Understanding the Health Impacts of Air Pollution in London

For: Transport for London and the Greater London Authority

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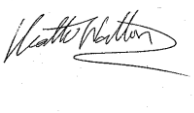
Heather Walton		
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Executive Summary

ES1 Background

The main purpose of this report, commissioned by TfL and the GLA, is to estimate the mortality burden of 2010 concentrations of fine particles ($PM_{2.5}$) in London (see key results box) as an update to the Institute of Occupational Medicine (IOM) report on $PM_{2.5}$ mortality using 2006 concentrations (Miller, 2010).

In addition, for the first time, emerging techniques have been used to assess the mortality burden of nitrogen dioxide (NO_2) in London, following on from WHO recommendations (WHO, 2013b). WHO acknowledged uncertainty in the evidence so the associated figures are considered approximate and need to be used with care[2].

The mortality burden is expressed as life-years lost across the population as a result of deaths in 2010[3] (a life year is one year lost for one person). This is the most accurate representation of the mortality burden, as it is when people die rather than whether they die that matters.

This result is also expressed as 'equivalent deaths at typical ages', the deaths that would account for the loss of life years if $PM_{2.5}$ or NO_2 were the sole cause[4].

The report extends the previous IOM work (Miller, 2010) to cover effects of short-term exposure to $PM_{2.5}$ and NO_2 as well as the economic valuation of short and long-term effects of both pollutants. The report does not cover effects of other pollutants such as ozone.

The results given in the key results box are for the burden of total pollution in 2010 but results were also calculated for the impact of future reductions. These calculations compared the impacts of predicted future reductions in $PM_{2.5}$ and NO_2 concentrations in 2012, 2015 and 2020 (maintaining 2020 concentrations until 2114), with the assumption of concentrations remaining at 2010 levels until 2114.

Other results given below include: the impact on life-expectancy from birth; apportionment of the health impacts to emission sources; the effects on health of trends in $PM_{2.5}$ or NO_2 concentrations from 2008-2012; London specific damage costs per tonne of transport emissions and a brief summary of methods. Reference is given to the main sections of the report for more details.

[2] See page 9.

[3] For long-term exposure and mortality, the effect in 2010 assumes pollution has been at 2010 levels for a long time. In practice, pollution-related mortality in 2010 is partly due to the effects of past concentrations and 2010 concentrations will have effects on mortality in later years.

[4] It is actually more likely that the loss of life years results from a partial contribution of these air pollutants to a larger numbers of deaths in combination with other risk factors, and that these smaller contributions 'add up' to the equivalent deaths.

ES2 Key results

$PM_{2.5}$ burden (long-term exposure): (**Section 2.1).** The total mortality burden of anthropogenic $PM_{2.5}$ for the year 2010 is estimated to be **52,630 life-years lost, equivalent to 3,537 deaths at typical ages[a]**. The result is similar but slightly larger than that estimated for London in 2010 by Public Health England (PHE), using methods designed for national comparisons (Gowers et al., 2014). The estimate for $PM_{2.5}$ attributable deaths has decreased from the previous estimate (4,267 deaths in 2008 based on 2006 concentrations) (Miller, 2010) partly due to a decrease in concentrations, to which policy interventions will have contributed, as well as some adjustments to the previous methods and inputs, such as using anthropogenic rather than total $PM_{2.5}$ and declines in baseline mortality rates. Further decreases should occur beyond 2010 as interventions have been put in place to reduce emissions further, although this may or may not be apparent in a specific year due to variations in weather conditions affecting concentrations.

New estimate of the NO_2 burden (long-term exposure): (**Section 2.1).** Whilst much less certain than for $PM_{2.5}$., the total mortality burden of long-term exposure to NO_2 is estimated to be **up to 88,113 life-years lost, equivalent to 5,879 deaths at typical ages[a]** (assuming the WHO value of up to a 30% overlap between the effects of $PM_{2.5}$ and NO_2). Some of this effect may be due to other traffic pollutants.

Can these effects be added? (**Section 2.1).** The total mortality burden in 2010 from $PM_{2.5}$ and NO_2 can be added to give a range from the 52,630 life-years lost, equivalent to 3,537 deaths at typical ages from $PM_{2.5}$ alone (if only including the most established effects) to as much as 140,743 life-years lost, equivalent to 9,416 deaths at typical ages[a] (assuming a 30% overlap between the effects of $PM_{2.5}$ and NO_2 and comparing with a zero concentration of NO_2). This potentially increases the estimated total mortality burden considerably, compared with both the previous IOM and PHE reports.

Short-term exposure and hospital admissions: (**Section 2.2).** Mortality is not the only air pollution related health effect – in 2010 $PM_{2.5}$ and NO_2 were associated with approximately 1990 and 420 respiratory hospital admissions respectively with an additional 740 cardiovascular hospital admissions associated with $PM_{2.5}$.

Economic costs: (**Section 4.2).** The estimated economic costs of the above health impacts ranged from **£1.4 billion** (long-term exposure to $PM_{2.5}$ and mortality; short-term exposure to $PM_{2.5}$ and hospital admissions; short-term exposure to NO_2 and both deaths brought forward and hospital admissions) to **£3.7 billion** (replacing short-term exposure to NO_2 and deaths brought forward with long-term exposure to NO_2 and mortality). Inclusion of other less well established health outcomes would increase the economic costs although this has not been estimated in this report.

[a] Rounded results 52,500 life-years lost, equivalent to 3,500 deaths at typical ages for $PM_{2.5,}$ 88,000 life-years lost, equivalent to 5,900 deaths at typical ages for NO_2 accounting for overlap with $PM_{2.5}$ and together up to as much as 141,000 life-years lost, equivalent to 9,500 deaths at typical ages (assuming a 30% overlap between the effects of $PM_{2.5}$ and NO_2 and comparing with a zero concentration of NO_2). Numbers shown here unrounded to show how the addition matches up rather than to suggest accuracy at a detail finer than a few hundred deaths or life years, given the uncertainties.

ES3 Derivation of estimates of the mortality burden of NO_2 and $PM_{2.5}$ in London

For the key results given above, the methods for $PM_{2.5}$ broadly followed those recommended for mortality burden in COMEAP (2010) and Gowers et al. (2014)[5], with some minor differences[6]. Methods for NO_2 followed the same principles but were based on coefficients recommended by the WHO HRAPIE project (WHO, 2013b)[7]. We chose the alternative based on an assumed 30% overlap with $PM_{2.5}$, as our main method[8], quantifying down to zero as the upper limit for the size of the effect. A sensitivity analysis quantifying only down to 20 μg m^{-3} is presented in the main report[9]. Subsequently, discussions in the field suggested support for a counter factual down to 5 μg m^{-3} (Annex 1). Rough scaling suggests that this would give figures about 10% smaller than the results given here, within the range from counter factuals at zero and 20 μg m^{-3}. Concentrations were modelled using the London Air Quality Modelling toolkit based on the London Atmospheric Emissions Inventory (LAEI) (GLA, 2013) and then weighted by the population aged 30+ at output area level. Estimates for individual London boroughs (provided in the main report section 2.1.3) were summed to give the London figure. The ranges around the estimates given in the key results are given in Table E1.

Table E1 Mortality burden of $PM_{2.5}$ and NO_2 in London

Pollutant (2010 concentrations)	Life years lost as a result of equivalent deaths in 2010	Equivalent deaths at typical ages in 2010
Anthropogenic* $PM_{2.5}$	52,630 (9287 to 98,648)[a]	3,537 (624 to 6,632)[a]
NO_2 (less certain) (30% overlap with $PM_{2.5}$)[10]	*Up to 88,113* *(51,629 to 121,918)[a]*	*Up to 5,879* *(3444 to 8138)[a]*
Total	52,630 *up to 140,743*	3,537 *up to 9,416*

* defined in glossary (Annex 11)

[a] Ranges based on plausibility intervals (statistical and other uncertainties) from COMEAP (2010) for $PM_{2.5}$ and 95% confidence intervals (statistical uncertainty) from WHO (2013b) for NO_2. The central estimates are added for the total but not the plausibility or confidence intervals because the probability of the estimate being at the same far end of the range in both cases is unlikely.

[5] Coefficient 6% increase in mortality per 10 μg m^{-3} $PM_{2.5}$, sensitivity 1%, 12%, applied to age 30+, assumes constant anthropogenic $PM_{2.5}$ at 2010 levels (lags ignored), life-years from deaths times baseline life expectancy by sex/age of death.

[6] Modelling by a different method at 20m not 1 km grid scale, different definition anthropogenic $PM_{2.5}$, population-weighting by borough, gender and 5 year age group (ca. 13.72 μg m^{-3}) not total population, life-years calculated by 5 year age group.

[7] Coefficient 3.9% (30% reduction from 5.5%) increase in mortality per 10 μg m^{-3} NO_2, 95% confidence interval 2.2%, 5.6%. Population-weighted concentration by borough, gender, 5 year age group varied around 36.42 μg m^{-3}.

[8] The exact size of the overlap is uncertain, where studied the maximum overlap was 33 %.

[9] If the effect was calculated from current levels down to 20 μg m^{-3} the mortality burden was 40,355 life-years lost, equivalent to 2650 attributable deaths at typical ages assuming a 30% overlap with $PM_{2.5}$, or 55,723 life-years lost, equivalent to 3661 attributable deaths assuming no overlap.

[10] If no overlap was assumed, the mortality burden of NO_2 was 119,999 (range 71,294 – 165,536) life-years lost, equivalent to 8,009 (range 4756 to 11,054) attributable deaths at typical ages.

The NO_2 results need to be interpreted cautiously. Whilst at least 70% of the effect of NO_2 in the original studies is independent of $PM_{2.5}$, it remains unclear to what degree NO_2 represents the effect of primary particles (or other traffic pollutants). This is because NO_2 concentrations are very closely correlated with traffic pollutants. For burden calculations, the total effect on mortality would be the same if NO_2 was acting as an indicator of other traffic pollutants and these other pollutants were present in London in the same proportions as in the original studies.

Apportionment of mortality burden by emissions source (section 2.3.1): The concentrations of pollutants derived from specific sources was modelled or estimated by difference. These concentrations were then used to calculate mortality burden as above. Transboundary $PM_{2.5}$ from outside London makes the largest contribution to the mortality burden of that pollutant, underlining the importance of national and European action to tackle air pollution sources. The largest contribution to the mortality burden of NO_2 is from sources within London (both road transport and other sources). Sources of NO_2 from outside London also make a significant contribution. As the sources of $PM_{2.5}$ within London make a less significant contribution to the mortality burden, it is clearly appropriate to focus on the mortality burden of NO_2 when designing policies to tackle local sources in London.

Life-expectancy from birth (section 2.1.4.3 and 2.1.5.2): The mortality burden can also be expressed as a loss of life expectancy from birth. This is calculated by assuming exposure to 2010 concentrations for a lifetime, for those born in 2010. This gives an average, some people will be unaffected and others will lose more. Adding these results together is not recommended as it is unknown whether or not the same people are affected by both $PM_{2.5}$and NO_2. Results are given in Table E2.

Table E2 Average loss of life-expectancy for those born in 2010, exposed to 2010 concentrations for a lifetime[11]

Pollutant	Male average loss of life expectancy	Female average loss of life expectancy
Anthropogenic $PM_{2.5}$	Around 9.5 months (294 days)	Around 9 months (270 days)
NO_2 (less certain) (30% overlap with $PM_{2.5}$)	*Up to around 17 months (515 days)*	*Up to around 15.5 months (468 days)*

Life year benefits of a sustained 1 μg m^{-3} reduction in $PM_{2.5}$ and NO_2 (section 2.1.4/2.1.5): As an abstract example of a potential policy reduction sustained until 2114[12], the mortality impact of a 1 μg m^{-3} reduction in $PM_{2.5}$ in 2010 was calculated. As this was a change, a full life table approach was used. The total results over the time period are given in Table E3. To put the results in context, note that this is for the whole population, followed up for 105 years, including new birth cohorts, which gives a total of over a billion life years lived. To compare

[11] Results for both $PM_{2.5}$ and NO_2 calculated using life tables with mortality rates and population based on an average for 2009/10/11 as a starting point and using the EPA recommended lag (COMEAP, 2010)

[12] This captures the full change in life years as the benefits are not realized immediately. It is equivalent to a policy reduction being sustained over time and then remaining as part of the policy baseline after further policies are implemented. It includes benefits to those born at a later date.

with the life years lost for no reduction in 2010 levels of $PM_{2.5}$ and NO_2 for a lifetime, see footnote 22. For the burden calculations, the modelled ambient concentration of NO_2 is higher than for $PM_{2.5}$. When the concentrations are the same, as in this example, the result for NO_2 is smaller.

Table E3 Life years gained across the population as a result of a 1 μg m^{-3} reduction in pollutant sustained 2010-2114

Pollutant	Life-years gained across the population from 2010-2114
Anthropogenic $PM_{2.5}$	573,145 (97,882 - 1,114,618)
NO_2 (less certain) (30% overlap with $PM_{2.5}$)	376,334 (214,064 - 535,961)
Total[13]	573,145 *up to 949,479*

ES4 Estimating the impact of short-term exposure to $PM_{2.5}$ and NO_2 in London (section 2.2)

Concentration-response functions from WHO (2013b) have been used to estimate the impact of short-term exposure to $PM_{2.5}$ and NO_2 on deaths brought forward[14] and hospital admissions using methods based on COMEAP (1998). The results are given in Table E4[15]. WHO recommended that the results for $PM_{2.5}$ and NO_2 can be added together, although only the NO_2 recommendations comment directly on the robustness to adjustment for other pollutants.

Table E4 Numbers of deaths brought forward and hospital admissions associated with short-term exposure to $PM_{2.5}$ and NO_2 in 2010[a]

Pollutant	Deaths brought forward[b]	Respiratory hospital admissions	Cardiovascular hospital admissions
Anthropogenic $PM_{2.5}$	787 (287 to 1,288)	1992 (-188[c] to 4,232)	740 (138 to 1,352)
NO_2	461 (273 to 650)	419 (-223[c] to 1,064)	-[d]

[a] Numbers in brackets represent the result for the 95% confidence interval around the concentration-response coefficients, representing statistical uncertainty.

[b] The estimated deaths brought forward should not be added to the deaths from long-term exposure. Results for total $PM_{2.5}$ and for PM_{10} are available in the main report.

[c] Negative values for the lower confidence intervals are regarded as indicating that the confidence intervals include the possibility of no effect not that air pollution has a beneficial effect.

[d] WHO did not recommend quantification of effects of NO_2 on cardiovascular admissions.

[13] These numbers are illustrative because if the change in risk from changes in $PM_{2.5}$ and NO_2 concentrations had been put into the same life table the answer would be different to some extent. (The risks from each pollutant would change the population size and age distribution which in turn would influence the effect of the other pollutant.)

[14] Deaths brought forward is a term used because short-term exposure studies may only reflect deaths brought forward by too short an amount of time to change the annual death rate, the design cannot determine this (COMEAP, 1998).

[15] The deaths brought forward as a result of short-term exposure to NO_2 are more certain than the results for long-term exposure and should therefore be regarded as an alternative result for numbers of attributable deaths.

Apportionment by emissions source (section 2.3.3): Around half of the deaths brought forward and respiratory hospital admissions due to short term exposure to NO_2 and $PM_{2.5}$ in London can be associated with $PM_{2.5}$ from sources outside London. Exposure to NO_2 makes a significant contribution, with the majority of these being associated with London sources. 75% of the cardiovascular hospital admissions associated with $PM_{2.5}$ result from sources outside London.

ES5 Trends in NO_2 and $PM_{2.5}$ concentrations in London and associations with health and mortality (section 3)

Changes in concentrations are best analysed with life tables and need to be followed up for a lifetime (105 years) to capture the full life years lived in those benefiting from reductions in pollution. For recent trends, the life years when modelled population weighted-mean concentrations remained the same as in 2008 for 105 years were compared with the life years for the modelled change in levels of pollution in 2010 and 2012[16], with levels in 2012 then remaining unchanged until 2112.

Long-term impact of concentration changes 2008-2012:

The modelled population-weighted annual mean concentrations of anthropogenic **$PM_{2.5}$** increased slightly from 2008 to 2010, decreasing to 2012 albeit still above that in 2008[17]. For **NO_2** there have been ongoing reductions in the modelled population-weighted annual mean concentrations since 2008[18].

The **$PM_{2.5}$** changes from 2008-2012 led to around 478,414 life years lost across the population followed up to 2112 (the minimum total result, if did not include NO_2).

However, this was offset by the ongoing reductions in **NO_2** from 2008-2012, giving up to around 1,062,063 life-years gained, assuming some overlap with the effects of $PM_{2.5}$.

Acknowledging the greater uncertainty in the effects of long-term exposure to NO_2, the net effect of 2008-2012 trends in both **$PM_{2.5}$ and NO_2**, assuming a 30% overlap, would be up to 583,649 life years gained[19].

Short-term impact of concentration changes 2008-2010[20]:
Estimates of respiratory hospital admissions in London associated with anthropogenic **$PM_{2.5}$:**

[16] Modelled concentrations were available for 2008, 2010 and 2012. The concentrations for 2009 and 2011 were assumed to be the same as the previous year.
[17] The year 2008 and 2010 were fully validated and modelled using their respective meteorology data, i.e. 2008 and 2010, while the year 2012 was projected forward from 2010 using the LAEI2010 and the most recent meteorology, i.e. 2010. Emissions were based on LAEI2010.
[18] The 2010 population used for population-weighting was larger than in 2008 but the trend from 2010 to 2012 is solely pollution derived as the 2010 population was used for weighting in both cases.
[19] See footnote 13.
[20] Deaths brought forward not included for $PM_{2.5}$ to avoid double-counting with long-term exposure and mortality. They are included for NO_2 so that they can be added to core summaries that exclude long-term exposure to NO_2 and mortality.

