HEALTH IMPACT ASSESSMENT OF AIR POLLUTION ON ASTHMA IN LONDON

For: Greater London Authority

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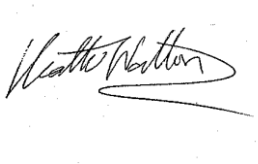
Signed by Dr Heather Walton



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Executive Summary and Key results

Previous studies have shown that numbers of asthma admissions are higher on days when pollution is higher. This report uses those previous studies to provide a modelled estimate of the impact of air pollution in London on asthma admissions, both at current levels and if particulate matter was reduced to the WHO Guideline level of 10 μg m^{-3}.

These estimates are obtained by combining the pollution concentrations in London with information from previous studies on the percentage change in asthma admissions on days with different air pollution concentrations. This percentage increase is then applied to the baseline numbers of asthma admissions in London. More specifically, the inputs were:

- Annual means[1] of 24-hour average fine particulate matter ($PM_{2.5}$) and nitrogen dioxide (NO_2) modelled at a 20x20m scale using the 2016 London Atmospheric Emissions Inventory (LAEI2016) with sea salt subtracted from $PM_{2.5}$ to represent anthropogenic $PM_{2.5}$. These annual means were then averaged by Ward (~13,000 residents). Ward level concentrations varied from 11.2 to 16.6 μg m^{-3} and from 25 to 55.7 μg m^{-3}, for anthropogenic $PM_{2.5}$ and NO_2 respectively.
- Percentage change in admissions per 10 μg m^{-3} change of pollutant concentration was derived by pooling the results of previous studies as part of this project. The chosen concentration response functions suggested percentage changes in admissions ranging from 1.2 to 3.9% depending on pollutant, age group and health outcome (chronic obstructive pulmonary disease (COPD) and asthma admissions combined were used for the elderly).
- Previous studies used to define the concentration-response functions were from locations with different ranges of pollutant concentrations. There was less evidence available for pollutant concentrations below 5 μg m^{-3} and below 10 μg m^{-3} for $PM_{2.5}$ and NO_2 respectively. The concentration-response functions were not applied below these cut-offs.
- Numbers of baseline asthma admissions for age 0-14 and 15-64, and COPD/asthma for age 65+ in each Ward summed across 2014-2016. These ranged for each Ward from 0 to 102 for asthma admissions in children, 0 to 127 for adults and 0 to 300 for asthma/COPD admissions in the elderly.

Calculations were then performed in each Ward down to 5 and 10 μg m^{-3} for $PM_{2.5}$ and NO_2 respectively, before summing the results for each local authority and the whole of London.

Results are summarised in the box below. The effects of air pollution on asthma admissions are evident, however there are many other factors driving variations in asthma admissions other than air pollution. There is also evidence of associations between air pollution and other types of asthma outcomes that are not covered here, such as asthma symptoms and

[1] Annual means were used because calculating the health impact for the annual mean is arithmetically equivalent to calculating it for each day and then summing the result, providing there is no threshold. There was a cut-off in this case but all concentrations were above it so this did not affect the arithmetic equivalence.

A&E visits. Further reductions in air pollution in London are likely to benefit asthmatic patients.

Key results

Exacerbation of asthma by air pollution is estimated to lead to around **1,000 asthma admissions from 2014 - 2016 in children** in London, 10% of all asthma admissions in children in London. (Asthma admissions may have more than one cause e.g. air pollution may worsen response to an allergen.)

Children are more sensitive than adults, so the numbers for adults are smaller (**over 600 adult asthma admissions from 2014-2016**)

Chronic obstructive pulmonary disease (COPD), another respiratory disease similar to asthma particularly found in smokers, is more common in the elderly and difficult to distinguish from asthma. Results for the elderly therefore combined asthma and COPD.

Exacerbation of asthma and COPD by air pollution is estimated to lead to **over 2,500 asthma/COPD admissions from 2014-2016 in the elderly** in London.

The total across these age groups is over **4,000 air pollution-associated asthma admissions**, with **asthma admissions in children accounting for approximately one quarter** of all admissions.

The above estimates are based on levels of nitrogen dioxide (NO_2) above 10 $\mu g\ m^{-3}$. Whether concentrations below 10 $\mu g\ m^{-3}$ have effects is much less certain given the more limited data at lower concentrations.

Calculations were also done for $PM_{2.5}$ concentrations above 5 $\mu g\ m^{-3}$. This gave smaller results that probably overlap to some extent with those for NO_2. In fact, as NO_2 is a traffic pollutant, it may represent traffic PM better than $PM_{2.5}$ does (total $PM_{2.5}$ is heavily but not totally influenced by regional sources).

As the background evidence for effects of air pollution on asthma is mainly based on nitrogen dioxide, diesel PM and proximity to traffic, using the results for NO_2 as an indicator for traffic pollution was chosen for the overall summary of the results.

This is not to say that calculations using $PM_{2.5}$ do not provide an indication of effects on asthma admissions. It was estimated that reduction of current $PM_{2.5}$ levels down to the WHO guideline of 10 $\mu g\ m^{-3}$ could have led to a reduction of 100 asthma admissions in children and around 850 asthma/COPD admissions in the elderly from 2014-2016. This may be a conservative estimate because policies reducing concentrations to 10 $\mu g\ m^{-3}$ would probably reduce concentrations further in some places.

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1 Introduction

In 2015, the Greater London Authority (GLA) commissioned King's College London (King's) to produce a health and economic impact assessment associated with air pollution levels in London (Walton et al., 2015). Following this report, the GLA asked King's to investigate the size of the link between asthma and air pollution in London. Reference was made to a health impact assessment study in New York (New York City Health, 2013) as an indication of the type of report that the GLA would find useful. It is accepted that air pollution is linked to exacerbation of asthma[2] (COMEAP, 1995; WHO, 2013; US EPA 2009, 2013, 2016) with ongoing debate on causation (COMEAP, 2010). This report concentrates on asthma admissions to hospital, particularly in children.

2 Method

Air Quality data

LAEI2016 reference air quality data

The emissions and air quality modelling reference year, created as part of the London Atmospheric Emissions Inventory (LAEI), was the latest version - LAEI 2016. For a complete description of the model, the reader should refer to the LAEI 2013 Methodology[3] pending publication of the LAEI 2016 methodology[4].

Pollutants

Annual average NO_2 concentration in 2016 at 20mx20m resolution.

Annual average anthropogenic $PM_{2.5}$ in 2016 at 20mx20m resolution: Non-anthropogenic $PM_{2.5}$ was derived by subtracting the modelled contribution from natural sources – here sea-salt - from the total $PM_{2.5}$ modelled as above to give anthropogenic $PM_{2.5}$.

From 20mx20m grid data to Ward concentration

Using the data of regular 20mx20m pollutant points, we created a raster layer (for every pollutant in 2016) in the R statistical analysis package. Mean spatially-weighted concentrations for each Ward were then calculated, using the Ward boundaries from the Governments Open Data portal (http://geoportal.statistics.gov.uk/.)

Note that some Wards in the City of London local authority had to be aggregated due to low numbers in the asthma hospital admission data.

$PM_{2.5}$ meeting WHO guidelines scenario

In the assumption that London met the $PM_{2.5}$ WHO guidelines of 10 $\mu g\ m^{-3}$, we created a scenario where all $PM_{2.5}$ concentration had a 10 $\mu g\ m^{-3}$ value in every Ward (note that in

[2] The clearest evidence is for sulphur dioxide and bronchoconstriction in human volunteer studies – an effect found at much lower concentrations in asthmatics compared with the general population (Johns et al, 2010) but sulphur dioxide concentrations are low.

[3] https://data.london.gov.uk/dataset/london-atmospheric-emissions-inventory-2013

[4] https://data.london.gov.uk/dataset/london-atmospheric-emissions-inventory--laei--2016

2016 average $PM_{2.5}$ concentration for each Ward was above 10 μg m^{-3}). Subsequently, sea-salt was removed from the 10 μg m^{-3} $PM_{2.5}$ modelled as above to give anthropogenic $PM_{2.5}$.

Health assessment

Choice of concentration-response function

Studies using 24-hour average NO_2 were examined as this is closer to the modelled concentrations than 1-hour maximum NO_2. As there are more studies including single-pollutant model results, these were pooled in the knowledge that there was likely to be some overlap between the pollutants. As the number of studies is relatively small, studies were not restricted to those in Europe.

Baseline health data

We included all emergency hospital admissions derived from Hospital Episode Statistics between 1 January 2014 and 31 December 2016. We included admissions with an asthma diagnosis (defined using the international classification of diseases 10th Revision (ICD-10) code J45) and chronic obstructive pulmonary disease (COPD) diagnosis (defined as ICD-10 code J40-J47). We stratified admissions by age group to differentiate between children (0-14 years), adults (15-65 years) and older ages (65 years and over). Based on the residential postcode at time of admission we aggregated data to Wards. Small number suppression was applied for Wards with less than 5 admissions (0-2 admissions set to 0, 3-5 admissions set to 5). [5]

Scenario design

The comparisons analysed were designed as follows. The burden of asthma admissions was assessed by calculating the effects of the increment from current levels of air pollution down to a cut-off representing the lower end of the range of the data in the original epidemiological studies. Although this is representative of the burden of concentrations upwards from (above) the cut-off value, in practical terms it was calculated as the reduction from current levels to the cut-off. This is because the baseline rates of asthma admissions already include the effects of air pollution and we would not know what baseline rate to use for levels of pollution much lower than are present in reality.

