for cardiovascular disease in Bristol is 0.5% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*On high air pollution days in Bristol, there are on average 10 more hospital admissions for cardiovascular disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 45.5% on high air pollution days in Bristol could save 10 hospital admissions for cardiovascular disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 45.5%.

*Each year on average, higher air pollution days in Bristol can send up to 19 more people to hospital for cardiovascular disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Liverpool**

*The risk of emergency hospitalisations for cardiovascular disease in Liverpool is 0.5% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*On high air pollution days in Liverpool, there are on average 14 more hospital admissions for cardiovascular disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 45.9% on high air pollution days in Liverpool could save 14 hospital admissions for cardiovascular disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels

these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 45.9%.

*Each year on average, higher air pollution days in Liverpool can send up to 25 more people to hospital for cardiovascular disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Manchester**

*The risk of emergency hospitalisations for cardiovascular disease in Manchester is 0.5% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*On high air pollution days in Manchester, there are on average 18 more hospital admissions for cardiovascular disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 49.6% on high air pollution days in Manchester could save 18 hospital admissions for cardiovascular disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 49.6%.

*Each year on average, higher air pollution days in Manchester can send up to 32 more people to hospital for cardiovascular disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Nottingham**

*The risk of emergency hospitalisations for cardiovascular disease in Nottingham is 0.5% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*On high air pollution days in Nottingham, there are on average 8 more hospital admissions for cardiovascular disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 41.6% on high air pollution days in Nottingham could save 8 hospital admissions for cardiovascular disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 41.6%.

*Each year on average, higher air pollution days in Nottingham can send up to 15 more people to hospital for cardiovascular disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Oxford**

*The risk of emergency hospitalisations for cardiovascular disease in Oxford is 0.4% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*On high air pollution days in Oxford, there are on average 3 more hospital admissions for cardiovascular disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 40.2% on high air pollution days in Oxford could save 3 hospital admissions for cardiovascular disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 40.2%.

*Each year on average, higher air pollution days in Oxford can send up to 5 more people to hospital for cardiovascular disease than lower air pollution days (short-term).*



Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Southampton**

*The risk of emergency hospitalisations for cardiovascular disease in Southampton is 0.4% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

*On high air pollution days in Southampton, there are on average 6 more hospital admissions for cardiovascular disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 41.0% on high air pollution days in Southampton could save 6 hospital admissions for cardiovascular disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 41.0%.

*Each year on average, higher air pollution days in Southampton can send up to 11 more people to hospital for cardiovascular disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **5.4.11 Coronary Heart Disease (CHD) Incidence (all ages)**

### **Birmingham**

Living near busy roads in Birmingham may contribute to a 0.2% greater chance of coronary heart disease (*long-term*).

Based on the difference between long term average PM<sub>10</sub> levels at roadsides compared to the long-term average at less polluted, quieter streets (the Birmingham background). Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

*Cutting air pollution in Birmingham by one fifth would decrease the risk of coronary heart disease by around 3.3% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

*Cutting air pollution in Birmingham by one fifth would result in 165 fewer cases of coronary heart disease each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

### **Bristol**

Living near busy roads in Bristol may contribute to an 8.0% greater chance of coronary heart disease (long-term).

Based on the difference between long term average PM<sub>10</sub> levels at roadsides compared to the long-term average at less polluted, quieter streets (the Bristol background). Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

*Cutting air pollution in Bristol by one fifth would decrease the risk of coronary heart disease by around 3.1% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

*Cutting air pollution in Bristol by one fifth would result in 62 fewer cases of coronary heart disease each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

### **Liverpool**

*Cutting air pollution in Liverpool by one fifth would decrease the risk of coronary heart disease by around 3.0% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

*Cutting air pollution in Liverpool by one fifth would result in 62 fewer cases of coronary heart disease each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

### **Nottingham**

Living near busy roads in Nottingham may contribute to a 1.0% greater chance of coronary heart disease (*long-term*).

Based on the difference between long term average PM<sub>10</sub> levels at roadsides compared to the long-term average at less polluted, quieter streets (the Nottingham background). Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

*Cutting air pollution in Nottingham by one fifth would decrease the risk of coronary heart disease by around 3.7% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

*Cutting air pollution in Nottingham by one fifth would result in 52 fewer cases of coronary heart disease each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

## **Oxford**

*Cutting air pollution in Oxford by one fifth would decrease the risk of coronary heart disease by around 2.7% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

*Cutting air pollution in Oxford by one fifth would result in 83 fewer cases of coronary heart disease each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

## **Southampton**

Living near busy roads in Southampton may contribute to a 5.6% greater chance of coronary heart disease (*long-term*).

Based on the difference between long term average PM<sub>10</sub> levels at roadsides compared to the long-term average at less polluted, quieter streets (the Southampton background). Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

*Cutting air pollution in Southampton by one fifth would decrease the risk of coronary heart disease by around 4.2% (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

*Cutting air pollution in Southampton by one fifth would result in 48 fewer cases of coronary heart disease each year (long-term).*

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

#### **5.4.12 Bronchitic symptoms (asthmatic children)**

##### **Birmingham**

Air pollution may contribute to asthmatic children that live near busy roads in Birmingham being subject to a 6.7% greater chance of developing bronchitic symptoms (long-term).

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Birmingham background). Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Birmingham by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 2.1% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Birmingham by one fifth would result in 328 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

##### **Bristol**

Air pollution may contribute to asthmatic children that live near busy roads in Bristol may experiencing a 4.5% greater chance of developing bronchitic symptoms (long-term).

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Bristol background). Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Bristol by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 1.9% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Bristol by one fifth would result in 94 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

## **Derby**

Cutting air pollution in Derby by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 2.6% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Derby by one fifth would result in 85 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

## **Liverpool**

Air pollution may contribute to asthmatic children that live near busy roads in Liverpool experiencing a 3.8% greater chance of developing bronchitic symptoms (long-term).

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Liverpool background). Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Liverpool by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 1.7% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Liverpool by one fifth would result in 85 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

## **Manchester**

Cutting air pollution in Manchester by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 2.1% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Manchester by one fifth would result in 134 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

### **Nottingham**

Air pollution may contribute to asthmatic children that live near busy roads in Nottingham experiencing a 2.3% greater chance of developing bronchitic symptoms (long-term).

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Nottingham background). Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Nottingham by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 2.3% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Nottingham by one fifth would result in 84 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

### **Oxford**

Air pollution may contribute to asthmatic children that live near busy roads in Oxford experiencing a 13.3% greater chance of developing bronchitic symptoms (long-term).

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Oxford background). Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Oxford by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 2.3% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Oxford by one fifth would result in 38 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

### **Southampton**

Air pollution may contribute to asthmatic children that live near busy roads in Southampton experiencing a 3.1% greater chance of developing bronchitic symptoms (long-term).

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Southampton background). Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Southampton by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 2.6% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

Cutting air pollution in Southampton by one fifth would result in 69 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

## **5.4.13 Acute bronchitis in children**

### **Birmingham**

Living near busy roads in Birmingham may contribute to a 0.0% greater risk of a chest infection (acute bronchitis) in children (long-term).

Based on the difference between long term average PM<sub>10</sub> levels at roadsides compared to the long-term average at less polluted, quieter streets (the Birmingham background). Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

Cutting air pollution in Birmingham by one fifth would decrease the risk of a chest infection (acute bronchitis) in children by around 0.3% (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

Cutting air pollution in Birmingham by one fifth would result in 371 fewer children with a chest infection (acute bronchitis) each year (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

### **Bristol**

Living near busy roads in Bristol may contribute to a 0.8% greater risk of a chest infection (acute bronchitis) in children (long-term).

Based on the difference between long term average PM<sub>10</sub> levels at roadsides compared to the long-term average at less polluted, quieter streets (the Bristol background). Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

Cutting air pollution in Bristol by one fifth would decrease the risk of a chest infection (acute bronchitis) in children by around 0.3% (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

Cutting air pollution in Bristol by one fifth would result in 114 fewer children with a chest infection (acute bronchitis) each year (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

### **Liverpool**

Cutting air pollution in Liverpool by one fifth would decrease the risk of a chest infection (acute bronchitis) in children by around 0.3% (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

Cutting air pollution in Liverpool by one fifth would result in 104 fewer children with a chest infection (acute bronchitis) each year (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

### **Nottingham**



Living near busy roads in Nottingham may contribute to a 0.1% greater risk of a chest infection (acute bronchitis) in children (long-term).

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Nottingham background). Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

Cutting air pollution in Nottingham by one fifth would decrease the risk of a chest infection (acute bronchitis) in children by around 0.4% (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

Cutting air pollution in Nottingham by one fifth would result in 97 fewer children with a chest infection (acute bronchitis) each year (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

## **Oxford**

Cutting air pollution in Oxford by one fifth would decrease the risk of a chest infection (acute bronchitis) in children by around 0.3% (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

Cutting air pollution in Oxford by one fifth would result in 31 fewer children with a chest infection (acute bronchitis) each year (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

## **Southampton**

Living near busy roads in Southampton may contribute to a 0.6% greater risk of a chest infection (acute bronchitis) in children (long-term).

Based on the difference between long term average nitrogen dioxide levels at roadsides compared to the long-term average at less polluted, quieter streets (the Southampton background). Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

Cutting air pollution in Southampton by one fifth would decrease the risk of a chest infection (acute bronchitis) in children by around 0.4% (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

Cutting air pollution in Southampton by one fifth would result in 81 fewer children with a chest infection (acute bronchitis) each year (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

#### **5.4.14 COPD admissions (all ages)**

##### **Birmingham**

*The risk of emergency hospitalisations for COPD in Birmingham is 2.3% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Birmingham, there are on average 69 more hospital admissions for COPD each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 31.7% on high air pollution days in Birmingham could save 69 hospital admissions for COPD each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 31.7%.

*Each year on average, higher air pollution days in Birmingham can send up to 103 more people to hospital for COPD than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

##### **Bristol**

*The risk of emergency hospitalisations for COPD in Bristol is 2.0% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Bristol, there are on average 20 more hospital admissions for COPD each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 27.7% on high air pollution days in Bristol could save 20 hospital admissions for COPD each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 27.7%.

*Each year on average, higher air pollution days in Bristol can send up to 30 more people to hospital for COPD than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Liverpool**

*The risk of emergency hospitalisations for COPD in Liverpool is 2.0% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Liverpool, there are on average 29 more hospital admissions for COPD each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 26.7% on high air pollution days in Liverpool could save 29 hospital admissions for COPD each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 26.7%.

*Each year on average, higher air pollution days in Liverpool can send up to 42 more people to hospital for COPD than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Manchester**

*The risk of emergency hospitalisations for COPD in Manchester is 2.1% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Manchester, there are on average 32 more hospital admissions for COPD each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 33.5% on high air pollution days in Manchester could save 32 hospital admissions for COPD each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 33.5%.

*Each year on average, higher air pollution days in Manchester can send up to 47 more people to hospital for COPD than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Nottingham**

*The risk of emergency hospitalisations for COPD in Nottingham is 2.2% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Nottingham, there are on average 17 more hospital admissions for COPD each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 34.2% on high air pollution days in Nottingham could save 17 hospital admissions for COPD each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 34.2%.

*Each year on average, higher air pollution days in Nottingham can send up to 25 more people to hospital for COPD than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Southampton**

*The risk of emergency hospitalisations for COPD in Southampton is 1.9% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Southampton, there are on average 12 more hospital admissions for COPD each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 27.7% on high air pollution days in Southampton could save 12 hospital admissions for COPD each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 27.7%.

*Each year on average, higher air pollution days in Southampton can send up to 18 more people to hospital for COPD than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **5.4.15 Pneumonia admissions in children**

### **Birmingham**

*The risk of emergency hospitalisations for pneumonia in children in Birmingham is 2.5% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Birmingham, there are on average 2 more hospital admissions for pneumonia in children each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 31.7% on high air pollution days in Birmingham could save 2 hospital admissions for pneumonia in children each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 31.7%.

*Each year on average, higher air pollution days in Birmingham can send up to 3 more people to hospital for pneumonia in children than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Bristol**

*The risk of emergency hospitalisations for pneumonia in children in Bristol is 2.2% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Bristol, there are on average 1 more hospital admission for pneumonia in children each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 27.7% on high air pollution days in Bristol could save 1 hospital admission for pneumonia in children each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 27.7%.

*Each year on average, higher air pollution days in Bristol can send up to 1 more people to hospital for pneumonia in children than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Liverpool**

*The risk of emergency hospitalisations for pneumonia in children in Liverpool is 2.2% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Liverpool, there are on average 1 more hospital admissions for pneumonia in children each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 26.7% on high air pollution days in Liverpool could save 1 hospital admissions for pneumonia in children each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 26.7%.

*Each year on average, higher air pollution days in Liverpool can send up to 1 more people to hospital for pneumonia in children than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Manchester**

*The risk of emergency hospitalisations for pneumonia in children in Manchester is 2.3% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Manchester, there are on average 1 more hospital admissions for pneumonia in children each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 33.5% on high air pollution days in Manchester could save 1 hospital admissions for pneumonia in children each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 33.5%.

*Each year on average, higher air pollution days in Manchester can send up to 1 more people to hospital for pneumonia in children than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Nottingham**

*The risk of emergency hospitalisations for pneumonia in children in Nottingham is 2.4% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Nottingham, there are on average 0 more hospital admissions for pneumonia in children each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 34.2% on high air pollution days in Nottingham could save 0 hospital admissions for pneumonia in children each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 34.2%.

*Each year on average, higher air pollution days in Nottingham can send up to 1 more child to hospital for pneumonia than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Southampton**

*The risk of emergency hospitalisations for pneumonia in children in Southampton is 2.0% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

*On high air pollution days in Southampton, there are on average 0 more hospital admission for pneumonia in children each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

*Lowering air pollution by 27.7% on high air pollution days in Southampton could save 0 hospital admission for pneumonia in children each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 27.7%.

*Each year on average, higher air pollution days in Southampton can send up to 1 more child to hospital for pneumonia than lower air pollution days (short-term).*



Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## 5.5 Statements for Poland Cities

As noted earlier, in producing statements for cities in Poland we have used the same methodology as used for the UK cities. Clearly the concentrations used are those relevant to the Polish cities (see sections 2 Air Pollution Exposures and ANNEX A: AIR POLLUTION CONCENTRATIONS IN THE UK AND POLAND) and the baseline rates for the health outcomes are those relevant to Poland as discussed in section 4. One point of interest is that the sources of the air pollutants in question are different in Poland from those in UK cities. In the latter the local sources in cities are dominated by transport emissions, with a further contribution to PM<sub>2.5</sub> concentrations from transboundary transport of secondary aerosols. In Poland however, while transport emissions are also important, there is a considerable amount of solid fuel still used in urban areas. We have used the same sources of CRFs in deriving the statements for Poland, but in some cases, because of the different pollutant mix compared with the UK, we have used a different pollutant where the impacts were calculated to be higher.

### 5.5.1 Heart attacks

Coronary heart disease is a looser term than ischaemic heart disease but is essentially the same. Coronary refers to the coronary blood vessels (usually arteries) that supply the heart muscle.

#### Bielsko-Biala

- *Cutting air pollution in Bielsko-Biala by one fifth would decrease the risk of coronary heart disease by around 6.6% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

- *Each year on average, higher air pollution days in Bielsko-Biala are responsible for 10 more cardiac arrests outside hospital each year than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

- *Cutting air pollution in Bielsko-Biala by one fifth may contribute to 47 fewer cases of coronary heart disease each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

## Poznan

- *Cutting air pollution in Poznan by one fifth would decrease the risk of coronary heart disease by around 6.1% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

- *Each year on average, higher air pollution days in Poznan are responsible for 29 more cardiac arrests outside hospital each year than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

- *Cutting air pollution in Poznan by one fifth may contribute to 139 fewer cases of coronary heart disease each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

## Warsaw

- *Cutting air pollution in Warsaw by one fifth would decrease the risk of coronary heart disease by around 7.5% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

- *Each year on average, higher air pollution days in Warsaw are responsible for 81 more cardiac arrests outside hospital each year than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

- *Cutting air pollution in Warsaw by one fifth may contribute to 542 fewer cases of coronary heart disease each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some

places. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

## **Wroclaw**

- *Cutting air pollution in Wroclaw by one fifth would decrease the risk of coronary heart disease by around 6.8% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

- *Each year on average, higher air pollution days in Wroclaw are responsible for 31 more cardiac arrests outside hospital each year than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>2.5</sub> concentrations. Calculation applies to all ages.