- increased from 1,658 in 2008
- to 1,992 in 2010,
- before declining to 1924 in 2012.

Similarly, estimated cardiovascular hospital admissions:

- increased from 654 in 2008
- to 740 in 2010
- before declining to 715 in 2012.

Deaths brought forward, as a result of short term exposure to **NO_2** (not to be included if effects of long-term exposure to NO_2 included)**:**

- declined from 499 in 2008,
- to 461 in 2010
- to 439 in 2012.

NO_2 associated respiratory hospital admissions in London:

- increased from 399 in 2008
- to 419 in 2010 (despite a decline in concentration, due to an increase in the population and baseline rate,)
- but declined again to 398 in 2012.

WHO did not recommend quantification of NO_2 and cardiovascular hospital admissions.

Long-term impact of projected concentration decreases 2010-2020:

The life years lived when pollution remained at 2010 levels for the next 105 years were compared with the life years lived for the projected changes in pollution for 2012, 2015 and 2020, with 2020 concentrations being maintained until 2114. Over this time period, population-weighted annual mean concentrations decline for both $PM_{2.5}$ and NO_2[21].

For anthropogenic **$PM_{2.5}$,** these projected changes would result in a gain of 901,466 life-years across the population followed up to 2114 (the minimum total result) compared with pollution remaining at 2010 levels[22].

For **NO_2** the predicted gain of up to 2,919,741 life years assuming a 30% overlap with $PM_{2.5}$ was substantially larger, although less certain.

The overall total could therefore be as much as 3,821,207 life years if these results are added to those from $PM_{2.5}$[23].

Short-term impact of projected concentration decreases 2010-2020[24]:

[21] These projected declines are driven entirely by the modelled concentrations, as the 2010 population was used for population-weighting in all cases.

[22] For context, leaving 2010 levels unreduced for 105 years compared with no anthropogenic $PM_{2.5}$ is estimated as leading to 7,853,982 life years lost. The equivalent, more uncertain result for not reducing 2010 levels of NO_2 is estimated as up to 13,677,155 life years lost.

[23] See footnote 13.

[24] 2010 populations and baseline rates were used in all future years.

For **$PM_{2.5}$**, respiratory hospital admissions in London were projected to:

- decrease from 1924 in 2012,
- to 1854 in 2015,
- to 1749 in 2020.

Similarly, cardiovascular hospital admissions were projected to:

- decrease from 715 in 2012,
- to 689 in 2015,
- to 650 in 2020.

For **NO_2**, deaths brought forward (not to be included if effects of long-term exposure to NO_2 included):

- declined from 439 in 2012,
- to 413 in 2015
- to 355 in 2020.

Respiratory hospital admissions in London were projected to:

- decrease from 398 in 2012,
- to 375 in 2015,
- to 323 in 2020.

ES6 Economic understanding of the costs of $PM_{2.5}$ and NO_2 in London

London specific damage costs[25] (£/tonne) for PM and NO_x transport emissions (section 4.1)[26]

The effect of a 10% reduction in transport emissions in central, inner or outer London on $PM_{2.5}$, PM_{10} and NO_2 concentrations across the whole of London in 2010 was modelled and the population-weighted concentration used to calculate health impacts, which were then valued in monetary terms. All values were updated to 2014 prices. The values were divided by the emissions change to give a cost per tonne (known as a damage cost), which can be used to approximately scale the economic benefits of emission changes. Damage costs were produced for a core set of quantified health outcomes following IGCB (2007) updated to include recommendations from COMEAP (2010) and results from a Department of Health funded systematic review (Atkinson et al., 2014; Mills et al. 2015). These were similar to the WHO core set. Damage costs were also produced for an extended set of quantified health outcomes that were more uncertain, as recommended for the WHO extended set.

$PM_{2.5}$ (core): Using the COMEAP (2010) recommended coefficient and lag profile, the life years gained were estimated by applying the pollutant reduction for 2010 only and following through

[25] Damage costs reflect the health impact of a tonne of emissions of a particular pollutant, expressed in monetary terms. They value impacts from the perspective of social welfare, and capture the wider costs to society as a whole (the environmental, social and economic impacts). For health impacts, this includes analysis of resource costs, opportunity costs and dis-utility.

[26] The damage costs exclude the effects of NO_X emissions on ozone (local and regional). The values excluded non-health impacts (materials) from PM and NO_X. Central estimates given here, with sensitivities in the main report. IGCB – Interdepartmental Group on Costs and Benefits of Air Quality. HMT Her Majesty's Treasury.

the impact over 105 years (as IGCB, 2007). These were then valued as life years lost, using the IGCB value in the Defra guidance (Defra, 2013). Future values of life years lost were calculated using Government guidance on uplift and discounting[27]. The valuation of respiratory and cardiovascular hospital admissions used the IGCB values (Defra, 2013). An adder was included to take account of the effects of London emissions on regional (UK) pollution. This was based on the rural damage cost values in the Defra damage costs (2011)[28]. The core PM damage costs were £125,329, £157,794 and £90,466 per tonne of emissions for central, inner and outer London respectively.

NO_x (core): Coefficients for NO_2 and deaths brought forward and respiratory hospital admissions from the Department of Health commissioned systematic review (Mills et al., 2015) were used (also used for the HRAPIE recommendations, limited set). The valuation of hospital admissions and deaths brought forward were undertaken using the IGCB values in the Defra guidance (Defra, 2013), updated to 2014 prices. The impacts of the London NO_2 contribution to regional (UK) nitrate as secondary $PM_{2.5}$ was included, using the secondary PM component of the Defra NO_X damage costs (Defra, 2011b). The core NO_x damage costs were £884, £910 and £861 per tonne for central, inner and outer London respectively.

PM_{10}/$PM_{2.5}$ (extended)[29]: WHO (2013b) included recommendations for other health outcomes with greater uncertainty[30]. The level of uncertainty varies but the coefficient may be based only on a single old study, or be a sensitivity 'in case' the overall conclusion of no effect is not correct. This is fine for screening proposals but would need detailed consideration when a proposal is analysed in full. Damage costs of £22,395, £27,598 and £14,224 per tonne (PM_{10}) and of £118,360, £152,884 and £79,540 ($PM_{2.5}$) each for central, inner and outer London respectively can be added to the central, inner and outer London core PM damage costs to reflect this.

NO_x (extended): This included damage costs for the analysis of mortality from long-term exposure to NO_2, with the 30% reduction to reduce double counting with $PM_{2.5}$ (based on several studies but hard to separate from other traffic pollutants), and also the effect of long-term exposure to NO_2 and prevalence of bronchitic symptoms in asthmatic children (based on one good study). The extended NO_x damage costs were £39,442, £52,344 and £27,948 per tonne for central, inner and outer London respectively for adding to the central, inner and outer London core NO_X damage costs.

[27] Future values were increased at 2% per annum, then discounted using the declining discount rate scheme in HMT Green Book (2011).

[28] Aligned to COMEAP (2010) assumptions.

[29] Valuation of endpoints was based on new valuation estimates (Watkiss and Hunt, forthcoming), updating previous values from CAFE (Hurley et al., 2005).

[30] PM_{10} related impacts from: infant mortality; asthmatic symptoms in asthmatic children; prevalence of bronchitis in children; incidence of adult bronchitis; and $PM_{2.5}$ related impacts from restricted activity days (avoiding overlap with hospital admission days), and bronchitis in children). The calculation method is given in the full report.

Current costs of $PM_{2.5}$ and NO_2 exposure in London (section 4.2)

The monetary values for life-years, hospital admissions and deaths brought forward were applied to the quantified effects of long- and short-term exposure to $PM_{2.5}$ and NO_2 in 2010 in London summarized earlier. A new method was developed to value the mortality burden by creating a profile of baseline life years over time for each five year age group and gender. These were multiplied by the appropriate future monetary values for a life year lost to give a weighted value-of-a-life-year (VOLY) for each gender and 5 year age group. These life years lost in each subgroup were then valued and summed to give the overall economic costs of the mortality burden.

The estimated annual monetised costs of air pollution related mortality for long-term exposure to **$PM_{2.5}$** (2010) for London was £1,358 million (in 2014 prices) with an additional £14 million for respiratory hospital admissions and £5 million for cardiovascular hospital admissions. The potential estimated annual costs of mortality from long-term exposure to **NO_2** (2010) for London (30% overlap with $PM_{2.5}$) was up to[31] £2,273 million (in 2014 prices), with an additional £3 million for respiratory hospital admissions.

The estimated annual costs across **both pollutants** ranges from a core result of £1,383 million (including all the hospital admission effects of $PM_{2.5}$, plus respiratory hospital admissions and deaths brought forward (£3 million) from short-term exposure to NO_2) to an extended result of £3,653 million, including long-term exposure to NO_2 and mortality[32]. The latter is the only outcome from the extended set of outcomes included. Inclusion of the other extended outcomes such as, for example, restricted activity days would increase the economic costs but further work is needed to consider this in detail so that the varying levels of uncertainty for each outcome can be fully described and their plausibility discussed.

Ready reckoner for use of transport emission damage costs (section 4.3)

A ready reckoner was produced to provide TfL with a simple emission-based tool to estimate the economic benefits of proposed road transport policies for London. The changes in transport emissions are input to a series of core and extended 'adder' spreadsheets and multiplied by the London specific damage costs to give a total present value (£). It can be used to scope the economic benefits of new proposals for policies that affect road transport emissions in London, at the early stage of new policy development.

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1 Introduction

1.1 Rationale and outline methodology

This report builds upon previous work (Miller, 2010), quantifying the impacts of exposure to fine particles on mortality in London. Transport for London (TfL) commissioned the current work as a more comprehensive health study in London, incorporating nitrogen dioxide (NO_2) and other pollutants that have an impact on health. Our ability to improve health impact assessments has been facilitated by research at the European level such as the World Health Organisation's (WHO) Review of evidence on health aspects of air pollution (REVIHAAP Project) published in 2013 (WHO, 2013a) and the subsequent WHO Health risks of air pollution in Europe (HRAPIE) project, recommending concentration-response functions for use in cost-benefit analysis (WHO, 2013b).

The Greater London Authority (GLA) and TfL require a more robust and accurate understanding of the economic cost of exposure to air pollution and associated public health impacts in London to support the economic case for action to improve air quality based on the cost:benefit ratio.

Work in this area requires good datasets and experience across disciplines. These include London's "gold standard' emissions inventory (LAEI), access to high quality air pollution modelling, knowledge of air quality policy in London, experience of environmental economic analysis and knowledge of the development of health impact assessment methods for the UK (COMEAP, 1998, 2010) and the WHO (REVIHAAP and its sister project HRAPIE) (WHO, 2013a, 2013b), as well as the underlying epidemiology.

1.2 Summary of health impact methods

The report uses methods based on those recommended by the Committee on the Medical Effects of Air Pollutants (COMEAP) and WHO, but within that framework have applied the most comprehensive analysis methods possible within the time available. Examples of these include:

- In section 4, inclusion of impact estimates based upon COMEAP recommendations as well as new revisions to the NO_2 risk estimates (HRAPIE), a crucial step for developing the 'ready reckoner', given the importance of NO_2 for London in the coming years.
- In estimating the effects of short-term exposure to particles and NO_2 in London, the report used (i) current COMEAP recommendations for PM_{10} and (ii) more up to date concentration-response functions for $PM_{2.5}$ and NO_2 from HRAPIE. The approach was to multiply the coefficients recommended by COMEAP or HRAPIE by the 2010 population-weighted annual average concentrations of PM_{10}, $PM_{2.5}$ and NO_2 in London, with the baseline rates for all age, all-cause mortality (excluding external causes) and for all age emergency respiratory and cardiovascular hospital admissions.
- The report assessed the economic costs of the health impacts of air pollution in London - building on the health burden calculations - and also produced marginal emission runs (10% decreases in transport emissions in central, inner and outer London) to derive new London-specific damage costs (£/tonne estimates).

This study therefore delivers an updated and state-of the art analysis of the health impacts in London, and provides a set of tools that will help TfL in future policy analysis.

During the course of the project a meeting was arranged to present the methods to a panel of public health experts and to consider opinions regarding what aspects were most valuable at the local level.

1.3 Definitions used in this document

A glossary is provided in Annex 11. However, some different terms are highlighted here to avoid potential confusion. The terms are commonly used in air pollution health impact assessment but may not be familiar to all readers.

The term **burden** is used for approximate 'snapshot' calculations of the health effects of the total amount of man-made air pollution in a particular year. The term is used generally for the total burden of a particular disease but in this context is applied to approximate calculations of deaths and life years lost from long-term exposure to air pollution (COMEAP, 2010). The term '**attributable deaths**' is used for the deaths calculated in this way. The intention of these calculations is to give a flavour of the rough overall effect of air pollution and for the calculations to be simple to do.

The term **impact** is used in UK air pollution health impact assessment for calculations of the health effects of changes in air pollution, such as those that might follow implementation of policies to reduce emissions of air pollutants. The calculations are generally more precise, for example in using life tables to take into account changes in health effects over time as a result of long-term exposure. Effects on mortality are expressed in terms of life years lost or gained (a more appropriate measure in the long-term as everyone dies eventually but may die earlier or later). A **life year** is one year lived for one person, usually added up over a population and over time.

Health effects of air pollution can arise from short- or long-term exposure. The knowledge of the effects of short-term exposure comes from studies of day to day changes in concentrations of air pollution. Studies showing effects on hospital admissions are of this type. Where this type of study finds increases in numbers of deaths, the deaths are described as '**deaths brought forward**' (COMEAP, 1998). This term relates to the fact that this type of study cannot distinguish whether the deaths are brought forward by only a short amount of time, and would not result in changes to the annual death rate, or whether the deaths are brought forward by a longer time.

1.4 Specification

The specification contained the following tasks:

- Updating estimates of the mortality burden and mortality impact of air pollution in London in 2010 (effects of long-term exposure to $PM_{2.5}$ and NO_2 including effects by London boroughs).

- Estimating the impact of air pollution on hospital admissions and deaths brought forward in London (effects of short-term exposure to PM_{10}, $PM_{2.5}$ and NO_2 in London).

- Apportionment of hospital admissions, death brought forward and mortality impacts of air pollution to broad sources (estimating the proportion of the effects of both short- and long-term exposure that are due to various sources e.g. road traffic, rural background etc).

- Understanding recent trends and the future impact of air pollution in London on health. Work on distinguishing the effects of meteorology from the effects of emissions changes in driving recent monitored trends will be reported separately.

- Developing a robust economic understanding of the costs of air pollution in London (defining updated London specific damage costs and the economic costs of the burden of air pollution in London).

- Developing a ready reckoner to help estimate health impacts of future policies (an Excel spreadsheet tool for use by TfL in screening future policies).

2 Health burden of 2010 levels of $PM_{2.5}$ and NO_2 in London

2.1 Updating estimates of the mortality burden and mortality impact of $PM_{2.5}$ and NO_2 in London

2.1.1 Background

Since the report by Miller (2010) that estimated the mortality impacts of particulate air pollution in London, COMEAP has published a report (COMEAP, 2010) on the mortality effects of long-term exposure to particulate pollution in the United Kingdom, which includes a detailed discussion of methodology and interpretation of the results of both the burden from the overall level of pollution and impact calculations of the health effects of changes in pollution. Both the burden and the impact methods applied in this report are closely related to those of Miller (2010) and consistent with the updates recommended by COMEAP (COMEAP, 2010, 2012) and those described in the recent Public Health England (PHE) document, Gowers et al. (2014), on the public health outcome indicator.

Whilst this study refined aspects of the method and updated the input data, the calculations for the burden of $PM_{2.5}$ followed previously established methods. The calculations for the mortality burden attributable to NO_2 were analogous to the $PM_{2.5}$ methods and used coefficients recommended by the WHO HRAPIE project (WHO, 2013b). However, the method to implement one aspect of the HRAPIE recommendations was not clear cut and required further discussion (Annex 1). The HRAPIE table of recommendations states that the concentration-response relationship should only be applied above 20 μg m^{-3}. This was aimed at calculations for small concentration reductions for policy analysis. Methods for burden calculations were not discussed. For burden calculations there is a need to discuss appropriate counter-factuals i.e. what is the baseline with which to compare the effects of current levels of pollution[33]? Counter-factuals can be chosen to be the lower end of the area of the concentration-response function with tight confidence intervals; the lowest concentration in the studies used to define the concentration-response function; the lowest concentration achievable with realistic policies; the lowest concentration occurring in the environment; or a zero concentration. One interpretation is to take 20 μg m^{-3} as the lower end of the area of the concentration-response function with tight confidence intervals (the justification used by HRAPIE for only quantifying concentration changes above 20 μg m^{-3}). However, some recent studies show tight confidence intervals down to lower concentrations and this is not the only option for choosing a counter-factual. Another option is to use the 5th percentiles or minimum concentrations in the key epidemiological studies (as for $PM_{2.5}$ in the Global Burden of Disease calculations, Lim et al., 2012). This would suggest using a counter factual at 10, 5 μg m^{-3} or 1.5-2 μg m^{-3} NO_2 (see Annex 1). Calculations down to zero and down to 20 μg m^{-3} were undertaken to encompass the range of possible assumptions, and we consider a counter factual towards the lower end of this range to be most appropriate.