For the WHO guideline comparison, the comparison was between current levels and a concentration of 10 μg m^{-3} everywhere. In practical terms, for convenience of analysis, sea salt and the cut-off value (see later) was subtracted from both the current levels and the 10 μg m^{-3} target. This, however, gives an equivalent increment as the sea salt and cut-off on both sides cancel each other out.

[5] The study uses data from the UK Small Area Health Statistics Unit (SAHSU), obtained from NHS Digital. The study was covered by national research ethics approval from the London-South East Research Ethics Committee - reference 17/LO/0846. Data access was covered by the Health Research Authority - Confidentiality Advisory Group under section 251 of the National Health Service Act 2006 and the Health Service (Control of Patient Information) Regulations 2002 - HRA CAG reference: 14/CAG/1039.

3 Processing of input data

Annual mean 24-hour average concentrations 2016

A summary of London anthropogenic $PM_{2.5}$ and NO_2 concentrations in 2016 have been estimated based on Wards concentrations and can be found in *Table 1*.

Table 1 Ward median (and inter-quartile range) concentration for annual average NO_2 concentration and anthropogenic $PM_{2.5}$ in London

Pollutant	Ward concentration (median/ inter-quartile range) in μg m^{-3}
Anthropogenic $PM_{2.5}$	12.85/ 1.04
NO_2	35.55/ 7.65

Concentration-response functions

While a small number of new meta-analyses had been published since those of Atkinson et al 2014 and Mills et al 2015, these were not entirely satisfactory for direct use. This is further discussed in Appendix 1. These were however used to identify new studies which were incorporated into updated meta-analyses. Details of these meta-analyses are given in Appendix 2[6] including results stratified by WHO geographic region.

Table 2 Concentration-response functions for air pollution and asthma and asthma/COPD admissions

Pollutant	% increase in hospital admissions per 10 μg m^{-3}		
	Children 0-14	Adults 15-64	Elderly 65+
	Asthma	Asthma	Asthma/COPD
$PM_{2.5}$	2.9% (1.6% - 4.2%)[a]	Evidence from 4 studies suggests no association[b]	3.93% (1.06% – 6.89%)[c]
NO_2	3.6% (1.8% - 5.4%)[d]	1.2% (1% – 2.3%)[e]	1.42% (1.07% - 1.76%)[f]

Footnote – COPD – chronic obstructive pulmonary disease.
[a]Source: meta-analysis of results from 11 studies, 22 cities for this report (see Appendix 2)
[b]Source: meta-analysis of results from 4 studies, 4 cities for this report (see Appendix 2)
[c]Source: meta-analysis by Atkinson *et al* 2014, 4 studies, 4 cities (see also Appendix 2)
[d]Source: meta-analysis of results from 8 studies, 24 cities for this report (see Appendix 2)
[e]Source: meta-analysis of results from 3 studies, 6 cities for this report (see Appendix 2)
[f]Source: meta-analysis by Mills *et al* 2015, 7 studies, 7 cities (see Appendix 2)

[6] The summaries in Appendix 2 are given as relative risks (the usual form in which they are reported in the studies). These are easily converted to percentage increases in risk by subtracting 1 and then multiplying by 100.

Cut-off concentrations (concentration range to which the concentration-response functions apply)

The original studies pooled to give the concentration-response function for asthma admissions (the largest grouping) in children were examined for the range of the concentration data in each study. Details are given in Appendix 1. It was concluded that above the selected cut-offs of 10 μg m^{-3} for NO_2 and 5 μg m^{-3} for $PM_{2.5}$, the selected concentration-response function was supported by many studies. Below these cut-offs there was only evidence from a smaller set of studies, and even in those studies there would be a much more limited set of datapoints at the concentrations below the cut-offs.

Baseline rates for asthma admissions by Ward

The median and inter-quartile ranges of the baseline data for asthma admissions is given in Table 3.

Table 3 Asthma admissions in children and adults and Asthma/COPD *admissions* in the elderly for 2014-2016 *in London by Ward*

Hospital admissions in London (median/ inter-quartile range)		
Children 0-14	Adults 15-64	Elderly 65+
Asthma	Asthma	Asthma/COPD
15/ 14	29/ 26	109/ 58

4 Health results

The burden for asthma admissions in children in London (around1,000 admissions for 2016) is larger for children than adults and for NO_2 than $PM_{2.5}$ (*Table 4*). The latter point was expected because the concentration-response function was larger for NO_2 than for $PM_{2.5}$ and the concentration increment for NO_2 is larger. Adding the results for the two pollutants is not recommended as there is likely to be overlap between the results. As the background evidence for effects of air pollution on asthma is mainly based on nitrogen dioxide (Brown 2015), diesel /traffic PM (COMEAP, 2010) and proximity to traffic (COMEAP, 2010), using the results for NO_2 as an indicator for traffic pollution was chosen for the overall summary of the results. These results are therefore shown in bold in Table 4.

Table 4 2014-2016 Asthma admissions (central and lower – upper CI estimate) in London from air pollution as indicated by either anthropogenic $PM_{2.5}$ (regional pollution, some local sources) or NO_2 (traffic pollution) – burden from lower end of range of concentrations in health studies to current 2016 levels of pollution

Pollutant[1]	Concentration increment[2] (median) in μg m^{-3}	Asthma admissions 0-14	Asthma admissions 15-64	Asthma/COPD admissions 65+
Anthropogenic $PM_{2.5}$ (regional pollution / some local)	7.85	248 (138 – 355)	n/a	2,102 (581 – 3,593)
NO_2 (traffic pollution)[3]	**25.55**	**965 (498 – 1,404)**	**634 (530 – 1,191)**	**2,506 (1900 – 3,087)**

[1] 24 hour-average

[2] Current concentrations to lowest concentrations covered in several studies (10 μg m^{-3} for NO_2, 5 μg m^{-3} for $PM_{2.5}$). If it is assumed that the relationship remains down to zero concentrations (i.e. outside the range of the data) then the results are about 35 % and 60 % larger for NO_2 and anthropogenic $PM_{2.5}$ respectively (see Appendix 3). Assumptions for lower cut-offs e.g. the lowest minimum in any study would be between the two results.

[3] As the background evidence for effects of air pollution on asthma is mainly based on nitrogen dioxide, diesel PM and proximity to traffic, using the results for NO_2 as an indicator for traffic pollution was chosen for the overall summary of the results.

Results for local authorities are given in Appendix 4. Results for local authorities (based on NO_2 2014-2016) ranged from 0 in the City of London to 69 and 37 for air pollution-associated asthma admissions in children and adults respectively in Croydon. For air pollution-associated asthma/COPD admissions in the elderly for 2014-2016, the range was from 2 in the City of London to 124 in Tower Hamlets. Note that variations across local authorities are not only influenced by variations in air pollution but also by variations in population size and in other risk factors affecting baseline rates for asthma. For COPD admissions baseline rates are influenced by smoking rates.

While uncertain due to small numbers, a map for asthma admissions in children associated with NO_2 and $PM_{2.5}$ has been produced – see Appendix 5.

For $PM_{2.5}$, an area of policy interest is the benefit that could be achieved by reducing levels of $PM_{2.5}$ so that they meet the WHO Air Quality Guideline of 10 μg m^{-3}. These are given in *Table **5***. using a rather simplistic scenario of assuming levels were 10 μg m^{-3} everywhere.

Table 5 2014-2016 Reduction in asthma admissions in London from air pollution if anthropogenic $PM_{2.5}$ is reduced to the WHO Air Quality Guideline of 10 μg m^{-3} compared with 2016 levels of $PM_{2.5}$

Pollutant[1]	Concentration increment (median) in μg m^{-3}	Asthma admissions 0-14	Asthma admissions 15-64	Asthma/COPD admissions 65+
Anthropogenic $PM_{2.5}$ (regional pollution/some local)	3.12	99 (55 – 141)	n/a	838 (233 – 1,422)
% reduction from $PM_{2.5}$ associated admissions from current levels		40%	n/a	40%

[1]24 hour-average

These results are smaller than for the overall burden but nonetheless indicate that important benefits could be realized by a reduction to 10 μg m^{-3}, with a reduction in asthma admissions due to $PM_{2.5}$ of 40% of the $PM_{2.5}$ associated admissions overall in Table 4. It would be a smaller proportion of the air pollution-associated overall figure (mainly represented by the NO_2 associated numbers which may pick up effects of traffic PM better than $PM_{2.5}$). The reduction to the WHO $PM_{2.5}$ guideline is a simplified scenario here. In practice, reductions could be achieved through a variety of policies, some of which will reduce both traffic PM and NO_2.

While the results in this report focus on admissions to hospital for asthma, these are not the only way of representing the effect of air pollution on asthma and do not represent the whole picture. They are one of the more reliable indicators, both from the point of view of the original research studies and due to the availability of routine statistics on hospital admissions. As part of this project, King's did some initial investigation of the potential for quantification of these outcomes as described in Appendix 6.

5 Discussion

These calculations have indicated that air pollution can have a marked impact on asthma admissions in children and adults and on asthma/COPD admissions in London. The study has a number of strengths:

- Performing updated meta-analyses of previous studies specially for this project. The meta-analyses were designed with the use of the summary estimates for quantification of asthma admissions in health impact assessments in mind. Combining emergency room visits and hospital admissions, as several meta-analyses do (Appendix 1) may be appropriate for a general view on whether there is an effect of air pollution on asthma exacerbations but is not appropriate for quantification because the baseline rates are very different.
- Modelled concentrations at a fine spatial scale (20 x 20m).
- Calculations done at Ward level before summing to local authority and to London. This allows the variations in baseline rates for asthma admissions to be taken into account as well as the variations in air pollution.