- *Cutting air pollution in Wroclaw by one fifth may contribute to 179 fewer cases of coronary heart disease each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Coronary heart disease (heart attacks and a type of angina (heart pain)) has many well-established causes e.g (fatty diet) but air pollution may contribute too.

## **5.5.2 Stroke**

### **Bielsko-Biala**

- *The risk of emergency hospitalisations for stroke in Bielsko-Biala is 2.3% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Cutting air pollution in Bielsko-Biala by one fifth would decrease the risk of hospitalisation for stroke by 0.9% (short-term).*

Based on a 20% reduction in the long-term average NO<sub>2</sub> levels in Poland. This is an arbitrary reduction chosen because it is the same as chosen for PM<sub>2.5</sub>. (For PM<sub>2.5</sub> it is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places.) Stroke has many well-established causes e.g (prior heart disease) but air pollution may contribute too.

- *Cutting air pollution in Bielsko-Biala by one fifth would decrease your risk of stroke by around 15.5% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Stroke has many well- established causes e.g (prior heart disease) but air pollution may contribute too.

- *On high air pollution days in Bielsko-Biala, there are on average 5 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Lowering air pollution by 41% on high air pollution days in Bielsko-Biala could save 5 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Cutting air pollution in Bielsko-Biala by one fifth could save 41 new cases of stroke each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Stroke has many well- established causes e.g (prior heart disease) but air pollution may contribute too.

- *Each year on average, higher air pollution days in Bielsko-Biala can send up to 8 more people to hospital for stroke than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

### **Poznan**

- *The risk of emergency hospitalisations for stroke in Poznan is 1.9 % higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Cutting air pollution in Poznan by one fifth would decrease the risk of hospitalisation for stroke by 1.1% (short-term).*

Based on a 20% reduction in the long-term average NO<sub>2</sub> levels in Poland. This is an arbitrary reduction chosen because it is the same as chosen for PM<sub>2.5</sub>. (For PM<sub>2.5</sub> it is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places.) Stroke has many well- established causes e.g (prior heart disease) but air pollution may contribute too.

- *Cutting air pollution in Poznan by one fifth would decrease your risk of stroke by around 14.1% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Stroke has many well- established causes e.g (prior heart disease) but air pollution may contribute too.

- *On high air pollution days in Poznan, there are on average 11 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Lowering air pollution by 31% on high air pollution days in Poznan could save 11 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Cutting air pollution in Poznan by one fifth could save 120 new cases of stroke each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Stroke has many well- established causes e.g (prior heart disease) but air pollution may contribute too.

- *Each year on average, higher air pollution days in Poznan can send up to 16 more people to hospital for stroke than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

## **Warsaw**

- *The risk of emergency hospitalisations for stroke in Warsaw is 2.1% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Cutting air pollution in Warsaw by one fifth would decrease the risk of hospitalisation for stroke by 1.7% (short-term).*

Based on a 20% reduction in the long-term average NO<sub>2</sub> levels in Poland. This is an arbitrary reduction chosen because it is the same as chosen for PM<sub>2.5</sub>. (For PM<sub>2.5</sub> it is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places.) Stroke has many well- established causes e.g (prior heart disease) but air pollution may contribute too.

- *Cutting air pollution in Warsaw by one fifth would decrease your risk of stroke by around 15.9% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Stroke has many well- established causes e.g (prior heart disease) but air pollution may contribute too.

- *On high air pollution days in Warsaw, there are on average 50 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Lowering air pollution by 22% on high air pollution days in Warsaw could save 49 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Cutting air pollution in Warsaw by one fifth could save 430 new cases of stroke each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Stroke has many well- established causes e.g (prior heart disease) but air pollution may contribute too.

- *Each year on average, higher air pollution days in Warsaw can send up to 76 more people to hospital for stroke than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure

given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

### **Wroclaw**

- *The risk of emergency hospitalisations for stroke in Wroclaw is 1.6% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of NO<sub>2</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average nitrogen dioxide concentrations. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Cutting air pollution in Wroclaw by one fifth would decrease the risk of hospitalisation for stroke by 1.4% (short-term).*

Based on a 20% reduction in the long-term average NO<sub>2</sub> levels in Poland. This is an arbitrary reduction chosen because it is the same as chosen for PM<sub>2.5</sub>. (For PM<sub>2.5</sub> it is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places.) Stroke has many well- established causes e.g (prior heart disease) but air pollution may contribute too.

- *Cutting air pollution in Wroclaw by one fifth would decrease your risk of stroke by around 15.3% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Stroke has many well- established causes e.g (prior heart disease) but air pollution may contribute too.

- *On high air pollution days in Wroclaw, there are on average 14 more hospital admissions for stroke each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Lowering air pollution by 21% on high air pollution days in Wroclaw could save 14 hospital admissions for stroke each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 22%. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

- *Cutting air pollution in Wroclaw by one fifth could save 150 new cases of stroke each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some

places. Stroke has many well- established causes e.g (prior heart disease) but air pollution may contribute too.

- *Each year on average, higher air pollution days in Wroclaw can send up to 21 more people to hospital for stroke than lower air pollution days. (short-term).*

Assumes half the year was at the average of the top half of the annual range of nitrogen dioxide levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function. Nitrogen dioxide may be acting as a marker for other traffic pollutants.

### **5.5.3 Asthma Admissions in Children**

#### **Bielsko-Biala**

- *In Bielsko-Biala, your child is 5.2% more likely to be hospitalised for asthma on days with high O<sub>3</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of ozone levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average ozone concentrations.

- *In Bielsko-Biala, we calculated a zero impact on children's hospital admissions for asthma on days of high air pollution compared to days with lower air pollution (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average ozone concentrations. Calculation applies to children aged 0-14.

#### **Poznan**

- *In Poznan, your child is 5.4% more likely to be hospitalised for asthma on days with high O<sub>3</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of ozone levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average ozone concentrations.

- *In Poznan, an additional 1 child is taken to hospital with asthma on days of high air pollution compared to days with lower air pollution (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average ozone concentrations. Calculation applies to children aged 0-14.

#### **Warsaw**

- *In Warsaw, your child is 5.5% more likely to be hospitalised for asthma on days with high O<sub>3</sub> pollution compared to days with lower air pollution (short-term).*



Assumes typical high air pollution days are at the average of the top half of the annual range of ozone levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average ozone concentrations.

- *In Warsaw, an additional 3 children are taken to hospital with asthma on days of high air pollution compared to days with lower air pollution (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average ozone concentrations. Calculation applies to children aged 0-14.

## **Wroclaw**

- *In Wroclaw, your child is 5.6% more likely to be hospitalised for asthma on days with high O<sub>3</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of ozone levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average ozone concentrations.

- *In Wroclaw, an additional 1 child is taken to hospital with asthma on days of high air pollution compared to days with lower air pollution (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average ozone concentrations. Calculation applies to children aged 0-14.

## **5.5.4 Asthma admissions in adults**

### **Bielsko-Biala**

- *In Bielsko-Biala, adults are 2.6% more likely to be hospitalised for asthma on days with high PM<sub>10</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>10</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>10</sub> concentrations.

- *In Bielsko-Biala, we calculated a zero impact on adult hospital admissions for asthma on days of high air pollution compared to days with low air pollution (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>10</sub> levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of PM<sub>10</sub> concentrations. Calculation applies to adults age 15-64.

### **Poznan**

- *In Poznan, adults are 2.0% more likely to be hospitalised for asthma on days with high PM<sub>10</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>10</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>10</sub> concentrations.

- *In Poznan, we calculated a zero impact on adult hospital admissions for asthma on days of high air pollution compared to days with low air pollution (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>10</sub> levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of PM<sub>10</sub> concentrations. Calculation applies to adults age 15-64.

### **Warsaw**

- *In Warsaw, adults are 1.7% more likely to be hospitalised for asthma on days with high PM<sub>10</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>10</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>10</sub> concentrations.

- *In Warsaw, one additional adult is taken to hospital with asthma on days of high air pollution compared to days with low air pollution (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>10</sub> levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of PM<sub>10</sub> concentrations. Calculation applies to adults age 15-64.

### **Wroclaw**

- *In Wroclaw, adults are 1.8% more likely to be hospitalised for asthma on days with high PM<sub>10</sub> pollution compared to days with lower air pollution (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>10</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average PM<sub>10</sub> concentrations.

- *In Wroclaw, one additional adult is taken to hospital with asthma on days of high air pollution compared to days with low air pollution (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>10</sub> levels and these days were reduced to the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of PM<sub>10</sub> concentrations. Calculation applies to adults age 15-64.

### 5.5.5 Reduced lung capacity

#### Bielsko-Biala

- *Cutting air pollution in Bielsko-Biala by one fifth would increase children's lung capacity by around 1.6% (long-term).*

Based on a 20% reduction in the long-term average NO<sub>2</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. We chose to use the same % reduction for NO<sub>2</sub>. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

- *Cutting air pollution in Bielsko-Biala by one fifth may contribute to a 2.4% greater chance of better lung function in children (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Refers to children aged 6-8.

- *Cutting air pollution in Bielsko-Biala by one fifth may contribute to around 124 fewer children with low lung function (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Refers to children aged 6-8.

#### Poznan

- *Cutting air pollution in Poznan by one fifth would increase children's lung capacity by around 2.0% (long-term).*

Based on a 20% reduction in the long-term average NO<sub>2</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. We chose to use the same % reduction for NO<sub>2</sub>. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

- *Cutting air pollution in Poznan by one fifth may contribute to a 2.2% greater chance of better lung function in children (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Refers to children aged 6-8.

- *Cutting air pollution in Poznan by one fifth may contribute to around 361 fewer children with low lung function (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Refers to children aged 6-8.

### **Warsaw**

- *Cutting air pollution in Warsaw by one fifth would increase children's lung capacity by around 3.2% (long-term).*

Based on a 20% reduction in the long-term average NO<sub>2</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. We chose to use the same % reduction for NO<sub>2</sub>. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

- *Cutting air pollution in Warsaw by one fifth may contribute to a 2.7% greater chance of better lung function in children (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Refers to children aged 6-8.

- *Cutting air pollution in Warsaw by one fifth may contribute to around 1,439 fewer children with low lung function (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Refers to children aged 6-8.

### **Wroclaw**

- *Cutting air pollution in Wroclaw by one fifth would increase children's lung capacity by around 2.6% (long-term).*

Based on a 20% reduction in the long-term average NO<sub>2</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. We chose to use the same % reduction for NO<sub>2</sub>. Compares the resulting predicted change in Forced Vital Capacity (a measure of the volume of the lungs) in children from age 11-15 with the theoretical normal values in children across the same age span.

- *Cutting air pollution in Wroclaw by one fifth may contribute to a 2.5% greater chance of better lung function in children (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Refers to children aged 6-8.

- *Cutting air pollution in Wroclaw by one fifth may contribute to around 470 fewer children with low lung function (long-term).*

Based on a 20% reduction in the long-term average PM<sub>10</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Refers to children aged 6-8.

### **5.5.6 Asthma symptoms in children**

#### **Bielsko-Biala**

- *In Bielsko-Biala, asthmatic children are 0.9% more likely to experience asthma symptoms on high air pollution days than on low pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness.

- *On high air pollution days, 4 more asthmatic children in Bielsko-Biala experience asthma symptoms than on low pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.

#### **Poznan**

- *In Poznan, asthmatic children are 0.7% more likely to experience asthma symptoms on high air pollution days than on low pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness.

- *On high air pollution days, 10 more asthmatic children in Poznan experience asthma symptoms than on low pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.

#### **Warsaw**

- *In Warsaw, asthmatic children are 0.6% more likely to experience asthma symptoms on high air pollution days than on low pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness.

- *On high air pollution days, 28 more asthmatic children in Warsaw experience asthma symptoms than on low pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.

### **Wroclaw**

- *In Wroclaw, asthmatic children are 0.7% more likely to experience asthma symptoms on high air pollution days than on low pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness.

- *On high air pollution days, 11 more asthmatic children in Wroclaw experience asthma symptoms than on low pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of particulate air pollution (PM<sub>10</sub>) levels and these days were reduced to the average of the bottom half of the range of levels. Asthmatic symptoms include cough, wheeze and breathlessness. Applies to children age 5-14.

## **5.5.7 Lung Cancer**

### **Bielsko-Biala**

- *Cutting air pollution in Bielsko-Biala by one fifth would decrease lung cancer cases by around 14.7% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland (there may be a lag between reduction of concentrations and reductions in lung cancer risk). This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

- *Cutting air pollution in Bielsko-Biala by one fifth may contribute to around 19 less lung cancer cases each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland (there may be a lag between reduction of concentrations and reductions in lung cancer risk). This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

### **Poznan**

- *Cutting air pollution in Poznan by one fifth would decrease lung cancer cases by around 13.4% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland (there may be a lag between reduction of concentrations and reductions in lung cancer risk). This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

- *Cutting air pollution in Poznan by one fifth may contribute to around 54 less lung cancer cases each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland (there may be a lag between reduction of concentrations and reductions in lung cancer risk). This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

### **Warsaw**

- *Cutting air pollution in Warsaw by one fifth would decrease lung cancer cases by around 15.1% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland (there may be a lag between reduction of concentrations and reductions in lung cancer risk). This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

- *Cutting air pollution in Warsaw by one fifth may contribute to around 194 less lung cancer cases each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland (there may be a lag between reduction of concentrations and reductions in lung cancer risk). This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

### **Wroclaw**

- *Cutting air pollution in Wroclaw by one fifth would decrease lung cancer cases by around 14.5% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland (there may be a lag between reduction of concentrations and reductions in lung cancer risk). This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

- *Cutting air pollution in Wroclaw by one fifth may contribute to around 68 less lung cancer cases each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland (there may be a lag between reduction of concentrations and reductions in lung cancer risk). This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Lung cancer develops through many steps and smoking is the major cause but air pollution may contribute too.

### 5.5.8 Respiratory admissions all ages

#### Bielsko-Biala

- *The risk of emergency hospitalisations for respiratory disease in Bielsko-Biala is 3.3% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum ozone concentrations.

- *On high air pollution days in Bielsko-Biala, there are on average 16 more hospital admissions for respiratory disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 49.4% on high air pollution days in Bielsko-Biala could save 16 hospital admissions for respiratory disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of xx%.

- *Each year on average, higher air pollution days in Bielsko-Biala can send up to 26 more people to hospital for respiratory disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

#### Poznan

- *The risk of emergency hospitalisations for respiratory disease in Poznan is 3.4% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum ozone concentrations.

- *On high air pollution days in Poznan, there are on average 54 more hospital admissions for respiratory disease each year than on lower air pollution days (short-term).*



Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 54.4% on high air pollution days in Poznan could save 54 hospital admissions for respiratory disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of xx%.

- *Each year on average, higher air pollution days in Poznan can send up to 86 more people to hospital for respiratory disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Warsaw**

- *The risk of emergency hospitalisations for respiratory disease in Warsaw is 3.4% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum ozone concentrations.

- *On high air pollution days in Warsaw, there are on average 243 more hospital admissions for respiratory disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 58.5% on high air pollution days in Warsaw could save xx hospital admissions for respiratory disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of xx%.

- *Each year on average, higher air pollution days in Warsaw can send up to 388 more people to hospital for respiratory disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Wroclaw**

- *The risk of emergency hospitalisations for respiratory disease in Wroclaw is 3.5% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum ozone concentrations.