[33] This question did not arise for $PM_{2.5}$ as COMEAP recommendations to concentrate on anthropogenic $PM_{2.5}$ are equivalent to using the level of non-anthropogenic $PM_{2.5}$ as a counter-factual.

2.1.2 Mortality burden from 2010 concentrations of $PM_{2.5}$ and NO_2 in London – input data and method

2.1.2.1 Processing of Input data

Modelled concentrations: $PM_{2.5}$ and NO_2 annual mean concentrations at 20m grid resolution were extracted from the LAEI2010[34] year 2010 air quality results and intersected with the latest (2011) Output Area (OA) layer from the Office of National Statistics (ONS) for the Greater London area (a total of 25,053 OAs). Each concentration grid point within each OA was further averaged at OA level, borough or Greater London area. A short description of the King's model used to predict all the annual mean concentrations used in this report can be found in Annex 4.

Anthropogenic source: PM source apportionment analysis was undertaken and measurement of Cl^- was used to trace sea salt, assuming it is NaCl only. Cl^- is not measured in $PM_{2.5}$, instead concentrations in PM_{10} were factored using size information from earlier studies in London (Davy, 2014). The annual mean contribution of sea salt within $PM_{2.5}$ was estimated to be 0.55 µg m^{-3} in 2010 and was removed from the total $PM_{2.5}$ concentration to generate anthropogenic $PM_{2.5}$ concentrations; consistent with EU guidance (European Commission, 2011).

Total NO_2 concentration was used as the state of knowledge (European Commission, 2011) does not allow for a natural part of NO_2 to be measured or quantified.

Population data: The population data was downloaded from ONS[35]. As the deaths had been averaged over 3 years the same was done for the population. The population data was given by single year of age at OA level and has been further averaged for 2009/2010/2011 to represent 2010. The population was summed by gender and 5 year age groups for aged 30 and above for each OA, each borough and for London overall. The American Cancer Society study (Pope et al. 2002) studied those aged 30 and above to derive the coefficient for $PM_{2.5}$ and this age span was also recommended for NO_2 by HRAPIE. The division of population by gender and 5 year age groups matched the deaths data.

Deaths data: The deaths data was extracted from ONS data by the PHE London Knowledge and Intelligence Team. The deaths data were given by 5 year age groups, averaged for 2009/2010/2011 at London borough level. This is taken to be a figure for 2010 with the random year-to-year variability in age groups with small numbers of deaths stabilised by averaging with the surrounding years.

Relative Risk (RR) for $PM_{2.5}$: A relative risk of 1.06 (COMEAP plausibility interval 1.01 to 1.12) has been used for the change in mortality as a result of long-term exposure to $PM_{2.5}$ derived from the American Cancer Society Study (Pope et al. 2002) and recommended for use in the UK by COMEAP (2010).

Relative Risk (RR) for NO_2: A relative risk of 1.055 (95% CI 1.031 - 1.080) for the change in mortality as a result of long-term exposure to NO_2 derived from a meta-analysis of the relevant

[34] http://data.london.gov.uk/datastore/package/london-atmospheric-emissions-inventory-2010

[35] http://www.ons.gov.uk/ons/about-ons/business-transparency/freedom-of-information/what-can-i-request/published-ad-hoc-data/pop/november-2013/index.html

studies by Hoek et al. (2013) was recommended by the WHO HRAPIE project (WHO, 2013b) but the HRAPIE report noted a potential overlap with the effects of $PM_{2.5}$ of up to around 30%[36]. We therefore used a relative risk of 1.039 (95% CI 1.022 – 1.056) derived by reducing the RR of 1.055 by 30%, with the original RR of 1.055 as an alternative. Note that downward adjustment by 30% to account for overlap with $PM_{2.5}$ does not remove any potential overlap with effects of other traffic pollutants.

Table 1 Concentration-response relationships for long-term exposure to $PM_{2.5}$ and NO_2

Pollutant	Relative Risk (RR)	Upper and lower plausibility (95% confidence interval for NO_2)
$PM_{2.5}$	1.06	1.01 / 1.12
NO_2	1.055	1.031 / 1.08
NO_2	1.039*	1.022 / 1.056

* Reduced from 1.055 by 30% to account for possible upper limit of overlap with $PM_{2.5}$

Lag: As burden calculations are intended as an approximate snapshot, they do not incorporate consideration of lags between exposure and effects. This can be regarded as either an assumption of no lag, or an assumption that air pollution concentrations have been stable at the same level for a sufficient time for the lag to be unimportant. Neither is strictly true but was regarded by COMEAP as an acceptable approximation in the context of producing simple, rough figures. This is discussed in COMEAP (2010).

Population-weighted average concentration ($PM_{2.5}$): The modelled concentration for each OA was multiplied by the population aged 30+ by gender and 5 year age group[37] in each OA. The result of this multiplication was then summed over all OAs in the borough and divided by the relevant gender and 5 year age group borough population to give the population-weighted average concentration for that gender and age group in the borough. The population-weighted average concentration for that gender and age group[38] was then used in the mortality burden calculations. Population-weighting was done at OA level as this was the smallest area for which population data was available. Where a single population-weighted average concentration for a borough is given in a table (for simplicity), this was produced by weighting across 5 year age groups above 30 years and across gender.

Population-weighted average concentration (NO_2): Population-weighting proceeded as for $PM_{2.5}$ for the lower bound counter factual of zero. For the sensitivity analysis, assuming a counter-factual of 20 $\mu g\ m^{-3}$, this value of 20 was subtracted from the 20 x 20 m grid concentrations in each OA and the difference was then averaged up to OA level to be used in

[36] HRAPIE recommended up to 33%. We used 30% to reflect the approximate nature of our knowledge of the size of the overlap.

[37] The upper age group was aged 85+.

[38] Available from the authors on request.

the population-weighting. For example, for a 20 x 20 m grid concentration of 30 μg m^{-3}, a concentration of 10 μg m^{-3} (30-20) was used in subsequent averaging up to OA level and then population weighting by gender and 5 year age group. Any 20 x 20 m grid concentrations that were negative after subtracting 20 μg m^{-3} were set to zero. This approach will not give the same result as subtracting 20 μg m^{-3} from the average at OA level. The best approach depends on exactly what was done in the original epidemiological studies. This is not always clear and, in any case, studies with different approaches have been pooled in the meta-analysis.

2.1.2.2 Calculations

The calculations followed COMEAP (2010) and Gowers et al. (2014). The relative risk (RR) per 10 μg m^{-3} was scaled to a new relative risk for the appropriate population-weighted average concentration (anthropogenic for $PM_{2.5}$) for each borough. The equation used (for the example coefficient of 1.06) was:

$RR(x) = 1.06^{x/10}$ where x is the population-weighted average concentration of interest (weighted by the relevant gender and 5 year age group aged above 30).

The new RR(x) was then converted to the attributable fraction (AF) using the following formula:

AF = (RR-1)/RR multiplied by 100 to give a percentage.

The attributable fraction was then multiplied by the number of deaths in the relevant gender and 5 year age group aged 30+ to give the number of attributable deaths.

The attributable deaths were then summed across the 5 year age groups above aged 30, for both males and females, to give a total for the borough. The attributable fraction for the borough is a weighted average across gender and age group. The attributable fraction for London overall was back calculated from the attributable deaths summed across boroughs divided by the total deaths in London.

To calculate the loss of life years associated with these deaths, the deaths and population data were input into the South East Public Health Observatory (SEPHO) Life Expectancy Calculator http://www.sepho.org.uk/viewResource.aspx?id=8943. This provides the expected remaining life expectancy for specified 5 year age groups. This was calculated separately for males and females[39]. (Note that this is the baseline life expectancy, representing how much an average person of that age group would have been expected to live if it had not been for the attributable deaths.) The relevant values for expected remaining life expectancy in an age group were then multiplied by the number of attributable deaths to estimate the total life years lost.

The calculations above were done at the borough level and the results for deaths and life years summed to give a total for London. This allows different death rates in different boroughs to influence the results. The use of population-weighting across the whole of London requires an assumption that the death rate is the same across London. A 'summary' attributable fraction

[39] Available from the authors on request.

for London was derived by inferring the attributable fraction that would have led to the results for attributable deaths that were totalled across the boroughs.

The process was repeated for the lower and upper confidence intervals around the relative risks and for both the full and the 30% reduced relative risks for NO_2.

Calculations were also done for the loss of life expectancy for those born in 2010 and exposed to $PM_{2.5}$ and NO_2 for a life time. The methods and results for this are given in section 2.1.4.3 and 2.1.5.2.

2.1.3 Mortality burden (attributable deaths and associated life-years) from 2010 concentrations of $PM_{2.5}$ and NO_2 in London - results

2.1.3.1 $PM_{2.5}$

The mortality burden of 2010 levels of anthropogenic $PM_{2.5}$ in London in 2010 was calculated as 52,630 (9,287 – 98,648) life-years lost[40], equivalent to 3,537 (range 624-6,632) attributable deaths at typical ages. The fraction of total mortality attributable to $PM_{2.5}$ across London was 7.6%.

The above figures are derived from summing the results in individual London boroughs. Results for the attributable fraction (the percentage of mortality attributable to $PM_{2.5}$) varied from 9.9% in the City of London to 7.1% in Havering. Taking into account the underlying mortality rate and the size of the population as well, the attributable deaths varied from 4 in the City of London to 182 in Bromley and the life years lost from 60 in the City of London to 2423 in Croydon. The rankings are not necessarily the same because the attributable fraction is derived directly from the population-weighted average concentration whereas the attributable deaths and life years lost are also affected by the mortality rate and age distribution in the boroughs. The results for the boroughs, using the central estimate, are given in Table 2, with the results for sensitivity analyses using the COMEAP plausibility intervals of 1% and 12% in Annex 2 Table 26. The sensitivity analysis results are roughly from a sixth to twice the results given in Table 2.

Table 2 London population, modelled population-weighted average concentration ($\mu g\ m^{-3}$) and estimated burden of effects on annual mortality in 2010 of 2010 levels of anthropogenic $PM_{2.5}$, using COMEAP's recommended concentration-response coefficient of a 6% increase in mortality per 10 $\mu g\ m^{-3}$ $PM_{2.5}$

Borough	Population* ($x10^3$)	$PM_{2.5}$ PWAC** ($\mu g\ m^{-3}$)	Baseline deaths	Attributable fraction (%)	Attributable deaths***	Life years lost
City of London	5.2	17.9	40	9.9	4	60

[40] These are the numbers of years across the population expected to be lived over time if the deaths to which particulate pollution contributed had not occurred.

Barking and Dagenham	98.3	13.2	1247	7.4	92	1268
Barnet	208.3	13.2	2353	7.4	174	2360
Bexley	142.3	13.0	1814	7.3	132	1772
Brent	171.3	13.7	1477	7.7	112	1855
Bromley	199.8	12.9	2524	7.2	182	2379
Camden	125.1	15.1	1085	8.4	91	1568
Croydon	212.0	13.2	2349	7.4	173	2423
Ealing	194.0	13.5	1867	7.6	142	2175
Enfield	177.3	13.0	1897	7.3	138	1944
Greenwich	141.2	13.6	1584	7.6	120	1659
Hackney	126.1	14.5	1015	8.1	82	1429
Hammersmith and Fulham	102.5	14.5	882	8.1	71	1166
Haringey	142.9	13.7	1106	7.7	85	1472
Harrow	142.4	12.8	1386	7.2	100	1544
Havering	150.1	12.6	2126	7.1	150	1968
Hillingdon	157.0	12.7	1754	7.1	125	1788
Hounslow	143.3	13.4	1362	7.5	102	1564
Islington	111.9	15.3	1035	8.5	88	1394
Kensington and Chelsea	101.5	15.1	803	8.4	67	1119
Kingston upon Thames	95.3	13.1	1000	7.4	74	1008
Lambeth	165.4	14.4	1360	8.1	109	1797
Lewisham	157.0	13.9	1552	7.8	120	1773
Merton	119.9	13.5	1143	7.6	86	1259
Newham	144.5	14.0	1221	7.9	96	1572
Redbridge	157.6	13.3	1716	7.4	128	1799
Richmond upon Thames	120.7	13.3	1131	7.4	84	1238

Southwark	157.7	14.9	1338	8.3	111	1793
Sutton	118.7	13.1	1391	7.3	102	1367
Tower Hamlets	118.6	15.5	983	8.7	85	1314
Waltham Forest	141.6	13.5	1356	7.6	102	1546
Wandsworth	173.4	14.1	1513	7.9	119	1686
Westminster	133.6	15.5	1071	8.6	92	1570
Total					3537	52,630

*Population and death rate, age 30+, based on 2009/2010/2011 average.

**PWAC - Population Weighted Average Concentration of $PM_{2.5}$, calculated for males and females and 5 year age groups separately, weighted average presented here.

***Attributable deaths and associated life years lost, age 30+ and calculated by 5 year age groups and gender.

2.1.3.2 $PM_{2.5}$ comparison with previous results

The mortality burden results calculated here are compared with previous results in Table 3 and Table 4. The public health outcome indicator for the fraction of mortality attributable to particulate air pollution[41] is the official indicator as it is important that the impact of particulate air pollution can be compared across the country. The results presented here should be considered alongside this. The work reported here includes refinements in method as well as differences in input data, taking advantage of more up to date data and the availability of data at a greater level of detail in London. This section compares the results and the methods/input data with both the Gowers et al. (2014) report and the Miller (2010) report which provided a previous estimate of the mortality impact of particulate air pollution in London.

Compared with the Miller (2010) report, the current work has incorporated the following changes in inputs and methodology:

- Updating mortality data from 2008 to mortality data for an average of 2009/2010/2011.
- Use of the latest population data for an average of 2009/2010/2011 at OA level, rather than 'High' population projection data for 2008 at Ward level as used in Miller (2010).
- The Miller (2010) report presented results for Greater London, subdivided by Ward. Population-weighting was done at borough level. To maintain a flexible approach to the future geographic output requirements of the GLA, we undertook population-weighted average concentration calculations at OA level and then combined these to borough scale for application of the $PM_{2.5}$ and NO_2 mortality calculations.
- Updating of modelled $PM_{2.5}$ concentrations from 2006 (LAEI2006) to 2010 (LAEI2010).

[41] www.phoutcomes.info

- Use of population-weighted average concentrations by age, sex and 5 year age groups. Population-weighting was by total population in Miller (2010).
- The use of anthropogenic $PM_{2.5}$ concentrations where previously total $PM_{2.5}$ concentrations were used.
- Attributable death calculations were done by age, sex and 5 year age groups in the current report and by total population in Miller (2010).

The Miller (2010) report estimated that fine particles have an impact on mortality equivalent to 4,267 deaths in London in 2008, with a range of 756 to 7,965. This is larger than our estimate of 3,537 (range 624-6,632) by 730 deaths. The differences can be considered in 3 categories:

a) A genuine decrease in $PM_{2.5}$ from 2006 to 2010.
b) The exclusion of non-anthropogenic $PM_{2.5}$ in this report.
c) Methodological/input changes of various sorts combined together.

The decrease in total $PM_{2.5}$ from 2006 to 2010 was 1.07 $\mu g\ m^{-3}$ (the sea salt is assumed to be part of the total $PM_{2.5}$ in 2006 and 2010 that cancels out as it is unlikely to change much). Scaling this using an approximate deaths per $\mu g\ m^{-3}$ factor (taking no account of non-linearities or distribution of deaths by age) gives 268-276 deaths, or 37-38% of the 730 death difference, depending on assumptions for the approximate factor. The non-anthropogenic $PM_{2.5}$ excluded was 0.55 $\mu g\ m^{-3}$ of sea salt; using the recent method this would account for about 137 deaths, or 18.5% of the 730 death difference. The remainder (317-325 deaths, or 43.5-44.5% of the 730 deaths difference, depending on the scaling factor) is due to methodological or input differences. The overall baseline mortality rate went down, for example, and this report uses concentrations population-weighted by the population aged 30+ separately by gender and age group rather than the total population.

Thus, although the main part (combining reasons a and b) of the reason for this decline is the difference in the population-weighted average $PM_{2.5}$ concentration of 15.34 $\mu g\ m^{-3}$ (total, 2006) compared with 13.72 $\mu g\ m^{-3}$ (anthropogenic, 2010) used here, there are also reasons unrelated to the concentration difference. The rankings by borough are similar with the largest population-weighted average concentrations, smallest population and smallest number of attributable deaths in the City of London, the largest numbers of attributable deaths in Bromley and the smallest population-weighted average concentrations in Havering.

The PHE report (Gowers et al., 2014) is more recent so the dates for the input data were closer. The population and mortality data were averaged over 3 years in both reports, for 2008/9/10 in Gowers et al. and 2009/10/11 in our report. $PM_{2.5}$ modelling was for 2010 in both cases. The coefficients used and the basic underlying methodology were the same as both reports follow COMEAP (2010). However, there are still some methodological differences:

- The mortality data for 2008/9/10 in Gowers et al. (2014) was grouped in 10 year age groups to match that available nationally, whereas this report used the original 5 year age grouping in the mortality data for 2009/10/11. These different choices also meant

that Gowers et al. (2014) calculated attributable deaths age 25+ (lowest age group 25-34) whereas this report used age 30+.

