



## **Parliament Hill Research**

# **The Impacts Of Air Pollution On Health A Summary Of The State Of Current Knowledge**

**A review of recent primary epidemiological evidence on the health risks of air pollution for the City of London Corporation, including quantitative estimates of the effects in Central London**

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### **About this report**

This report summarises the current understanding of the health impacts of air pollution, in particular new evidence that has been developed since 2005, when the World Health Organisation (WHO) undertook the last major review of evidence. The report comprises an Executive Summary, a summary (in Part A) of the recent advances in understanding of how air pollution affects health, a review (Part B) of how air pollution compares to other causes of disease based on WHO, DH and PHE statistics, and a quantitative analysis of public health effects in Central London (Part C). A full list of references and appendices detailing the methodology used to conduct the review conclude the end of the report.

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## Executive Summary

Since the last major World Health Organisation (WHO) review in 2005 several thousand new studies have been published on the effects of air pollution on health. This evidence was drawn together in 2013, again by WHO, in a comprehensive review specifically to inform policymakers, public health officials and to a lesser extent health practitioners and scientists. This review has established that there is much better evidence on the health impacts of air pollution than in 2005, and that Particulate Matter (PM), nitrogen dioxide (NO<sub>2</sub>) and ozone (O<sub>3</sub>) are certain causes of death and disease, rather than probable causes as previously understood. Though people with cardiovascular (CVD) or respiratory disease can be hospitalised by air pollution episodes, the average effect on most individuals is small. However since very many people are exposed this adds up to a large effect on the population. **Public Health England's (PHE) figures suggest that PM<sub>2.5</sub> is a major cause of disease in London, and at least as important as road accidents, communicable disease, liver disease and suicide, and ranks up to 5<sup>th</sup> overall.**

There is now convincing evidence<sup>1</sup> that PM contributes quantifiably to premature death, both from long term and short term exposure. **Quantifiable mortality outcomes of long term exposure are mortality due to cardiovascular disease (CVD), cardiopulmonary disease, lung cancer and all-cause mortality<sup>2</sup>.** Long term CVD effects have been found after exposure of 6 months-2 years. **Quantifiable long-term morbidity<sup>3</sup> includes bronchitis in children and adults, asthma attacks, CVD and respiratory hospital admissions, urgent care due to asthma and CVD, and restricted activity days (e.g. working days lost).** Other effects include adverse birth outcomes and childhood respiratory disease. There is probable evidence that NO<sub>2</sub> and O<sub>3</sub> quantifiably cause disease. **Quantifiable outcomes of short term exposure to NO<sub>2</sub> are cardiovascular mortality, hospital admissions, asthma and bronchitis in children.**

There is probable evidence that coarse (2.5-10 µm), fine (0-2.5 µm) and ultrafine (< 0.1µm) PM fractions and NO<sub>2</sub> each harm in different ways and cause different diseases. Coarse PM is associated with CVD and respiratory disease, fine is associated with CVD and lung cancer, ultrafine PM possibly contributes to inflammation of the central nervous system, problems in pregnancy, Alzheimer's and Parkinson's diseases, Type II diabetes and poor school outcomes. NO<sub>2</sub> is associated with respiratory diseases and asthma. There is possible evidence that the effects of PM<sub>2.5</sub> are exacerbated by obesity, female gender, and lifestyle conditions associated with less educated populations [Hoek et al, 2013].

**Calculations of air pollution's impacts in Central London suggest that PM<sub>2.5</sub> pollution is responsible for the annual equivalent of 620 deaths, 70 respiratory hospital admissions, 90 CVD hospital admissions, and 656,900 sick days. Air pollution from**

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<sup>1</sup> Definitions of convincing, probable, possible and insufficient evidence are given in Appendix 3.

<sup>2</sup> All-cause mortality is defined as the incidence of deaths within a population or the number of deaths in a population

<sup>3</sup> Morbidity is defined as the incidence of disease within a population

**NO<sub>2</sub> was estimated to cause 182 respiratory hospital admissions.** Of these figures the most certain is that for deaths; the least certain is that for sick days.