- *On high air pollution days in Wroclaw, there are on average 80 more hospital admissions for respiratory disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 54.8% on high air pollution days in Wroclaw could save xx hospital admissions for respiratory disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of xx%.

- *Each year on average, higher air pollution days in Wroclaw can send up to 129 more people to hospital for respiratory disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of ozone levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## 5.5.9 Cardiovascular admissions all ages

### Bielsko-Biala

- *The risk of emergency hospitalisations for cardiovascular disease in Bielsko-Biala is 1.9% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

- *On high air pollution days in Bielsko-Biala, there are on average 26 more hospital admissions for cardiovascular disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 58.9% on high air pollution days in Bielsko-Biala could save 26 hospital admissions for cardiovascular disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 58.9%.

- *Each year on average, higher air pollution days in Bielsko-Biala can send up to 48 more people to hospital for cardiovascular disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Poznan**

- *The risk of emergency hospitalisations for cardiovascular disease in Poznan is 1.6% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

- *On high air pollution days in Poznan, there are on average 56 more hospital admissions for cardiovascular disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 58.4% on high air pollution days in Poznan could save 56 hospital admissions for cardiovascular disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 58.4%.

- *Each year on average, higher air pollution days in Poznan can send up to 101 more people to hospital for cardiovascular disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Warsaw**

- *The risk of emergency hospitalisations for cardiovascular disease in Warsaw is 1.4% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

- *On high air pollution days in Warsaw, there are on average 214 more hospital admissions for cardiovascular disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 51.0% on high air pollution days in Warsaw could save 214 hospital admissions for cardiovascular disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 51.0%.

- *Each year on average, higher air pollution days in Warsaw can send up to 390 more people to hospital for cardiovascular disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Wroclaw**

- *The risk of emergency hospitalisations for cardiovascular disease in Wroclaw is 1.5% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of PM<sub>2.5</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily average particulate matter concentrations.

- *On high air pollution days in Wroclaw, there are on average 79 more hospital admissions for cardiovascular disease each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 53.5% on high air pollution days in Wroclaw could save 79 hospital admissions for cardiovascular disease each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 53.5%.

- *Each year on average, higher air pollution days in Wroclaw can send up to 145 more people to hospital for cardiovascular disease than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of PM<sub>2.5</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **5.5.10 Term low birthweight**

### **Bielsko-Biala**

- *Cutting air pollution in Bielsko-Biala by one fifth would decrease the risk of babies being born underweight by around 0.4% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland using the Pedersen et al (2013) study for the CRF. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

- *Cutting air pollution in Bielsko-Biala by one fifth would result in 6 fewer babies born underweight each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

### **Poznan**

- *Cutting air pollution in Poznan by one fifth would decrease the risk of babies being born underweight by around 0.4% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

- *Cutting air pollution in Poznan by one fifth would result in 18 fewer babies born underweight each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

### **Warsaw**

- *Cutting air pollution in Warsaw by one fifth would decrease the risk of babies being born underweight by around 0.4% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

- *Cutting air pollution in Warsaw by one fifth would result in 64 fewer babies born underweight each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

### **Wroclaw**

- *Cutting air pollution in Wroclaw by one fifth would decrease the risk of babies being born underweight by around 0.4% (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

- *Cutting air pollution in Wroclaw by one fifth would result in 22 fewer babies born underweight each year (long-term).*

Based on a 20% reduction in the long-term average PM<sub>2.5</sub> levels in Poland. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3), although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Babies born underweight refers to babies born at term with a birthweight less than 2,500g.

### **5.5.11 Bronchitic symptoms (asthmatic children)**

#### **Bielsko-Biala**

- Cutting air pollution in Bielsko-Biala by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 1.3% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

- Cutting air pollution in Bielsko-Biala by one fifth would result in 12 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

#### **Poznan**

- Cutting air pollution in Poznan by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 1.7% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

- Cutting air pollution in Poznan by one fifth would result in 48 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

## **Warsaw**

- Cutting air pollution in Warsaw by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 2.7% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

- Cutting air pollution in Warsaw by one fifth would result in 247 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

## **Wroclaw**

- Cutting air pollution in Wroclaw by one fifth would decrease the risk of bronchitic symptoms in asthmatic children each year by around 2.2% (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

- Cutting air pollution in Wroclaw by one fifth would result in 72 fewer asthmatic children with bronchitic symptoms each year (long-term).

20% is an arbitrary number for a reduction in long-term NO<sub>2</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Bronchitic symptoms in asthmatic children refers to symptoms of cough and phlegm. While less well known, these are also symptoms of asthma along with wheeze and breathlessness. Refers to children aged 5-14.

### **5.5.12 Acute bronchitis in children**

#### **Bielsko-Biala**

- Cutting air pollution in Bielsko-Biala by one fifth would decrease the risk of a chest infection (acute bronchitis) in children by around 0.7% (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in

practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

- Cutting air pollution in Bielsko-Biala by one fifth would result in 79 fewer children with a chest infection (acute bronchitis) each year (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

### **Poznan**

- Cutting air pollution in Poznan by one fifth would decrease the risk of a chest infection (acute bronchitis) in children by around 0.6% (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

- Cutting air pollution in Poznan by one fifth would result in 233 fewer children with a chest infection (acute bronchitis) each year (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

### **Warsaw**

- Cutting air pollution in Warsaw by one fifth would decrease the risk of a chest infection (acute bronchitis) in children by around 0.8% (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

- Cutting air pollution in Warsaw by one fifth would result in 909 fewer children with a chest infection (acute bronchitis) each year (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.



## Wroclaw

- Cutting air pollution in Wroclaw by one fifth would decrease the risk of a chest infection (acute bronchitis) in children by around 0.7% (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

- Cutting air pollution in Wroclaw by one fifth would result in 300 fewer children with a chest infection (acute bronchitis) each year (long-term).

20% is an arbitrary number for a reduction in long-term PM<sub>10</sub> concentrations. This is roughly the reduction that would be needed to reach the WHO Interim Target 3 (IT3) for PM<sub>2.5</sub>, although in practice, to reach that Target everywhere would result in concentrations well below IT3 in some places. Acute bronchitis means transient inflammation of the upper airways of the lung as a result of a chest infection. (There are other types of chest infections as well). Refers to children aged 6-12.

### 5.5.13 COPD admissions all ages

#### Bielsko-Biala

- *The risk of emergency hospitalisations for COPD in Bielsko-Biala is 4.9% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

- *We calculated a zero impact on high air pollution days in Bielsko-Biala, on hospital admissions for COPD each year.*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 49.4% on high air pollution days in Bielsko-Biala could save 0 hospital admissions for COPD each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 49.4%.

- *Each year on average, higher air pollution days in Bielsko-Biala can send up to 1 more person to hospital for COPD than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

#### Poznan

- *The risk of emergency hospitalisations for COPD in Poznan is 5.0% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

- *On high air pollution days in Poznan, there are on average 6 more hospital admissions for COPD each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 54.4% on high air pollution days in Poznan could save 6 hospital admissions for COPD each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 54.4%.

- *Each year on average, higher air pollution days in Poznan can send up to 10 more people to hospital for COPD than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Warsaw**

- *The risk of emergency hospitalisations for COPD in Warsaw is 5.1% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

- *On high air pollution days in Warsaw, there are on average 38 more hospital admissions for COPD each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 58.5% on high air pollution days in Warsaw could save 38 hospital admissions for COPD each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 58.5%.

- *Each year on average, higher air pollution days in Warsaw can send up to 56 more people to hospital for COPD than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

#### **Wroclaw**

- *The risk of emergency hospitalisations for COPD in Wroclaw is 5.3% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

- *On high air pollution days in Wroclaw, there are on average 7 more hospital admissions for COPD each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 54.8% on high air pollution days in Wroclaw could save 7 hospital admissions for COPD each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 54.8%.

- *Each year on average, higher air pollution days in Wroclaw can send up to 10 more people to hospital for COPD than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **5.5.14 Pneumonia admissions in children**

#### **Bielsko-Biala**

- *The risk of emergency hospitalisations for pneumonia in children in Bielsko-Biala is 5.4% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

- *On high air pollution days in Bielsko-Biala, there are on average 3 more hospital admissions for pneumonia in children each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 49.4% on high air pollution days in Bielsko-Biala could save 3 hospital admissions for pneumonia in children each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 49.4%.

- *Each year on average, higher air pollution days in Bielsko-Biala can send up to 4 more person to hospital for pneumonia in children than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Poznan**

- *The risk of emergency hospitalisations for pneumonia in children in Poznan is 5.5% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

- *On high air pollution days in Poznan, there are on average 14 more hospital admissions for pneumonia in children each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 54.4% on high air pollution days in Poznan could save 14 hospital admissions for pneumonia in children each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 54.4%.

- *Each year on average, higher air pollution days in Poznan can send up to 23 more people to hospital for pneumonia in children than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

### **Warsaw**

- *The risk of emergency hospitalisations for pneumonia in children in Warsaw is 5.6% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

- *On high air pollution days in Warsaw, there are on average 48 more hospital admissions for pneumonia in children each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 58.5% on high air pollution days in Warsaw could save 48 hospital admissions for pneumonia in children each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 58.5%.

- *Each year on average, higher air pollution days in Warsaw can send up to 77 more people to hospital for pneumonia in children than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## **Wroclaw**

- *The risk of emergency hospitalisations for pneumonia in children in Wroclaw is 5.8% higher on high air pollution days than on lower air pollution days (short-term).*

Assumes typical high air pollution days are at the average of the top half of the annual range of O<sub>3</sub> levels and typical low air pollution days were at the average of the bottom half of the range of levels. In more technical terms, this is the difference between the 75<sup>th</sup> and 25<sup>th</sup> percentile of daily 8-hour maximum concentrations.

- *On high air pollution days in Wroclaw, there are on average 17 more hospital admissions for pneumonia in children each year than on lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels.

- *Lowering air pollution by 54.8% on high air pollution days in Wroclaw could save 17 hospital admissions for pneumonia in children each year (short-term/alternative).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. (The 75<sup>th</sup> to the 25<sup>th</sup> percentile). This is a change in air pollution level on high days of 54.8%.

- *Each year on average, higher air pollution days in Wroclaw can send up to 27 more people to hospital for pneumonia in children than lower air pollution days (short-term).*

Assumes half the year was at the average of the top half of the annual range of O<sub>3</sub> levels and these days were reduced to the average of the bottom half of the range of levels. Figure given uses the upper 95% confidence interval of the concentration-response function.

## 6 Discussion, conclusions and further work

### 6.1 Discussion and conclusions

The aim of this work was to develop statements relating to the potential adverse effects of ambient outdoor air pollution on a range of diseases and health outcomes other than premature mortality. The motivation for this was to make statements which are more relevant to those sections of the public which may be vulnerable to specific health outcomes. Moreover, we also aimed to make the statements specific to individual cities to add to the relevance to the public. For the first time therefore, we have assessed the risks to a range of outcomes in individual towns and cities in the UK and in Poland.

In doing this we have relied on the epidemiological literature and while there are a large number of studies in these areas we have scrutinised papers to ensure that we have used credible robust studies which we felt were generally applicable to our task. Wherever possible we have relied on consensus assessments like those produced by the WHO in their REVIHAAP/HRAPIE reports (WHO 2013a and b) and the expert advice from the UK Committee on the Medical Effects of Air Pollutants (COMEAP).

There are a number of advances in the work presented here. Some of the outcomes such as respiratory hospital admissions and cardiovascular hospital admissions have been quantified previously on quite a regular basis. Others based on recommendations from WHO (2013a) have been included in European work and within cost-benefit analyses but not necessarily in statements for the public. Others have not been quantified previously, although some are subsets of broader health outcomes that are more usually quantified e.g. pneumonia admissions are part of respiratory hospital admissions. Some outcomes only required new inputs to established methods but others needed methodological development such as expressing a change in Forced Vital Capacity in millilitres as a % change in lung function growth compared with the ideal.

Other organisations and researchers have done work in this broad area of air pollution impacts on health. Public Health England (PHE, 2018) have published a report setting out a toolkit for estimating the costs to the National Health Service of some health impacts of air pollution. The study did not however deal with as many health outcomes as our current work. In the EU, a cost-benefit analysis was carried out by Holland and published in 2017 European Union, 2017, M. Amman ed.), again analysing some health outcomes but fewer than we consider here. Similarly at a global scale, the Global Burden of Disease study (Cohen et al, 2017) dealt with the impact of air pollution and several types of cause-specific mortality - COPD, lung cancer, ischaemic heart disease, and stroke but again fewer than we consider here, and moreover the study concentrated mainly on mortality and loss of life expectancy.

We have made clear that this study has relied on our own judgement of the literature – chiefly epidemiological in choosing the CRFs, rather than it being a consensus view of a group of experts as has been done by WHO for example (WHO, 2013b). However wherever sound studies were available we have used meta-analyses rather than rely on single papers. Where meta-analyses were not available, we used what we considered the most comprehensive and sound single studies.

We recognise too that we have not considered the issue of causality here and so in that regard our findings could be regarded as preliminary. The quantification of the impacts that we have carried out will of course be subject to updating as newer studies appear and as further meta-analyses and consensus assessments of the literature are made.

As in a large proportion of time-series epidemiological studies, we have used fixed-point monitoring data to characterise personal exposure. It is well known that there are uncertainties attached to this but at present this approach represents the best available method of quantifying exposures.

One area of uncertainty which we have sought to reduce is that of year-on-year variability of air pollutant concentrations due to variable meteorology. We have minimised this by taking three-year averages of the pollutant concentrations at all sites

In carrying out this work we have collaborated closely with Purpose UK, an organisation with considerable expertise in public communication, to ensure the statements that we produce are relevant and meaningful to the public at large. This has meant a merging of sound science with text which is nonetheless understandable by the lay public. We believe therefore that we have produced a collection of statements which are not only scientifically credible but which convey quantitative assessments of risk to the population from ambient air pollution in a range of cities.

## **6.2 Further work**

Considerable detailed work has been invested in producing the results in the present report. Inevitably though, some aspects could be improved, and the work could be further extended.

One of the aims was to be really clear about the sources for the evidence used to provide the statements. This would allow users of the statements to know the exact date of the evidence used. Some evidence comes from an earlier date than others which gives some indication of what statements might need to be considered for updating first. On the other hand, some areas of the literature are more active than others. Air pollution and birth outcomes is an active research area at present and COMEAP is preparing a report on this subject.

The timing or frequency of updates to the statements would probably be determined primarily by significant advances in the epidemiological literature, either by consensus assessments of the kind carried out by WHO, or by the emergence of important large meta-analyses. Even if pollutant concentrations were changing by a relatively small year-on-year amount, the variability due to changing meteorology would be difficult to distinguish from the effects of policy measures so these effects could always be allowed for by taking (as we have done here) averages over several historical years. An exception of course which would need to be considered would be major changes in air quality as the result of strong policy measures such as the large reductions in the sulphur content of petrol and diesel, when the concentrations of ultrafine particles decreased dramatically in a matter of months. A historical example would be the removal of lead in petrol when ambient concentrations of lead reduced by very large amounts, also in a matter of a few months. Further work could include an evolving database of new epidemiological studies relevant to the outcomes considered here (including those we chose not to take forward because of insufficient evidence). Such a database would allow timely updates to the statements published here.

We used monitoring data rather than modelled data in this project. As explained in the limitations section, it would be better to use modelled concentrations for the long-term exposure data calculations if sufficient resources were available.

The methods used here can be applied to other locations if the appropriate input data is available. This would be most effective if combined with focus group work to see which particular health outcome statements were of most interest in specific new locations.

We are aware that due to the wide scope of our work, the report is quite long. It would be possible to design software and an interface that allowed users to select their city and outcome of interest. They could then be provided with a printout or electronic document with the specific statement, together with its technical justification. At first this might be limited to the statements present in the report, but it could be used in future to allow periodic updates. Note that this would not be a direct health impact assessment tool – such tools are already available for some outcomes e.g. WHOAIRQ+, but other tools do not produce an output in the form of user-friendly statements.