- Population data for the life years lost calculations was for 2008/9/10 in Gowers et al. (2014) by 10 year age group and by 5 year age group for 2009/10/11 in this report.
- There were differences in the modelling approaches. Gowers et al. (2014) used the pollution climate mapping (PCM) model estimating concentration on a 1km x 1km grid square basis using information from the National Atmospheric Emissions Inventory (NAEI); this report used the London Air Quality Toolkit dispersion model (Annex 4) on a 20m x 20m grid basis using information from the LAEI.
- The definition of non-anthropogenic $PM_{2.5}$ differed – the Gowers et al. (2014) report subtracted sea salt and the residual from the PCM model that could not be allocated to known sources, for this report only sea salt was subtracted.
- The population-weighting in the Gowers et al. report was done on a 1km x 1km grid square basis and used the total population from the 2001 census (because some of the NAEI emissions are based on the 2001 census); this report used concentrations from 20m x 20m grid points averaged up to OA level and population-weighted separately by gender and 5 year age groups above 30+ from population data for 2009/10/11 (revised following the 2011 Census).

The PHE figures were similar but smaller - 41,404 life-years lost, equivalent to 3,388 attributable deaths at typical ages (Table 3 and Table 4) compared with 52,630 life-years lost, equivalent to 3,537 attributable deaths at typical ages in this report. This is probably mainly due to differences in estimated levels of anthropogenic $PM_{2.5}$ since these estimates were smaller in the PHE report (Table 3) whereas the baseline population (Table 3) and baseline numbers of deaths (Table 4) were larger. The differences in the estimated levels of anthropogenic $PM_{2.5}$ were lower in the PHE report due to a larger proportion of $PM_{2.5}$ being assumed to be non-anthropogenic but it may also have been influenced by the finer scale modelling in this report. It is not entirely clear whether or not the latter is an advantage – it will be more influenced by roadside sources (although this was lessened by subsequent averaging up to OA level) but the original studies modelled at a broad city wide scale. However, fine scale modelling was particularly important for NO_2 and it was helpful to use the same scale for both NO_2 and $PM_{2.5}$.

The rankings by borough were similar between the two reports. The PHE report included the City of London with Hackney but the borough with the smallest number of attributable deaths in the PHE report (Kensington and Chelsea) had the second smallest number of attributable deaths in this report after the City of London (Table 3). The borough with the smallest number of life years lost (Kingston upon Thames) had the second smallest number of life-years lost in this report after the City of London (Table 4). The attributable fraction which is less affected by the size and age distribution in each borough, and more directly related to the pollution level

was smallest in Havering and Bromley in the PHE report compared with Havering and Hillingdon in this report. The largest attributable fraction was Westminster and Kensington and Chelsea in the PHE report, followed by Tower Hamlets and the largest after the City of London in this report, was Tower Hamlets followed by Westminster and Islington (Table 4).

Table 3 London population, modelled population-weighted average concentration µg m^{-3} and estimated effects on annual mortality in 2010 of 2010 levels of anthropogenic $PM_{2.5}$, using COMEAP's recommended concentration-response coefficient of 6%, compared to PHE (Gowers, Miller and Stedman, 2014) and IOM (Miller, 2010) estimates.

	Population* ($x10^3$)			$PM_{2.5}$ PWAC** (µg m^{-3})			Attributable deaths***		
Borough	KCL (30+) 2009/10/11	PHE (25+) 2008/9/10	IOM (total) 2008	KCL anth. 2010	PHE anth. 2010	IOM Total 2006	KCL	PHE	IOM
City of London	5.2		9.2	17.9		17.6	4		4
Barking and Dagenham	98.3	109.7	172.4	13.2	12.6	15.0	92	93	120
Barnet	208.3	235.8	312.7	13.2	12.0	15.1	174	162	191
Bexley	142.3	154.7	235.0	13.0	11.8	14.9	132	122	171
Brent	171.3	178.3	277.9	13.7	12.9	15.4	112	111	133
Bromley	199.8	217.9	302.5	12.9	11.1	14.7	182	161	217
Camden	125.1	165.5	207.2	15.1	13.8	16.2	91	87	107
Croydon	212.0	233.4	341.0	13.2	11.5	14.9	173	155	205
Ealing	194.0	219.4	317.7	13.5	12.8	15.4	142	137	167
Enfield	177.3	195.5	291.3	13.0	11.8	15.0	138	133	178
Greenwich	141.2	150.1	236.5	13.6	12.7	15.3	120	119	150
Hackney†	126.1	151.9	223.4	14.5	14.0	15.7	82	86	96
Hammersmith and Fulham	102.5	121.9	178.7	14.5	14.1	15.8	71	72	86
Haringey	142.9	156.0	235.1	13.7	12.7	15.3	85	81	99
Harrow	142.4	155.8	219.0	12.8	11.3	14.8	100	90	119
Havering	150.1	163.2	230.5	12.6	11.1	14.6	150	137	182
Hillingdon	157.0	172.4	253.4	12.7	11.6	14.9	125	118	154
Hounslow	143.3	159.0	229.9	13.4	12.7	15.3	102	99	121
Islington	111.9	135.3	195.1	15.3	14.1	15.9	88	84	100
Kensington and Chelsea	101.5	128.0	169.0	15.1	14.9	16.2	67	68	75
Kingston upon Thames	95.3	113.1	154.2	13.1	11.9	15.0	74	68	91
Lambeth	165.4	200.8	291.8	14.4	13.7	15.7	109	112	139
Lewisham	157.0	181.5	269.0	13.9	12.7	15.3	120	116	153
Merton	119.9	146.6	198.1	13.5	12.3	15.2	86	82	107
Newham	144.5	147.2	261.7	14.0	13.5	15.4	96	98	121

Redbridge	157.6	177.9	252.6	13.3	12.4	15.1	128	123	153
Richmond upon Thames	120.7	133.5	184.5	13.3	12.0	15.0	84	77	97
Southwark	157.7	197.0	276.8	14.9	14.1	15.8	111	113	136
Sutton	118.7	133.6	185.2	13.1	11.4	14.9	102	92	124
Tower Hamlets	118.6	151.8	231.7	15.5	14.5	16.0	85	85	102
Waltham Forest	141.6	149.0	226.7	13.5	12.9	15.3	102	103	129
Wandsworth	173.4	121.3	289.1	14.1	13.1	15.6	119	116	148
Westminster	133.6	182.5	214.8	15.5	14.9	16.6	92	88	96
Total							3537	3388	4271

*Population: KCL, age 30+, based on 2009/2010/2011 average separately by gender and 5 year age groups
PHE, age 25+, based on averaging 2008/2009/2010.
IOM, total population based on 'High' projections for 2008.

** PWAC - population weighted average concentration of $PM_{2.5}$.

KCL, calculated for males and females separately and 5 year age groups, weighted average presented here.

PHE modelling undertaken on a 1 x 1km scale and uses anthropogenic $PM_{2.5}$ as in our calculations. Population-weighting used the total population from the 2001 census.

IOM uses (20 x 20m) modelling (scale) for 2006 based on total rather than anthropogenic $PM_{2.5}$. Here PWC for each ward has been averaged to obtain a Borough estimate.

*** Attributable deaths: KCL, based on deaths in the population age 30+ and calculated by summing gender and 5 year age groups results by borough.

PHE, based on deaths in the population age 25+, summing across 10 year age groups also calculated by borough.

IOM calculated by Ward, cumulated to Borough level here, used total population. The total of 4271 in the table is probably slightly different from the 4267 quoted in the Miller (2010) report because of this process.

† Hackney includes the City of London in the PHE report.

Table 4 Estimated effects on annual mortality in 2010 of anthropogenic $PM_{2.5}$, attributable fraction and life years lost, using COMEAP's recommended concentration-response coefficient of 6%, compared to PHE (Gowers, Miller and Stedman, 2014) estimates.

	Baseline deaths		Attributable fraction* (%)		Life years lost**	
Borough	KCL 2009/10/11	PHE 2008/9/10	KCL	PHE	KCL	PHE
City of London	40		9.9		60	
Barking and Dagenham	1247	1317	7.4	7.1	1268	1027
Barnet	2353	2397	7.4	6.8	2360	1701
Bexley	1814	1846	7.3	6.6	1772	1255
Brent	1477	1530	7.7	7.2	1855	1561
Bromley	2524	2571	7.2	6.3	2379	1621
Camden	1085	1126	8.4	7.7	1568	1157
Croydon	2349	2391	7.4	6.5	2423	1749
Ealing	1867	1905	7.6	7.2	2175	1773
Enfield	1897	2000	7.3	6.6	1944	1509
Greenwich	1584	1658	7.6	7.2	1659	1312

Hackney†	1015	1097	8.1	7.9	1429	1397
Hammersmith and Fulham	882	907	8.1	7.9	1166	1070
Haringey	1106	1142	7.7	7.1	1472	1215
Harrow	1386	1410	7.2	6.4	1544	1100
Havering	2126	2174	7.1	6.3	1968	1397
Hillingdon	1754	1794	7.1	6.5	1787	1335
Hounslow	1362	1382	7.5	7.1	1564	1167
Islington	1035	1069	8.5	7.9	1394	1084
Kensington and Chelsea	803	824	8.4	8.3	1119	1164
Kingston upon Thames	1000	1082	7.4	6.7	1008	730
Lambeth	1360	1454	8.1	7.7	1797	1520
Lewisham	1552	1628	7.8	7.2	1773	1331
Merton	1143	1186	7.6	6.9	1259	974
Newham	1221	1302	7.9	7.6	1572	1322
Redbridge	1716	1757	7.4	7	1799	1376
Richmond upon Thames	1131	1144	7.4	6.8	1238	897
Southwark	1338	1426	8.3	7.9	1793	1651
Sutton	1391	1424	7.3	6.4	1367	949
Tower Hamlets	983	1047	8.7	8.1	1314	1121
Waltham Forest	1356	1420	7.6	7.3	1546	1205
Wandsworth	1513	1587	7.9	7.3	1686	1331
Westminster	1071	1061	8.6	8.3	1570	1403
Total	46482	48058			52630	41404

* Attributable fraction for KCL calculated from population-weighted average concentration of anthropogenic $PM_{2.5}$, calculated at OA level separately for males and females 30+ by 5 year age group from 2009/10/11 population data, weighted average presented here. PHE attributable fraction based on population-weighted average anthropogenic $PM_{2.5}$ weighted by total population from the 2001 census at 1km x 1km grid level.

** Associated life years lost, KCL, age 30+ and calculated by gender and 5 year age groups, by Borough. PHE, age 25+, 10 year age groups, also calculated by borough.

† Hackney includes the City of London in the PHE report.

2.1.3.3 NO_2

The mortality burden of NO_2 in London was up to 88,113 life-years lost, equivalent to up to 5,879 (3,444-8,138) attributable deaths at typical ages (results for the central estimate are shown in Table 5, with sensitivities based on confidence intervals of 2.2% and 5.6% around the central coefficient in Annex 2 Table 27). The fraction of total mortality attributable to NO_2 across London was up to 12.6%. These figures are an upper bound as based on a counter factual of zero but the result is expected to be closer to these values than to the sensitivity analysis with a counter factual at 20 $\mu g\ m^{-3}$ (Table 7). For a 5 $\mu g\ m^{-3}$ counter factual (Annex 1), rough scaling (dividing the difference between counter factual at 0 and 20 by 4 to represent a 5 $\mu g\ m^{-3}$ change and subtracting this from the counter factual at zero figure) suggests a burden about 10% lower than the upper bound. These figures are all maximum figures for a burden of NO_2 *per se* as there may also be contributions from other traffic pollutants.

These figures were based on assuming a 30% overlap of effects with $PM_{2.5}$. The size of this overlap is uncertain - it has only been examined in a few studies with findings of overlaps up to 33%. Despite this uncertainty, it seems likely that there is at least some overlap, so we prefer this estimate.

Assuming a 30% overlap, results for the attributable fraction of mortality attributable to NO_2 varied from up to 20% in the City of London to up to 9.8% in Havering. Taking into account the underlying mortality rate and the size of the population, the attributable deaths varied from up to 8 in the City of London to up to 279 in Barnet and the life years lost from up to 120 in the City of London to up to 3797 in Croydon.

The figures if no overlap was assumed were up to 119,999 life-years lost, equivalent to up to 8,009 (4,756-11,054) attributable deaths at typical ages (Table 6), with sensitivities based on confidence intervals of 3.1% and 8% around the central coefficient in Annex 2 (Table 28).

The borough figures are also available assuming no overlap and for ranges around the concentration-response relationship, as shown for the London total. Results by borough for the fraction of mortality attributable to NO_2 varied from up to 26.8% in the City of London to up to 13.4% in Havering (Table 6). Taking into account the underlying mortality rate and the size of the population, the attributable deaths varied from up to 10 in the City of London to up to 381 in Barnet and the life years lost from up to 160 in the City of London to up to 5,188 in Croydon.

Table 5 London population, modelled population-weighted average concentration ($\mu g\ m^{-3}$) and estimated maximum burden of effects on annual mortality in 2010 NO_2, using the concentration-response coefficient of a 3.9% increase in mortality per 10 $\mu g\ m^{-3}$ NO_2 reflecting a 30% reduction due to overlap with $PM_{2.5}$

Borough	Population * ($x10^3$)	NO_2 PWAC** ($\mu g\ m^{-3}$)	Deaths	Attributable fraction (%)	Attributable deaths***	Life years lost
City of London	5.2	58.2	40	20.0	8	119
Barking and Dagenham	98.3	31.9	1247	11.5	142	1954
Barnet	208.3	33.1	2353	11.9	279	3784
Bexley	142.3	30.6	1814	11.1	201	2693
Brent	171.3	37.3	1477	13.3	193	3195
Bromley	199.8	29.9	2524	10.8	271	3532
Camden	125.1	45.7	1085	16.0	173	2983
Croydon	212.0	32.5	2349	11.7	271	3797
Ealing	194.0	36.9	1867	13.2	245	3760
Enfield	177.3	31.3	1897	11.3	212	2999
Greenwich	141.2	35.6	1584	12.7	200	2763
Hackney	126.1	41.4	1015	14.7	148	2572
Hammersmith and Fulham	102.5	42.6	882	15.0	132	2162

Haringey	142.9	36.7	1106	13.1	144	2510
Harrow	142.4	30.2	1386	10.9	150	2331
Havering	150.1	27.0	2126	9.8	207	2722
Hillingdon	157.0	30.3	1754	10.9	188	2709
Hounslow	143.3	35.6	1362	12.7	174	2657
Islington	111.9	45.2	1035	15.9	164	2590
Kensington and Chelsea	101.5	47.5	803	16.6	133	2204
Kingston upon Thames	95.3	32.6	1000	11.7	117	1609
Lambeth	165.4	41.6	1360	14.7	198	3273
Lewisham	157.0	37.4	1552	13.3	204	3028
Merton	119.9	34.8	1143	12.5	141	2072
Newham	144.5	38.2	1221	13.6	165	2716
Redbridge	157.6	32.4	1716	11.7	200	2818
Richmond upon Thames	120.7	33.8	1131	12.1	136	2013
Southwark	157.7	44.1	1338	15.5	206	3346
Sutton	118.7	31.4	1391	11.3	157	2110
Tower Hamlets	118.6	46.5	983	16.3	158	2463
Waltham Forest	141.6	34.7	1356	12.4	165	2515
Wandsworth	173.4	39.4	1513	14.0	210	2976
Westminster	133.6	49.5	1071	17.2	184	3139
Total					5879	88113

*Population and death rate, age 30+, based on 2009/2010/2011 average.

**PWAC - Population Weighted Average Concentration of NO_2, calculated for males and females and 5 year age groups separately, weighted average presented here.

***Attributable deaths and associated life years lost, age 30+ and calculated by 5 year age groups and gender.

Table 6 London population, modelled population-weighted average concentration (μg m^{-3}) and estimated maximum burden of effects on annual mortality in 2010 of NO_2, using the recommended concentration-response coefficient of 5.5% increase in mortality per 10 μg m^{-3} NO_2 (assuming no overlap with $PM_{2.5}$)

Borough	Population* (x10^3)	NO_2 PWAC** (μg m^{-3})	Deaths	Attributable fraction (%)	Attributable deaths***	Life years lost
City of London	5.2	58.2	40	26.8	10	160
Barking and Dagenham	98.3	31.9	1247	15.7	194	2670
Barnet	208.3	33.1	2353	16.2	381	5168
Bexley	142.3	30.6	1814	15.1	275	3683
Brent	171.3	37.3	1477	18.1	263	4351
Bromley	199.8	29.9	2524	14.8	371	4835
Camden	125.1	45.7	1085	21.7	234	4037
Croydon	212.0	32.5	2349	16.0	370	5188
Ealing	194.0	36.9	1867	17.9	333	5120
Enfield	177.3	31.3	1897	15.4	291	4101

Greenwich	141.2	35.6	1584	17.3	273	3767
Hackney	126.1	41.4	1015	19.9	200	3492
Hammersmith and Fulham	102.5	42.6	882	20.4	180	2932
Haringey	142.9	36.7	1106	17.8	196	3418
Harrow	142.4	30.2	1386	14.9	206	3189
Havering	150.1	27.0	2126	13.4	284	3734
Hillingdon	157.0	30.3	1754	15.0	258	3708
Hounslow	143.3	35.6	1362	17.4	237	3621
Islington	111.9	45.2	1035	21.5	222	3506
Kensington and Chelsea	101.5	47.5	803	22.4	179	2979
Kingston upon Thames	95.3	32.6	1000	16.0	160	2197
Lambeth	165.4	41.6	1360	20.0	269	4442
Lewisham	157.0	37.4	1552	18.1	278	4122
Merton	119.9	34.8	1143	17.0	193	2826
Newham	144.5	38.2	1221	18.5	225	3696
Redbridge	157.6	32.4	1716	15.9	273	3850
Richmond upon Thames	120.7	33.8	1131	16.6	186	2747
Southwark	157.7	44.1	1338	21.0	280	4534
Sutton	118.7	31.4	1391	15.5	215	2884
Tower Hamlets	118.6	46.5	983	22.0	214	3332
Waltham Forest	141.6	34.7	1356	16.9	225	3431
Wandsworth	173.4	39.4	1513	19.0	285	4045
Westminster	133.6	49.5	1071	23.3	249	4235
Total					8009	119999

*Population and death rate, age 30+, based on 2009/2010/2011 average.