## PART A: THE HEALTH IMPACTS OF AIR POLLUTION

### 1. The Health Effects of Particulate Matter (PM)

All the studies reviewed examined the effects of PM to some extent (especially PM<sub>2.5</sub>) as previous studies identified it as the pollutant that health outcomes were best understood for. The new evidence has improved the understanding of the effects of PM<sub>2.5</sub> and reduced the uncertainty in quantifiable effects of PM<sub>2.5</sub>. The evidence suggests that reducing PM concentrations will reduce CVD mortality within a few years [Brook et al, 2010; WHO, 2013]. Evidence has also been reviewed concerning distinct effects of black ultrafine, fine and coarse particles [WHO, 2013]. Coarse and fine particles deposit at differing locations in the respiratory tract, and have different sources and compositions, and there is possible evidence that they act partly through different biological mechanisms and result in different health outcomes [WHO, 2013]. **Brook et al [2010] recommend that all patients with CVD should be educated about the risks posed by air pollution and consideration should be given to educating those at high risk of CVD. This education should advise on reducing exposure and limiting activity on the basis of air pollution forecasts [Brook et al, 2013].** There is now evidence that PM<sub>2.5-10</sub> and PM<sub>2.5</sub> have distinct effects, due to the different locations in which they lodge in the airways. The ultrafine components of PM<sub>2.5</sub> are believed to cause systemic harm.

#### *Quantifiable long term effects of PM<sub>2.5</sub>*

An authoritative review of PM<sub>2.5</sub> [Brook et al, 2010] concluded that long-term exposure to PM<sub>2.5</sub> is a cause of both CVD mortality and morbidity [WHO, 2013], and the evidence is convincing. Brook et al (2010) reached consensus that the mechanism is systemic pro-inflammatory response, vasoconstriction, vascular dysfunction, increased coagulation potential and atherosclerosis [WHO, 2013]. There is probable evidence that there is a linear association between long term exposure to PM<sub>2.5</sub> and all-cause mortality and morbidity [WHO, 2013], that association continues to levels well below the current annual WHO guideline level of 10µg/m<sup>3</sup> annual average, and that there is no lower threshold. Thus there is probable evidence that disease still occurs due to PM<sub>2.5</sub> at levels well below EU limits and at-and-below levels experienced throughout London.

A Health Impact Assessment based on long term PM<sub>2.5</sub> can quantify the following health outcomes [WHO, 2013a], with Relative Risks (RRs)<sup>4</sup> and Hazard Ratios (HRs)<sup>5</sup> and 95% confidence intervals (CI) drawn from cited or recent reviews. For people over 30 years

- Attributable deaths due to all-causes, RR=1.062 (CI 1.04-1.083) per 10µg/m<sup>3</sup> [Fann et al, 2011; Hoek et al, 2013]
- Attributable deaths due to CVD, COPD and trachea, bronchus and lung cancer, RRs

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<sup>4</sup> Relative Risk (RR) is the probability of an event occurring. It compares a risk in two different groups of people, e.g. those exposed to air pollution vs. those in clean air. It can be expressed as fractional value, such as 1.06, or as a percentage, such as 6%, both meaning the same thing.

<sup>5</sup> Hazard Ratio (HR) is the measure of relative probability of an event in two groups over time. It is similar to a relative risk, but takes account of the fact that once people have certain types of event, such as death, they are no longer at risk of that event.

should be obtained from [Lim et al, 2012]

The RR for all-cause mortality is consistent with COMEAP (2009) but substantially reduces the uncertainty from a 75% CI of 1.01-1.12, to a 95% CI of 1.040-1.083.

#### *Non-quantifiable long term effects of PM<sub>2.5</sub>*

Long term exposure to elevated concentrations of PM<sub>2.5</sub> at levels typically experienced in urban areas reduces life expectancy by several months to a few years [Brook et al, 2010, p.2331]. There is possible evidence of atherosclerosis (increased intima media thickness) after exposure periods of six months to two years in human and animal studies [Brook et al, 2010; WHO, 2013]. Long term exposure is linked to atherosclerosis, adverse birth outcomes and childhood respiratory disease [WHO, 2013], at the level of possible evidence. And there is emerging - but insufficient - evidence of links between long term PM<sub>2.5</sub> exposure and the health of the central nervous system, the progression of Alzheimer's and Parkinson's diseases, developmental outcomes in children, and such reproductive health outcomes as low birth weight, as well as other chronic conditions such as diabetes [WHO, 2013].

#### *Quantifiable short term effects of PM<sub>2.5</sub>*

There is probable evidence that short-term (hours-days) exposure to PM<sub>2.5</sub> increases daily mortality, hospital admissions due to CVD and respiratory diseases [WHO, 2013a].

- Daily all-cause mortality, 1.23% per 10µg/m<sup>3</sup>
- Increased respiratory hospital admissions, all ages, 0.91% per 10µg/m<sup>3</sup> 24hr mean
- Increased CVD hospital admissions, all ages, 1.9% per 10µg/m<sup>3</sup> 24hr mean.