Some baseline rates could not be obtained in the time available but might be able to be obtained or inferred using population data with more time. Short term exposure and myocardial infarction is an example which also had methodological challenges with the particular design of studies that could potentially be used.

Some health outcomes discussed in Chapter 3 and the associated annex were not taken forward at this particular time despite a concentration-response function being available. Further work on these aspects could be done. Some were not pursued because they were not a priority in focus group testing, but this could vary in different location.

There are some broader issues in health impact assessment that might be possible to take into account in future. One particular aspect is the overlap between the effects of different pollutants in the original studies. Statistical techniques such as multi-pollutant models are available for this but can be difficult to interpret in the presence of measurement error and close correlations between pollutants. Work is ongoing to improve statistical approaches to this issue which might change the method we chose of selecting results for just one pollutant.

There is some literature on health effects of air pollution in susceptible groups other than asthmatics (the main sensitive group apart from children that we addressed here). It is not necessarily available in a summary form in a published meta-analysis. Sometimes this is because of a lack of studies; otherwise it may be that enough studies are available, but no meta-analysis has been done yet. Potentially higher risks may occur in these groups e.g. further hospital admissions in those that already have heart disease, individuals with particular forms of antioxidant enzymes that are less able to protect the lung from pollutants. This is an interesting area for further work.

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## ANNEX A: AIR POLLUTION CONCENTRATIONS IN THE UK AND POLAND

*Table A2 - Median, 25th and 75th percentiles (IQR) of the overall air pollution data for a yearly time series dataset averaging the concentrations from 2015 to 2017 in various UK and Polish cities.*

City	NO <sub>2</sub> (µg/m <sup>3</sup> ) Median (IQR)	PM <sub>2.5</sub> (µg/m <sup>3</sup> ) Median (IQR)	O <sub>3</sub> (µg/m <sup>3</sup> ) Median (IQR)	PM <sub>10</sub> (µg/m <sup>3</sup> ) Media (IQR)	CO (µg/m <sup>3</sup> ) Median (IQR)	SO <sub>2</sub> (µg/m <sup>3</sup> ) Median (IQR)
London	47.2 (41.8-53.6)	11.1 (9.1-14.5)	45.5 (35.7-54.1)	20.6 (17.8-24.6)	0.30 (0.27-0.34)	3.9 (3.4-4.4)
Birmingham	29.2 (23.9-35.2)	9.3 (7.6-13.3)	52.3 (43.4-63.5)	14.2 (11.7-18.5)	NA	0.7 (0.4-1.0)
Bristol	26.4 (21.6-33.7)	8.7 (6.6-12.1)	55.9 (47.3-65.4)	13.6 (11.4-17.5)	NA	NA
Oxford	31.6 (27.1-36.7)	8.8 (7.0-11.7)	NA	11.1 (8.7-14.8)	NA	NA
Southampton	36.2 (30.1-43.1)	8.6 (6.9-11.7)	51.6 (43.3-59.9)	18.2 (14.9-23.1)	NA	1.5 (1.1-1.9)
Liverpool	24.4 (19.7-30.8)	7.8 (6.0-11.0)	58.4 (49.9-68.1)	12.9 (10.2-16.5)	NA	1.8 (1.5-2.4)
Manchester	29.3 (24.0-36.1)	8.2 (12.1-6.1)	46.8 (37.1-55.6)	NA	NA	1.5 (1.1-1.8)
Derby	35.4 (27.7-44.8)	NA	NA	NA	NA	NA
Nottingham	32.3 (25.8-40.1)	9.8 (8.0-13.7)	48.1 (38.1-57.9)	16.0 (13.4-21.1)	NA	2.0 (1.7-2.4)
Warsaw	37.0 (32.0-41.1)	21.2 (15.3-31.2)	56.5 (32.6-78.5)	31.8 (25.8-41.2)	0.49 (0.44-0.57)	3.7 (2.7-5.5)
Wroclaw	30.1 (27.1-34.2)	20.5 (14.7-31.6)	61.6 (38.7-85.7)	29.1 (23.2-39.8)	0.47 (0.39-0.62)	3.1 (2.4-4.7)
Poznan	23.2 (18.6-27.0)	19.0 (12.8-30.8)	60.8 (37.6-82.4)	26.1 (20.3-38.3)	0.34 (0.26-0.45)	2.5 (2.0-3.8)
Bielsko-Biala	18.7 (14.3-24.1)	20.7 (14.4-35.0)	64.3 (44.7-88.4)	28.3 (20.8-44.5)	0.40 (0.24-0.62)	6.6 (3.7-11.3)



**Table A3 - Median, 25<sup>th</sup> and 75<sup>th</sup> percentiles (IQR) of air pollution data by type of monitor(urban background; roadside) for a yearly time series dataset averaging the concentrations from 2015 to 2017 in various UK cities<sup>13</sup>.**

City	NO <sub>2</sub> (µg/m <sup>3</sup> ) Median (IQR)		PM <sub>2.5</sub> (µg/m <sup>3</sup> ) Median (IQR)		O <sub>3</sub> (µg/m <sup>3</sup> ) Median (IQR)		PM <sub>10</sub> (µg/m <sup>3</sup> ) Median (IQR)		CO (µg/m <sup>3</sup> ) Median (IQR)		SO <sub>2</sub> (µg/m <sup>3</sup> ) Median (IQR)	
	Backgrnd	Roads	Backgrnd	Roads	Backgrnd	Roads	Backgrnd	Roads	Backgrnd	Roads	Backgrnd	Roads
London	29.7 (25.3-36.0)	58.3 (52.5-64.4)	9.7 (7.6-13.2)	12.5 (10.4-15.9)	51.4 (40.4-61.4)	34.4 (26.6-41.8)	16.4 (13.9-20.4)	21.8 (18.9-26.2)	0.21 (0.19-0.25)	0.47 (0.40-0.54)	3.2 (2.7-3.6)	5.0 (4.2-5.8)
Birmingham	22.9 (17.3-28.3)	40.4 (33.6-47.0)	9.0 (7.2-13.1)	10.2 (8.2-13.7)	56.5 (47.5-68.4)	44.7 (36.0-54.8)	13.9 (11.2-17.8)	14.1 (11.6-19.6)	NA	NA	0.7 (0.4-1.0)	NA
Bristol	23.9 (18.3-31.4)	36.0 (25.8-47.6)	8.7 (6.6-12.1)	NA	55.9 (47.3-65.4)	NA	13.5 (11.3-17.2)	20.3 (15.4-25.6)	NA	NA	NA	NA
Oxford	12.9 (9.6-17.0)	45.2 (39.1-52.2)	8.8 (7.0-11.7)	NA	NA	NA	11.1 (8.7-14.8)	NA	NA	NA	NA	NA
Southampton	29.7 (25.0-36.3)	38.3 (31.2-44.9)	8.6 (6.9-11.7)	NA	51.6 (43.3-59.9)	NA	14.9 (11.7-19.3)	19.7 (16.2-25.3)	NA	NA	1.5 (1.1-1.9)	NA
Liverpool	20.8 (14.7-26.0)	31.2 (25.1-39.1)	7.8 (6.0-11.0)	NA	58.4 (49.9-68.1)	NA	12.9 (10.2-16.5)	NA	NA	NA	1.8 (1.5-2.4)	NA
Nottingham	28.8	35.3	9.8 (8.0-13.7)	NA	48.1 (38.1-57.9)	NA	15.6 (13.0-20.4)	16.5	NA	NA	2.0 (1.7-2.4)	NA

<sup>13</sup> Manchester and Derby have not been included in this table as there is no extra information from the previous table.

	(23.7- 37.4)	(28.0- 46.8)						(13.3- 22.2)				
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## ANNEX B: CONCENTRATION RESPONSE FUNCTIONS, THEIR SELECTION AND JUSTIFICATION

### B1. Cardiovascular hospital admissions (short-term exposures, all ages and elderly)

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)	Comments
<b>PM<sub>2.5</sub></b>	Short-term	Atkinson et al 2014	Percentage increase: 0.91% (0.17, 1.66) per 10µg/m <sup>3</sup>	Based on estimates for WHO EUR A region (various lags). All ages
<b>PM<sub>10</sub></b>	Short-term	Katsouyanni et al 2009	Percentage increase: 0.60% (0.20, 1.00) per 10µg/m <sup>3</sup>	Based on estimates from 8 European cities. Ages 65+. 8df/year, lag 0-1
<b>NO<sub>2</sub></b>	Short-term	Mills et al 2015	Percentage increase: 0.42% (0.23-0.62) per 10µg/m <sup>3</sup>	Based on European estimates. various lags. All ages
<b>NO<sub>2</sub></b>	Short-term	Mills et al 2015	Percentage increase: 1.02% (0.08-1.97) per 10µg/m <sup>3</sup>	Based on European estimates. various lags. Elderly
<b>O<sub>3</sub></b>	Short-term	Walton et al 2014	Percentage increase: 0.44% (0.01- 0.88) per 10 µg/m <sup>3</sup> 8-hour ozone	Based on estimates for WHO EUR A region. Various lags. Elderly
<b>O<sub>3</sub></b>	Short-term	APHENA (HRAPIE, 2013)	Percentage increase: 0.89% (0.50–1.27) per 10 µg/m <sup>3</sup> 8-hour ozone	Age: 65+, Excluding STROKE

**Justification:** The Atkinson review (like other papers) notes that there is wide heterogeneity across the globe in estimates. While this heterogeneity is perhaps not surprising given the possible differing sensitivities in populations across the world, and, taking as well into account that health care systems vary widely and there are identified effect modifiers (Requia et al 2018), we choose European estimates when possible.

Specifically, for each pollutant: or  $PM_{2.5}$  the CRF of choice is that reported by Atkinson et al (2014), which is also recommended by the WHO HRAPIE report 2013b. For  $PM_{10}$  the only paper is the Requia et al (2018) estimate and we chose not to use it, because it is global and because it merges CVD and Respiratory causes. From the HEI APHENA report (as reported in HRAPIE) we have  $PM_{10}$  per  $10\mu g/m^3$  based on 8 EU cities and we chose to use this one. For  $SO_2$ , we had CRFs but we choose not to use them, because  $SO_2$  is generally low in Europe. For  $NO_2$  we choose the European estimate from Mills et al. For CO the estimate from the Requia study is only based on 2 studies from Brazil and China and we chose not to use it. For ozone, we used two estimates both of which are for the elderly: The estimate from the APHENA study recommended by the WHO HRAPIE Report from European cities, and also the paper by Walton 2014 which is for the WHO EURA region.

## B2. Respiratory hospital admissions (short-term exposures, all ages and elderly)

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)	Comments
$PM_{2.5}$	Short-term	Atkinson et al 2014	Percentage increase: 1.90% (-0.18-4.02) per $10\mu g/m^3$	For the WHO EUR A region. Various lags. All ages
$PM_{2.5}$	Short-term	Atkinson et al 2014	Percentage increase: 0.99% (-0.90-2.92) per $10\mu g/m^3$	For the WHO EUR A region. Various lags. Elderly (ages 65+)
$PM_{10}$	Short-term	Katsouyanni et al 2009	Percentage increase: 0.60% (0.25-0.95) per $10\mu g/m^3$	Based on 8 European cities Elderly, ages 65+
$NO_2$	Short-term	Mills et al 2015	Percentage increase: 0.52% (0.09-0.95) per $10\mu g/m^3$	Based on estimates from European studies. Various lags. All ages.
$O_3$	Short-term	APHENA (HRAPIE, 2013)	Percentage increase: 0.44% (0.07–0.83) per $10\mu g/m^3$ 8-hr max	Based on estimates from European studies. Ages 65+ years
		Walton 2014	Percentage increase: 0.47% (-0.21-1.15) per $10\mu g/m^3$ 8-hr max	For the WHO EUR A region. Ages 65+ years.
		COMEAP Report 2015 recommendation	Percentage increase: 0.75% (0.30-1.20) per $10\mu g/m^3$ 8-hour ozone	All ages

**Justification:** The Atkinson review (like other papers) notes that there is wide heterogeneity in estimates across the globe. Taking this into account we chose not to use global estimates if possible. Specifically, by pollutant: For PM<sub>2.5</sub> we chose the CRF from Atkinson et al 2014, which is also recommended by HRAPIE. From the HEI APHENA report (2009) we chose the CRF for PM<sub>10</sub> based on 8 EU cities among 65+ years old individuals. For SO<sub>2</sub> we do not suggest using CRFs because SO<sub>2</sub> is generally low in Europe. For NO<sub>2</sub> we chose the EU estimate from Mills et al (2015). For CO the estimate from the Requia paper is only based on 2 studies from Brazil and China and we do not suggest using it. For ozone: we have two estimates for the elderly, i.e. the APHENA (Katsouyanni 2009) also recommended by HRAPIE (2013) and Walton 2014. In magnitude they are practically identical, so either may be used. For all ages we adopt the COMEAP, 2015, recommendation.

### B3. COPD hospital admissions

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)	Comments
PM <sub>10</sub>	Short-term	Moore et al 2016	Odds Ratio of 1.01 (1.00-1.01) per 10 µg/m <sup>3</sup>	All ages, only European studies, n= 12
*NO <sub>2</sub>	Short-term	Moore et al 2016	Odds Ratio: 1.01 (1.00-1.02) per 10 µg/m <sup>3</sup>	European studies n=13
NO <sub>2</sub>	Short-term	Anderson et al 1997 & Colais 2009 from Mills et al 2015	Percentage increase: 0.81% (0.03-1.59) per 10 µg/m <sup>3</sup>	all ages
PM <sub>2.5</sub>	Short-term	Moore et al 2016	Odds Ratio: 1.02 (0.99-1.04) per 10 µg/m <sup>3</sup>	All ages European studies, only 3
		Atkinson et al 2014	Percentage increase: 3.93% (1.06-6.89) per 10 µg/m <sup>3</sup>	EUR A 65+ without asthma, 2 studies
O <sub>3</sub>	Short term	Walton et al 2014	Percentage increase: 1.12% (0.59-1.66) per 10 µg/m <sup>3</sup>	All ages EURA 6 cities 8 hr average
CO	Short-term	Moore et al 2016	Odds Ratio: 1.04 (1.02-1.06) per 1 mg/m <sup>3</sup>	Based on 6 European studies
<b>*In the paper it is not specified whether the NO<sub>2</sub> concentrations are 24-h or something else. We have assumed that they are 24-h</b>				

**Justification:** We have selected, where possible, European study estimates. We do not refer to SO<sub>2</sub> estimates because generally levels are low in Europe. For PM<sub>2.5</sub> there are only 3 studies and the increase (2%) is not statistically significant but is nearly significant. For ozone effects Moore 2016 and Walton 2014 are practically identical, so we use Walton et al. because Moore et al mixed hospital admissions and emergency department visits.

#### B4. Lung function decrements and symptoms in COPD patients

Pollutant	Health outcome	Time reference	CRF Source	CRF (95% confidence interval)
PM <sub>10</sub>	FEV1	Short-term	De Hartog 2010 from Bloemsma et al 2016	Decrease in FEV1 20-35 mL per 50 µg/m <sup>3</sup> , corresponds to 1-2% of typical population mean FEV1 of adults with COPD
PM <sub>10</sub>	Shortness of breath	Short-term	Karakatsani 2012 from Bloemsma et al 2016	Odds Ratio: 1.04 (1.00-1.07) per 10 µg/m <sup>3</sup>

**Justification:** The available studies are: de Hartog 2010 with 4 cities (Amsterdam, Athens, Birmingham, Helsinki) across Europe; one in Rome which shows a decrease of -23.5, very far from the combined estimate; one in London with a null result. We chose to consider the estimate from de Hartog (which is in magnitude near to the overall combined value and spans cities across Europe). However, further investigation of the exact definition of subjects (for the population at risk needed for later calculations) revealed that 69% of the patients in de Hartog et al were asthmatics (even more in some cities). Particularly at older ages, it is difficult to distinguish between COPD and asthma (Miravittles et al 2012), but de Hartog et al used subjects for a full range of ages. A mixed patient group can be a challenge for defining the population at risk, and we already have other concentration-response functions for health outcomes in asthmatic patients, at least. We chose not to pursue this outcome further at this stage.