There are also aspects that could be improved in further work:

- Ozone is not included but there is evidence of associations with asthma admissions (Walton *et al*, 2014).
- As COPD is difficult to distinguish from asthma in the elderly, admissions for these causes were combined in the 65+ age group. However, there are also COPD admissions at the older end of the 15-64 year age group. These are omitted in this analysis.
- While the modelling was at a 20 x 20m scale, it needed to be averaged up to Ward level to be matched with the health data. While the health data itself is unlikely to be available at a finer scale than Ward level (there were already some Wards with sufficiently small numbers to have to suppress them), it would be possible to population-weight the concentrations at output-area level.
- The meta-analyses used studies in whatever regions of the world were available. This had the advantage of increasing the strength of the evidence in terms of the number of studies but the disadvantage of including studies from locations with higher concentrations and a different composition from the air pollution mixture found in London.
- The meta-analyses were based on single pollutant model results. Multi-pollutant model results which aim to identify the independent effects of the pollutants were not reviewed but are likely to be too small in number for meta-analysis.
- Additional sensitivity analyses could be done on varying assumptions for the concentration-response functions or the lowest concentrations that are regarded as providing evidence for associations in the epidemiological studies (cut-offs).

We dealt with the overlap between pollutants by basing the summary of results on studies using NO_2, arguing that this represented not only NO_2 itself but also traffic PM, the part of PM with the greatest evidence for links with asthma exacerbations. PM can also contain pollen fragments, lipopolysaccharides (derived from bacterial cell walls) (at very low levels in $PM_{2.5}$) and fungal spores (Robinson et al, 2013), all of which can act as allergens/triggers

of inflammation. These are found to a greater extent in the coarse fraction but if small fragments are in the $PM_{2.5}$ fraction, then they would not be expected to be particularly highly correlated with NO_2. This might argue for some of the $PM_{2.5}$ associations being independent of traffic pollution.

It is not known whether the increased number of asthma admissions on higher air pollution days would not have happened at all if pollution levels had been lower or whether the air pollution accelerated an already existing decline in disease status in asthmatic patients that would have resulted in a hospital admission at a later date. It is still likely that reductions in air pollution would reduce numbers of hospital admissions but there is some uncertainty as to what degree assuming the hospital admissions are additional results in an overestimate of the reductions.

It is important to put these figures in context. For example, the total number of asthma admissions in children over 2014-2016 is 11,000, compared with around 1,000 estimated to be linked to air pollution here, about 10%. The proportion is smaller for adults (around 3%). There are other important triggers for asthma exacerbations such as respiratory infections and allergens. The studies on NO_2 and airway hypersensitivity show that prior exposure to NO_2 increases the response of the airways to later exposure to histamine, a chemical involved in the allergenic response (Brown, 2015). So, it is entirely possible for more than one trigger to contribute to an admission to hospital for asthma.

Further work is needed both in terms of expanding the range of original research studies and in developing health impact assessment of the effects of air pollution in London.

- If more time-series studies were available to derive the concentration response functions it might be possible to base a concentration-response function on studies in Europe.
- The analysis done here of reducing $PM_{2.5}$ to the level of the WHO Guideline was a rather simplistic analysis. It assumed an instantaneous drop in pollution and a level of 10 µg m^{-3} $PM_{2.5}$ everywhere. In practice, policies to reduce $PM_{2.5}$ to 10 µg m^{-3} everywhere would be likely to lead to levels below 10 µg m^{-3} in some parts of London. But this could be addressed with more specific policy modelling and analysis of changes over time.
- There is substantial evidence from the US of associations between air pollution and emergency room visits (Orellano et al 2017). These studies cannot be used directly as the healthcare systems are different (emergency room visits are the equivalent of a combination of GP visits and A&E visits in the UK). There is one study in London of A&E visits (Atkinson et al 1999) although using results of a single study is less robust. The baseline data on A&E visits was considered in this project but needed further investigation (Appendix 6).
- There is also evidence from previous studies for other outcomes related to asthma – asthma symptoms in asthmatic children (Weimnayr et al, 2010; McConnell et al 2003), asthma prevalence (Favorato et al, 2013) and asthma incidence (Bowatte et al 2014, Khreis et al, 2017). For the latter, COMEAP (2010) concluded that the evidence mainly related to proximity to roads with heavy diesel traffic. There will be some overlap between these different outcomes that would need to be thought through. More work is needed to check the latest evidence on these outcomes

(initial discussion in Appendix 6) and to design and implement the methodology to quantify the effects on these outcomes in London.

6 Appendices

Appendix 1: Literature search methods

Selecting concentration-response functions

The starting point was a DH commissioned project reviewing studies up to May 2011 published as Atkinson et al 2014 and Mills et al 2015 for $PM_{2.5}$ and NO_2.

Search string

The search of literature databases used the same search string as in the project above although it omitted terms relating to mortality and to cardiovascular disease to concentrate on asthma.

((((((((((air pollution) OR pollution) OR ozone) OR nitrogen dioxide) OR nitrogen oxide*) OR particulate matter)) AND (((((timeseries) OR time series) OR time-series) OR daily) OR case-crossover)) AND (((((((((hospital admission*) OR admission*) OR emergency room) OR visit*) OR attendance*) OR a AND e) OR (a and e)) OR (accident and emergency)) OR emergency department*)) AND ("2011"[Date - Publication] : "3000"[Date - Publication]))) AND asthma) OR J45

Reviews were checked for additional studies.

The studies were sifted for quality using the same protocol as Atkinson et al 2014 and Mills et al 2015. This included omitting time-series studies with less than 12 months of data and ensuring studies had appropriate control for temperature and season.

Inclusion criteria: Time-series studies or case-crossover studies, asthma admissions, children, adults or elderly separately with quantitative information on single-pollutant model relative risks or odds ratios for NO_2 or $PM_{2.5}$

Exclusion criteria: Other study designs, time-series studies with less than 12 months of data (including episode studies), studies without description of control for season or temperature, studies of emergency room visits that did not separate inpatients from outpatient visits, pollutant metrics that were unclear e.g. 'dust storm $PM_{2.5}$', studies of PM components or sources without $PM_{2.5}$ as a metric, studies of temperature on mortality that controlled for the effects of pollutants.

363 studies were picked up by the literature search, 228 studies after removing duplicates and screening by title. Sub-searches by age-group were used to assist the screening process by title and abstract e.g. 29 studies provided results for adults. Studies of emergency room visits were not screened out at this stage as it is often not clear from the abstract whether

they separate out inpatient admissions or not. Screening by title and abstract left the full papers for 55 studies to be screened. Screening out studies that only used total emergency room visits, without separating out hospital admissions, left 7 studies for hospital admissions in adults and 8 studies in children. These were subsequently supplemented by 15 studies of emergency room visits or hospital admissions identified through reading the reviews which led to 1 additional study of hospital admissions in adults and 3 in children. These were screened down further by pollutant metric to give a final set of 6 studies in adults and 8 in children. A sub-search on reviews identified 3 reviews.

Of the 4 reviews identified, 1 was a qualitative narrative review (Delzell 2013). The other 3 were:

Zheng et al (2015) Emergency room visits and hospital admissions combined, all ages. Sub-group analysis indicated larger estimates for children and the elderly, but these were still for emergency room visits and hospital admissions combined. Stratification of the all ages result into hospital admissions and emergency room visits was reported to result in larger estimates for hospital admissions but no quantitative information was given in the main paper and there was no additional separation by age group.

Lim et al (2016) Mainly emergency room visits and hospital admissions combined, children only, $PM_{2.5}$ only. A separate summary estimate for hospital admissions was given, although which studies were included was not specified. These were inferred by screening the total list of studies in the combined emergency room visits and hospital admission analysis. This indicated that the summary allowed more than one study per city (it could be argued that these are not independent of each other – a requirement for the meta-analytical approach). It also included more than one estimate per study (for different age groups for example). It did not include 6 studies that were included in Atkinson et al (2014). It was therefore unclear that it provided an update to the earlier study rather than just a different approach. It was therefore decided not to use the summary estimate in Lim et al direct but to add any new studies identified in this analysis not picked up in the literature search in a new meta-analysis.

Orellano et al (2017) Combined emergency room visits and hospital admissions in their summary estimates. Separate summary estimates were provided for children and adults, but emergency room visits and hospital admissions were not separated. This review was screened for additional studies, some of which did in fact separate out hospital admissions.

For combined asthma/COPD admissions in the elderly, a sub search of the above search on COPD identified 12 studies. Further screening did not identify any new studies. A review by Moore et al (2016) was identified but it covered COPD admissions alone not combined with asthma. So, the summary estimates from Atkinson et al 2014 ($PM_{2.5}$) and Mills et al 2015 (NO_2) were used.

For asthma admissions in adults and children for NO_2 and $PM_{2.5}$ new meta-analyses were performed.

The updated meta-analyses used the same protocol as Atkinson et al (2014) and Mills et al (2015) e.g. 1 estimate per study location (priority was given to a city analysis within a multi-city study, otherwise the most recent study was used unless there were specific reasons against). A hierarchical approach was used for single and multi-city estimates as explained in Appendix 2.

Odds ratios were converted to relative risks using prevalence data for asthma admissions if possible, if not general asthma prevalence in the relevant city[7] was checked to assess whether prevalence of asthma hospital admissions was likely to be sufficiently low for the odds ratio to be similar to the relative risk. This was indeed the case.