#### *Non-quantifiable short term effects of PM<sub>2.5</sub>*

Short-term exposure to PM<sub>2.5</sub> over a period of a few hours to weeks can trigger CVD-related mortality and non-fatal events, including myocardial ischemia and myocardial infarctions (MI, heart attacks), acute decompensated MI (heart failure), arrhythmias and strokes. This increase in risk is for susceptible - though not necessarily critically ill - individuals. The elderly and those with diabetes are at greater risk, and women and obese individuals may be at greater risk [Brook et al, 2010]. RRs are provided for working days lost (RR=4.6% per 10µg/m<sup>3</sup>) but are recommended for use only to test the range of effects [WHO, 2013a], as are those for Restricted Activity Days and Incidence of Asthma in children.

#### *Non-quantifiable effects of exposure to the PM coarse fraction*

There is possible evidence that short-term exposure to coarse particles (2.5-10µm) is associated with respiratory and cardiovascular effects, including premature mortality, and the differences from the effects of PM<sub>2.5</sub> can be explained partly by differences in intake and biological mechanisms [WHO, 2013].

#### *Non-quantifiable effects of exposure to ultrafine PM*

There is possible evidence of an association between ultrafine (< 0.1µm) PM and cardiorespiratory health and the health of the central nervous system [WHO, 2013]. Studies suggest that the body ingests 66%-88% of the ultrafine particles in each in-breath. These

are then distributed to other organs after entering through the lungs and cause effects on those organs also, such as in the central nervous system [WHO, 2013].

## **2. The health effects of NO<sub>2</sub>**

Researchers have difficulty separating the long-term effects of NO<sub>2</sub> from those of PM as the two are highly correlated being from the same sources. Recent very large cohort studies now suggest that NO<sub>2</sub> levels at or below the current Limit Value lead to significant health effects in the short and long term, independent of PM [WHO, 2013].

### *Quantifiable short term effects of NO<sub>2</sub>*

The WHO expert panel states that short term NO<sub>2</sub> exposure is associated with adverse cardiovascular and respiratory outcomes and all-cause mortality independent of PM. Evidence of appropriate biological mechanisms supports this with airway hyper-responsiveness and lung cell changes observed in animal and human studies at concentrations widely experienced [WHO, 2013], and typical of London roadsides. Some studies suggest stronger effects by NO<sub>2</sub> than by PM on asthma [WHO, 2013].

WHO [2013] recommends that short-term NO<sub>2</sub> effects should be included for sensitivity analysis in a two-pollutant Health Impact Assessment based on PM<sub>2.5</sub>, with the strongest evidence being for respiratory hospital admissions and some evidence for all-cause mortality. In WHO (2013a) the following RRs are listed:

- Increased all-cause mortality, RR=0.27%<sup>6</sup> (CI 0.16%-0.38%) per 10µg/m<sup>3</sup> 1hr mean
- Increased respiratory hospital admissions, all ages, 1.8% per 10µg/m<sup>3</sup> 24hr mean

Given the very high concentrations of NO<sub>2</sub> found at roadsides in London, the all-cause mortality coefficient is of particular note.

### *Non-quantifiable long term effects of NO<sub>2</sub>*

Some studies suggest associations between long-term NO<sub>2</sub> exposure and CVD and respiratory mortality, children's respiratory symptoms and lung function, independent of PM. There is concern that NO<sub>2</sub> in this case may be a proxy for other pollutants, but evidence on the biological mechanisms by which NO<sub>2</sub> affects the airways suggests supports the case for a direct causal relationship with NO<sub>2</sub> [WHO, 2013]. The following RRs are listed but considered to be of high uncertainty [WHO, 2013a]:

- Long term all-cause mortality, RR=1.055 (CI 1.031-1.080) per 10µg/m<sup>3</sup>.

This is not controlled for PM so does not meet NO<sub>2</sub> HIA guidelines in WHO (2013).

## **3. Health effects of O<sub>3</sub> exposure**

There is probable evidence that health effects of short-term exposure to ozone are experienced below the current EU information threshold level of 180µg/m<sup>3</sup> [WHO, 2013] but that a threshold for ozone harm exists somewhere below 90µg/m<sup>3</sup>. A number of coefficients

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<sup>6</sup> We show very small RRs as percent for clarity, e.g. 0.27% = 1.0027

are available for different outcomes (see [WHO, 2013]), such as this example.

- All cause mortality due to short term exposure over  $35\mu\text{g}\text{m}^{-3}$ , all ages, 0.29% (CI 0.14%-0.43%) per  $10\mu\text{g}/\text{m}^3$  (8hr mean) of ozone.