**PWAC – Population-Weighted Average Concentration of NO_2, calculated for males and females and 5 year age groups separately, weighted average presented here.

***Attributable deaths and associated life years lost, age 30+ and calculated by 5 year age groups and gender.

Results using the alternative (less likely) counter factual at 20 µg m^{-3} are given in Table 7. Assuming a 30% overlap of effects with $PM_{2.5,}$ this gave the mortality burden of NO_2 in London to be up to 40,355 life-years lost, equivalent to up to 2,650 attributable deaths at typical ages. The figures, if no overlap was assumed, were up to 55,723 life-years lost, equivalent to up to 3,661 attributable deaths at typical ages (Table 8). These numbers are similar or smaller than the effects of $PM_{2.5}$. The ranking by borough differed from the main approach in that while the City of London still had the smallest number of attributable deaths and life years lost, the boroughs with the largest numbers of attributable deaths were now Ealing and Southwark and the borough with the largest numbers of life years lost was now Westminster. This change is probably because the distribution of 20m x 20m grid concentrations (from which the 20 µg m^{-3} was subtracted) around the OA level average differs in different boroughs.

Table 7 London population, modelled population-weighted average concentration (μg m^{-3}) and sensitivity approach to calculation of the estimated maximum burden of effects on annual mortality in 2010 of NO_2, where 20 μg m^{-3} was subtracted from 2010 levels of NO_2, using concentration-response coefficient of a 3.9% increase in mortality per 10 μg m^{-3} NO_2

Borough	Population* ($x10^3$)	NO_2 PWAC** μg m^{-3})	Baseline deaths	Attributable fraction (%)	Attributable deaths***	Life years lost
City of London	5.2	38.2	40	13.6	5	80
Barking and Dagenham	98.3	11.9	1247	4.5	54	747
Barnet	208.3	13.0	2353	4.9	114	1543
Bexley	142.3	10.6	1814	4.0	73	974
Brent	171.3	17.3	1477	6.4	91	1515
Bromley	199.8	9.9	2524	3.7	92	1197
Camden	125.1	25.7	1085	9.4	100	1735
Croydon	212.0	12.5	2349	4.7	106	1487
Ealing	194.0	16.9	1867	6.3	116	1777
Enfield	177.3	11.3	1897	4.2	78	1112
Greenwich	141.2	15.6	1584	5.8	90	1247
Hackney	126.1	21.4	1015	7.9	79	1373
Hammersmith and Fulham	102.5	22.6	882	8.3	73	1188
Haringey	142.9	16.7	1106	6.2	67	1178
Harrow	142.4	10.2	1386	3.8	52	810
Havering	150.1	7.0	2126	2.6	54	719
Hillingdon	157.0	10.3	1754	3.9	64	931
Hounslow	143.3	15.6	1362	5.8	79	1210
Islington	111.9	25.2	1035	9.2	95	1496
Kensington and Chelsea	101.5	27.5	803	10.0	79	1320
Kingston upon	95.3	12.6	1000	4.7	47	646

Thames						
Lambeth	165.4	21.7	1360	8.0	106	1756
Lewisham	157.0	17.4	1552	6.4	97	1446
Merton	119.9	14.8	1143	5.5	62	908
Newham	144.5	18.2	1221	6.7	81	1339
Redbridge	157.6	12.4	1716	4.6	79	1117
Richmond upon Thames	120.7	13.8	1131	5.1	57	846
Southwark	157.7	24.1	1338	8.8	116	1889
Sutton	118.7	11.4	1391	4.3	59	792
Tower Hamlets	118.6	26.5	983	9.6	93	1448
Waltham Forest	141.6	14.7	1356	5.5	70	1081
Wandsworth	173.4	19.4	1513	7.1	106	1509
Westminster	133.6	29.5	1071	10.7	114	1940
Total					2650	40355

*Population and death rate, age 30+, based on 2009/2010/2011 average.

** PWAC – Population-Weighted Average Concentration of NO_2, calculated for males and females separately by 5 year age group after subtraction of 20 µg m^{-3} from 20 x 20m grid concentrations averaged up to OA level, weighted average presented here.

***Attributable deaths and associated life years lost, age 30+ and calculated by 5 year age groups and gender.

Table 8 London population, modelled population-weighted average concentration (µg m^{-3}) and sensitivity approach to calculation of the estimated maximum burden of effects on annual mortality in 2010 of NO_2, where 20 µg m^{-3} was subtracted from 2010 levels of NO_2, using concentration-response coefficient of a 5.5% increase in mortality per 10 µg m^{-3} NO_2

Borough	Population* (x10^3)	NO_2 PWAC** (µg m^{-3})	Baseline deaths	Attributable fraction (%)	Attributable deaths***	Life years lost
City of London	5.2	38.2	40	18.5	7	110
Barking and Dagenham	98.3	11.9	1247	6.2	75	1036
Barnet	208.3	13.0	2353	6.7	158	2139
Bexley	142.3	10.6	1814	5.5	101	1352

Brent	171.3	17.3	1477	8.8	125	2094
Bromley	199.8	9.9	2524	5.2	127	1663
Camden	125.1	25.7	1085	12.8	138	2382
Croydon	212.0	12.5	2349	6.5	146	2061
Ealing	194.0	16.9	1867	8.6	160	2455
Enfield	177.3	11.3	1897	5.8	109	1543
Greenwich	141.2	15.6	1584	8.0	125	1726
Hackney	126.1	21.4	1015	10.8	108	1891
Hammersmith and Fulham	102.5	22.6	882	11.4	100	1635
Haringey	142.9	16.7	1106	8.6	93	1628
Harrow	142.4	10.2	1386	5.3	72	1125
Havering	150.1	7.0	2126	3.7	76	1001
Hillingdon	157.0	10.3	1754	5.4	89	1293
Hounslow	143.3	15.6	1362	8.0	110	1673
Islington	111.9	25.2	1035	12.6	130	2054
Kensington and Chelsea	101.5	27.5	803	13.7	109	1810
Kingston upon Thames	95.3	12.6	1000	6.5	65	896
Lambeth	165.4	21.7	1360	10.9	146	2418
Lewisham	157.0	17.4	1552	8.9	134	1997
Merton	119.9	14.8	1143	7.6	86	1256
Newham	144.5	18.2	1221	9.3	112	1848
Redbridge	157.6	12.4	1716	6.4	110	1548
Richmond upon Thames	120.7	13.8	1131	7.1	79	1172
Southwark	157.7	24.1	1338	12.1	160	2596
Sutton	118.7	11.4	1391	5.9	82	1099
Tower Hamlets	118.6	26.5	983	13.2	127	1987

Waltham Forest	141.6	14.7	1356	7.6	97	1497
Wandsworth	173.4	19.4	1513	9.9	147	2082
Westminster	133.6	29.5	1071	14.6	156	2656
Total					3661	55723

*Population and death rate, age 30+, based on 2009/2010/2011 average.

** PWAC – Population-Weighted Average Concentration of NO_2, calculated for males and females separately by 5 year age group after subtraction of 20 μg m^{-3} from 20 x 20m grid concentrations averaged up to OA level, weighted average presented here.

***Attributable deaths and associated life years lost, age 30+ and calculated by 5 year age groups and gender.

2.1.3.4 Total mortality burden of $PM_{2.5}$ and NO_2

The three approaches to calculating the total mortality burden of $PM_{2.5}$ and NO_2 used in the section above are summarised again here with the WHO recommendations as this guides how to add together the results.

- Only using the mortality burden of $PM_{2.5}$ as there is more certainty over the size of this effect. This is the WHO recommendation for the 'limited set' of more certain concentration-response functions.

- Adding together the effects of $PM_{2.5}$ and the effects of NO_2, but reducing the effects of NO_2 by 30% to account for the possible maximum size of the overlap between NO_2 and $PM_{2.5}$. WHO stated that the full effects of long-term exposure to NO_2 on mortality (in the 'extended set' could not be added to those of $PM_{2.5}$ as there was up to a 33% overlap between their effects. The alternative of adding them together taking account of the overlap (rounded to 30%) is preferred in this report.

- Adding together the effects of $PM_{2.5}$ and the effects of NO_2, assuming no overlap between NO_2 and $PM_{2.5}$. Assuming no overlap is the WHO recommendation for the 'extended set' of concentration-response functions but not adding the effects of $PM_{2.5}$ and NO_2 together when the effects of NO_2 are calculated this way.

Whilst at least 70% of the calculated effect of NO_2 is independent of $PM_{2.5}$, it remains unclear to what degree NO_2 represents the effect of primary particles (or other traffic pollutants). For burden calculations, the total effect on mortality would be the same if NO_2 was acting as an indicator of other traffic pollutants but the degree of potential overlap is important for assessing the effects of policies directed at specific pollutants.

In summary, the total burden of air pollution in 2010 is probably more than the 52,630 life-years lost, equivalent to 3,537 deaths at typical ages (WHO 'limited set' accounting for $PM_{2.5}$ only [similar to the 2010 IOM report analysis]). The total could be as much as 140,743 life-years lost,

equivalent to 9,416 deaths at typical ages (WHO 'extended set' including both $PM_{2.5}$ and NO_2, assuming a 30% overlap between their effects) or even higher[42] if no overlap is assumed.

The counter factual at 20 µg m^{-3} does not need to feature in the total as it falls between the results assuming only effects of $PM_{2.5}$ and the results assuming effects of both $PM_{2.5}$ and NO_2 with a counter factual at zero. Although the latter is an upper bound of the range for a 30% overlap between $NO_2$and $PM_{2.5}$, it is likely that, if moving beyond the most established result with $PM_{2.5}$ alone, the total is well towards the upper end of the range (Annex 1).

Totals are not given for boroughs but the results can be added in a similar way with the same caveats i.e. from the figures for $PM_{2.5}$ in Table 2 up to the total from Table 2 and Table 5.

2.1.4 Mortality impact of changes in $PM_{2.5}$ and NO_2 on life-expectancy and life-years lost in London (life table calculations) - input data and method

The methods and results discussed in the sections above used a 'short-cut' methodology to give an approximate view of the burden of 2010 levels of pollution on mortality. This and the following section use life table calculations to assess the effect of changes in pollution on mortality rates going forward in time. This is done for an illustrative 1 µg m^{-3} reduction.

While the previous section used life tables to derive the average remaining life-expectancy in specific 5 year age groups of the general population (irrespective of pollution) and then multiplied this by the pollution attributable deaths, this section actually takes the pollution changes into account within the life tables. There are two key differences from the burden calculations - one is that a lag between a change in exposure and effect is taken into account, and the second is that the calculations take into account the changes in the size and age-distribution of the population as a result of more people surviving from one year to the next when pollution is reduced.

The calculations are designed to compare results from a baseline scenario where mortality rates remain as in 2010 compared with a scenario in which the mortality rates are changed according to the pollution changes. They followed the impact methodology described in COMEAP (2010). The method is also used to calculated changes in life-expectancy from birth for a 1 µg m^{-3} reduction and for maintaining concentrations at 2010 levels for a life time.

2.1.4.1 Processing of Input data

Data updates (impact methodology): The input data for calculating changes in life-expectancy and life years lost for a permanent 1 µg m^{-3} reduction in $PM_{2.5}$ and NO_2 was as follows:

Modelled concentrations: As in section above using anthropogenic $PM_{2.5}$ and NO_2.

[42] 172,500 life years lost, equivalent to 11,500 attributable deaths at typical ages.

Population data: This was as in the section above i.e. given by sex and by single year of age at OA level aggregated up to London level and further averaged for 2009/2010/2011 to represent 2010. The population data was used for two purposes - for population-weighting, which used an amalgamation of populations age 30+ by gender, and for input to the life tables which kept the population data by sex and single year of age.

Population-weighted average concentrations: average concentration in each OA multiplied by the total population age 30+ by gender within each individual OA, furthermore summed across London and divided by the total population age 30+ by gender in London.

Mortality data: The deaths data for all causes was extracted from ONS data by the PHE London Knowledge and Intelligence Team. The deaths data were given by single year of age and gender, with an upper age of 90+, averaged for 2009/2010/2011 for London. This is taken to be a figure for 2010 with the random year-to-year variability in age groups with small numbers of deaths stabilised by averaging with the surrounding years.

Life tables: These were compiled with mortality rates generated from the population and mortality data described above. A computer programme coded in SQL was used to project forward from a 2010 starting point based on the IOMLIFET system[43] (Miller and Hurley, 2003). The IOMLIFET system subtracts neonatal deaths and then calculates survival probabilities from the non-neonatal deaths as in other years. We included neonatal deaths as we did not have these defined separately but followed the SEPHO template[44] and Gowers et al. (2014) in taking into account the uneven distribution of deaths over the course of the first year in calculating the survival probability[45]. The years 90 - 105 were allocated the pooled mortality rate for age 90+ as in IOMLIFET. The mortality rates for each age in 2010 were also assumed to apply in future years for the baseline scenario. New birth cohorts of the same size as in 2010 came into the life table each year.

Follow up: Life tables were run through for 105 years to 2114 - this is important because those that survive as a result of reduced pollution could survive for many years and the years of life saved cannot be counted fully without modelling the future time patterns of deaths of the survivors. (In the UK over 13,000 people live beyond 100 and over 600 beyond 105[46]).

Delay between exposure and effect: The recommended distribution of lags from COMEAP (2010) (based on that recommended by the US EPA) was used i.e. 30% of the effect in the first year, 12.5% in each of years 2-5 and 20% spread over years 5-20.

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[43] http://www.iom-world.org/research/research-expertise/statistical-services/iomlifet/

[44] http://www.sepho.org.uk/viewResource.aspx?id=8943

[45] The survival probability (the ratio of the number alive at the end of the year to the number alive at the beginning) is derived by the equivalent of adding half the deaths back onto the mid-year population to give the starting population and subtracting half the deaths from the mid-year population to give the end population, assuming deaths are distributed evenly across the year. This is not the case in the first year where a weighting factor based on 90% of the deaths occurring in the first half of the year and 10% in the second half is used instead. After rearrangement the actual formula is (1- 0.1 x hazard rate)/(1+ 0.9 x hazard rate) rather than the (1- 0.5 x hazard rate)/(1+ 0.5 x hazard rate) used in other years

[46] http://www.ons.gov.uk/ons/rel/mortality-ageing/estimates-of-the-very-old--including-centenarians-/2002---2012--united-kingdom/stb-2002-2012-uk.html

2.1.4.2 Calculations for 1 μg m^{-3} reduction

We updated the Miller (2010) figures for changes in life-expectancy and life years gained for a permanent 1 μg m^{-3} reduction in anthropogenic $PM_{2.5}$ from 2010 sustained to 2114 compared with pollution remaining unchanged at 2010 concentrations until 2114. The 1 μg m^{-3} was a population-weighted average concentration reduction.

The relative risk of 1.06 per 10 μg m^{-3} was scaled for the 1 μg m^{-3} reduction as in section 2.1.2.2 except that a lag was applied. The lag means that the full change in the hazard rate does not apply immediately but builds up. However, the SQL programme is organized to change the hazard rate only via an input as change in concentration. Thus, as an arithmetic device, the concentrations were changed in order to change the hazard rate, although exposure to the full concentration would still occur from the beginning in reality, with it being the biological effect that is delayed. This arithmetic device involved scaling the coefficient as if a 0.3 μg m^{-3} reduction applied in the first year (the EPA lag is for 30% of the effect in the first year). For the second year, a 0.125 μg m^{-3} reduction was applied to represent the delayed effect from the first year but, as it was a sustained reduction of 1 μg m^{-3}, a 0.3 μg m^{-3} reduction was also applied representing the portion of the effect of exposure in the second year that had an immediate effect[47]. This process was continued year on year (it stabilises to the equivalent of the full effect of a 1 μg m^{-3} reduction after 20 years as the partial delayed effects of the past 20 years add up to 100%). This gave a relative risk (hazard ratio) of 0.994190 for the year 2030 and beyond.

The scaled relative risks for each of the relevant years, taking into account the lag, were then multiplied by the hazard rates in all the age groups above age 30 to give a new set of hazard rates. The life table with the new hazard rates was then used to derive survival probabilities, deaths in each year and life years lived in the usual way. The matrix of life years lived for the baseline scenario was then subtracted from the matrix for the impacted scenario with the sustained 1 μg m^{-3} reduction to give the life years gained.