There is a debate regarding whether it is best to use local or regional studies that have a more relevant pollutant mixture and population characteristics or wider global groups of studies. There is quite a bit of variation across studies just by chance (repeated studies within the same city can vary substantially too) so generally it is better to use a larger number of studies. Usually there are not sufficient numbers of studies from one country (the aim is to have at least 4 studies for meta-analysis). Ideally, we would use studies from Europe but, in practice, the number of studies in Europe was small and we chose to use all studies from across the world.

Application of the above protocols reduced the number of studies providing estimates further. The final set of studies contributing estimates is given in Appendix 2.

Selection of Cut-offs

The original studies pooled to give the concentration-response function for asthma admissions (the largest grouping) in children were examined for the range of the concentration data in each study. Various groups have approached this in different ways. The approach followed by the Global Burden of Disease project for $PM_{2.5}$ was to use a counterfactual bounded by the minimum value and 5th percentile of the concentrations in the largest cohort study used to derive the coefficient (Burnett et al., 2014, Lim et al., 2013). COMEAP (2018) in its report on nitrogen dioxide, examined minimums, 5^{th} and 10^{th} percentiles in the range of studies used in the meta-analysis of long-term exposure to nitrogen dioxide and mortality. We took a similar approach.

We looked at the studies included in our meta-analyses (Appendix 2) for the descriptive statistics of the air pollution data that were used in their epidemiological models. In particular, we searched for minimum concentrations and 5^{th}, 10^{th} and 25^{th} percentiles to get an idea of the lower part of the distribution of the air pollution exposure data. However, most studies did not report all the statistics mentioned above, but rather mainly the minimum and 25^{th} percentiles were reported. Thus, we examined the range of these statistics in order to select appropriate cut-off values for the two pollutants.

[7] Thanks to Li Yan King's College, London for checking asthma prevalence in Chongqing.

For NO_2, we identified a range in the minimum values from approximately 4.5 to 36 μg m^{-3}. The upper limit of this range though was an outlier, while most of the minimum values were between 4.5 and 13.3. Moreover, the 25th percentile of the reported concentrations ranged from 15 to 55 μg m^{-3}, excluding an extreme value of 105 μg m^{-3} reported by Lee et al 2003 in their study in Seoul, South Korea. Therefore, we chose a cut-off value of 10 μg m^{-3} as representative of the lower end of the range of NO_2 daily concentrations.

For $PM_{2.5}$, minimum values ranged from 0.25 to 18 μg m^{-3}, but 18 was an outlier as the majority of values were between 0.25 and 2.3 μg m^{-3}. Similarly, 25th percentiles ranged from 4.5 to 35.4 μg m^{-3}, but 6 out of 11 studies reported numbers below 8 μg m^{-3}. Thus, we regarded that a value between 3 and 8 μg m^{-3} for the lower end of the range of $PM_{2.5}$ concentrations was relatively plausible.

These calculations and assumptions were based on the epidemiological asthma studies in children used for the quantification of the concentration-response functions. We, also, checked the summary measures of the exposure data in the studies in other age groups included in our meta-analyses and their reported statistics are on average within the same ranges.

It was concluded that above the selected cut-offs of 10 μg m^{-3} for NO_2 and 5 μg m^{-3} for $PM_{2.5}$, the selected concentration-response function was supported by many studies. Below these cut-offs there was only evidence from a smaller set of studies, and even in those studies there would be a much more limited set of datapoints at the concentrations below the cut-offs.

Appendix 2: Meta-Analyses

A hierarchical, two-stage approach was followed (Atkinson *et* al 2014) in order to get a pooled estimate for the relative risk. Firstly, a summary estimate from single-city studies within each WHO region was calculated. Then, these estimates were combined with the multi-city study estimates and pooled region-specific estimates and then a global relative risk were calculated.

$PM_{2.5}$ and Asthma Hospital Admissions

In total, 11 studies were included in the meta-analysis from 4 different WHO regions, i.e. 5 from the Americas (AMR A), 2 from Europe (EUR A) and 4 from Western Pacific (1 WPR A and 3 WPR B). The studies are:

City, Author, Year	WHO Region	Single- or Multi-City Study	RR (per 10 μg m^{-3})	95% CI
Anchorage, Chimonas, 2007	AMR A	Single	0.876	(0.637, 1.206)
Toronto, Lin, 2002	AMR A	Single	0.936	(0.872, 1.004)
New York, Goodman, 2017	AMR A	Single	1.022	(1.001, 1.043)
St Louis, Winquist, 2012	AMR A	Single	1.056	(0.984, 1.133)
California, Ostro, 2009	AMR A	Multi	1.023	(0.994, 1.054)
West Midlands, Anderson, 2001	EUR A	Single	1.033	(0.995, 1.074)
Copenhagen, Iskandar, 2012	EUR A	Single	1.192	(1.084, 1.285)
Australia & New Zealand, Barnett, 2005	WPR A	Multi	1.027	(0.936, 1.126)
Shanghai, Hua, 2014	WPR B	Single	1.043	(1.034, 1.052)
Chongqing, Ding, 2017	WPR B	Single	1.021	(0.992, 1.051)
Hong Kong, Ko, 2007	WPR B	Single	1.024	(1.013, 1.035)

First stage: Pooling single-city study estimates

WHO Region	Single-city study Pooled RR	95% CI
AMR A	1.003	(0.950, 1.059)
EUR A	1.104	(0.960, 1.269)
WPR A	No study	-
WPR B	1.032	(1.016, 1.047)

Weights:

AMR A: Chimonas=2.71%, Lin= 26.24%, Goodman= 44.67%, Winquist= 26.38%. **Heterogeneity**: I^2=59.7%

EUR A: Anderson=53.7%, Iskandar=46.3% **Heterogeneity**: I^2=88.9%

WPR B: Hua=42.80, Ding=17.71%, Ko=39.49% **Heterogeneity**: I^2=74.3%

Second stage: Pooling multi-city study and previous estimates

WHO Region	Multi-city study Pooled RR	95% CI
AMR A	1.019	(0.993, 1.045)
EUR A	1.104	(0.960, 1.269)
WPR A	1.027	(0 .936, 1.126)
WPR B	1.032	(1.016, 1.047)

Weights:

AMR A: Single-city studies=22.84%, Ostro=77.16% **Heterogeneity**: I^2=0%

EUR A: Single-city studies=100%

WPR A: Barnett=100%

WPR B: Single-city studies=100%

Global summary estimate

WHO Region	Pooled RR	95% CI
Global	1.029	(1.016, 1.042)

Weights:

AMR A=24.97%, EUR A=0.86%, WPR A=1.93%, WPR B=72.24%

Heterogeneity:

I^2=0%

TRIM 'N' FILL

First stage: Pooling single-city study estimates

WHO Region	Single-city study Pooled RR	95% CI
AMR A	1.003	(0.950, 1.059)
EUR A	1.034	(0.908, 1.177)
WPR A	No study	-
WPR B	1.032	(1.016, 1.047)

Weights:

AMR A: Chimonas=2.71%, Lin= 26.24%, Goodman= 44.67%, Winquist= 26.38%. **Heterogeneity**: I^2=59.7% **NO TRIMMING PERFORMED**

EUR A: Anderson=35.98%, Iskandar=32.01%, **1 Filled study**=32.01% **Heterogeneity**: I^2=90.8%

WPR B: Hua=42.80, Ding=17.71%, Ko=39.49% **Heterogeneity**: I^2=74.3% **NO TRIMMING PERFORMED**

Second stage: Pooling multi-city study and previous estimates

WHO Region	Multi-city study Pooled RR	95% CI
AMR A	1.019	(0.993, 1.045)
EUR A	1.034	(0.908, 1.177)
WPR A	1.027	(0 .936, 1.126)
WPR B	1.032	(1.016, 1.047)

Weights:

AMR A: Single-city studies=22.84%, Ostro=77.16% **Heterogeneity**: I^2=0% **NO TRIMMING PERFORMED**

EUR A: Single-city studies=100% **NO TRIMMING PERFORMED**

WPR A: Barnett=100% **NO TRIMMING PERFORMED**

WPR B: Single-city studies=100% **NO TRIMMING PERFORMED**

Global summary estimate

WHO Region	Pooled RR	95% CI
Global	1.028	(1.015, 1.042)

Weights:

AMR A=24.97%, EUR A=0.86%, WPR A=1.93%, WPR B=72.24%

Heterogeneity:

I^2=0%

$PM_{2.5}$ and Asthma Hospital Admissions – Adults

We have only 4 studies in total:

City, Author, Year	WHO Region	Single- or Multi-City Study	RR (per 10 μg m^{-3})	95% CI
New York, Goodman, 2017	AMR A	Single	0.995	(0.981, 1.010)
St Louis, Winquist, 2012	AMR A	Single	1.031	(0.972, 1.094)
West Midlands, Anderson, 2001	EUR A	Single	0.952	(0.904, 1.001)
Hong Kong, Ko, 2007	WPR B	Single	1.018	(1.008, 1.028)

The same hierarchical, two-stage approach as in the previous meta-analysis was followed, but in this case we had only single-city studies and only two were from the same WHO region. We have:

First stage: Pooling single-city study estimates

WHO Region	Single-city study Pooled RR	95% CI
AMR A	1.001	(0.976, 1.027)
EUR A	0.952	(0.904, 1.001)
WPR A	No study	-
WPR B	1.018	(1.008, 1.028)

Weights:

AMR A: Goodman= 83.59%, Winquist= 16.41%. **Heterogeneity**: I^2=23.7%

EUR A: Anderson=100%

WPR B: Ko=100%

Second stage: Pooling multi-city study and previous estimates – Omitted, NO multi-city studies