#### **4. Other findings**

##### *Documented improvements in public health with reduction in air pollution*

Both WHO (2013) and Fann (2010) conclude that there is significant evidence from many interventions (both deliberate and accidental) that have reduced  $\text{PM}_{2.5}$  concentration have lead to improved public health outcomes.

##### *Vehicle exhaust is a Group 1 Carcinogen causing lung cancer*

In 2012 The WHO's International Agency for Research on Cancer (IARC) classified diesel exhaust *per se* as a Group 1 carcinogen causing lung cancer, the same class as smoking and asbestos. In 2013, IARC went on to classify all vehicle exhaust as a Group 1 carcinogen.

##### *Bio-diesel exhaust more carcinogenic*

Several studies suggest that bio-diesel exhaust may be 4-10 times more mutagenic than fossil-fuel diesel exhaust, though at the level of insufficient evidence [WHO, 2013].

## **PART B: AIR POLLUTION COMPARED TO OTHER CAUSES OF DISEASE**

### **5. The effects of PM<sub>2.5</sub> air pollution compared with other causes of disease**

Three studies directly or indirectly compare levels of disease due to air pollution with diseases from other causes, at local, national and international scales [PHE, 2103; DH, 2011; Lim et al, 2012]. All three estimated the effects of air pollution using mean long term concentrations of PM<sub>2.5</sub> over large geographic areas, reflecting the scientific consensus on the understanding of air pollution as it was until the most recent reviews. PM<sub>2.5</sub> was used as it is the pollutant with the best understood correlation with all-cause mortality. Ambient (that is outdoor) PM<sub>2.5</sub> air pollution is estimated to be the 9th cause of disease worldwide and 11th overall in Western Europe, according to the WHO's burden of disease study [Lim et al, 2012] (Table 1, Global and Western Europe columns).

For England, the Department of Health conducted a calibration analysis of the PHOF indicators. This ranked ambient PM<sub>2.5</sub> as joint 5<sup>th</sup> cause of premature death (or strictly speaking joint 5<sup>th</sup> factor where an improvement would increase life expectancy), however this analysis excludes many important causes of disease that lack the background data needed for the calibration process, e.g. diabetes. Notwithstanding this caveat, ambient PM<sub>2.5</sub> came out ahead of smoking prevalence (as opposed to individual smoking), infant mortality, chronic liver disease, road death, communicable diseases and cancer diagnosed at stage 1 and 2 in this ranking (Table 1, England column).

Using the PHOF dataset [PHE, 2013] it is also possible to compare causes of death in London, as the indicators include 13 mortality rate measures (e.g. mortality rate due to cancer in the under 75s), which include the PM<sub>2.5</sub> Indicator 3.01. We converted these to a common set of units (see Appendix 1) for the year 2011, and developed three variations of the PM<sub>2.5</sub> Indicator:

1. The risk to a population exposed to PM<sub>2.5</sub> concentrations found in the City
2. The risk to a population exposed 50% of the time to City concentrations and 50% of the time to London average PM<sub>2.5</sub> concentrations
3. The average value of mortality risk due to PM<sub>2.5</sub> across London.

Bearing in mind the issues described earlier in applying Concentration Response Functions (CRFs) to small populations or small areas, it is important that (1) and (2) be considered as levels of mortality risk due to air pollution not numbers of deaths, and these will be more uncertain than (3). As (3) is calculated over a large population it can be considered as an estimate of the mortality rate due to PM<sub>2.5</sub>. On this basis, compared with other mortality figures in the PHOF, we find that air pollution ranks as the joint 5<sup>th</sup> major cause of premature death in London, with City Residents and City Workers having a somewhat higher risk of mortality due to PM<sub>2.5</sub> than the average Londoner (see Table 1, London column, and Appendix 1).

Overall, these studies present probable evidence that PM<sub>2.5</sub> air pollution is one of the major causes of disease in the World, in Western Europe, in England and in London, and that PM<sub>2.5</sub> has a somewhat higher impact in the City than in the rest of London, as might be

expected due to its somewhat higher concentrations of PM<sub>2.5</sub>. They suggest that reducing PM<sub>2.5</sub> concentrations and exposure should be prioritised at least as much as preventing respiratory mortality, communicable diseases, liver disease and suicide.