For symptoms, the Bloemsma (2016) review gives an estimate for shortness of breath from Karakatsani et al from Europe, for the same 4 cities as above; Peacock from London (1995-97) with an estimate of 1.06 for dyspnoea (breathlessness) and Alahmari (2011-13) with null effect also for dyspnoea. For dyspnoea we decided that we would not use an estimate as the Peacock study is very old and the 2 London studies are heterogeneous.

#### B5. Myocardial Infarction (short- and long-term exposures, all ages)

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)
PM <sub>2.5</sub>	Short-term: all lags, up to 7 days but most were 0	Mustafic et al 2012	Relative Risk: 1.025 (1.015-1.036) per 10µg/m <sup>3</sup>
	Long-term: annual average	Cesaroni et al 2014	Hazard Ratio: 1.13 (0.98-1.30) per 5µg/m <sup>3</sup>
PM <sub>10</sub>	Short-term: up to 7 days	Mustafic et al 2012	Relative Risk: 1.006 (1.002-1.009) per 10 µg/m <sup>3</sup>
	Short term: lag 0	Lanki et al 2006	Relative Risk: 1.003 (0.995 – 1.011) per 10 µg/m <sup>3</sup>
	Short term (6 hour average)	Bhaskaharan et al 2011	Relative Risk: 1.012 (1.003 – 1.021) per 10 µg/m <sup>3</sup>

	Long-term: annual average	Cesaroni et al 2014	Hazard Ratio: 1.12 (1.01-1.25) per 10µg/m <sup>3</sup>
<b>NO<sub>2</sub></b>	Short-term: up to 7 days	Mustafic et al 2012	Relative Risk: 1.011 (1.006-1.016) per 10 µg/m <sup>3</sup>
	Short term: lag 0	Lanki et al 2006	Relative Risk: 0.996 (0.988 – 1.008) per 8 µg/m <sup>3</sup>
	Short term (6 hour average)	Bhaskaharan et al 2011	Relative Risk: 1.011 (1.003 – 1.018) per 10µg/m <sup>3</sup>
	Long-term: annual average	Cesaroni et al 2014	Hazard Ratio: 1.03 (0.97-1.08) per 10µg/m <sup>3</sup>
<b>O<sub>3</sub></b>	Short term: lag 0	Lanki et al 2006	Relative Risk: 0.991 (0.979 – 1.003) per 15 µg/m <sup>3</sup>
	Short term (6 hour average)	Bhaskaharan et al 2011	Relative Risk: 0.998 (0.992 – 1.004) per 10 µg/m <sup>3</sup>
<b>SO<sub>2</sub></b>	Short term (6 hour average)	Bhaskaharan et al 2011	Relative Risk: 1.000 (0.978 – 1.023) per 10 µg/m <sup>3</sup>
<b>CO</b>	Short-term: up to 7 days	Mustafic et al 2012	Relative Risk: 1.048 (1.026-1.070) per 1 µg/m <sup>3</sup>
	Short term: lag 0	Lanki et al 2006	Relative Risk: 1.005 (1.000 – 1.010) per 0.2 mg/m <sup>3</sup>
	Short term (6 hour average)	Bhaskaharan et al 2011	Relative Risk: 1.002 (0.997 – 1.007) per 0.1 mg/m <sup>3</sup>

**Justification:** For PM<sub>2.5</sub> the meta-analysis of Mustafic et al (2012) could be considered as it includes 13 studies and Europe is well represented. The study does not give a separate EU estimate. However, it mixes studies of hospital admissions and studies of deaths within the same meta-analysis. This makes use in health impact assessment difficult as the appropriate baseline rate is hard to define (perhaps some mixture of admissions and deaths weighted according to relative study weights in the meta-analysis) and the result would be hard to express simply. The Argacha et al 2016 paper, based on a large Belgian study, gives a very similar result 1.028 (1.003, 1.054), but is only based on one country. Consequently we decided not to use the Mustafic et al paper.

PM<sub>10</sub>: Here too the meta-analysis of Mustafic et al (2012) could have been considered as it includes 17 studies and Europe is well represented but has problems with outcome definition. The Argacha et al 2016 paper gives a larger effect 1.026 (1.005, 1.048), but it is only based on one country., Bhaskaharan et al 2011 is an interesting study of the timing of air pollution associated myocardial

infarction or acute coronary syndrome across 15 conurbations in England and Wales. This found a positive and statistically significant association with PM<sub>10</sub> within the first 6 hours but not after a longer lag. After correspondence with the authors, we concluded that this was too complicated to incorporate into a health impact assessment context at this stage. Lanki et al (2006) is a study of hospital admissions for first myocardial infarction across 5 European cohorts showing an association with PM<sub>10</sub> that was almost statistically significant. This could potentially be selected if appropriate baseline rates could be obtained.

For NO<sub>2</sub> the meta-analysis of Mustafic et al (2012) includes 21 studies and Europe is well represented but uses a mixture of hospital admissions and deaths studies. Again we rejected the Argacha et al 2016 paper as although it gives a larger effect 1.051 (1.018, 1.084), it is only based on one country. Bhaskaharan et al 2011 showed a positive and statistically significant association but would be too complicated to implement in HIA. Lanki et al 2006 did not find an association between admission for first MI and NO<sub>2</sub>.

SO<sub>2</sub> was not considered because SO<sub>2</sub> levels are mostly low in European countries.

For ozone the Mustafic estimate is not statistically significant. Nor were the Lanki et al or Bhaskaharan et al estimates. We conclude that at this point there is insufficient evidence to consider ozone.

For CO there is a statistically significant effect based on 20 studies from Mustafic et al And a marginally significant association in Lanki et al and Bhaskharan et al. Lanki et al could potentially be taken forward if baseline rates were available.

The paper by Cesaroni *et al* (2014) is part of the ESCAPE project based on 11 European cohorts and is quite robust. The association with PM<sub>10</sub> is statistically significant. The associations with PM<sub>2.5</sub> and NO<sub>2</sub> are close to statistical significance.

## B6. Cerebrovascular Disease (Stroke)

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)
PM <sub>2.5</sub>	Short-term: varying lags but most between 0 and 2, up to 7 days	Shah et al 2015	Relative Risk: 1.011 (1.010-1.012) per 10 µg/m <sup>3</sup>
	Long-term: annual average	Stafoggia et al 2014	Hazard Ratio: 1.19 (0.88-1.62) per 5µg/m <sup>3</sup>
PM <sub>10</sub>	Short-term: varying lags but most between 0 and 2, up to 7 days	Shah et al 2015	Relative Risk: 1.002 (1.000 -1.003) per 10 µg/m <sup>3</sup>
	Long-term: annual average	Stafoggia et al 2014	Hazard Ratio: 1.11 (0.90-1.36)per 10µg/m <sup>3</sup>
NO <sub>2</sub>	Short-term: varying lags but most between 0 and 2, up to 7 days	Shah et al 2015	Relative Risk: 1.012 (1.005 -1.018) per 10 ppb
O <sub>3</sub>	Short-term: varying lags but most between 0 and 2, up to 7 days	Shah et al 2015	Relative Risk: 1.001 (1.000 -1.002) per 10 ppb



<b>CO</b>	Short-term: varying lags but most between 0 and 2, up to 7 days	Shah et al 2015	Relative Risk: 1.011 (0.999 -1.023) per 1 ppm
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**Justification:** Short-term exposures: For PM<sub>2.5</sub>, PM<sub>10</sub>, NO<sub>2</sub>, Ozone and CO the Shah et al 2015 paper is based on 6.2 million events across 28 countries (103 articles) but most are from Europe and N. America. For PM<sub>2.5</sub> overall 41 estimates. For PM<sub>10</sub> there are overall 78 estimates, for NO<sub>2</sub> 70 estimates in all, for ozone 53 estimates, and for CO 37 estimates in all. We excluded SO<sub>2</sub> as before. We have chosen the estimate for admissions excluding mortality. In particular, we used the effect estimates for hospital admissions from a stratified analysis in the supplementary material of the paper (appendix 7 of the supplementary material to the Shah paper). The number of studies included in the meta-analysis of the hospital admissions-air pollution associations is smaller than the studies included in the overall analysis. However, these figures can still be regarded as enough for a pooled effect estimate (29, 42, 46, 35 and 29 number of estimates included in the meta-analysis for PM<sub>2.5</sub>, PM<sub>10</sub>, NO<sub>2</sub>, Ozone and CO respectively). This choice of the CRFs used for our calculations made finding baseline rates easier because we searched for emergency hospital admissions rather than admissions plus cause-specific deaths.

Long-term: We used the estimates from Stafoggia et al 2014, from the ESCAPE project based on European cohorts. ESCAPE only considered PM and NO<sub>2</sub>. The estimates for PM are not statistically significant at the nominal level, but are positive. For NO<sub>2</sub> there was no evidence of effect in the paper.

## B7. Heart Failure

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)
<b>PM<sub>2.5</sub></b>	Short-term: varying lags but most between 0 and 2, up to 7 days	Shah et al 2013	Percentage increase: 2.12% (1.42-2.82) per 10 µg/m <sup>3</sup>
<b>PM<sub>10</sub></b>	Short-term: varying lags but most between 0 and 2, up to 7 days	Shah et al 2013	Percentage increase: 1.63% (1.20-2.07) per 10 µg/m <sup>3</sup>
	Long-term: annual average	Atkinson et al 2013	Hazard Ratio: 1.06 (1.01–1.11) per 3.0 µg/m <sup>3</sup> (IQR)
<b>NO<sub>2</sub></b>	Short-term: varying lags but most between 0 and 2, up to 7 days	Shah et al 2013	Percentage increase: 1.70% (1.25-2.16) per 10 ppb
	Long-term: annual average	Combined estimate from Atkinson et al 2013 and Sorensen et al 2017	Combined Hazard Ratio: 1.073 (1.026-1.121) per 10 µg/m <sup>3</sup>
<b>SO<sub>2</sub></b>	Short-term: varying lags but most between 0 and 2, up to 7 days	Shah et al 2013	Percentage increase: 2.36% (1.35-3.38) per 10 ppb

	Long-term: annual average	Atkinson et al 2013	Hazard Ratio: 1.04 (1.01–1.08) per 2.2 $\mu\text{g}/\text{m}^3$ (IQR)
<b>O<sub>3</sub></b>	Short-term: varying lags but most between 0 and 2, up to 7 days	Shah et al 2013	Percentage increase: 0.46% (-0.10-1.02) per 10 ppb
<b>CO</b>	Short-term: up to 7 days	Shah et al 2013	Percentage increase: 3.52% (2.52-4.54) per 1 ppm

**Justification:** Short-term exposures: For PM<sub>2.5</sub>, PM<sub>10</sub>, NO<sub>2</sub>, Ozone and CO we used the Shah et al 2013 paper which gives an estimate from many studies from the whole world (4 million events 35 articles). Heart failure hospitalisation or death is reported. The number of estimates for PM<sub>2.5</sub> is 26, for PM<sub>10</sub> 11, for NO<sub>2</sub> 28 for SO<sub>2</sub> 23, for ozone 25 and for CO 27. About one third to half of the studies come from the U.S. and when the authors calculated separate U.S. and non-U.S. estimates there is geographical modification. However, the authors do not give European estimates separately. Also, they do not give separate estimates only for admissions but the graph in the paper suggests that there is little difference between US and non-US estimates. The consistency and magnitude of effects indicates that heart failure may be significantly affected by air pollution to a large extent.

Long-term exposures: PM<sub>2.5</sub> and CO were not considered by Atkinson et al, 2013b. For PM<sub>10</sub> the Atkinson et al estimate is chosen based on a large UK administrative cohort. For NO<sub>2</sub> we combined the results from 2 studies, one from the UK (Atkinson et al 2013 as before) and one from Denmark (Sorensen et al 2017), using random effects meta-analysis. Atkinson et al 2013 considered ozone and found no effects.

## B8. Hypertension (H) and Diastolic Blood Pressure (DBP)

Pollutant	Time reference	CRF Source	CRF
<b>PM<sub>2.5</sub></b>	Short-term: up to 7 days	Yang et al 2018	Odds Ratio for H: 1.10 (1.06 -1.13), Mean increase in DBP (mmHg):0.20 (0.02, 0.38) per 10 $\mu\text{g}/\text{m}^3$
	Long-term: annual average	Yang et al 2018	Odds Ratio for H: 1.05 (1.01,1.09), Mean increase in DBP (mmHg):0.47 (0.12, 0.82) per 10 $\mu\text{g}/\text{m}^3$
<b>PM<sub>10</sub></b>	Short-term: up to 7 days	Yang et al 2018	Odds Ratio for H: 1.06 (1.02, 1.10), Mean increase (mmHg) in DBP:0.15 (0.01, 0.29) per 10 $\mu\text{g}/\text{m}^3$
	Long-term: annual average	Yang et al 2018	Odds Ratio for H: 1.04 (0.99 -1.09), Mean increase in DBP (mmHg):0.86 (0.37, 1.35) per 10 $\mu\text{g}/\text{m}^3$
<b>NO<sub>2</sub></b>	Short-term: up to 7 days	Yang et al 2018	Odds Ratio for H: 1.05 (1.02 -1.08), Mean increase in

	Long-term: annual average		DBP (mmHg) 0.28 (0.15, 1.13) per 10 µg/m <sup>3</sup>
		Cai et al 2015	Odds Ratio for H: 1.046 (1.012, 1.081) per 10 µg/m <sup>3</sup>
		Yang et al 2018	Mean increase in DBP (mmHg) :0.77 (0.38, 1.17) per 10 µg/m <sup>3</sup>
O <sub>3</sub>	Short-term: up to 7 days	Yang et al 2018	Mean increase in DBP:0.13 (-0.01, 0.28) per 10 µg/m <sup>3</sup>
	Long-term		Mean increase in DBP:0.17 (-0.02, 0.36) per 10 µg/m <sup>3</sup>

**Justification:** There are 2 meta-analyses on air pollution and hypertension, Cai et al 2016 and Yang et al 2018, both addressing effects of short and long-term exposures. They both have a major disadvantage: for effects of short-term exposures, there are practically no results from European studies. However, because increases in blood pressure and in the risk of hypertension are very important for health, we decided to choose the most recent paper Yang et al 2018. An additional advantage of the Yang et al paper is that it provides not only the risk for hypertension but also the increase in systolic (SBP) and diastolic blood pressure (DBP). Between the 2, DBP was associated with more consistent and SS associations and thus we concentrated on DBP. We noted that the comparable results between the 2 papers were consistent with no major differences. For long-term NO<sub>2</sub> exposure, however, the estimate of Cai for hypertension was significant whilst that of Yang was null, when Yang finds an effect of long-term exposure on DBP. So, we kept the estimate of Cai for hypertension. The SO<sub>2</sub> effects were not considered because SO<sub>2</sub> is lower in Europe. There was no evidence for an effect of CO.