Global summary estimate

WHO Region	Pooled RR	95% CI
Global	0.999	(0.971, 1.028)

Weights:

AMR A=34.86%, EUR A=19.14%, WPR B=46.00%

Heterogeneity:

I^2=73.4%

TRIM 'N' FILL

First stage: Pooling single-city study estimates

WHO Region	Single-city study Pooled RR	95% CI
AMR A	0.995	(0.969, 1.021)
EUR A	0.952	(0.904, 1.001)
WPR A	No study	-
WPR B	1.018	(1.008, 1.028)

Weights:

AMR A: Goodman= 68.55%, Winquist= 15.72%, **1 Filled study**=15.72%. **Heterogeneity**: I^2=28.3%

EUR A: Anderson=100%, **NO TRIMMING PERFORMED**

WPR B: Ko=100% **Heterogeneity**: I^2=100% **NO TRIMMING PERFORMED**

Second stage: Pooling multi-city study and previous estimates – Omitted, NO multi-city studies

Global summary estimate

WHO Region	Pooled RR	95% CI
Global	0.996	(0.966, 1.027)

Weights:

AMR A=35.08%, EUR A= 20.32%, WPR B= 44.61%

Heterogeneity:

I^2=76.6%

$PM_{2.5}$ and COPD/Asthma Hospital Admissions – Elderly

Used estimate from Atkinson et al 2014 given in the supplementary material. The estimate labelled COPD excluding asthma is in fact the one for COPD including asthma (the original studies were checked). This included the following:

City, Author, Year	WHO Region	Single- or Multi-City Study	RR (per 10 μg m^{-3})	95% CI
Andersen 2008	EUR A	Single, Copenhagen	1.000	(0.9025, 1.108)
Halonen, 2009	EUR A	Single, Helsinki	1.0417	(1.0125, 1.0709)
Moolgavkar, 2000	AMR A	Single, Los Angeles County	1.02	(1.0037, 1.0363)
Ito, 2003 in Health Effects Institute, 2003 (Update of Lippmann et al 2000)	AMR A	Single, Wayne County (Detroit)	1.0117	(0.9714,1.052)

Pooled overall summary estimate 1.0236 (1.01, 1.0373)), I^2: 32% (EUR A 1.0393 (1.0106, 1.0689) only 2 studies; AMR A 1.019 (1.0037, 1.0346) only 2 studies)

NO_2 and Asthma Hospital Admissions in Children

In total, 8 studies were included in the meta-analysis from 3 WHO regions, i.e. 3 from Europe (EUR A), 2 from WPR A and 3 from WPR B. The studies are:

City, Author, Year	WHO Region	Single- or Multi-City Study	RR (per 10 μg m^{-3})	95% CI
3 European Cities, Sunyer, 1997	EUR A	Multi	1.005	(1.001, 1.009)
EpiAir Italy, Colais, 2009	EUR A	Multi	1.013	(0.989, 1.038)
Copenhagen, Iskandar, 2012	EUR A	Single	1.078	(1.032, 1.125)
Fukuoka, Ueda, 2010	WPR A	Single	1.057	(1.011, 1.105)
Australia & New Zealand, Barnett, 2005	WPR A	Multi	1.062	(1.002, 1.125)
Seoul, Lee, 2003	WPR B	Single	1.018	(1.000, 1.036)
Chongqing, Ding, 2017	WPR B	Single	1.168	(1.011, 1.350)
Hong Kong, Ko, 2007	WPR B	Single	1.039	(1.028, 1.050)

First stage: Pooling single-city study estimates

WHO Region	Single-city study Pooled RR	95% CI
AMR A	No study	-
EUR A	1.078	(1.033, 1.126)
WPR A	1.057	(1.011, 1.105)
WPR B	1.033	(1.008, 1.058)

Weights:

EUR A: Iskandar=100%

WPR A: Fukuoka=100%

WPR B: Lee=44.74%, Ko=52.65%, Chongqing=2.61% **Heterogeneity**: I^2=70.2%

Second stage: Pooling multi-city study and previous estimates

WHO Region	Multi-city study Pooled RR	95% CI
AMR A	No Study	-
EUR A	1.024	(0.994, 1.055)
WPR A	1.059	(1.022, 1.096)
WPR B	1.033	(1.008, 1.058)

Weights:

EUR A: Single-city studies=22.63%, Sunyer=43.67%, Colais=33.70%. **Heterogeneity**: I^2=80.8%

WPR A: Fukuoka=63.09%, Single-city studies=36.91%. **Heterogeneity**: I^2=0.0%

WPR B: Single-city studies=100%

Global summary estimate

WHO Region	Pooled RR	95% CI
Global	1.036	(1.018, 1.054)

Weights:

EUR A=31.32%, WPR A=22.62%, WPR B=46.06%

Heterogeneity:

I^2=6.8%

NO_2 and Asthma Hospital Admissions in Adults

In total, 3 studies were included in the meta-analysis from 2 WHO regions. Two studies were from Europe (EUR A and 1 from WPR B. The studies are:

City, Author, Year	WHO Region	Single- or Multi-City Study	RR (per 10 μg m^{-3})	95% CI
4 European Cities, Sunyer, 1997	EUR A	Multi	1.006	(1.001, 1.011)
Rome, Michelozzi, 2000	EUR A	Single	1.024	(0.991, 1.058)
Hong Kong, Ko, 2007	WPR B	Single	1.018	(1.007, 1.029)

First stage: Pooling single-city study estimates

WHO Region	Single-city study Pooled RR	95% CI
AMR A	No study	-
EUR A	1.024	(0.991, 1.058)
WPR A	No study	-
WPR B	1.018	(1.007, 1.029)

Weights:

EUR A: Michelozzi=100%

WPR A: Ko=100%

Second stage: Pooling multi-city study and previous estimates

WHO Region	Multi-city study Pooled RR	95% CI
AMR A	No Study	-
EUR A	1.007	(0.999, 1.015)
WPR A	No Study	-
WPR B	1.018	(1.007, 1.029)

Weights:

EUR A: Single-city studies=5.85%, Sunyer=94.15%. **Heterogeneity**: I^2=7.3%

WPR B: Single-city studies=100%

Global summary estimate

WHO Region	Pooled RR	95% CI
Global	1.012	(1.001, 1.023)

Weights:

EUR A=55.61%, WPR B=44.39%

Heterogeneity:

I^2=61.3%

NO_2 and COPD/Asthma Hospital Admissions – Elderly

Used estimate from Mills et al 2015 given in the supplementary material. This included the following:

City, Author, Year	WHO Region	Single- or Multi-City Study	RR (per 10 μg m^{-3})	95% CI
Andersen 2008	EUR A	Single, Copenhagen	1.0508	(1.0087, 1.0929)
Halonen, 2009	EUR A	Single, Helsinki	1.0237	(1.0025, 1.045)
Moolgavkar, 2000	AMR A	Single, Cook County	1.0105	(1.0036, 1.0173)
Moolgavkar, 2000	AMR A	Single, Los Angeles County	1.0131	(1.0097, 1.0164)
Moolgavkar, 2000	AMR A	Single, Maricopa	1.023	(1.0057, 1.0403)
Lippmann, 2000	AMR A	Single, Wayne County (Detroit)	1.0117	(0.9714,1.052)
Health Effects Institute, 2010	WPR B	Single, Hong Kong	1.0151	(1.0108, 1.0193

Pooled overall summary estimate 1.0142 (1.0107, 1.0176)) I^2 30.8% (EUR A 1.0314 (1.0076, 1.0558) only 2 studies; AMR A 1.0128 (1.0099, 1.0158) (only 2 studies, but 4 areas)

Appendix 3: Results with no cut-off

Table 6 2014-2016 Asthma admissions in London from air pollution as indicated by either anthropogenic $PM_{2.5}$ (regional pollution, some local sources) or NO_2 (traffic pollution) – burden from concentrations of zero to current 2016 levels of pollution

Pollutant[1]	Concentration increment (median) in $\mu g\ m^{-3}$	Asthma admissions 0-14	Asthma admissions 15-64	Asthma/COPD admissions 65+
Anthropogenic $PM_{2.5}$ (regional pollution/some local)	12.85	401 (225 – 573)	n/a	3,398 (946 – 5,768)
NO_2 (traffic pollution)	35.55	1,317 (685 – 1,900)	869 (728 – 1,625)	3,450 (2,621 – 4,244)

[1]24 hour-average

Appendix 4: Asthma outcomes for local authorities

Table 7 Central estimate asthma admissions in London from anthropogenic $PM_{2.5}$ (regional pollution, some local sources) air pollution – burden from lower end of range of concentrations in health studies to current 2016 levels of pollution

Local authority	Asthma admissions 0-14	Asthma admissions 15-64	Asthma/COPD admissions 65+
City of London	0	n/a	2
Barking and Dagenham	8	n/a	56
Barnet	6	n/a	74
Bexley	6	n/a	66
Brent	10	n/a	91
Bromley	6	n/a	66
Camden	4	n/a	56
Croydon	19	n/a	111
Ealing	10	n/a	95
Enfield	9	n/a	67
Greenwich	13	n/a	58
Hackney	7	n/a	50
Hammersmith and Fulham	2	n/a	75
Haringey	6	n/a	53
Harrow	7	n/a	65
Havering	4	n/a	68
Hillingdon	7	n/a	45
Hounslow	5	n/a	91
Islington	7	n/a	71
Kensington and Chelsea	2	n/a	38
Kingston upon Thames	3	n/a	29
Lambeth	12	n/a	79
Lewisham	10	n/a	49
Merton	7	n/a	61
Newham	15	n/a	67
Redbridge	10	n/a	64
Richmond upon Thames	4	n/a	35
Southwark	12	n/a	83
Sutton	5	n/a	45
Tower Hamlets	11	n/a	93
Waltham Forest	11	n/a	70
Wandsworth	7	n/a	86
Westminster	4	n/a	46