**Table A: Ranking of risk from PM<sub>2.5</sub> air pollution vs. other causes of disease or death**

Rank	Global [Lim et al, 2012], burden of disease	Western Europe [Lim et al, 2012], disease	England [DH, 2011], Premature mortality <sup>7</sup>	London [PHE, 2013] mortality rate/0.1M
1	High blood pressure	Smoking	Cancer	Cancer in the under 75s (<75s)
2	Smoking	High blood pressure	CVD	Preventable* deaths
3	Indoor air pollution	High Body Mass Index	Adult excess weight	CVD (<75s)
4	Low fruit diets	Physical inactivity	Over 18s smoking prevalence	Preventable* cancer
5	Alcohol use	High fasting blood glucose	<b>Ambient PM (=5<sup>th</sup>)</b>	<b>Ambient PM</b>
6	High Body Mass Index	Alcohol use	Vaccination coverage (=5 <sup>th</sup> )	Preventable* CVD
7	High fasting blood glucose	Low fruit diets	Respiratory disease (=5 <sup>th</sup> )	Road accidents, killed & seriously injured
8	Childhood underweight	High total cholesterol	Infant mortality	Communicable diseases
9	<b>Ambient PM</b>	Low nuts & seeds diet	Premature death of people with mental illness	Respiratory disease (<75s)
10	Physical inactivity	High sodium	Cancer diagnosed at stage 1 & 2 (=10 <sup>th</sup> )	Liver disease (<75s)
11	High sodium	<b>Ambient PM</b>	Chronic Liver Disease (=10 <sup>th</sup> )	Preventable* liver disease (<75s)
12	Low nuts & seeds diet	High processed meat diet	Suicide (=10 <sup>th</sup> )	Preventable* respiratory disease (<75s)
13	Iron deficiency	Low vegetables	Road death & injuries (=10 <sup>th</sup> )	Suicide
14	Suboptimal breastfeeding	Drug use	Communicable diseases (=10 <sup>th</sup> )	
15	High total cholesterol	Low omega-3	Excess winter deaths	
16	Low whole grains	Low whole grains	Drug treatment	
17	Low vegetables	Occupational low back pain	Falls in over 65s	
18	Low omega-3 diet	Low fibre diet	Smoking at time of delivery	
19	Drug use	Low polyunsaturated fatty acids diet	NHS Health Check	
20	Occupational injury	Low bone mineral density	Under 18 conceptions, Homelessness, Fuel Poverty	* See Page 17 for definition of preventable

<sup>7</sup> The ONS defines premature mortality to be death under the age of 75. For more information see ONS report “Trends in premature mortality in England and Wales, 1950-2004.”

## Part C Quantitative Estimates Of The Impacts in Central London

The WHO has published recommendations and guidelines for conducting Health Impact Assessment on the effects of air pollution on health [WHO, 2013a], which include RR coefficients for use in assessments. The pollutant-outcome pairs comprising the recommended minimum assessment include

- A. All-cause mortality in the over 30s and Annual mean PM<sub>2.5</sub>
- B. Hospital admissions (CVD & strokes) and Daily mean PM<sub>2.5</sub>
- C. Hospital admissions (Respiratory disease) and Daily Mean PM<sub>2.5</sub>
- D. Hospital admissions (Respiratory disease) and Daily Mean NO<sub>2</sub>

Coefficients are also provided for

- E. Lost working days and 14-day mean PM<sub>2.5</sub>
- F. Asthma attacks in children and Daily mean PM<sub>10</sub>
- G. Bronchitis symptoms in asthmatic children and Annual mean NO<sub>2</sub>

We have used air pollution measurements from the urban background measurements stations in the AURN (Automatic and Rural Network) and LAQN (London Air Quality Network) networks in the eight central London boroughs to obtain relevant data to estimate A-G and the potential effects of reductions.

### A. All-cause mortality in the over 30s and Annual mean PM<sub>2.5</sub>

This quantity has been calculated by PHE for PHOF i3.01, expressed as an attributable percentage of mortality, for the elimination of all anthropogenic sources of PM<sub>2.5</sub>. Converted to deaths in 2011, this amounts to an equivalent mortality of 617 of the 12,195 deaths in 2011, comparable to the 720 deaths due to respiratory disease but less than 1/3 of the 2,101 deaths due to cardiovascular disease.

Using the results of Hoek et al. (2013) as recommended in WHO (2013a), and the APHEKOM counterfactual approach to the calculation (Pascal et al, 2011) of life tables, we obtained a slightly higher figure for all-cause mortality attributable to PM<sub>2.5</sub> of 646 in Central London. This difference can be accounted by differences between PCM (Pollution Climate Mapping) data used in i3.01 and actual urban background measurements. The data used was for the period 2010-2012 and produced an average concentration of 17µgm<sup>-3</sup>. In addition, since some of the anthropogenic sources of the air pollution experienced in London are from non-London sources, local authorities in London cannot reduce all the anthropogenically sourced PM<sub>2.5</sub> arriving in London. To explore the effects of reducing air pollution partially, we have considered two scenarios (based on recommendations from [Pascal, 2011]):

1. Reducing PM<sub>2.5</sub> concentrations by 5µgm<sup>-3</sup>
2. Reducing PM<sub>2.5</sub> concentrations to the WHO guideline of 10µgm<sup>-3</sup>.