## B9. Out of hospital cardiac arrest

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)
PM <sub>2.5</sub>	Short-term: varying lags but most between 0 and 3, up to 7 days	Zhao et al 2017	Relative Risk: 1.04 (1.01,1.07) per 10 µg/m <sup>3</sup>
PM <sub>10</sub>	Short-term: varying lags but most between 0 and 2, up to 7 days	Zhao et al 2017	Relative Risk: 1.02 (1.01,1.04) per 10 µg/m <sup>3</sup>
NO <sub>2</sub>	Short-term: varying lags but most between 0 and 2, up to 7 days	Zhao et al 2017	Relative Risk: 1.02(1.00,1.03) per 10 ppb
O <sub>3</sub>	Short-term: varying lags but most between 0 and 2, up to 7 days	Zhao et al 2017	Relative Risk: 1.02(1.01,1.02) per 10 ppb

<b>CO</b>	Short-term: varying lags but most between 0 and 2, up to 7 days	Zhao et al 2017	Relative Risk: 1.06 (1.00,1.14) per 1 ppm
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**Justification:** The Zhao et al 2017 review of the effects of short-term exposures was used. The estimates are from the worldwide analysis, but some studies are from Europe. There was no Statistically Significant evidence for SO<sub>2</sub>. The PM<sub>2.5</sub> estimate is based on 12 studies, the PM<sub>10</sub> on 9 studies, the NO<sub>2</sub>, ozone and CO on 11 studies.

#### B10. Cardiac arrhythmias (hospitalisation)

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)
<b>PM<sub>2.5</sub></b>	Short-term varying lags but most between 0 and 2, up to 7 days	Song et al 2016	Relative Risk: 1.015 (1.005, 1.025) per 10 µg/m <sup>3</sup>
<b>PM<sub>10</sub></b>	Short-term varying lags but most between 0 and 2, up to 7 days	Song et al 2016	Relative Risk: 1.009 (1.004, 1.015) per 10 µg/m <sup>3</sup>
<b>NO<sub>2</sub></b>	Short-term varying lags but most between 0 and 2, up to 7 days	Song et al 2016	Relative Risk: 1.036 (1.018, 1.055) per 10 ppb
<b>CO</b>	Short-term varying lags but most between 0 and 2, up to 7 days	Song et al 2016	Relative Risk: 1.040 (1.017, 1.065) per 1 ppm

**Justification:** The review by Song et al provided estimates for mortality and hospitalisation. The hospitalisation effects were selected here. They also provided estimates only for Europe, which in some instances differed, but were based on few studies and were not chosen. For ozone and SO<sub>2</sub> the evidence was for a borderline effect and we decided not to consider it. Specifically, for PM<sub>2.5</sub> 17 studies were included, for PM<sub>10</sub> and CO 12 studies and for NO<sub>2</sub> 13.

#### B11. Atrial fibrillation

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)
<b>PM<sub>2.5</sub></b>	Short-term	Shao et al 2016	Relative Risk: 1.009 (1.002,1.016) per 10 µg/m <sup>3</sup>
<b>NO<sub>2</sub></b>	Short-term	Shao et al 2016	Relative Risk: 1.012 (1.007,1.017) per 10 ppb
<b>SO<sub>2</sub></b>	Short-term	Shao et al 2016	Relative Risk: 1.010 (1.006,1.013) per 10 ppb

<b>O<sub>3</sub></b>	Short-term	Shao et al 2016	Relative Risk: 1.011 (1.002,1.019) per 10 ppb
<b>CO</b>	Short-term	Shao et al 2016	Relative Risk: 1.006 (1.002,1.011) per 1 ppm

**Justification:** The Shao et al review involved 4 studies with 461,441 participants (3 for SO<sub>2</sub> and 2 for CO). All case-crossover designs. Among the studies one was in England and Wales, whilst the 3 were in North America.

## B12. Lung cancer incidence

Pollutant	Time reference	CRF Source	CRF
PM <sub>2.5</sub>	Long-term	Raaschou-Nielsen et al 2013	Hazard Ratio: 1.18 (0.96, 1.46) per 5µg/m <sup>3</sup> Hazard Ratio: 1.39 (0.91, 2.13) per 10µg/m <sup>3</sup>
PM <sub>10</sub>	Long-term	Raaschou-Nielsen et al 2013	Hazard Ratio: 1.22 (1.03, 1.45) per 10 µg/m <sup>3</sup>

**Justification:** A paper by Hamra et al 2014 deals with incidence and mortality together. The authors consider multicentre studies as a single study with one estimate and give it a weight according to the number of events. They do not take into account the multi country representation. Therefore, we chose not to use their estimates.

Studies with incidence data alone are relatively few. In the EU, for PM<sub>10</sub>, only the Raaschou-Nielsen 2013 study includes incidence, so we used this.

In the EU, for PM<sub>2.5</sub> there are 2 studies which include incidence, by Beelen et al and by Raaschou-Nielsen. The one from Beelen et al is based only in the Netherlands, whilst Raaschou-Nielsen includes 22 cohorts across the EU. For PM<sub>2.5</sub> the CRF from Raaschou-Nielsen and from the Hamra et al study are not very different in magnitude so we used the values quoted by Raaschou-Nielsen.

For NO<sub>2</sub>, the ESCAPE paper (Raaschou-Nielsen) reports null effects. The Hamra 2015 paper analyses mortality and incidence endpoints together and while the relative risk of 1.04 is statistically significant, it is dominated by mortality studies. The paper reports an EU estimate of 1.02 which is not statistically significant. In view of this weak evidence, we have not pursued NO<sub>2</sub> a pollutant associated with lung cancer incidence.

For ozone the only paper is Atkinson 2016 which analyses mortality and finds no evidence for ozone effects on lung cancer.

## B13. Pneumonia admissions in children

Pollutant	Time reference	CRF Source	CRF % change
PM <sub>10</sub>	Short-term	Nhung et al 2017	Percentage increase: 1.50% (0.6-2.4) per 10 µg/m <sup>3</sup>
NO <sub>2</sub>	Short-term	Nhung et al 2017	Percentage increase: 0.60% (-0.20-1.40) per 10 ppb

<b>PM<sub>2.5</sub></b>	Short-term	Nhung et al 2017	Percentage increase: 1.50% (-0.20-3.10) per 10 µg/m <sup>3</sup>
<b>O<sub>3</sub> (maximum 8-hour average)</b>	Short-term	Nhung et al 2017	Percentage increase: 2.40% (1.00-3.80) per 10 ppb

**Justification:** The CO association has not been used as there is no consistent evidence according to the authors.

As noted above, we have not pursued SO<sub>2</sub> because SO<sub>2</sub> is generally low in Europe.

For PM<sub>10</sub> there was no effect modification by country with high/non-high income, so we are using the overall estimate as in the table above. It is based on 13 studies.

For PM<sub>2.5</sub> the results reported effect modification by country with high/non-high income and thus we used the HIE effect estimate (based on 9 studies) as given in Fig 2. Too few studies were from EU to enable the use of an EU estimate. The estimate is not statistically significant, but close to significance. For NO<sub>2</sub> there was effect modification by country with high/non-high income and thus we used the HIE effect estimate.

For O<sub>3</sub> there as above we used the HIE effect estimate (based on 7 studies).

## B14. Lung function in children

Pollutant	Time reference	CRF Source	CRF
PM <sub>2.5</sub>	Long- term	Gehring et al 2013 ESCAPE project	% change of FEV1 -2.49 (-4.57 - -0.36) per 5 µg/m <sup>3</sup>
NO <sub>2</sub>	Long-term	Gehring et al 2013 ESCAPE project	% change of FEV1 -0.98 (-1.70 - -0.26) per 10 µg/m <sup>3</sup>
NO <sub>2</sub>	Long-term	Gehring et al 2013 ESCAPE project	Odds Ratio for "low lung function" 1.35 (1.06-1.73) per 10 µg/m <sup>3</sup>
PM <sub>10</sub>	Long-term	Gehring et al 2013 ESCAPE project	Odds Ratio for "low lung function" 1.69 (1.04-2.74) per 10 µg/m <sup>3</sup>
PM <sub>2.5</sub>	Long-term	Gehring et al 2013 ESCAPE project	Odds Ratio for "low lung function" 1.41 (0.74-2.71) per 5 µg/m <sup>3</sup>

**Justification:** Here we use the Gehring et al paper, from the ESCAPE project, which is based on 5 European birth cohorts from Sweden, Germany, U.K., and the Netherlands. They use estimated exposure (using the ESCAPE methodology, see e.g. Eeftens et al, Atmos Environ, 2012, 62: 303-17) at current address. They give also birth address but report smaller effects. CRFs from the more adjusted models are chosen. The odds ratio for PM<sub>2.5</sub> "low lung function" (FEV<sub>1</sub><85% predicted) is not statistically significant.

They find no evidence for associations between PM<sub>10</sub> or the coarse fraction and decrease in FEV<sub>1</sub> but find an association of PM<sub>10</sub> with the risk of "low lung function", which in fact may be more clinically relevant. In the ESCAPE study they did not consider ozone or SO<sub>2</sub> or CO.

**B15. Lung function growth (associated with long-term decrease in pollutants from the California Children's Health Study)**

	Time reference	CRF Source	CRF for Difference in FEV <sub>1</sub> /FVC at 15 years of age	CRF for difference in FEV <sub>1</sub> /FVC growth from 11 to 15 years of age
<b>PM<sub>2.5</sub></b>	Long-term	Gauderman et al 2015	FEV <sub>1</sub> , per 12.6µg/m <sup>3</sup> : 165.5ml (95.4-235.6ml)	FEV <sub>1</sub> , per 12.6µg/m <sup>3</sup> : 65.5ml (17.1-113.8ml)
<b>PM<sub>2.5</sub></b>	Long-term	Gauderman et al 2015	FVC, per 12.6µg/m <sup>3</sup> : 237.0ml (147.2-326.7ml)	FVC, per 12.6µg/m <sup>3</sup> : 126.9ml (65.7-188.1ml)
<b>PM<sub>10</sub></b>	Long-term	Gauderman et al 2015	FEV <sub>1</sub> , per 8.7µg/m <sup>3</sup> : 153.2ml (97.7-208.6ml)	FEV <sub>1</sub> , per 8.7µg/m <sup>3</sup> : 65.5ml (27.2-103.7ml)
<b>PM<sub>10</sub></b>	Long-term	Gauderman et al 2015	FVC, per 8.7µg/m <sup>3</sup> : 206.8ml (124.6-289.1ml)	FVC, per 8.7µg/m <sup>3</sup> : 113.0ml (60.0-166.1ml)
<b>NO<sub>2</sub></b>	Long-term	Gauderman et al 2015	FEV <sub>1</sub> , per 14.1ppb: 210.6ml (156.0-265.2ml)	FEV <sub>1</sub> , per 14.1ppb: 91.4 ml (47.9-134.9ml)
<b>NO<sub>2</sub></b>	Long-term	Gauderman et al 2015	FVC, per 14.1ppb: 300.2ml (240.0-360.3ml)	FVC, per 14.1ppb: 168.9ml (127.0-210.7ml)

**Justification:** The California Children's study (Gauderman et al, 2015) is an extensive long-term investigation and is a study widely recognised for its quality and robustness and we have therefore used the findings here for growth from 11 to 15 years of age. We did not propose to use the difference in FEV<sub>1</sub> and FVC at age 15 at this stage because the exact basis of the concentration-response function was not clear from the paper. In addition, millilitres of FVC is not an easily communicated outcome. A later outcome on numbers of children with low lung function was preferable (see section 5.3.5 Reduced lung growth and low lung function).

**B16. Asthma admissions in children (short-term)**

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)
<b>PM<sub>2.5</sub></b>	Short-term: varying lags	Walton et al 2019	Relative Risk: 1.029 (1.016-1.042) per 10 µg/m <sup>3</sup>
<b>PM<sub>10</sub></b>	Short-term: lag 0-1	Atkinson et al 2001 (APHEA 2)	Relative Risk: 1.012 (1.002-1.023) per 10 µg/m <sup>3</sup>
<b>NO<sub>2</sub> 24 hour</b>	Short-term: varying lags	Walton et al 2019	Relative Risk: 1.036 (1.018-1.054) per 10 µg/m <sup>3</sup>



<b>O<sub>3</sub> 8-hour max</b>	Short-term: varying lags	Walton et al 2014 updated for this project	Relative Risk:1.012 (0.992-1.032) per 10 µg/m <sup>3</sup>
<b>SO<sub>2</sub> 24 hour</b>	Short-term: varying lags/lag 0-1	Sunyer et al 2003 (multi-city study)	Relative Risk:1.013 (1.004-1.0221) per 10 µg/m <sup>3</sup>

For PM<sub>2.5</sub> and NO<sub>2</sub>, we have chosen Walton et al 2019. The paper includes studies worldwide (11 studies, 14 cities for PM<sub>2.5</sub>; 8 studies, 24 cities for NO<sub>2</sub>). The authors found low heterogeneity in the global estimate (suggesting this particular group of global studies do not vary much by region). A variety of ages was studied, broadly in the range 0-14 years.

PM<sub>10</sub> was not considered in Atkinson et al 2014 or Walton et al 2019, and there was only 1 new study in a post 2011 literature search. Consequently we chose to use Atkinson et al 2001, a European multi-city study analysed according to a common protocol. The ages studied ranged from 0-14 years.

For ozone, we updated the Walton et al 2014 meta-analysis including 12 studies, 19 cities in total, (4 studies, 6 cities from Europe). More specifically, in addition to the studies included in the Walton et al 2014 study, we added another four studies in our meta-analysis which have influenced the size of the previous estimate<sup>14</sup>. However, the new pooled CRF remained not statistically significant.

The CRF for SO<sub>2</sub> was based on areas where the concentrations are generally low. We chose Sunyer et al (2003) rather than a meta-analysis of single city studies by Anderson et al (2007) because the former is from Europe with cities analysed according to a common protocol. A further review (without a meta-analysis) was done for the REVIHAAP report (WHO, 2013a). This noted that further studies published since 2006 on asthma admissions in children are also positive and statistically significant in two studies (Lee, Wong & Lau, 2006; Samoli et al., 2011a), with a range from 1.3% to 6% per 10 µg/m<sup>3</sup> SO<sub>2</sub> respectively. The latter (Samoli et al) is from a European study not included in Sunyer et al. 2003. There have been no new studies since 2011. We did not use this CRF as SO<sub>2</sub> levels are low but if it was used, the new studies could be acknowledged, or incorporated into a new meta-analysis.

There were no studies available for CO.

### B17. Asthma admissions in adults (short-term)

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)
<b>PM<sub>10</sub></b>	Short-term: lag 0-1	Atkinson et al 2001 (APHEA 2)	Relative Risk: 1.011 (1.003-1.018) per 10 µg/m <sup>3</sup>
<b>NO<sub>2</sub> 24 hour</b>	Short-term: varying lags	Walton et al 2019	Relative Risk: 1.012 (1.01-1.023) per 10 µg/m <sup>3</sup>

<sup>14</sup> The extra studies included in the updated meta-analysis are: Winkvist et al 2012, Ding et al 2017, Zu et al 2017 and Goodman et al 2017.

<b>O<sub>3</sub> 8-hour max</b>	Short-term: varying lags	Walton et al 2014	Relative Risk: 1.0215 (0.9982, 1.0447) per 10 µg/m <sup>3</sup>
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**Justification:** PM<sub>2.5</sub> is not listed in the table because a meta-analysis of 4 studies (Walton et al, 2019) did not suggest any association.

SO<sub>2</sub> is not listed for the same reason – the relative risk was close to the null and not statistically significant in meta-analysis of studies to 2006 (Anderson et al 2007) and a European multi-city study (Sunyer et al, 2003).

PM<sub>10</sub> was not considered in the Atkinson et al (2014) or Walton et al (2019) papers, and there were no new studies in a post 2011 literature search. Consequently we chose to use the results of Atkinson et al 2001, a European multi-city study analysed according to a common protocol.

For NO<sub>2</sub>, we have chosen to use the results of Walton et al (2019) as it is a recent study and includes studies worldwide.

For ozone, there is no statistically significant evidence to enable us to consider this pollutant.

There were no studies available for CO.

Asthma admissions in children and in adults 15-64 are considered above. Asthma in the elderly is not considered here because asthma and COPD are difficult to distinguish in the elderly. There is a separate CRF for COPD admissions which can be used instead.