Calculations were also done for the 1% and 12% COMEAP plausibility intervals for $PM_{2.5}$.
The gain in life expectancy for the 2010 birth cohort was extracted from the above calculation by dividing the cumulative life years lived for those born in 2010 by the size of the birth cohort (64,220 for males and 61,290 for females).

A specific calculation was done for the $PM_{2.5}$ central estimate only, in order to provide a comparison with one of the results in Miller (2010). This was to calculate the life years gained for the 2010 population followed up over 105 years after a 1 μg m^{-3} reduction, without new birth cohorts, and to calculate the average gain in life expectancy by dividing the total life years gained by the whole of the 2010 population. This division of the total life years gained by the total population is not done for the standard calculation with new birth cohorts because the new birth cohorts included are not followed up for a whole lifetime, and this is needed for an appropriate value for life expectancy changes on a per person basis. This does not prevent calculation of life years gained rather than overall life expectancy for new birth cohorts in the future as life years are gained for every year survived.

[47] This approach gives the same result as the IOM lag tool available from the IOMLIFET website.

Calculations were also done for NO_2 using the concentration-response functions in Table 1. Taking into account a counter factual at 20 μg m^{-3}, zero or somewhere in between was not required as the 1 μg m^{-3} reduction was above 20 μg m^{-3}. In these calculations the counter factual is the concentration without the 1 μg m^{-3} reduction.

2.1.4.3 Loss in life-expectancy from birth

We also used the impact methodology to estimate the loss in life-expectancy from birth for the current population as a result of 2010 levels of anthropogenic $PM_{2.5}$. These calculations followed the methods described above for a 1 μg m^{-3} reduction except that the reduction was for the full population-weighted average concentration for 2010 e.g. 13.75 μg m^{-3} population-weighted for males for $PM_{2.5}$. This was then taken to be the loss of life expectancy from the 2010 concentration. Note that, unlike for the burden calculations, this is for a sustained exposure to a reduction by 2010 levels of pollution from birth for a lifetime, not just in the year 2010, but it only applies to the birth cohort.

The calculation for NO_2 used the same method as that for $PM_{2.5}$. As discussed in section 2.1.1 and Annex 1, there are a variety of possible counter factuals for burden calculations. The results given here assume a counter factual of zero as an upper bound and the previous comments regarding the result being nearer to the upper bound apply here. The calculation used a reduction from the baseline mortality rate at current concentrations as an analytical device[48] rather than an increase from the mortality rate without NO_2 as that is unknown. A reduction gives a life expectancy improvement but this was regarded as equivalent to the life expectancy loss related to current concentrations of NO_2.

2.1.5 Mortality impact of changes in $PM_{2.5}$ and NO_2 on life-expectancy and life-years lost in London (life table calculations) – results

2.1.5.1 1 μg m^{-3} reduction

The results showed that a 1 μg m^{-3} reduction in $PM_{2.5}$ in 2010, sustained until 2114 would result in a gain of 573,145 (97,882 - 1,114,618) life years over 105 years across the population (Table 9). This includes benefits to those born at a later date. To put the results in context, note that this is for the whole population, followed up for 105 years, including new birth cohorts, which gives a total of over a billion life years lived. To compare with the life years lost for no reduction in 2010 levels of $PM_{2.5}$ and NO_2 for a lifetime, see section 3.2.2. Part of the 1 μg m^{-3} reduction calculation (that relating to the birth cohort) indicates that there would be an average gain in life-expectancy for people born in 2010 of 19.7 (3.4 - 38.3) days in females and 21.4 (3.4 - 41.6) days in males. For those alive in 2010, excluding future new birth cohorts, 416,490 life years were gained for the central estimate and dividing this by the size of the 2010 population gave an average gain in life expectancy of 18.1 days in females and 19.6 days in males. The number of life years gained is smaller without the benefits to future generations of a sustained 1 μg m^{-3} reduction. The average gain in life expectancy is smaller than for the birth cohort as there is less

[48] Because we only know the baseline mortality rate including the effect of NO_2.

time for the older parts of the population in 2010 to gain life years. Thus, the figures of 18.1 and 19.6 days are averages of gains that vary by age.

These results were similar to the results from Miller (2010) where the estimate was that a permanent reduction in $PM_{2.5}$ concentrations of 1 μg m^{-3} would gain 405,659 years of life for the current population (2008) in London and a further 192,674 years for those born during that period, followed for the lifetime of the current population. The total including new birth cohorts was 598,333 years of life gained. For the 2008 population, the 405,659 years of life was estimated to be equivalent to an average 3 weeks per member of the 2008 population, with the expected gains differing by age.

The concentration change (1 μg m^{-3}), concentration-response relationship and basic methodology were the same for this work and the previous report. The population used for this work was larger (section 2.1.3.2) which would increase the life years gained and the mortality data used is different (which could increase or decrease the results depending on direction, with greater effects for higher baseline mortality rates and therefore greater gains for a reduction). The baseline scenario in the Miller (2010) report assumed future age-specific mortality rates based on the 2008 data for all London, calculated from all-cause death numbers, excluding neonatal deaths, and the total population figures totalled over all Wards. The deaths data were available only in five-year groups (plus <1 and the 4-year group 1-4) so the Miller (2010) report allocated the same mortality rate for each year within these age groups so that the IOMLIFET spreadsheets could be operated in 1-year age-groups. This contrasts with the current report, which had deaths available by single year of age, although they were averaged over the three years 2009/10/11 and the population averaged over 2009/10/11 was used to derive the mortality rates.

For NO_2, assuming an up to 30% overlap with $PM_{2.5}$, a 1 μg m^{-3} reduction in NO_2 in 2010, sustained until 2114 would result in a gain of up to 376,334 (214,064 - 535,961) life years over 105 years across the population (Table 9). This includes benefits to those born at a later date. For people born in 2010, this 1 μg m^{-3} reduction would result in an average gain in life-expectancy of up to 12.9 (7.3 – 18.4) days in females and 14 (8 -20) days in males.

For NO_2, assuming no overlap with $PM_{2.5}$, a 1 μg m^{-3} reduction in NO_2 in 2010, sustained until 2114 would result in a gain of up to 526,642 (300,306 - 756,981) life years over 105 years across the population (Table 9). This includes benefits to those born at a later date. For people born in 2010, this 1 μg m^{-3} reduction would result in an average gain in life-expectancy of up to 18.1 (10.3 – 26) days in females and 19.6 (11.2 - 28.2) days in males.

It can be seen that for the 1 μg m^{-3} reduction, the size of the effect is similar for $PM_{2.5}$ and NO_2 without assuming an overlap. The result for NO_2 assuming an overlap is a bit smaller.

Table 9 Gain in life years and life expectancy from birth from a reduction of 1 μg m^{-3} in $PM_{2.5}$ and NO_2 in 2010, sustained until 2114, EPA lag

Pollutant	Relative Risk (RR)	Gender	life-years gained	life-expectancy from birth gained (number of days)
$PM_{2.5}$	Central (1.06)	Female	270,092	19.7
		Male	303,053	21.4
		Total	573,145	
$PM_{2.5}$	Lower (1.01)	Female	46,127	3.4
		Male	51,754	3.6
		Total	97,882	
$PM_{2.5}$	Upper (1.12)	Female	525,249	38.3
		Male	589,370	41.6
		Total	1,114,618	
NO_2	Central (1.055)	Female	248,178	18.1
		Male	278,464	19.6
		Total	526,642	
NO_2	Lower (1.031)	Female	141,519	10.3
		Male	158,787	11.2
		Total	300,306	
NO_2	Upper (1.08)	Female	356,722	26
		Male	400,259	28.2
		Total	756,981	
NO_2	Central (1.039)	Female	177,347	12.9
		Male	198,987	14
		Total	376,334	
NO_2	Lower (1.022)	Female	100,878	7.3
		Male	113,186	8
		Total	214,064	

NO_2	Upper (1.056)	Female	252,569	18.4
		Male	283,391	20
		Total	535,961	

2.1.5.2 Loss of life expectancy from 2010 levels of anthropogenic $PM_{2.5}$ and 2010 levels of NO_2

The results showed that a 13.75 μg m^{-3} population-weighted concentration of $PM_{2.5}$ for males in 2010, and of 13.69 μg m^{-3} for females , sustained until 2114 would result in an average loss in life-expectancy for people born in 2010 of around 9.5 months (294 days) in males and around 9 months (270 days) in females.

For a 36.63 μg m^{-3} population-weighted concentration of NO_2 for males in 2010, and of 36.21 μg m^{-3} for females, sustained until 2114, the average loss in life-expectancy for people born in 2010 would be around 17 months (515 days) in males and around 15.5 months (468 days) in females.

2.2 Estimating the impact of $PM_{2.5}$, PM_{10} and NO_2 on hospital admissions and deaths brought forward in London

2.2.1 Background

The COMEAP recommendations for concentration-response coefficients for hospital admissions and deaths brought forward are given in a report published in 1998 (COMEAP, 1998) and a statement on particulate matter and cardiovascular hospital admissions published in 2001 (COMEAP, 2001). These concentration-response functions are commonly used in health impact assessment in the UK and cover PM_{10}, SO_2, ozone and NO_2.

The Department of Health has recently commissioned a systematic review and meta-analysis of time-series studies on $PM_{2.5}$, ozone and NO_2 to be provided to COMEAP to assist them in updating the concentration-response functions recommended in 1998. This work was led by St. George's, University of London with the participation of King's. The final report has been submitted to the Department of Health[49]. Papers on concentration-response functions for $PM_{2.5}$ and NO_2 have been published (Atkinson et al. 2014; Mills et al., 2015). Concentration-response functions from the above work (Annex 3 Table 29) have been used to inform WHO HRAPIE recommendations for $PM_{2.5}$ and NO_2 and we used these recommendations for $PM_{2.5}$ and NO_2 in the calculations below. We also used the 1998 COMEAP recommendation for PM_{10} as an alternative to $PM_{2.5}$.

[49] Summary available at http://www.prp-ccf.org.uk/PRPFiles/SFR_April_2011/0020037%20SFR_Atkinson.pdf

2.2.2 Deaths brought forward and hospital admissions - input data and method

Processing of Input data

Modelled concentrations: PM_{10}, $PM_{2.5}$ and NO_2 annual mean concentrations at 20m grid resolution were extracted from the LAEI2010 year 2010 air quality results and processed as above in section 2.1.2.1.

Anthropogenic source: The contribution of sea salt within the PM_{10} mass was measured to be 1.5 $\mu g\ m^{-3}$ in 2010 and removed from total PM_{10} concentration to generate anthropogenic PM_{10} concentrations. Anthropogenic $PM_{2.5}$ was calculated as in section 2.1.2.1.

Population data: The population data was used across all ages by single year of age at OA level and was further averaged for 2009/2010/2011 to represent 2010. The populations in each OA were also summed to give the total population for London overall. The 3 year average was used to give the same population base as for the mortality burden calculations but the difference from the 2010 population alone was in any case only 0.1%.

Deaths data: The total deaths data and deaths from external causes (ICD10 V01 - Y89 and U509) were extracted from ONS data by PHE London Knowledge and Intelligence Team. The deaths data were given by 5 year age groups, averaged for 2009/2010/2011 at London borough level. The deaths from external causes were subtracted from total deaths. The baseline rates for deaths and for the types of hospital admissions specified below are given in Annex 3 Table 29.

Emergency respiratory hospital admissions: Emergency respiratory hospital admissions all ages ICD 10 J00-J99 (first episode, finished consultant episode, London residents) for London for 2010 were extracted from Hospital Episode Statistics by the PHE London Knowledge and Intelligence team.

Emergency cardiovascular hospital admissions: All cardiovascular emergency hospital admissions all ages ICD 10 I00-I99 (first episode, finished consultant episode, London residents) for London for 2010 extracted from Hospital Episode Statistics by the PHE London Knowledge and Intelligence team.

Relative Risk: Relative risks for $PM_{2.5}$ and NO_2 were as recommended by HRAPIE for deaths brought forward, respiratory and cardiovascular hospital admissions (Annex 3 Table 29). The relative risk for daily maximum 1 hour average NO_2 and respiratory hospital admissions (rather than for 24 hour average) was used as the modelling is validated against 1 hour average monitoring data. Relative risks for PM_{10} as recommended by COMEAP (1998) and COMEAP (2001).

Population-weighted average concentration: The population-weighted average concentration was calculated as above in 2.1.2.1 but using the whole population (average of 2009/2010/2011) rather than the population aged 30+, as the relative risks are based on all ages, and then summing across all OAs straight to the Greater London area.

2.2.2.1 Calculations

The coefficients for these outcomes are derived from Poisson regression that plots the natural log (LN) of the relative risk against concentration. Therefore to convert the relative risk per 10 μg m^{-3} to a new relative risk for the relevant population-weighted average concentration requires (i) taking the natural log of the relative risk; (ii) dividing this by 10 to get back the original slope per μg m^{-3}, (iii) multiplying by the population-weighted average concentration to give the new LN RR and (iv) taking the antilog (exponential) of this to give the new RR. Subtracting 1 from this and multiplying by 100 gives the new % increase in the outcome for that pollutant.

Multiplying this % increase by the baseline number of deaths brought forward or hospital admissions gives the final result. While the original studies are based on daily concentrations, if there is no threshold, as is currently assumed, performing one calculation on the annual mean is arithmetically equivalent to performing calculations for each day of the year and adding them up.

2.2.3 Deaths brought forward and hospital admissions - results

This section provides results for the total numbers of deaths brought forward, respiratory hospital admissions and cardiovascular hospital admissions in London due to 2010 concentrations of PM_{10}, $PM_{2.5}$ and NO_2 for (i) current COMEAP recommendations for PM_{10} and (ii) more up to date concentration-response functions for $PM_{2.5}$ and NO_2 from HRAPIE. Deaths brought forward from short-term exposure should not be added to the mortality burden from long-term exposure. WHO recommended that the results for $PM_{2.5}$ and NO_2 can be added together, although only the NO_2 recommendations comment directly on the robustness to adjustment for other pollutants.

2.2.3.1 $PM_{2.5}$

The original COMEAP recommendations for calculating total effects of short-term exposure did not suggest use of anthropogenic levels of pollution and were based on PM_{10}. However, the Department of Health commissioned a review of concentration-response relationships for $PM_{2.5}$ to assist COMEAP in updating their recommendations and the published results of this review have been used by WHO to recommend concentration-response functions for health impact assessments. We have therefore used these here, although results for PM_{10} are also available in Annex 5 Table 30. Results for both anthropogenic $PM_{2.5}$ (to match the effects of long-term exposure) and total $PM_{2.5}$ are presented.

The estimate for the effects of short-term exposure to 2010 levels of anthropogenic $PM_{2.5}$ in London in 2010 (13.76 μg m^{-3}) is 787 (287-1,288) deaths brought forward, 1,992 (-188-4,232)[50]

[50] We have retained negative values where the lower confidence intervals for the CRFs are below a relative risk of 1. We do not regard this as meaning air pollution has a beneficial effect but rather as indicating that the confidence intervals include the possibility of no effect.

respiratory hospital admissions and 740 (138-1,352) cardiovascular hospital admissions. The results for deaths brought forward should not be added to the deaths from long-term exposure.

Using the total level of $PM_{2.5}$ (14.30 μg m^{-3}), the estimate for the effects of short-term exposure to $PM_{2.5}$ in London is 818 (299-1,340) deaths brought forward, 2,072 (-195-4,405) respiratory hospital admissions and 769 (144-1,406) cardiovascular hospital admissions.

2.2.3.2 NO_2

The estimate for the effects of short-term exposure to 2010 levels of NO_2 in London (36.67 μg m^{-3}) is 461 (273-650) deaths brought forward, and 419 (-223-1,064) respiratory hospital admissions. The results for deaths brought forward as a result of short-term exposure to NO_2 are more certain than the results for long-term exposure and should therefore be regarded as an alternative result for numbers of deaths. WHO did not recommend quantification of effects of NO_2 on cardiovascular admissions.

2.3 Apportionment of hospital admissions, death brought forward and mortality impacts of $PM_{2.5}$ and NO_2 to broad sources

2.3.1 Apportionment of health burden from $PM_{2.5}$ and NO_2 - input data and method

Processing of Input data

Total concentrations: Total $PM_{2.5}$ and NO_2 annual mean concentrations in 2010 were extracted as in 2.1.2.1 above.

London road only concentrations: For $PM_{2.5}$ only, the road source (London road transport only) annual mean concentrations were extracted from the source apportionment year 2010 air quality results (commissioned by TfL as part of the LAEI2010; available on request).

Other (non-road) London sources only concentrations: For $PM_{2.5}$ only, the other London sources (all London sources except road traffic) annual mean concentrations were extracted from the source apportionment year 2010 air quality results (commissioned by TfL as part of the LAEI2010). Note that the other London sources annual mean concentrations account for the other London sources emissions (from LAEI2010) and an additional 1.05 μg m^{-3} accounting for all biomass sources in London. (Biomass sources are not in the emissions inventory but are added into the air quality modelling, LAEI2010 (GLA, 2013).

Non-London sources concentrations: $PM_{2.5}$ rural and regional concentrations have been derived from measurements at rural monitoring sites as part of air quality networks operated by DEFRA and King's College, London. These were estimated to be an annual mean of 9.85 μg m^{-3} in the year 2010. The annual mean NO_2 rural concentration was similarly determined as 11.3 μg m^{-3} in the year 2010.