#### *Reducing Annual PM<sub>2.5</sub> concentrations by 5µgm<sup>-3</sup>*

This could have the benefit of avoiding 222 deaths annually, equivalent to halving deaths from preventable liver disease. Of these 115 deaths would have been due to cardiovascular disease.

*Reducing Annual PM<sub>2.5</sub> concentrations to the WHO guideline of 10µg/m<sup>-3</sup>*

This could have the benefit of avoiding 294 deaths annually, of which 152 would have been due to cardiovascular disease.

### **B, C and D. Hospital admissions for CVD and respiratory disease and Daily mean PM<sub>2.5</sub> or NO<sub>2</sub>**

Again for these it is necessary to consider multiple hypotheses. Those used were

1. Reducing daily PM<sub>2.5</sub> by 5µg/m<sup>3</sup>
2. Reducing daily PM<sub>2.5</sub> to the WHO guideline
3. Reducing daily NO<sub>2</sub> by 7µg/m<sup>3</sup> to bring it below the EU Annual Limit Value
4. Reducing daily NO<sub>2</sub> to 10µg/m<sup>3</sup>

The data used was urban background measurements of PM<sub>2.5</sub> and NO<sub>2</sub> in Central London from four monitoring stations, over the period 2010-2012. The mean PM<sub>2.5</sub> value was 17µg/m<sup>3</sup>, while that for NO<sub>2</sub> was 46µg/m<sup>3</sup>.

*Reducing Daily PM<sub>2.5</sub> concentrations by 5µg/m<sup>-3</sup>*

This could have the benefit of avoiding 22 respiratory hospital admissions in Central London out of a total of 2335 annually, or about 1% of respiratory admissions. It could reduce CVD hospitalizations by 30 per year, or about 0.4% of the 7004 annually.

*Reducing Daily PM<sub>2.5</sub> concentrations to the WHO guideline of 10µg/m<sup>-3</sup>*

This could have the benefit of avoiding 37 respiratory hospital admissions in Central London annually, or about 1.5% of respiratory admissions. It could reduce CVD hospitalizations by 50 per year, or about 0.7% of the 7004 annually.

*Reducing Daily NO<sub>2</sub> concentrations by 7µg/m<sup>-3</sup>*

This could have the benefit of avoiding 29 respiratory hospital admissions in Central London, or about 1% of respiratory admissions.

*Reducing Daily NO<sub>2</sub> concentrations to 10µg/m<sup>-3</sup>*

This could have the benefit of avoiding 143 respiratory hospital admissions in Central London annually, or about 6% of respiratory admissions.

### **E. Lost working days and 14-day mean PM<sub>2.5</sub>**

For this calculation as the outcome has no terminal health outcome, and the published data is considered to be uncertain, a simplified calculation was used. A RR of 1.046 per 10µg/m<sup>3</sup> [WHO, 2013a] was used with the PM<sub>2.5</sub> data described previously. Underlying data on sick days was obtained from the PHOF indicator set (i1.09ii) and - after some manipulation - was estimated to be 4.9million lost days at work per year amongst the Central London population, or 3 sick days per person per year. Using this RR the number of sick days attributable to air pollution was estimated to be 656,900 per year in Central London. It should be noted that WHO (2013a) considers that this statistic should only be used to test the extended impacts of air pollution.

**F & G. Asthma and respiratory incidence in children**

It was not possible to calculate these statistics as the underlying baseline data were not available from ONS or other sources.

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## Appendix 1

### The evidence reviewed, the scientific process behind it & the method applied

In 2012 and again in 2013 the WHO International Agency for Research on Cancer (IARC) classified diesel exhaust fumes as a “Group 1 carcinogen” and in 2013 WHO published their Review of Evidence of the Health Aspects of Air Pollution (REVIHAAP, [WHO 2013]). The IARC classification means that ***a positive relationship has been observed between the exposure [to diesel exhaust] and [lung cancer] in studies in which chance, bias and confounding could be ruled out with reasonable confidence*** [IARC, 2012]. The REVIHAAP review is a more wide ranging study covering the health aspects of many pollutants including PM, NO<sub>2</sub> and O<sub>3</sub> and was commissioned by the EU as part of its review of air pollution regulations in EU countries.