## B18. Symptoms in asthmatic children

Pollutant	Outcome	Time reference	CRF Source	CRF (95% confidence interval)
NO <sub>2</sub> annual average	Bronchitic symptoms	Long-term:	WHO (2013b) based on McConnell et al 2003	Odd Ratio: 1.021 (0.990–1.060) per 1 µg/m <sup>3</sup> change in annual mean NO <sub>2</sub>
PM <sub>10</sub> annual average	Asthma symptoms	Daily average	WHO (2013b) based on Weinmayr et al 2010	Odds Ratio: 1.028 (1.006-1.051) per 10 µg/m <sup>3</sup>

**Justification:** The McConnell paper is a good quality study but is the only one for this health outcome from long-term exposures. It deals with bronchitic symptoms, such as cough, phlegm and (less likely) asthma symptoms, wheeze and breathlessness. The work used several pollutants but NO<sub>2</sub> was stable to adjustment for other pollutants. The associations were not quite statistically significant but very close.

The Weinmayr et al (2010) paper has combined evidence from 36 panel studies of asthmatic children aged 5–19 years, using meta-analysis. They included 51 populations, 36 of which from Europe. The asthma symptoms included cough, wheeze, shortness of breath, asthma attacks or asthma symptoms. The WHO HRAPIE 2013 project (WHO 2013b) recommend the use of the PM<sub>10</sub> CRF for the effects of short-term exposure and the association is statistically significant.

## B19. Asthma incidence and prevalence

### *Asthma incidence*

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)	Comments
PM <sub>2.5</sub>	Long-term:	Bowatte et al 2015	Odds Ratio: 1.014 (1.00 – 1.30) per 2 µg/m <sup>3</sup>	From 4 cohorts (2 from Europe). Good quality meta-analysis. Focus on birth cohorts and childhood onset. Predates publication of Gehring et al (2015).
		Jacquemin et al 2015	Odds Ratio: 1.04 (0.88 - 1.23) per 5 µg/m <sup>3</sup>	6 cohorts from ESCAPE, omit not statistically significant (incidence adult-onset asthma)
		Khreis et al 2017	Odds Ratio: 1.03 (1.01, 1.05) per 1 µg/m <sup>3</sup>	From 9 cohorts and 1 case control study 'at any age' but times of follow up were mostly in childhood (1 was to age 21). Incidence or life-time

				prevalence in childhood. While mixing study types and outcome definitions was done carefully, it is better suited to a qualitative conclusion. It is complicated to implement estimates from a mixed approach in health impact assessment.
		Gehring et al 2015	Odds Ratio: 1.25 (0.94-1.66) per 5 $\mu\text{g}/\text{m}^3$	ESCAPE study of 4 European cohorts incidence asthma in childhood and adolescence. $\text{PM}_{2.5}$ was not statistically significant but $\text{PM}_{2.5}$ absorbance was. So traffic pollution statements via $\text{NO}_2$ may be more appropriate?
$\text{PM}_{10}$	Long-term:	Jacquemin et al 2015	Odds Ratio: 1.04 (0.88 -1.23) per 10 $\mu\text{g}/\text{m}^3$	ESCAPE, omit not statistically significant (incidence adult-onset asthma)
		Khreis et al 2017	Odds Ratio: 1.05 (1.02 – 1.08) per 2 $\mu\text{g}/\text{m}^3$	From 12 studies. Incidence or life-time prevalence in childhood. While mixing study types and outcome definitions was done carefully, it is better suited to a qualitative conclusion. It is complicated to implement estimates from a mixed approach in health impact assessment.
		Gehring et al 2015	Odds Ratio: 1.08 (0.77-1.51) per 10 $\mu\text{g}/\text{m}^3$	ESCAPE study of 4 European cohorts incidence asthma in childhood and adolescence. Select this study but do not use – not statistically significant.
$\text{NO}_2$	Long-term:	Bowatte et al 2015	Odds Ratio: 1.09 (0.96 – 1.23) per 10 $\mu\text{g}/\text{m}^3$	From 5 cohorts (3 from Europe). Good quality meta-analysis. Focus on birth cohorts and childhood onset. Predates publication of Gehring et al (2015).
		Jacquemin et al 2015	Odds Ratio: 1.10 (0.99 - 1.2) per 10 $\mu\text{g}/\text{m}^3$	6 cohorts from ESCAPE, almost statistically significant (incidence adult-onset asthma) but concept of childhood incidence on exposure from birth gives clearer statements.
		Khreis et al 2017	Odds Ratio: 1.05 (1.02 –	From 20 studies. Incidence or life-time prevalence in childhood. While mixing study

			1.07) per 4 $\mu\text{g}/\text{m}^3$	types and outcome definitions was done carefully, it is better suited to a qualitative conclusion. It is complicated to implement estimates from a mixed approach in health impact assessment.
		Gehring et al 2015	Odds Ratio: 1.13 (1.02-1.25) per 10 $\mu\text{g}/\text{m}^3$	ESCAPE study of 4 European cohorts incidence asthma in childhood and adolescence. Select this study – consistent analysis across cohorts, European.
<b>O<sub>3</sub></b>	Long-term:	Zu et al (2018)	Mixed evidence	Systematic review not meta-analysis, although did scale results to a common metric. No studies from Europe. Studies were inconsistent in direction. Paper argues there is biological plausibility. Evidence for adult onset asthma from Seventh Day Adventist study but some methodological debate about it. Suggest do not select this pollutant/outcome combination.
<b>SO<sub>2</sub></b>	Long-term:	No reviews identified, not included in ESCAPE		Some plausibility for a link – short-term exposure leads to rapid bronchoconstriction, some evidence from occupational exposure. But concentrations are low. Suggest do not select as a pollutant/outcome combination.
<b>CO</b>	Long-term:	No reviews identified, not included in ESCAPE		Some studies show associations e.g. Pennington et al 2018 but this is thought to be due to correlations with other traffic pollutants. No particular mechanistic reason to expect carbon monoxide to be linked to asthma incidence. Suggest do not select as a pollutant/outcome combination.

**Justification:** For PM<sub>2.5</sub>, the Bowatte et al paper derives from 4 cohorts (2 from Europe) and is a good quality meta-analysis. The paper focusses on birth cohorts and childhood onset. The paper predates publication of Gehring et al (2015). The Jacquemin paper reports odds ratios which are not statistically

significant and is not considered further here. The Khreis et al paper uses results from 9 cohorts and 1 case control study ‘at any age’ but times of follow up were mostly in childhood (1 was to age 21). Studies included incidence or life-time prevalence in childhood. While mixing study types and outcome definitions was done carefully, it is better suited to a qualitative conclusion. It is complicated to implement estimates from a mixed approach in health impact assessment. The Gehring et al paper is from the ESCAPE study of 4 European cohorts looking at the incidence of asthma in childhood and adolescence. PM<sub>2.5</sub> was not statistically significant but PM<sub>2.5</sub> absorbance was. PM<sub>2.5</sub> absorbance is likely to be predominantly a measure traffic pollution so this study may be more suited to traffic related statements incorporating NO<sub>2</sub>.

For PM<sub>10</sub>, similar comments apply to the Jacquemin and Khreis papers and the Gehring paper reported odds ratios which were not statistically significant for PM<sub>10</sub>.

For NO<sub>2</sub>, the Bowatte et al paper reports results from 5 cohorts (3 from Europe) and is a good quality meta-analysis. It focusses on birth cohorts and childhood onset and predates publication of Gehring et al (2015). The Jacquemin et al paper gives results from 6 cohorts from the ESCAPE study, reporting odds ratios which are almost statistically significant (incidence adult-onset asthma) but the concept of childhood incidence on exposure from birth gives clearer statements. For the Khreis et al paper, similar comments apply as discussed under PM<sub>2.5</sub> above. The Gehring et al paper, part of the ESCAPE study of 4 European cohorts, deals with incidence of asthma in childhood and adolescence. This study could be selected – consistent analysis across cohorts and European but the ESCAPE cohort also found no association with asthma prevalence (Molter *et al* (2015). While not impossible (if cases are remitting), it is a bit contradictory to have evidence for an increase in new cases with increased concentrations and no increase in total cases. In addition, knowledge of the biology and genetics of asthma is changing considerably with many different types being identified (Siroux et al (2014) and studies of associations between air pollution and the different types of asthma (which is a better method) is much more limited (Lau et al, 2018) We therefore decided not to quantify effects on asthma incidence at this time.

For ozone, Zu et al paper is a systematic review rather than a meta-analysis, although the authors did scale their results to a common metric. There were no studies from Europe and the studies used were inconsistent in the direction of the association. The authors argued there is biological plausibility, and there was evidence for adult onset asthma from a Seventh Day Adventist study, but there is some methodological debate about this. We did not select this pollutant/outcome combination.

For SO<sub>2</sub>, there is some plausibility for a link – short-term exposure leads to rapid bronchoconstriction, and there is evidence from occupational exposure. But concentrations are low. We did not select this as a pollutant/outcome combination.

For CO, some studies show associations e.g. Pennington et al 2018 but this is thought to be due to correlations with other traffic pollutants. There is no particular mechanistic reason to expect carbon monoxide to be linked to asthma incidence. We did not select this as a pollutant/outcome combination.

#### *Asthma prevalence*

	Time reference	CRF Source	CRF (95% confidence interval)	Comments
PM <sub>2.5</sub>	<b>Long-term:</b>	Molter et al (2015)	Odd Ratio: 1.23 (0.78-	ESCAPE study of 5 European cohorts asthma prevalence at

			1.95) per 5 $\mu\text{g}/\text{m}^3$	age 8 vs PM2.5 at birth address. PM2.5 absorbance also non-significant.
		Khreis et al 2017	Odd Ratio: 1.03 (1.01, 1.05) per 1 $\mu\text{g}/\text{m}^3$	From 9 cohorts and 1 case control study 'at any age' but times of follow up were mostly in childhood (1 was to age 21). Incidence or life-time prevalence in childhood. While mixing study types and outcome definitions was done carefully, it is better suited to a qualitative conclusion. It is complicated to implement estimates from a mixed approach in health impact assessment.
PM <sub>10</sub>	Long-term:	Molter et al (2015)	Odd Ratio:0.88 (0.63-1.24) per 10 $\mu\text{g}/\text{m}^3$	ESCAPE study of 5 European cohorts asthma prevalence at age 8 vs PM10 at birth address
		Khreis et al 2017	Odd Ratio:1.05 (1.02 – 1.08) per 2 $\mu\text{g}/\text{m}^3$	From 12 studies. Incidence or life-time prevalence in childhood. While mixing study types and outcome definitions was done carefully, it is better suited to a qualitative conclusion. It is complicated to implement estimates from a mixed approach in health impact assessment.
NO <sub>2</sub>	Long-term:	Favarato et al 2014	Odd Ratio:1.06 (1.00 – 1.11) per 10 $\mu\text{g}/\text{m}^3$	14 studies of 12-month period prevalence of asthma in children of within city contrasts dominated by traffic pollution
		Molter et al (2015)	Odd Ratio:1.10 (0.81 – 1.49) per 10 $\mu\text{g}/\text{m}^3$	ESCAPE study of 5 European cohorts asthma prevalence at age 8 vs NO2 at birth address
		Khreis et al 2017	Odd Ratio: 1.05 (1.02 – 1.07) per 4 $\mu\text{g}/\text{m}^3$	From 20 studies. Incidence or life-time prevalence in childhood. While mixing study types and outcome definitions was done carefully, it is better suited to a qualitative conclusion. It is complicated to implement estimates from a mixed approach in health impact assessment.

<b>O<sub>3</sub></b>	<b>Long term</b>	No reviews identified, not included in ESCAPE		Individual studies exist suggesting a relationship e.g. Akinbami et al (2010).
<b>SO<sub>2</sub></b>	<b>Long term</b>	No reviews identified, not included in ESCAPE		Some plausibility for a link – short-term exposure leads to rapid bronchoconstriction, some evidence from occupational exposure. But concentrations are low. Suggest do not select as a pollutant/outcome combination.
<b>CO</b>	<b>Long term</b>	No reviews identified, not included in ESCAPE		

Long-term exposure to traffic pollution (nitrogen dioxide) and asthma prevalence was recommended for quantification in future in WHO (2013b) as the relevant meta-analysis was completed but not published at the time of the WHO report. This is now available (Favarato et al, 2014). This found that NO<sub>2</sub> as a marker of traffic increased the summary odds ratio and was borderline statistically significant. More recently a large study pooling data from 5 birth cohorts in Europe (Molter et al (2015) did not find an association between NO<sub>2</sub> or PM<sub>2.5</sub> and asthma prevalence. Several of these birth cohorts had been examined in earlier publications, using different exposure metrics, and these publications were included in Favarato et al 2014. As Molter et al (2015) study supersedes Favarato et al (2014), focusses on European data, and is not statistically significant, we chose not to pursue quantification of asthma prevalence.

## **B20. (Term) Low birthweight**

<b>Pollutant</b>	<b>Time reference</b>	<b>CRF Source</b>	<b>CRF (95% confidence interval)</b>
<b>PM<sub>2.5</sub></b>	Long-term	Pedersen et al 2013	Odds Ratio: 1.18 (1.06-1.33) per 5 µg/m <sup>3</sup>
<b>PM<sub>10</sub></b>	Long-term	Pedersen et al 2013	Odds Ratio: 1.16 (1.00-1.33) per 10 µg/m <sup>3</sup>
<b>CO</b>	Long-term	Stieb et al 2012	Odds Ratio: 1.07 (1.02-1.12) per 1 ppm
<b>NO<sub>2</sub></b>	Long-term	Pedersen et al 2013	Odds Ratio: 1.09 (1.00-1.19) per 10 ppb
<b>SO<sub>2</sub></b>	Long-term	Stieb et al 2012	Odds Ratio: 1.03 (1.02-1.05) per 5 ppb

The Pedersen et al 2013 study pools raw data from 14 European cohorts in the ESCAPE study, and hence we use this for PM<sub>2.5</sub>, PM<sub>10</sub> and NO<sub>2</sub>. For other pollutants, SO<sub>2</sub> and CO we could use the Stieb et al (2012) results which are based on 6 studies. There are no new meta-analyses. Stieb et al is a



good quality meta-analysis. Note there is good causal support for CO and term low birthweight from the mechanistic point of view.

For ozone there is no statistically significant evidence to allow us to consider this pollutant here.

We have decided not to propose the use of other birth outcomes. Specifically:

**Preterm Birth:** A study from the ESCAPE project (Giorgis-Allemand et al 2017) provided up to date European data from 13 cohorts across 11 countries so this was chosen. It did not find an association with PM<sub>2.5</sub>, PM<sub>10</sub> or NO<sub>2</sub>. The association for ozone from the only meta-analysis available (Stieb et al 2012) also found no association. Suggest not including SO<sub>2</sub> as levels are low.

**Birthweight:** While concentration-response functions exist, simple statements in terms of grams of birthweight are not particularly user friendly. It is better to express the effect in terms of term low birthweight and explain that there is some support from studies of birthweight as a continuous variable.

**Stillbirth:** There are too few studies for firm conclusions on this outcome.

## B21. Bronchitis prevalence in children

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)
PM <sub>10</sub>	Long-term	WHO (2013b) based on Hoek et al 2012	1.08 (0.98-1.19) per 10 µg/m <sup>3</sup>

**Justification:** For this outcome we used the consensus CRF from the WHO (2013b) HRAPIE study, based on the PATY study (Hoek et al (2012)). PATY study included data from about 40,000 children living in nine countries. The association reported was not quite statistically significant but very close.

## B22. Incidence of chronic bronchitis in adults

Pollutant	Time reference	CRF Source	CRF (95% confidence interval)
PM <sub>10</sub>	Long-term	WHO (2013b) based on AHSMOG study (Abbey et al 1995a; 1995b) and SAPALDIA study (Schindler et al 2009)	Odds Ratio: 1.117 (1.040-1.189) per 10 µg/m <sup>3</sup>
PM <sub>10</sub>	Long-term	COMEAP 2016	Odds Ratio: 1.32 (1.02-1.71) per 10 µg/m <sup>3</sup>
PM <sub>10</sub>	Long-term	Cai et al 2014	Odds Ratio: 1.35 (0.97-1.88) per 10 µg/m <sup>3</sup> . Never-smokers.