Natural sources: $PM_{2.5}$ natural source was measured as sea salt to be 0.55 μg m^{-3} in 2010. NO_2 does not allow for a natural part to be measured or quantified as described further in 2.1.2.1.

2.3.1.1 $PM_{2.5}$

The methods set out in section 2.1 and 2.2 have been used to calculate the health burden of pollution from the total anthropogenic $PM_{2.5}$ annual mean concentrations, its London road traffic source and the other London sources only using the population-weighted average concentration of each source in turn.

The non-London sources have been derived as described above. The total London source can be defined as the difference between the anthropogenic $PM_{2.5}$ annual mean concentrations and the non-London sources or the sum of the London road traffic source and the other London sources.

2.3.1.2 NO_2

The methods set out in 2.1 and 2.2 have been used to calculate the health burden of pollution from the total NO_2 annual mean concentrations. Further apportionment presumes that NO_2 itself is responsible for the whole of the effect. If considering NO_2 as an indicator, at least in part, it should be noted that the correlations with other constituents potentially contributing to the effect are likely to differ by source.

The non-London sources have been derived as described above.

The total London source can be defined as the difference between the total NO_2 annual mean concentrations and the non-London sources.

In the case of NO_2 and in accordance with DEFRA guidelines, the London sources cannot be apportioned further into road traffic and other source components. It is not possible to calculate an unambiguous source apportionment for annual mean NO_2 concentrations as there is no simple linear relationship between NO_2 concentrations and NO_X emissions or concentrations (DEFRA 2011a).

2.3.2 Apportionment of health burden from $PM_{2.5}$ and NO_2 - results

By combining the existing modelled estimates separately with the methods described in 2.1 and 2.2 we have produced the following results for London for $PM_{2.5}$ and NO_2:

- The percentage change in mortality, attributable deaths and years of life lost.
- The total numbers of deaths brought forward, respiratory hospital admissions and cardiovascular hospital admissions ($PM_{2.5}$ only) in London, using the recommendations of HRAPIE.

2.3.2.1 $PM_{2.5}$

The mortality burden of 2010 levels of $PM_{2.5}$ is

- 52,630 life-years lost, equivalent to 3537 attributable deaths at typical ages for total anthropogenic $PM_{2.5}$.
- 5147 life-years lost, equivalent to 346 attributable deaths at typical ages from London road transport sources.
- 9913 life-years lost, equivalent to 666 attributable deaths at typical ages from other (non-road transport) London sources.
- 15,060 life-years lost, equivalent to 1012 attributable deaths at typical ages from London (road transport + other) sources.
- 37,570 life-years lost, equivalent to 2525 attributable deaths at typical ages from non-London sources.

2.3.2.2 NO_2

The mortality burden of 2010 levels of NO_2 is

- Up to 119,999 and up to 88,113 life-years lost, equivalent to 8009 and 5879 attributable deaths at typical ages for total NO_2 and assuming a 30% overlap, respectively.
- 79,441 and 58,332 life-years lost, equivalent to 5302 and 3892 attributable deaths at typical ages from London (road transport + other) sources and assuming a 30% overlap, respectively.
- 40,558 and 29,781 life-years lost, equivalent to 2707 and 1987 attributable deaths at typical ages from non-London sources and assuming a 30% overlap, respectively.
- In the case of NO_2 and in accordance with DEFRA guidelines, the London sources cannot be apportioned further into road traffic and other sources components. The mortality burden above and the impact of air pollution (on deaths brought forward and hospital admissions) below have been calculated by scaling the NO_2 concentration from non-London sources in 2010 (derived from King's measurements) to the total NO_2 annual mean concentrations in 2010. The mortality data associated with London (road transport + other) sources was calculated by difference.

2.3.2.3 $PM_{2.5}$ and NO_2 Apportionment

Figure 1 shows the breakdown into broad source categories of the mortality burden from 2010 concentrations of $PM_{2.5}$ and NO_2[51]. This breakdown assumes a 30% overlap of effect between NO_2 and $PM_{2.5}$. Figure 2 shows the breakdown with no assumed overlap of effect.

[51] Assuming the effects of NO_2 and $PM_{2.5}$ can be added. It also assumes that the nature and/or potency of the health effects does not vary by source.

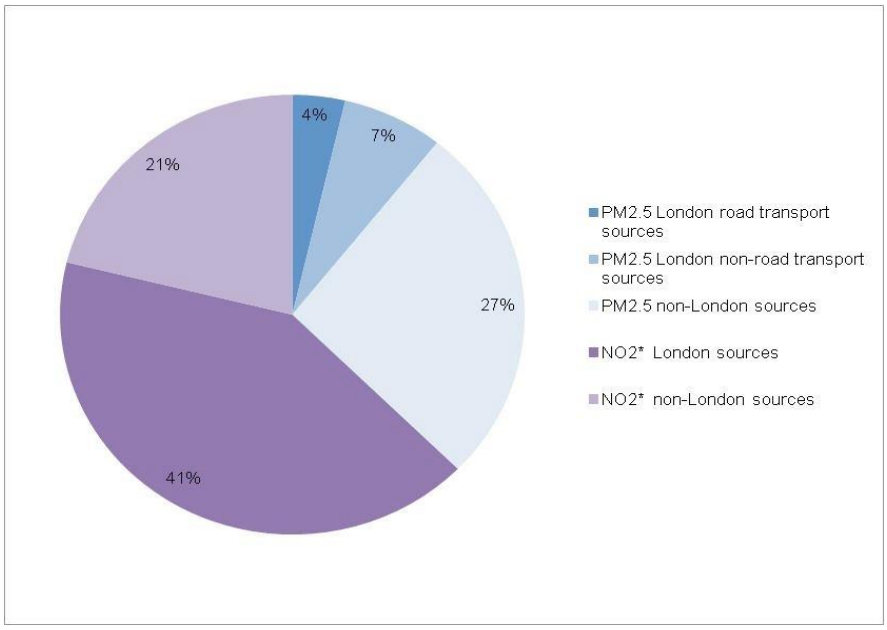


Figure 1 Apportionment of the mortality burden (life years lost and equivalent attributable deaths) of 2010 levels of pollution to emissions sources *effect of NO_2 assumes a 30% overlap with the effects of $PM_{2.5}$

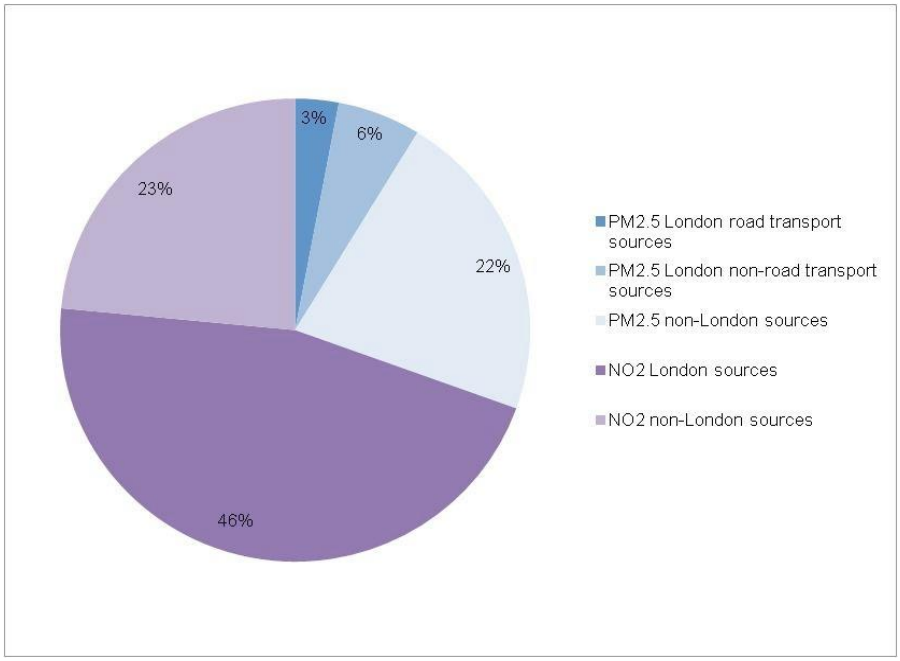


Figure 2 Apportionment of the mortality burden (life years lost and equivalent attributable deaths) of 2010 levels of pollution to emissions sources, effect of NO_2 with no assumed overlap of effects of $PM_{2.5}$

Both with and without an overlap, the largest contribution to the mortality burden of NO_2 is from sources within London (both road transport and other sources). Sources of NO_2 from outside London also make a significant contribution and are similar to the contribution of $PM_{2.5}$ sources outside London. The sources of $PM_{2.5}$ within London make a less significant contribution

to the mortality burden. The results in Figure 1 and Figure 2 highlight the importance of considering the mortality impact of NO_2 (and/or specific traffic pollutants) in London.

2.3.3 Source apportionment of the impact of $PM_{2.5}$ and NO_2 on deaths brought forward and hospital admissions

Deaths brought forward from short-term exposure and mortality burden from long-term exposure not to be added.

2.3.3.1 $PM_{2.5}$

Using the total level, the estimate for the effects of short-term exposure to $PM_{2.5}$ is

- 818 deaths brought forward, 2072 respiratory hospital admissions and 769 cardiovascular hospital admissions for total $PM_{2.5}$.
- 76 deaths brought forward, 192 respiratory hospital admissions and 72 cardiovascular hospital admissions from London road transport sources.
- 146 deaths brought forward, 368 respiratory hospital admissions and 137 cardiovascular hospital admissions from other (non-road transport) London sources.
- 222 deaths brought forward, 560 respiratory hospital admissions and 209 cardiovascular hospital admissions from London (road transport + other) sources.
- 596 deaths brought forward, 1512 respiratory hospital admissions and 560 cardiovascular hospital admissions from non-London sources.

2.3.3.2 NO_2

The estimate for the effects of short-term exposure to NO_2 is

- 461 deaths brought forward and 419 respiratory hospital admissions for total NO_2.
- 305 deaths brought forward and 277 respiratory hospital admissions from London (road transport + other) sources.
- 156 deaths brought forward and 142 respiratory hospital admissions from non-London sources.

2.3.3.3 $PM_{2.5}$ and NO_2 Apportionment

Figure 3 and Figure 4 indicate that around half of the deaths brought forward and respiratory hospital admissions due to short term exposure to pollution in London can be associated with $PM_{2.5}$ from sources outside London. Exposure to NO_2 makes a significant contribution, with the majority of these being associated with London sources. Quantification of cardiovascular hospital admissions attributed to short-term exposure to pollution was only recommended by WHO HRAPIE for $PM_{2.5}$, as illustrated in Figure 5, 75% of which can be associated with $PM_{2.5}$ from outside London.

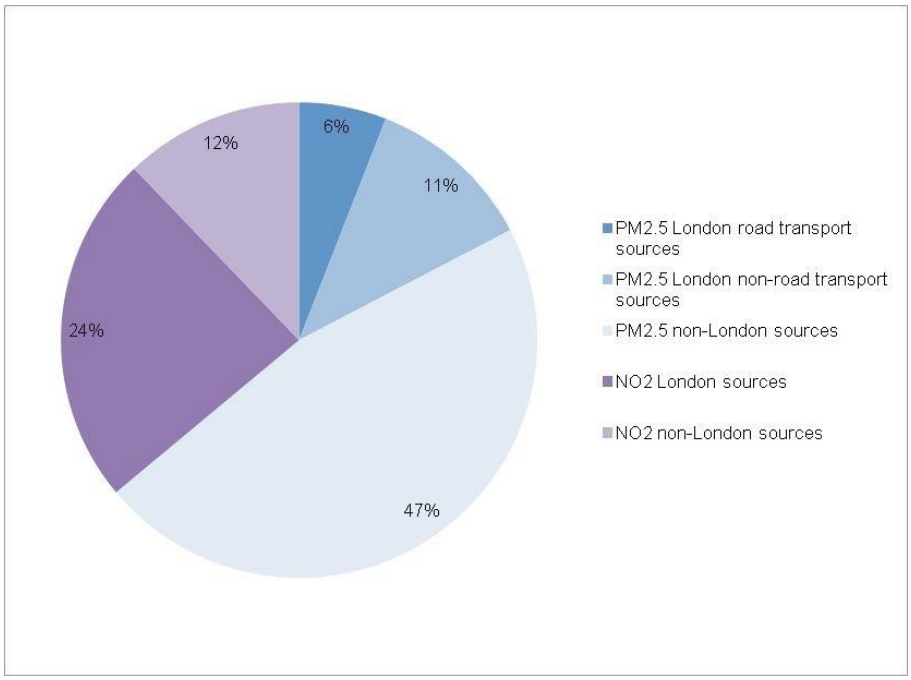


Figure 3 Apportionment of the effects of short-term exposure (deaths brought forward) to 2010 levels of $PM_{2.5}$ and NO_2 to emissions sources

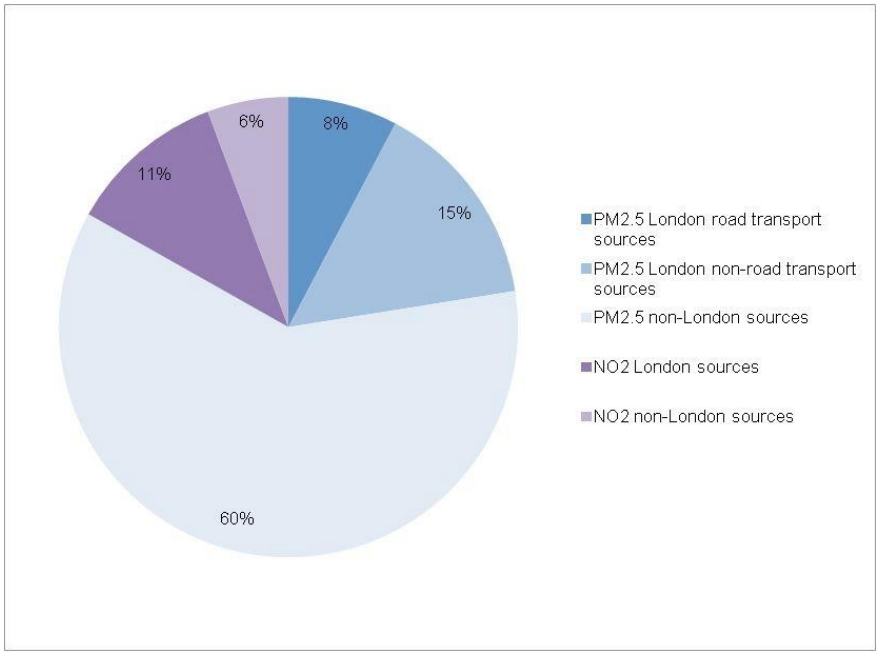


Figure 4 Apportionment of the effects of short-term exposure (respiratory hospital admissions) of 2010 levels of $PM_{2.5}$ and NO_2 to emissions sources

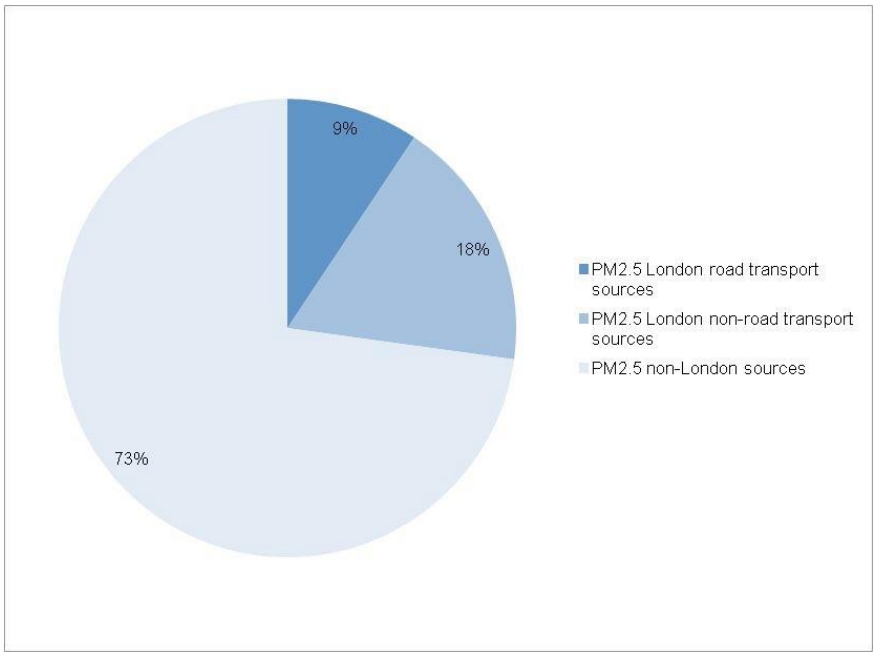


Figure 5 Apportionment of the effects of short-term exposure (cardiovascular hospital admissions) of 2010 levels of $PM_{2.5}$ to emissions sources

3 Understanding recent trends and the future impact of $PM_{2.5}$ and NO_2 in London on health

3.1 Recent trends and the impact of $PM_{2.5}$ and NO_2 in London on health

3.1.1 Introduction

Section 2 examined the burden of mortality attributable to 2010 levels of anthropogenic $PM_{2.5}$ or NO_2 in 2010 but, of course, the pollutants are present every year, and in addition the concentrations fluctuate from year to year. Some of this fluctuation is due to different weather conditions, but over a longer time period as emissions are reduced, concentrations should also reduce and doing so, lessen health impacts.