Major scientific reviews and announcements do not occur in isolation but rather follow established processes to reach a consensus view. These comprise the gathering of very large volumes of evidence using an including and impartial mechanism, drafting of a review of that evidence by a panel of experts based on the evidence and their expert knowledge and sometimes using statistical techniques to pool the results of different studies, criticism of these drafts by a much wider panel of experts, amendments to the drafts, often in several iterations, and final agreement of a consensus view. In the case of REVIHAAP, the report was written and commented on by 74 authors, and they reviewed some 2100 scientific reports covering studies of several million people worldwide. As this laborious process typically takes 3-7 years the reviews are inevitably somewhat out of date by the time they are published. To mitigate this they often refer to smaller, quicker and more recent reviews that include more recent evidence on a specific sub-topic and are expected to be published around the same time.

We began by studying REVIHAAP, then a number of the major reviews cited in it or associated with it by its lead authors, and evidence from DH and PHE. We first present the evidence from WHO, DH and PHE on how the effects of PM<sub>2.5</sub> compare with other causes of disease from the following publications:

- 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, published in The Lancet in 2012 [Lim et al, 2012]
- Impact Assessment of the Public Health Outcomes Framework (PHOF), UK Dept. of Health, 2011 [DH, 2011]
- The Public Health Outcomes Framework Observatory, Public Health England, 2013 [PHOF, 2013]

We then present evidence on the separate health aspects of PM, NO<sub>2</sub> and O<sub>3</sub>. Primarily, this is drawn from two studies that set out to test the case for convincing or probable evidence of a quantifiable and causal link between air pollutants and disease. These review evidence

from all of the following areas of research: epidemiological research into time series, cohorts and panels, clinical animal and human studies into toxicology and biological mechanisms.

- The REVIHAAP Technical Report [WHO, 2013]
- Health Risks of Air Pollution in Europe [WHO, 2013a]
- Particulate Matter Air Pollution and Cardiovascular Disease: An Update to the Scientific Statement From the American Heart Association, a review published in *Circulation* in 2010 [Brook et al, 2010]

These sections of this report also draw on evidence from reviews cited in these two, or published in 2013 that were linked to the findings of REVHAAP:

- Estimating the National Public Health Burden Associated with Exposure to Ambient PM<sub>2.5</sub> and Ozone, published in *Risk Analysis* in 2012 [Fann et al, 2012]
- Long-term air pollution exposure and cardio-respiratory mortality: a review, published in *Environmental Health* in 2013 [Hoek et al, 2013]
- Global association of air pollution and heart failure: a systematic review and meta-analysis, published in *The Lancet* in 2013 [Shah 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), a meta-study published in *Lancet Oncology* in 2013 [Raaschou-Nielson, 2013].

In addition we have drawn on evidence and tools developed by COMEAP and in the APHEKOM air pollution and epidemiology research project:

- Long-Term Exposure to Air Pollution: Effect on Mortality, Dept. of Health Committee on the Medical Effects of Air Pollutants, 2009 [COMEAP, 2009]
- Local city report for London, [Atkinson, 2011]
- Guidelines for Assessing the health impacts of air pollution in European Cities, [Pascal et al, 2011]

We have also consulted directly with three leading epidemiologists. We are most grateful to Prof F. Kelly, Dr M. Nieuwenhuijsen and Dr A. de Nazelle for their generous advice, but emphasise that this report's contents and errors are entirely due to the authors.

### *Diseases and Health Outcomes*

It is important to note the distinction between the terms **disease** and the epidemiological term **health outcome**. Disease refers to a medical ailment of individuals, while health outcome is a technical epidemiological term meaning “a measured change in the health of an individual, group of people or population which is attributable to an intervention or series of interventions.” Health outcomes include measured individual cases of disease, but also include broader population effects such as measured hospital admissions, asthma attacks or overall mortality. Health outcomes must also be linked to a specific cause or intervention.

### *Statistical Associations, Quantifiable vs. Non-quantifiable health outcomes*

Epidemiologists also routinely refer to **associations** by which they mean statistical associations between a cause and an effect. These are separated into statistically significant associations where the estimated are believed to be correct to within a certain margin of error, and those that are not statistically significant where the margin of error is so

large that the estimates are not considered meaningful. These allow epidemiologists to separate their findings into those where:

- A. There is convincing or probable evidence that a statistically significant **quantifiable estimate** can be made of specific mortality and morbidity outcomes (such as deaths or rates of certain illnesses) in a population due to air pollutants
- B. There is probable or possible evidence of an association between air pollutants and specific mortality and morbidity outcomes in a population, but evidence is **insufficient to be quantifiable with statistical significance.**

#### *Concentration-Response Functions and Coefficients*

To quantify the health outcomes associated with an air pollutant, epidemiologists use Concentration-Response Functions (CRFs). These specify the rate of a certain health outcome in a population exposed to a given concentration of air pollution over a specified period. These are calculated over very large populations, often over multiple studies and geographic locations. For small geographic areas or populations like a single borough application of CRFs are better considered an estimate of risk, rather than an accurate diagnosis of the number of deaths or illnesses. This approach has been used by PHE and we have used their data at Borough level for PM<sub>2.5</sub>.