Table 8 Lower estimate asthma admissions in London from anthropogenic $PM_{2.5}$ (regional pollution, some local sources) air pollution – burden from lower end of range of concentrations in health studies to current 2016 levels of pollution

Local authority	Asthma admissions 0-14	Asthma admissions 15-64	Asthma/COPD admissions 65+
City of London	0	n/a	0
Barking and Dagenham	4	n/a	15
Barnet	3	n/a	20
Bexley	3	n/a	18
Brent	5	n/a	25
Bromley	3	n/a	18
Camden	2	n/a	15
Croydon	11	n/a	31
Ealing	6	n/a	26
Enfield	5	n/a	18
Greenwich	7	n/a	16
Hackney	4	n/a	14
Hammersmith and Fulham	1	n/a	21
Haringey	3	n/a	15
Harrow	4	n/a	18
Havering	2	n/a	19
Hillingdon	4	n/a	13
Hounslow	3	n/a	25
Islington	4	n/a	20
Kensington and Chelsea	1	n/a	10
Kingston upon Thames	2	n/a	8
Lambeth	7	n/a	22
Lewisham	6	n/a	13
Merton	4	n/a	17
Newham	8	n/a	19
Redbridge	6	n/a	18
Richmond upon Thames	2	n/a	10
Southwark	7	n/a	23
Sutton	3	n/a	12
Tower Hamlets	6	n/a	26
Waltham Forest	6	n/a	19
Wandsworth	4	n/a	24
Westminster	2	n/a	13

Table 9 Upper estimate asthma admissions in London from anthropogenic $PM_{2.5}$ (regional pollution, some local sources) air pollution – burden from lower end of range of concentrations in health studies to current 2016 levels of pollution

Local authority	Asthma admissions 0-14	Asthma admissions 15-64	Asthma/COPD admissions 65+
City of London	0	n/a	3
Barking and Dagenham	11	n/a	95
Barnet	9	n/a	126
Bexley	8	n/a	113
Brent	14	n/a	156
Bromley	8	n/a	112
Camden	6	n/a	95
Croydon	28	n/a	190
Ealing	15	n/a	162
Enfield	13	n/a	114
Greenwich	18	n/a	99
Hackney	11	n/a	85
Hammersmith and Fulham	3	n/a	128
Haringey	8	n/a	91
Harrow	10	n/a	112
Havering	5	n/a	116
Hillingdon	9	n/a	78
Hounslow	8	n/a	155
Islington	10	n/a	121
Kensington and Chelsea	3	n/a	64
Kingston upon Thames	5	n/a	49
Lambeth	18	n/a	134
Lewisham	15	n/a	83
Merton	10	n/a	104
Newham	21	n/a	115
Redbridge	15	n/a	109
Richmond upon Thames	5	n/a	60
Southwark	17	n/a	141
Sutton	7	n/a	77
Tower Hamlets	15	n/a	158
Waltham Forest	15	n/a	120
Wandsworth	10	n/a	146
Westminster	6	n/a	79

Table 10 Central estimate asthma admissions in London from NO_2 (traffic pollution) air pollution – burden from lower end of range of concentrations in health studies to current 2016 levels of pollution

Local authority	Asthma admissions 0-14	Asthma admissions 15-64	Asthma/COPD admissions 65+
City of London	0	0	2
Barking and Dagenham	28	13	60
Barnet	23	17	86
Bexley	20	9	68
Brent	40	34	113
Bromley	19	8	66
Camden	19	17	74
Croydon	69	38	119
Ealing	40	34	116
Enfield	32	14	75
Greenwich	48	13	67
Hackney	32	23	65
Hammersmith and Fulham	9	20	98
Haringey	23	17	66
Harrow	25	20	70
Havering	12	9	65
Hillingdon	25	19	50
Hounslow	22	24	111
Islington	30	26	94
Kensington and Chelsea	9	9	51
Kingston upon Thames	12	7	32
Lambeth	51	33	100
Lewisham	41	17	58
Merton	25	15	70
Newham	59	34	82
Redbridge	38	19	72
Richmond upon Thames	13	7	40
Southwark	50	32	110
Sutton	16	12	48
Tower Hamlets	47	25	124
Waltham Forest	41	24	83
Wandsworth	28	25	106
Westminster	17	20	63

Table 11 Lower estimate asthma admissions in London from NO_2 (traffic pollution) air pollution – burden from lower end of range of concentrations in health studies to current 2016 levels of pollution

Local authority	Asthma admissions 0-14	Asthma admissions 15-64	Asthma/COPD admissions 65+
City of London	0	0	2
Barking and Dagenham	15	11	45
Barnet	12	14	65
Bexley	10	8	52
Brent	21	28	86
Bromley	10	7	50
Camden	10	15	56
Croydon	36	31	90
Ealing	21	28	88
Enfield	17	12	57
Greenwich	25	11	51
Hackney	16	19	50
Hammersmith and Fulham	5	16	74
Haringey	12	14	50
Harrow	13	16	53
Havering	6	7	49
Hillingdon	13	16	38
Hounslow	11	20	84
Islington	16	22	72
Kensington and Chelsea	5	8	39
Kingston upon Thames	6	5	24
Lambeth	27	27	76
Lewisham	21	14	44
Merton	13	12	53
Newham	31	28	63
Redbridge	20	16	55
Richmond upon Thames	7	6	30
Southwark	26	26	83
Sutton	8	10	36
Tower Hamlets	24	21	94
Waltham Forest	21	20	63
Wandsworth	14	21	80
Westminster	9	17	48

Table 12 Upper estimate asthma admissions in London from NO_2 (traffic pollution) air pollution – burden from lower end of range of concentrations in health studies to current 2016 levels of pollution

Local authority	Asthma admissions 0-14	Asthma admissions 15-64	Asthma/COPD admissions 65+
City of London	0	1	3
Barking and Dagenham	41	24	74
Barnet	34	32	106
Bexley	30	17	84
Brent	58	63	139
Bromley	27	15	82
Camden	27	33	91
Croydon	101	71	146
Ealing	59	63	143
Enfield	47	27	92
Greenwich	70	24	83
Hackney	46	42	80
Hammersmith and Fulham	13	37	120
Haringey	34	32	82
Harrow	36	37	86
Havering	18	17	80
Hillingdon	36	36	62
Hounslow	31	45	137
Islington	44	49	116
Kensington and Chelsea	13	17	63
Kingston upon Thames	18	12	40
Lambeth	74	61	123
Lewisham	59	32	71
Merton	37	28	86
Newham	86	63	102
Redbridge	55	36	89
Richmond upon Thames	19	14	50
Southwark	73	59	135
Sutton	24	24	59
Tower Hamlets	68	48	152
Waltham Forest	60	46	102
Wandsworth	41	47	130
Westminster	25	37	78

Appendix 5: Mapping of asthma admission in children

The data on asthma admissions in children associated with air pollution by Ward is shown in Figure 1. (Note that the wide band for NO2 >3-8 is because there few Wards with number of admissions >3).

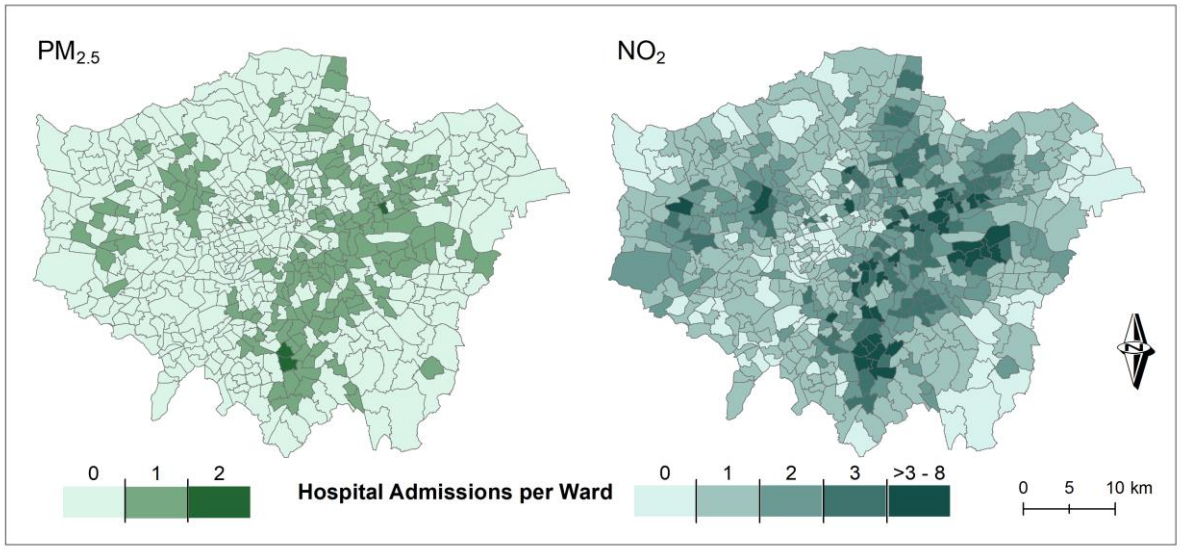

Figure 1 Asthma admissions in children (0-14 years) associated with anthropogenic $PM_{2.5}$ and NO_2 respectively (by Ward) (NB Uncertain due to small numbers)

The pattern for asthma admissions in children are similar to a map of baseline asthma admissions (not shown) i.e. the geographical variation is driven more by the geographical variation in baseline asthma admissions rates than by air pollution variations. This indicates that while there is an association of air pollution with asthma admissions, there are other factors driving variation in asthma admissions that might be more important.