**Justification:** The first health effect estimate is the consensus CRF from the WHO (2013b) HRAPIE study. It was based on two studies from the United states (AHSMOG) and Switzerland (SAPALDIA). Both studies modelled the probability of a new case of chronic bronchitis in adults over an approximately 10-year period and related it with particulate pollution, adjusting for other factors. In the ESCAPE study the associations between chronic bronchitis and various pollutants, i.e. particles and gases, were investigated. All the associations were not statistically significant in the general population. We report here the findings for never-smokers and PM<sub>10</sub>, which was the pollutant included

in the HRAPIE and COMEAP reports and was marginally not statistically significant. The COMEAP 2016 report that the majority of the available studies have focused on the associations of chronic bronchitis with particulate matter. They recommended the use of the chronic phlegm in never-smokers CRF from the ESCAPE study, because this estimate was found statistically significant and very similar to the same study's estimate for chronic bronchitis in never-smokers (see table above). However, COMEAP only recommended this for use in sensitivity analysis because it was considered that the evidence overall was unconvincing. Based on this, we decided not to produce quantitative statements for this health outcome.

## **ANNEX C: BASELINE RATES AND THEIR SOURCES IN THE UK**

### **C1. Term low birthweight**

Data for the births in England and Wales in 2017 were obtained from the Office for National Statistics (ONS) website ([link](#)). The datasets from the same source for previous years did not report the information needed, i.e. they reported only total live births by region (not by city) and no figures about the live births with more than 37 weeks of gestation. From the 2017 dataset, city-specific figures were collected based on the mother's area of usual residence. Also, total live births with known birthweight (>37 weeks gestation) were collected and background rates, as annual prevalence, were calculated, i.e. the ratio of the number of low birth weights over the number of total births). We used the average across all cities and rounded it to one decimal place: 2.8%.

Data were downloaded from:

<https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/livebirths/datasets/birthcharacteristicsinenglandandwales>

### **C2. Numbers of asthmatic children and prevalence of asthmatic\bronchitic symptoms**

(see also section C9. Population at risk)

In order to estimate the number of asthmatic children in the UK cities we combined population data and the asthma prevalence. Regarding the latter, we assumed that 10% of the population of children in the UK, and 5.3% of children in Poland are asthmatic, based on the findings of Lai et al (2009) (supplementary material - severe asthma, age 13-14). This was the choice recommended by WHO (2013b) for the number of asthmatic children for the PM<sub>10</sub> and asthmatic symptoms in asthmatic children pollutant-outcome pair. WHO (2013b) recommended use of 'ever asthma' for NO<sub>2</sub> and bronchitic symptoms in asthmatic children but we chose to use the same figure for numbers of asthmatic children for the two different statements, to avoid confusion. This means the population at risk for bronchitic symptoms in asthmatic children is underestimated. Regarding the asthmatic and bronchitic symptoms, we used information provided by the World Health Organisation in the HRAPIE report (WHO 2013b). The daily incidence of the former was assumed to be 17%, while the prevalence of the latter was 21.1%.

### **C3. Lung Cancer cases**

The number of lung cancer for England and Wales in 2016 and for London for 2015-2017 were available online from Cancer Research UK ([link](#)) and the Office for National Statistics (ONS, [link](#)) respectively. Also, population data for all the cities included in the statements were collected. We calculated the number of lung cancer cases in all the UK cities (except for London) by assuming that the percentage of cases over the city population is the same across cities and equal to the England-wide figure.

### **C4. Lung Growth**

The Gauderman et al 2015 study investigated the relationship between air pollution and lung growth and the CRFs reported are expressed as difference in ml change from age 11 to age 15 for a 10 µg/m<sup>3</sup> difference in NO<sub>2</sub> (specifically an improvement for a decline in NO<sub>2</sub>) for forced vital capacity (FVC). FVC

is a lung function test measuring the maximum amount of air a person can exhale after a maximum inhalation. In order to perform health impact calculations for this outcome, we used data from Quanjer et al 2012 as baseline data (worked examples for calculating lung function using tables from their supplementary material - Caucasian ethnic group assumed). More specifically, we calculated the predicted FVC for age 11 and age 15 (boys and girls) and calculated the predicted change in FVC between the two age groups. Finally, the average of the predicted change for boys and girls was calculated and used as baseline value for lung growth.

## **C5. Myocardial infarction (short-term)**

Lanki et al (2006) used hospital admissions for first myocardial infarction. This is not routinely available – the standard statistics are for all myocardial infarction admissions. Initial investigation did not reveal data on the proportion of total myocardial infarction admissions that are for first MI. An alternative would be to use the baseline numbers of admissions for first MI from the study itself, convert these to a rate per unit population and assume these applied in the relevant cities. This would need population data for a specific age range (e.g. over 35 or 35-74 for some) in the study cities. This might be available with more investigation but not within the time constraints of this project.

Myocardial infarction (long-term). Cesaroni *et al* (2014) used both hospital admissions and mortality data sources to identify incidence of acute coronary events. Acute myocardial infarction and 'other acute and sub-acute forms of ischaemic heart disease (ICD 10 I20.0, I21, I23 and I24) as an outcome for the hospital admissions data and deaths from ischaemic heart disease (I20-I25) as an outcome for the mortality data. The data could be linked to avoid double counting e.g. deaths where there was a hospital admission for an MI within 28 days of the death were excluded. Ideally, the baseline rates used would match these definitions. The study was analysed using Cox Proportional Hazards modelling. Follow-up varied by cohort from 3 years upwards. For incidence data care usually needs to be taken that the new cases are being calculated over the same time period for the health impacts as in the original study. For Cox proportional hazards modelling however, it is assumed in the analysis that the hazard ratio does not vary with age (increasing time). Thus, the same hazard ratio would apply to a 1-year period as to a longer period. Therefore, this can be applied to baseline rates for annual incidence.

## **C6. CHD Incidence**

Incidence of coronary heart disease (ICD 10 code I20-25.9) for 2017 was available from the British Heart Foundation website for the UK and constituent nations and regions ([link](#) - see Morbidity Table 2.6). This in turn was derived from the Institute of Health Metrics and Evaluation Global Health Data Exchange website ([link](#)). Formal statistics are not collected on incidence of coronary heart disease. It is not entirely clear how the data on the BHF website was compiled from the IHME data, which is a collection of data from different studies. However, it was possible to check that these different studies were studies of events (mortality and morbidity) and not hospital admissions or mortality alone. Thus, it seems reasonable to use the BHF data, with caveats and noting that, while the ICD codes match that used for the mortality element in Cesaroni *et al* (2014), it is wider than that used for the morbidity element. This may result in a slight overestimate.

## **C7. Low lung function**

To estimate the number of children with low lung function from this group we applied a 7.7% factor for the low lung function prevalence (MAAS cohort in ESCAPE, Gehring 2013)

## **C8. Cardiac Arrest**

Similarly to the lung cancer figures, we collected data available online for the number of out-of-hospital cardiac arrests in the United Kingdom in 2016 (British Heart Foundation 2015 - [link](#)). Also, we used population data for all the cities included in the analysis for the same year. The incidence of cardiac arrests in each city was estimated by assuming that the percentage of cases over the UK population is the same across cities.

## **C9. Population at risk**

Some of the statements produced in this report were on health outcomes for which only a subset of the total population can be regarded at risk. To approximate these subsets of people by age, we used yearly data from the Office for National Statistics (ONS) website ([link](#)). In particular, we calculated the 2015-2017 average population of different age groups, such as children aged 5 to 14 years old for asthma symptoms in asthmatic children, children aged 6 to 8 years old for low lung function and children aged 6 to 12 years old for acute bronchitis. Moreover, for London specifically we were able to approximate the percentage of the total population that live near busy, polluted roads which was 33% (see also section 5.1 Scenarios). This percentage was used for the statements produced for the comparison of the health impact on people living near busy roads compared with those living near quieter roads. The method for estimating numbers of asthmatic children has already been explained in section 4.1.4.

## C10. Stroke Incidence

Incidence of first-ever stroke for 2016 in England was available online from the Office for National Statistics (ONS) website ([link](#)). As above, we collected population data for all the cities included in the health statements and calculated city-specific incidence of first-ever stroke. More specifically, we assumed that the percentage of strokes over the city population is the same across cities and equal to the England-wide figure.

*Table C1 -Baseline rates for the health outcomes related to hospital admissions in 9 cities in the UK.*

Outcome	Birmingham	Bristol	Derby	Liverpool	London	Manchester	Nottingham	Oxford	Southampton
All respiratory (Total)	19735	6295	4044	8991	99536	9762	4808	1959	4278
All respiratory (65+)	10819	3451	2217	4929	54569	5352	2636	1074	2345
Asthma (0-14)	361	115	74	164	1821	179	88	36	78
Asthma (15-64)	822	262	169	375	4148	407	200	82	178
COPD without asthma (Total)	3085	984	632	1406	15562	1526	752	306	669
COPD without asthma (65+)	2352	750	482	1072	11863	1163	573	234	510
Pneumonia	151	48	31	69	763	75	37	15	33
All cardiovascular (Total)	13072	4170	2679	5956	65934	6466	3185	1298	2834
All cardiovascular (65+)	8653	2760	1773	3942	43645	4280	2108	859	1876
Cardiac Arrhythmias	1960	625	402	893	9885	969	477	195	425
Stroke	2104	671	431	958	10610	1041	513	209	456

**Table C2 -Baseline rates for the health outcomes not involving hospital admissions in 9 cities in the UK.**

<b>Outcome</b>	<b>Birmingham</b>	<b>Bristol</b>	<b>Derby</b>	<b>Liverpool</b>	<b>London</b>	<b>Manchester</b>	<b>Nottingham</b>	<b>Oxford</b>	<b>Southampton</b>
<b>Incidence of first stroke in 2016</b>	1160	456	13	482	9063	544	326	705	259
<b>Incidence of CHD in 2017</b>	5046	1986	57	2098	39432	2366	1418	3065	1129
<b>Incidence of cardiac arrest in 2016</b>	1033	406	12	429	8069	484	290	627	231
<b>Lung cancer cases 2015/17 average</b>	781	307	9	325	4017	366	219	474	175

**Table C3 -Population at risk in the UK for various health outcomes included in the report.**

<b>Outcome</b>	<b>Birmingham</b>	<b>Bristol</b>	<b>Derby</b>	<b>Liverpool</b>	<b>London</b>	<b>Manchester</b>	<b>Nottingham</b>	<b>Oxford</b>	<b>Southampton</b>
<b>Live births with known birthweight in 2017 (&gt;37 weeks of gestation)</b>	15003	5325	2866	5362	114926	6588	3775	1488	2941
<b>Population of children 5-14 years old 2015/17 average</b>	156730	50000	33358	48757	1067956	64063	36123	16687	26566

<b>Population of children 6-8 years old 2015/17 average</b>	49094	16494	10804	15669	349143	21061	11687	5245	8888
<b>Population of children 6-12 years old 2015/17 average</b>	111228	35673	23869	34411	759572	45758	25718	11882	18943



## **ANNEX D: BASELINE RATES AND THEIR SOURCES IN POLAND**

### **D1. Numbers of asthmatic children and prevalence of asthmatic\bronchitic symptoms in asthmatic children**

Population data for children was obtained as in section 4.1. The source for the numbers of asthmatic children was again the supplementary material for Lai et al (2009), 13-14 year old children, severe asthma. The average for Krakow and Poznan was used (5.3%). The prevalence of asthmatic symptoms and for bronchitic symptoms was as recommended by WHO (2013b).

### **D2. Lung Cancer Cases**

Population data for all ages were collected as described above. However, for the whole country population we collected data for 2018 because we used the same year Globocan report ([link](#)) to obtain the lung cancer cases. To approximate the lung cancer cases in each of the four Polish cities, we assumed that the incidence rate is the same as the country-specific one. Data were downloaded from <http://gco.iarc.fr/today/data/factsheets/populations/616-poland-fact-sheets.pdf>

### **D3. Term low birthweight**

Birth data for 2017 were downloaded from the Statistics Poland website:

<https://stat.gov.pl/en/topics/population/population/demographic-situation-in-poland-up-to-2017-births-and-fertility,8,1.html>

Only total live births for the whole country were reported. We assumed that the live birth annual figures in the Polish cities were proportionate to the city's population. Then, to estimate the percentage of term low birthweights over the total number of live births we used the corresponding proportion from the England data, i.e. 2.8%, and apply it as a scaling factor.

### **D4. Cardiac Arrest**

We used an incidence rate of 146 per 100,000 persons per year, based on a previously published study (Gräsner et al 2016). Then we combined this incidence rate with the population figures and approximated the annual number of cases of out-of-hospital cardiac arrests for each Polish city.

### **D5. Stroke Incidence**

Country-wide incidence rate was collected from the literature, but from a relatively old paper (Członkowska & Ryglewicz 1999). Population data were collected as described above and the annual number of stroke cases were calculated for all the four Polish cities assuming the rate is the same across the cities (equal to the approximation of the country incidence).

### **D6. CHD Incidence**

We could not find any source of information for this outcome, so we approximated the coronary heart disease incidence in Poland by the corresponding estimates from Lithuania. More specifically, we used an incidence rate found in the literature (Veronesi et al 2016) and applied it in the Polish population

data to approximate the number of CHD cases in the Polish cities. We decided to do this because the aforementioned paper provided also a number of fatal CHD events for cohorts from Poland and Lithuania which were found to be very similar (113 in 10.7 years, 114 in 11.7 years respectively, with 2072 men and 2152 women in the cohort).

## D7. Population at risk:

Similar to the UK section, Polish total population figures were collected from Statistics Poland for 2015 and 2016 and the average of the two years was used ([link](#)) in order to calculate the subset of people at risk for various health outcomes. Age groups 3-6 and 7-14 years old were also reported in the same source, thus we divided the values of the first group by 2 and added the numbers for the second group to have an estimate for children 5-14 years old. For age groups 6-8 and 6-12, we performed similar approximations. Regarding the city-specific populations, only total population could be obtained for the four Polish cities ([example link](#)). We assumed that the proportion of the various age groups over the total population is the same between these cities and the whole country, so we applied a scaling factor based on the country population. We were not able to collect any data about the proportion of the total population that lives near busy roads.

**Table D1** - Baseline rates for the health outcomes related to hospital admissions in 4 cities in Poland.

Outcome	Bielsko-Biała	Poznań	Warsaw	Wrocław
All respiratory (Total)	1000	3216	14090	4565
All respiratory (65+)	147	1169	5366	1900
Asthma (0-14)	4	50	105	37
Asthma (15-64)	2	33	151	89
COPD without asthma (Total)	15	256	1473	267
COPD without asthma (65+)	12	196	1131	197
Pneumonia	100	520	1725	590
All cardiovascular (Total)	2802	6784	29545	10310
All cardiovascular (65+)	1811	4685	21765	7698
Cardiac Arrhythmias	203	883	5801	1253
Stroke	451	1092	4754	1659

**Table D2** -Baseline rates for the health outcomes *not* involving hospital admissions in 4 cities in Poland.

Outcome	Bielsko-Biała	Poznań	Warsaw	Wrocław
Incidence of first stroke in 2016	265	847	2703	976
Incidence of CHD in 2016	712	2275	7256	2620
Incidence of cardiac arrest in 2016	249	797	2541	918
Lung cancer cases 2016	126	404	1287	465

**Table C3** - Population at risk in Poland for various health outcomes included in the report.

Outcome	Bielsko-Biała	Poznań	Warsaw	Wrocław
Live births with known birthweight in 2016 (>37 weeks of gestation)	1548	4949	15785	5700
Population of children 5-14 years old 2015/16 average	17098	54651	174330	62952
Population of children 6-8 years old 2015/16 average	5164	16505	52650	19013
Population of children 6-12 years old 2015/16 average	11848	37870	120802	43623