## Appendix 2

### Mortality rates due to various diseases and causes of diseases in the Public Health England PHOF for the London average, Westminster and Camden.

Indicator	London	Westminster	Camden
4.05i - Cancer in the under 75s (< 75)	104.9	95.1	102.4
4.03 - Preventable deaths	104.3	132.9	148.2
4.04i - CVD < 75	64.3	61.5	65.6
4.05ii - Preventable* cancer < 75	60.8	54.4	63.1
3.01 Mortality due to air pollution PM <sub>2.5</sub> **	50.8	40.9	39.6
4.04ii - Preventable* CVD <75	40.2	34.0	38.0
1.10 - Road accidents (KSI)	35.5	81.8	49.4
4.08 - Communicable diseases	31.6	25.9	24.5
4.07i - Respiratory disease < 75	22.7	18.9	17.6
4.06i - Liver disease < 75	15.7	15.3	22.1
4.06ii - Preventable* Liver disease < 75	13.4	13.7	19.5
4.07ii - Preventable* Resp'y disease < 75	11.5	8.5	9.6
4.10 - Suicide rate (provisional)	6.9	9.1	7.7

\* A death is preventable if, in the light of understanding of the determinants of health at the time of death, all or most deaths from that cause (subject to age limits if appropriate) could be avoided by public health interventions in the broadest sense - from Office of National Statistics publication "Definition of avoidable mortality."

\*\* i3.01 has been converted to deaths per 100,000 population per year.

Of the 13 indicators listed above, 12 are given in the PHOF as mortality rates per 100,000, except for the PM<sub>2.5</sub> Indicator 3.01 (I3.01) which is given as a percentage of all-cause mortality. Indicator 3.01 is given for all boroughs, including the City, but the other 12 mortality indicators are not. To make all 13 indicators comparable, we converted I3.01 for each London borough to a death rate per 100,000 population using ONS mortality and population data for 2011. From this we calculated a London average mortality rate due to PM<sub>2.5</sub>. We also calculated the mortality risk to a population exposed all the time to the City levels of PM on the basis of the indicator value given for the City, and for a population exposed 50% of the time to the City levels and 50% to London levels. As stated in Sections 2 and 3, it is important that these latter two be considered an estimate of mortality risk, not a diagnosis of mortality, as CRFs must be applied over large populations to give meaningful diagnoses.

## **Appendix 3**

### **Definitions of the standard of evidence**

Each of the review examined this this report itself uses different approaches to grade the quality of evidence available to support the consensus view. For this report we have attempted to follow [Lim et al, 2012] by using the schema of the World Cancer Research Fund grading system [WCRF, 2007] based on the evidence given in the documents listed above. These definitions are:

#### **Convincing evidence**

Evidence based on epidemiological studies showing consistent associations between exposure and disease, with little or no evidence to the contrary. The available evidence is based on a substantial number of studies including prospective observational studies and where relevant, randomised controlled trials of sufficient size, duration, and quality showing consistent effects. The association should be biologically plausible.

#### **Probable evidence**

Evidence based on epidemiological studies showing fairly consistent associations between exposure and disease, but for which there are perceived shortcomings in the available evidence or some evidence to the contrary, which precludes a more definite judgment. Shortcomings in the evidence may be any of the following: insufficient duration of trials (or studies); insufficient trials (or studies) available; inadequate sample sizes; or incomplete follow-up. Laboratory evidence is usually supportive. The association should be biologically plausible.

#### **Possible evidence**

Evidence based mainly on findings from case-control and cross-sectional studies. Insufficient randomised controlled trials, observational studies, or non-randomised trials are available. Evidence based on non-epidemiological studies, such as clinical and laboratory investigations, is supportive. More trials are needed to support the tentative associations, which should be biologically plausible.

#### **Insufficient evidence**

Evidence based on findings of a few studies which are suggestive, but insufficient to establish an association between exposure and disease. Little or no evidence is available from randomised controlled trials. Better-designed research is needed to support the tentative associations.