In general, mapping data at Ward level for small subsets (in this case, children not the whole population, asthma admissions not all respiratory admissions and air pollution-associated asthma admissions not all asthma admissions) can be misleading because of the very small numbers. It needs to be borne in mind that for confidentiality reasons where there were fewer than 5 baseline asthma admissions in a Ward these details were suppressed by rounding up to 5 or down to zero. This subsequently affects the air pollution associated numbers of admissions. Thus, differences between categories could be slightly misleading in that it shows variation which might be due to data errors rather than actual differences. Because the rounding up and rounding down cancels itself out to some extent across all Wards, this is less of an issue when results are summed to local authorities and to London.

Appendix 6: Other asthma outcomes

A&E attendances

There are substantial numbers of studies on emergency room visits for asthma in the US (New York City Health, 2013, US EPA, 2009,2013,2016) but this does not have an exact health care system equivalent in the UK (somewhere between an A&E visit and a GP consultation). There is a single study in London of air pollution and accident and emergency attendances (Atkinson et al, 1999). While we would not usually use a single study, it could be argued that the literature on emergency room visits provides conceptual, if not quantitative, support for the study in London. While there is a need to consider this issue further, the vast majority of new studies of emergency room visits were from the US. Consideration of studies in other countries needs additional work to check whether they have similar health care systems. Regarding baseline rates for A&E visits, Appendix 5 already illustrates that the observed spatial patterns in admissions data are driven by factors other than air pollution. This aspect was stronger in A&E data with spatial differences most likely due to factors such as reporting differences between hospitals and differences in arrangements of local health care services. At this stage it was not considered possible to include baseline A&E data in air pollution health impact assessment without understanding these other variations better.

Asthma symptoms

WHO (2013) has recommendations for PM_{10} and asthma symptoms in children (based on Weinmayr et al 2010) and for NO_2 and bronchitic symptoms in asthmatic children (sensitivity analysis as there was only one study). These endpoints have previously been calculated within the damage cost calculations in the Health Impacts of Air Pollution in London report (Walton et al, 2015) (within the Extended set acknowledged as more uncertain), although they were not reported separately. Weinmayr et al 2010 (a meta-analysis) also gives a summary estimate for NO_2 and asthma symptoms in children. There is a need to check whether there are any relevant new meta-analyses, multi-centre studies or reasonable numbers of new individual studies.

Calculations on asthma symptoms in children require 2 aspects of baseline data – the numbers of asthmatic children and the rate of asthma symptoms within asthmatic children. Data on numbers of asthmatic children can be obtained from the International Study on Asthma and Allergies in Childhood (ISAAC) (Lai et al, 2009) which included London as one of its centres. Baseline rates for asthma symptoms in asthmatic children and bronchitic symptoms in asthmatic children could be based on WHO (2013) – themselves based on inference from several panel studies (asthma symptoms) or on the original study plus one other (Migliore et al 2009) in the case of bronchitic symptoms. Details of baseline rate assumptions are given in Table 40 of Walton et al (2015). There would be a need to check whether there were any more up to date sources of baseline rates that might reduce the uncertainties involved in using baseline rates from research studies in other locations.

Asthma prevalence

The outcomes discussed above are related to short-term exposure but there are also studies relating long-term exposure to traffic pollution (nitrogen dioxide) and asthma prevalence. This was recommended for quantification in future in WHO (2013) 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_2 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_2 or $PM_{2.5}$ and asthma prevalence. Several of these birth cohorts had been examined in earlier publications, using different exposure metrics, and these publications were included in Favorato et al 2014. A quick check did not reveal any new meta-analyses combining this more recent study with previous ones. This would be important for coming to an overall conclusion.

Asthma incidence

There are some newer meta-analyses addressing air pollution and asthma incidence (Bowatte et al 2014, Khreis et al, 2017). For asthma incidence, COMEAP (2010) concluded that the evidence mainly related to proximity to roads with heavy diesel traffic. Future work should consider whether these new meta-analyses are robust and might be expected to lead to any change in the COMEAP view.

7 References

Andersen, Z. J., P. Wahlin, O. Raaschou-Nielsen, M. Ketzel, T. Scheike and S. Loft (2008). "Size distribution and total number concentration of ultrafine and accumulation mode particles and hospital admissions in children and the elderly in Copenhagen, Denmark." Occupational and Environmental Medicine **65**(7): 458-466.

Anderson, H. R., S. A. Bremner, R. W. Atkinson, R. M. Harrison and S. Walters (2001). "Particulate matter and daily mortality and hospital admissions in the west midlands conurbation of the United Kingdom: associations with fine and coarse particles, black smoke and sulphate." Occupational and Environmental Medicine **58**(8): 504-510.

Atkinson, R. W., H. R. Anderson, D. P. Strachan, J. M. Bland, S. A. Bremner and A. P. deLeon (1999). "Short-term associations between outdoor air pollution and visits to accident and emergency departments in London for respiratory complaints." European Respiratory Journal **13**(2): 257-265.

Atkinson, R. W., S. Kang, H. R. Anderson, I. C. Mills and H. A. Walton (2014). "Epidemiological time series studies of PM2.5 and daily mortality and hospital admissions: a systematic review and meta-analysis." Thorax **69**(7): 660-665.

Barnett, A. G., G. M. Williams, J. Schwartz, A. H. Neller, T. L. Best, A. L. Petroeschevsky and R. W. Simpson (2005). "Air pollution and child respiratory health - A case-crossover study in Australia and new Zealand." American Journal of Respiratory and Critical Care Medicine **171**(11): 1272-1278.

Bowatte, G., C. Lodge, A. J. Lowe, B. Erbas, J. Perret, M. J. Abramson, M. Matheson and S. C. Dharmage (2015). "The influence of childhood traffic-related air pollution exposure on asthma, allergy and sensitization: a systematic review and a meta-analysis of birth cohort studies." Allergy **70**(3): 245-256.

Brown, J. S. (2015). "Nitrogen dioxide exposure and airway responsiveness in individuals with asthma." Inhal Toxicol **27**(1): 1-14.

Burnett, R. T., Pope Iii, C. A., Ezzati, M., Olives, C., Lim, S. S., Mehta, S., Shin, H. H., Singh, G., Hubbell, B. & Brauer, M. (2014). “An integrated risk function for estimating the global burden of disease attributable to ambient fine particulate matter exposure”. Environmental Health Perspectives, 122, 397.

Chimonas, M. A. R. and B. D. Gessner (2007). "Airborne particulate matter from primarily geologic, non-industrial sources at levels below National Ambient Air Quality Standards is associated with outpatient visits for asthma and quick-relief medication prescriptions among children less than 20 years old enrolled in Medicaid in Anchorage, Alaska." Environmental Research **103**(3): 397-404.

Colais, P., M. Serinelli, A. Faustini, M. Stafoggia, G. Randi, R. Tessari, M. Chiusolo, B. Pacelli, S. Mallone, M. A. Vigotti, A. Cernigliaro, C. Galassi, G. Berti and F. Forastiere (2009). "Air pollution and urgent hospital admissions in nine Italian cities. Results of the EpiAir Project." Epidemiologia & Prevenzione **33**(6): 77-94.

COMEAP (1995) “Asthma and Outdoor Air Pollution”: London: HMSO. Available at http://webarchive.nationalarchives.gov.uk/20140505110006/http://www.comeap.org.uk/documents/reports

COMEAP (2010) “Does outdoor air pollution cause asthma?” http://webarchive.nationalarchives.gov.uk/20140505111639/http://www.comeap.org.uk/documents/statements/39-page/linking/53-does-outdoor-air-pollution-cause-asthma

COMEAP (2018) “Associations of long-term average concentrations of nitrogen dioxide with mortality” https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/734799/COMEAP_NO2_Report.pdf

Delzell, J. E., Jr. (2013). "Common lung conditions: asthma." FP Essent **409**: 11-16.

Ding, P. H., G. S. Wang, Y. L. Guo, S. C. Chang and G. H. Wan (2017). "Urban air pollution and meteorological factors affect emergency department visits of elderly patients with chronic obstructive pulmonary disease in Taiwan." Environ Pollut **224**: 751-758.

Favarato, G., H. R. Anderson, R. Atkinson, G. Fuller, I. Mills and H. Walton (2014). "Traffic-related pollution and asthma prevalence in children. Quantification of associations with nitrogen dioxide." Air Qual Atmos Health **7**(4): 459-466.

Goodman, J. E., C. T. Loftus, X. Liu and K. Zu (2017). "Impact of respiratory infections, outdoor pollen, and socioeconomic status on associations between air pollutants and pediatric asthma hospital admissions." PLoS One **12**(7): e0180522.

Halonen, J. I., T. Lanki, T. Yli-Tuomi, P. Tiittanen, M. Kulmala and J. Pekkanen (2009). "Particulate Air Pollution and Acute Cardiorespiratory Hospital Admissions and Mortality Among the Elderly." Epidemiology **20**(1): 143-153.

HEI Public Health and Air Pollution in Asia Program. (2010) Public Health and Air Pollution in Asia “(PAPA): Coordinated Studies of Short-Term Exposure to Air Pollution and Daily Mortality in Four Cities”. HEI Research Report 154. Health Effects Institute, Boston, MA. https://www.healtheffects.org/publication/public-health-and-air-pollution-asia-papa-coordinated-studies-short-term-exposure-air-0

Hua, J., Y. Yin, L. Peng, L. Du, F. Geng and L. Zhu (2014). "Acute effects of black carbon and PM(2).(5) on children asthma admissions: a time-series study in a Chinese city." Sci Total Environ **481**: 433-438.

Iskandar, A., Z. J. Andersen, K. Bonnelykke, T. Ellermann, K. K. Andersen and H. Bisgaard (2012). "Coarse and fine particles but not ultrafine particles in urban air trigger hospital admission for asthma in children." Thorax **67**(3): 252-257.

Ito K (2003) ”Associations of Particulate Matter Components with Daily Mortality and Morbidity in Detroit, Michigan” in Health Effects Institute. 2003. “Revised Analyses of Time-Series Studies of Air Pollution and Health”. Special Report. Health Effects Institute, Boston MA. https://www.healtheffects.org/publication/revised-analyses-time-series-studies-air-pollution-and-health

Johns, D. O., D. Svendsgaard and W. S. Linn (2010). "Analysis of the concentration-respiratory response among asthmatics following controlled short-term exposures to sulfur dioxide." Inhal Toxicol 22(14): 1184-1193.

Khreis, H., C. Kelly, J. Tate, R. Parslow, K. Lucas and M. Nieuwenhuijsen (2017). "Exposure to traffic-related air pollution and risk of development of childhood asthma: A systematic review and meta-analysis." Environ Int **100**: 1-31.

Ko, F. W. S., W. Tam, T. W. Wong, C. K. W. Lai, G. W. K. Wong, T. F. Leung, S. S. S. Ng and D. S. C. Hui (2007). "Effects of air pollution on asthma hospitalization rates in different age groups in Hong Kong." Clinical and Experimental Allergy **37**(9): 1312-1319.

Lai C et al. (2009). "Global variation in the prevalence and severity of asthma symptoms: phase three of the International Study of Asthma and Allergies in Childhood (ISAAC)". Thorax, 64(6):476–483.

Lee, J. T. (2003). "Associations between air pollution and asthma related hospital admissions in children in Seoul, Korea: A case crossover study." Korean Journal of Preventative Medicine **36**: 47-53.

Lim, S. S., Vos, T., Flaxman, A. D., Danaei, G., Shibuya, K., Adair-Rohani, H., Almazroa, M. A., Amann, M., Anderson, H. R. & Andrews, K. G. (2013). "A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010". The Lancet, 380, 2224-2260

Lim, H., H. J. Kwon, J. A. Lim, J. H. Choi, M. Ha, S. S. Hwang and W. J. Choi (2016). "Short-term Effect of Fine Particulate Matter on Children's Hospital Admissions and Emergency Department Visits for Asthma: A Systematic Review and Meta-analysis." J Prev Med Public Health **49**(4): 205-219.

Lin, M., Y. Chen, R. T. Burnett, P. J. Villeneuve and D. Krewski (2002). "The influence of ambient coarse particulate matter on asthma hospitalization in children: Case-crossover and time-series analyses." Environmental Health Perspectives **110**(6): 575-581.

Lippmann, M., K. Ito, A. Nadas and R. T. Burnett (2000). "Association of particulate matter components with daily mortality and morbidity in urban populations", Health Effects Institute. **95:** 5-72.

McConnell, R., K. Berhane, F. Gilliland, J. Molitor, D. Thomas, F. Lurmann, E. Avol, W. J. Gauderman and J. M. Peters (2003). "Prospective study of air pollution and bronchitic symptoms in children with asthma." American journal of respiratory and critical care medicine **168**(7): 790-797.

Michelozzi, P., F. Forastiere, C. A. Perucci, D. Fusco, A. Barca and T. Spadea (2000). "Acute effects of air pollution in Rome. [Italian]." Annali dell Istituto Superiore di Sanita **36**(3): 297-304.

Migliore E et al. (2009). Respiratory symptoms in children living near busy roads and their relationship to vehicular traffic: results of an Italian multicenter study (SIDRIA 2). Environmental Health, 8:27.

Mills, I. C., R. W. Atkinson, S. Kang, H. Walton and H. R. Anderson (2015). "Quantitative systematic review of the associations between short-term exposure to nitrogen dioxide and mortality and hospital admissions." BMJ Open **5**(5): e006946.

Molter, A., A. Simpson, D. Berdel, B. Brunekreef, A. Custovic, J. Cyrys, J. de Jongste, F. de Vocht, E. Fuertes, U. Gehring, O. Gruzieva, J. Heinrich, G. Hoek, B. Hoffmann, C. Klumper, M. Korek, T. A. Kuhlbusch, S. Lindley, D. Postma, C. Tischer, A. Wijga, G. Pershagen and R. Agius (2015). "A multicentre study of air pollution exposure and childhood asthma prevalence: the ESCAPE project." Eur Respir J **45**(3): 610-624.

Moolgavkar, S. H. (2000). "Air pollution and hospital admissions for diseases of the circulatory system in three US metropolitan areas." Journal of the Air & Waste Management Association **50**(7): 1199-1206.

Moore, E., L. Chatzidiakou, M. O. Kuku, R. L. Jones, L. Smeeth, S. Beevers, F. J. Kelly, B. Barratt and J. K. Quint (2016). "Global Associations between Air Pollutants and Chronic Obstructive Pulmonary Disease Hospitalizations. A Systematic Review." Ann Am Thorac Soc **13**(10): 1814-1827.

New York City Health, (2013). "Air Pollution and the Health of New Yorkers: The Impact of Fine Particles and Ozone". https://www1.nyc.gov/assets/doh/downloads/pdf/eode/eode-air-quality-impact.pdf. (Accessed 15 October 2018).

Orellano, P., N. Quaranta, J. Reynoso, B. Balbi and J. Vasquez (2017). "Effect of outdoor air pollution on asthma exacerbations in children and adults: Systematic review and multilevel meta-analysis." PLoS One **12**(3): e0174050.

Ostro, B., L. Roth, B. Malig and M. Marty (2009). "The Effects of Fine Particle Components on Respiratory Hospital Admissions in Children." Environmental Health Perspectives **117**(3): 475-480.

Robinson NH, Allan JD, Huffman JA, Kaye PH, Foot VE and Gallagher M (2013) "Cluster analysis of WIBS single-particle bioaerosol data" Atmos. Meas. Tech., 6, 337–347.

Sunyer, J., C. Spix, P. Quenel, A. Ponce-de-Leon, T. Barumandzadeh, G. Touloumi, L. Bacharova, B. Wojtyniak, J. Vonk, L. Bisanti, J. Schwartz and K. Katsouyanni (1997). "Urban air pollution and emergency admissions for asthma in four European cities: The APHEA project." Thorax **52**(9): 760-765.

Ueda, K., H. Nitta and H. Odajima (2010). "The effects of weather, air pollutants, and Asian dust on hospitalization for asthma in Fukuoka." Environmental Health and Preventive Medicine **15**(6): 350-357.

US Environmental Protection Agency Integrated Science Assessments for Nitrogen Dioxide (Health Criteria) (2016); Ozone and Related Photochemical Oxidants (2013) and Particulate Matter (2009) Available via https://www.epa.gov/isa

Walton HA, Kang S, Atkinson RW, Mills IC, Anderson HR(2014). "Quantitative systematic review of the associations between short-term exposure to ambient ozone and mortality and hospital admissions". In: Atkinson RW, Mills IC, Walton HA, Kang S, Anderson HR, editors. "Systematic Review and Quantitative Meta-analysis of the Evidence for Associations between Chronic and Short-term Exposure to Outdoor Air Pollutants and Health" Department of Health Policy Research Programme Project: 002/0037 http://www.sgul.ac.uk/images/Research/Pop_Health/DHreport_chronic_shortterm_exposure_to_outdoor_pollutants092015.pdf

Walton H, Dajnak D, Beevers SD, Williams ML, Watkiss P, Hunt A, (2015) "Understanding the Health Impacts of Air Pollution in London", King's College London available at: https://www.london.gov.uk/sites/default/files/hiainlondon_kingsreport_14072015_final.pdf. (Accessed 1 October 2018).

Weinmayr, G., E. Romeo, M. De Sario, S. K. Weiland and F. Forastiere (2010). "Short-term effects of PM10 and NO2 on respiratory health among children with asthma or asthma-like symptoms: a systematic review and meta-analysis." Environ Health Perspect **118**(4): 449-457.

WHO (2013) "Health Risks of Air Pollution in Europe HRAPIE project. Recommendations for concentration–response functions for cost–benefit analysis of particulate matter, ozone and nitrogen dioxide." http://www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications/2013/health-risks-of-air-pollution-in-europe-hrapie-project-recommendations-for-concentrationresponse-functions-for-costbenefit-analysis-of-particulate-matter,-ozone-and-nitrogen-dioxide.

Winquist, A., M. Klein, P. Tolbert, W. D. Flanders, J. Hess and S. E. Sarnat (2012). "Comparison of emergency department and hospital admissions data for air pollution time-series studies." Environ Health **11**: 70.

Zheng, X. Y., H. Ding, L. N. Jiang, S. W. Chen, J. P. Zheng, M. Qiu, Y. X. Zhou, Q. Chen and W. J. Guan (2015). "Association between Air Pollutants and Asthma Emergency Room Visits and Hospital Admissions in Time Series Studies: A Systematic Review and Meta-Analysis." PLoS One **10**(9): e0138146.