In examining long-term exposure, it is most appropriate to use the full life table impact methodology as this can take the sequential changes from year to year into account. In addition, follow-up is needed over a lifetime (105 years). This is because survivors from a pollution reduction can die decades later, and the life years lost or gained cannot be counted until the deaths in these survivors have occurred at the relevant later date. Use of the life table methodology also allows the lag between exposure and effect to be taken into account.

A variety of assumptions need to be made about the starting baseline mortality rate and whether future mortality rate and population size changes, for reasons other than pollution, should be taken into account. In addition, air pollution concentrations may not have been fully modelled in every year. In estimating the changes in life years as a result of recent trends in concentrations of anthropogenic $PM_{2.5}$ and of NO_2, we have used the concentrations for the years where full modelling was available. Concentrations for 2010 and beyond were projected from 2010 emissions as the 2010 emissions inventory was the most recent available – thus, although 2012 is a past year, it was still based on projections from 2010. This is discussed further in section 3.1.2.1. For simplicity, and to isolate the changes as a result of pollution we used the 2008 population and mortality rates as a starting point and assumed that the mortality rates were unchanged in the baseline going forward. Any population and mortality rate changes in the pollution scenarios were a result only of the pollution changes.

3.1.2 Input data and method for the impact of recent trends in $PM_{2.5}$ and NO_2 (2008-2012) on health and mortality in London

3.1.2.1 Processing of Input data

Modelled concentrations: 2010 PM_{10}, $PM_{2.5}$ and NO_2 concentrations were as in section 2.1. PM_{10}, $PM_{2.5}$ and NO_2 annual mean concentrations were extracted from the LAEI2010 for the year 2008 and 2012 air quality results and processed as above for section 2.1.2.1 to produce population-weighted average concentrations. Note that the year 2008 and 2010 were fully validated and modelled using their respective meteorology data, i.e. 2008 and 2010, while the year 2012 was projected forward from 2010 using the LAEI2010's most recent meteorology, i.e.

2010. 2009 concentrations were assumed to be as in 2008; 2011 as in 2010 and concentrations beyond 2012 as in 2012.

Population data: As provided by GLA demographics and averaged for 2007/2008/2009 to represent 2008, then adjusted according to the life table for future years. This approach will also include new birth cohorts assuming the numbers of new births as in the 3 year average based 2008 life table - people born after the start of the pollution reductions will benefit from lower levels of pollution maintained into the future.

Mortality data: Life tables with London specific mortality rates in 1 year age groups have been used, averaged over the years 2007/2008/2009 and allocated as the starting point in 2008 (see section 2.1.4). These mortality rates were assumed to apply in future years.

Population-weighted average concentrations: average concentration in each OA multiplied by the total population age 30+ by gender within each individual OA furthermore summed across London and divided by the total population age 30+ by gender in London. The 2008 population-weighted average concentration used 2007/2008/2009 average population data; the 2010 and 2012 population-weighted average concentration used 2009/2010/2011 averaged population data. The latter was done to provide consistency with the future trends calculations (section 3.2) which used 2009/2010/2011 averaged population data for population-weighting of concentrations projected forward from 2010.

3.1.2.2 Calculations

The method for calculating mortality burdens described in section 2.1 provides an approximate snapshot of an effect in 1 year but ignores the effect of $PM_{2.5}$ (and NO_2) in previous years, other than to assume the concentrations were the same in previous years. A more sophisticated approach recommended by COMEAP (2010) for changes in pollutant concentrations takes into account the fact that the level of pollution in 2008 and the resulting deaths will change the baseline population for 2010 and indeed for 2012 as well. This is an updated version of the approach taken in the economic analysis of the Air Quality Strategy (IGCB, 2007).

Our approach to the calculation of future health impacts is to start in 2008 and feed in changes in the size and age structure of the population from year to year; adjusting the mortality rates according to the projected concentrations of anthropogenic $PM_{2.5}$ (and NO_2) in 2010 and 2012. Essentially, this approach combines the health benefits of changes in pollution between 2008 and 2012.

We considered different ways of defining the scenarios for comparison. It might seem at first that an increase in pollution equivalent to the level of anthropogenic $PM_{2.5}$ in 2008 over and above a baseline mortality rate without pollution should be calculated. However, the baseline mortality rate without pollution is not known because in real life, the baseline mortality rate includes the effect of pollution. Hence, the impact for the counterfactual scenario was actually calculated as the impact on the baseline mortality rate of removing an amount of anthropogenic $PM_{2.5}$ equivalent to the level in 2008, for 105 years beyond 2008. This gives a gain in life years

that was then taken to be equivalent to the loss of life years as a result of 2008 levels of anthropogenic $PM_{2.5}$ sustained for 105 years[52].

This counterfactual scenario (scenario 1) was then compared with a second scenario in which the effects of changes in pollution for 2010 and 2012 on the life years were calculated by changing the mortality rate in accordance with removing 2008 concentrations in 2008/9, 2010 concentrations in 2010/11, and then 2012 concentrations each year until 2112.

The difference in life years between the 2 scenarios was then taken as the mortality impact of recent trends in anthropogenic $PM_{2.5}$.

This approach is illustrated Figure 6.

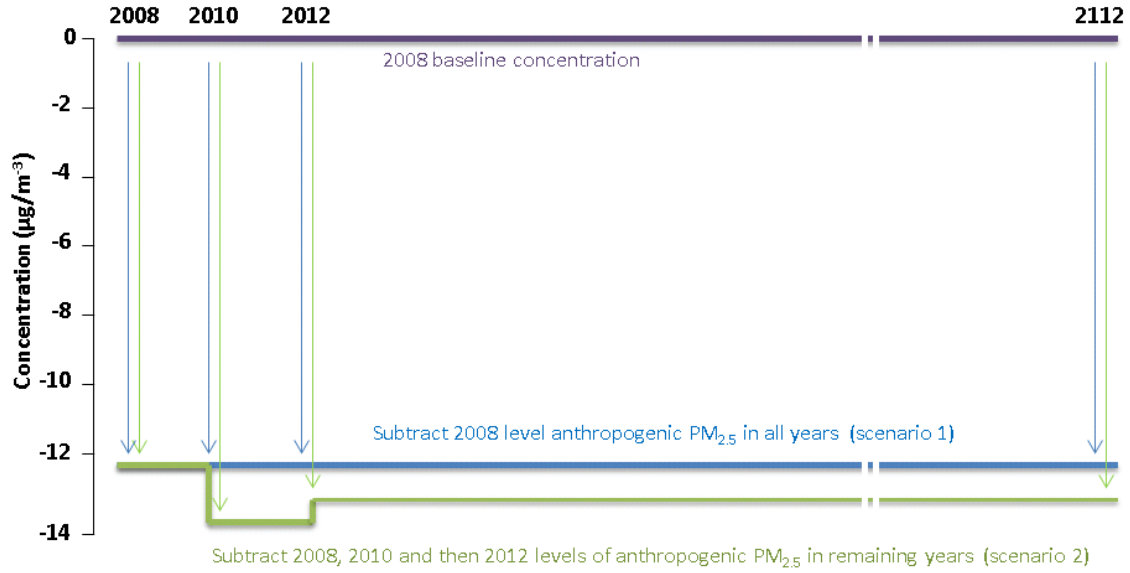


Figure 6 Recent trends and counterfactual scenarios for population-weighted anthropogenic $PM_{2.5}$ (example for males, 30+)

[52] Strictly for a log-linear relationship, a decrease in concentration does not give the same result as for an increase in concentration as the curve changes shape when moving up or down. However, this had to be set against using current baseline mortality rates as if they did not include the effects of current pollution, which they do.

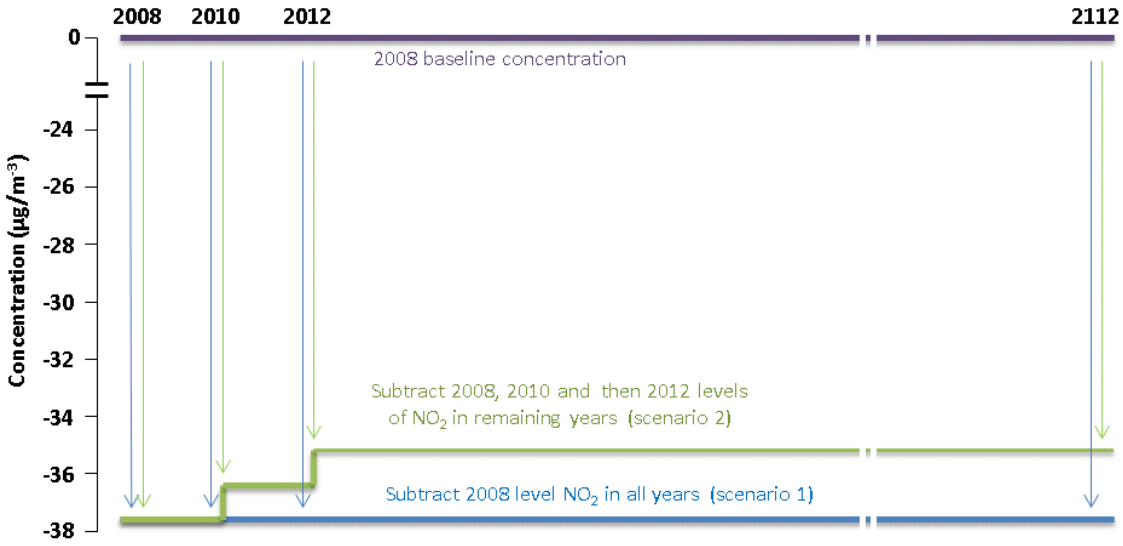


Figure 7 Recent trends and counterfactual scenarios for population-weighted NO_2 (example for males, 30+)

An analogous approach was taken for NO_2. As the end result was a small difference between two scenarios, representing small differences in NO_2 population-weighted annual average concentrations for which no output areas were below 20 µg m^{-3}, we did not need to take a counter-factual at 20 µg m^{-3} (or other concentration) into account. (The removal of 2008 levels in the counterfactual scenario for example, was regarded as a conceptual analytical mechanism to ultimately derive the small difference between the two scenarios). The approach for NO_2 is illustrated in Figure 7. (Note break in y-axis).

Follow-up: Life tables were run through from 2008 to 2112 - this is important because those that survive as a result of reduced pollution could survive for many years and the years of life saved cannot be counted fully without modelling the future time patterns of deaths of the survivors.

Counterfactual: A baseline scenario in which 2008 concentrations are subtracted, representing 2008 concentrations remaining unchanged over time.

Delay between exposure and effect: The approach allowed for a delay between exposure and effect using the recommended distribution of lags from COMEAP (2010) and recommended by the US EPA (see section 2.1.4.1). An analogous approach was used for the effects of long-term exposure to NO_2. HRAPIE recommended that, in the absence of information on likely lags between long-term exposure to NO_2 and mortality, calculations should follow whatever lags are chosen for $PM_{2.5}$.

3.1.3 Results of the impact of recent $PM_{2.5}$ and NO_2 trends (2008-2012) on health and mortality in London

3.1.3.1 Effects of the changes in $PM_{2.5}$ and NO_2 from 2008 to 2012 on total life years

The population-weighted average concentrations used are shown in Table 10. Concentrations of $PM_{2.5}$ increased slightly from 2008 to 2010, then decreased to 2012, albeit still above that in 2008. For NO_2 there have been ongoing reductions since 2008.

Table 10 Population-weighted average concentration (PWAC) for the population aged 30 and over of anthropogenic $PM_{2.5}$ and total NO_2 (μg m^{-3})

Year	Anthropogenic $PM_{2.5}$ PWAC (μg m^{-3}) male	Anthropogenic $PM_{2.5}$ PWAC (μg m^{-3}) female	Total NO_2 PWAC (μg m^{-3}) male	Total NO_2 PWAC (μg m^{-3}) female
2008-2009	12.43	12.37	37.85	37.4
2010-2011	13.75	13.69	36.63	36.21
2012-2112	13.29	13.23	34.87	34.47

The population-weighted average concentration $PM_{2.5}$ changes in Table 10 give an estimate of around 478,414 life years lost rather than gained as a result of recent $PM_{2.5}$ trends (Table 11) followed up to 2112. The ongoing reductions in NO_2 since 2008, give an estimate of up to around 1,062,063 life-years gained as a result of recent trends in NO_2 followed up to 2112, assuming some overlap with the effects of $PM_{2.5}$. Up to around 1,483,070 life years have or will be gained if no overlap were assumed.

The same issues apply to adding the effects of $PM_{2.5}$ and NO_2 as discussed in section 2.1.3.4. In total, the life years saved as a result of recent trends in $PM_{2.5}$ and NO_2 in London followed up to 2112 were estimated as probably between 478,414 life years lost (WHO 'limited set' covering $PM_{2.5}$ only) and 583,649 life years gained (WHO 'extended set' including both $PM_{2.5}$ and NO_2, assuming a 30% overlap between their effects) or even up to 1,004,656 life years gained if there was no overlap[53].

[53] These numbers are illustrative because if the change in risk from changes in $PM_{2.5}$ and NO_2 concentrations had been put into the same life table the answer would be different to some extent. (The risks from each pollutant would change the population size and age distribution which in turn would influence the effect of the other pollutant.)

Table 11 Total life years saved over time as a result of the changes in pollution from 2008 to 2012 then sustained to 2112; with new birth cohorts; EPA lag compared with 2008 concentrations maintained over time

Gender	Life Years gained Anthropogenic $PM_{2.5}$	Life Years gained Total NO_2 (accounting for overlap)	Life Years gained Total NO_2 (assuming no overlap)
Female	-226,019	495,180	690,926
Male	-252,395	566,884	792,144
Total	-478,414	1,062,063	1,483,070

It is important to emphasise that the life years lost or life years gained are spread over a long time period, both because there is a lag of up to 20 years for a proportion of the direct effect to show as changes in mortality and because, even after this, mortality changes as a result of the indirect effects on the size and age structure of the population. The distribution of the above totals over time, expressed as the difference between the cumulative life years lost for each scenario (also shown), is demonstrated in the following Figure 8, Figure 9 and Figure 10 for males (m) and females (f) combined (note differences in scale between figures). (Although calculations were originally for a gain in life years from reductions equivalent to e.g. 2008 concentrations, the results are expressed here as the impact on life years lost i.e. the adverse impact for scenarios 1 and 2). It is worth referring back to the diagrams of the scenarios in interpreting these graphs, as, for example, the scenario in which 2008 concentrations remain the same is contrasted with the scenario in which 2012 concentrations are maintained beyond 2012. This is appropriate for isolating the 2008-2012 trend but ignores any improvements beyond 2012.

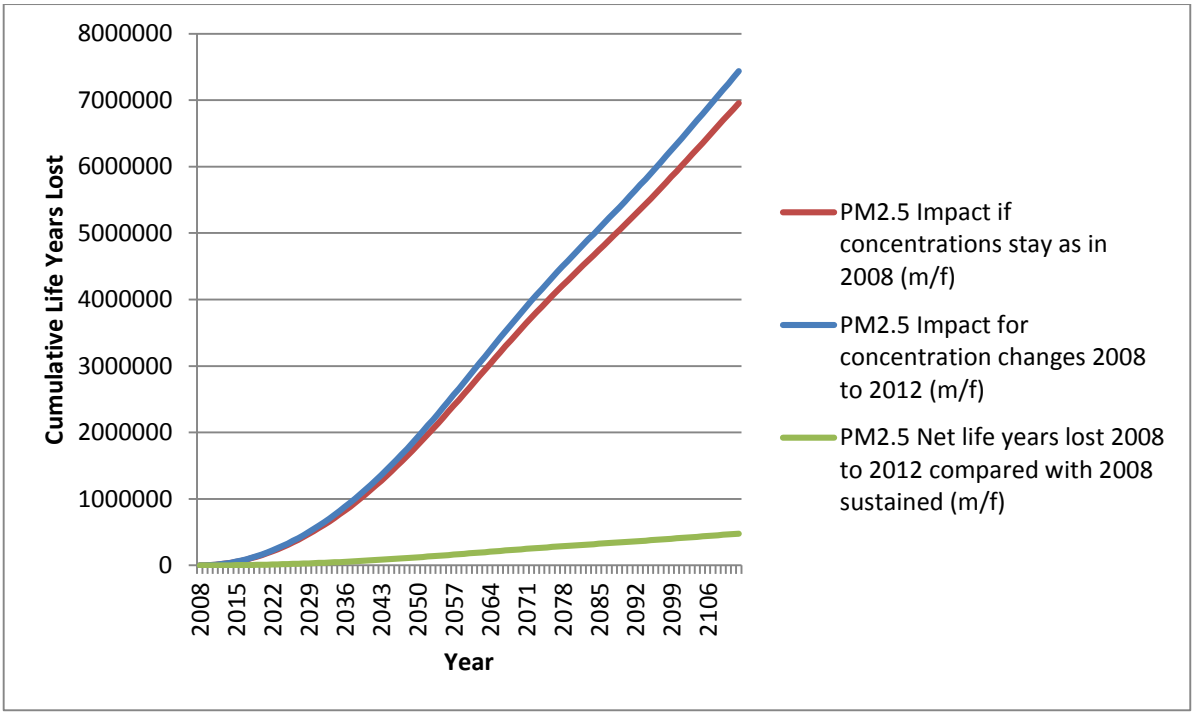


Figure 8 Impact of $PM_{2.5}$ concentration changes 2008-2012, compared with 2008 concentrations maintained over time

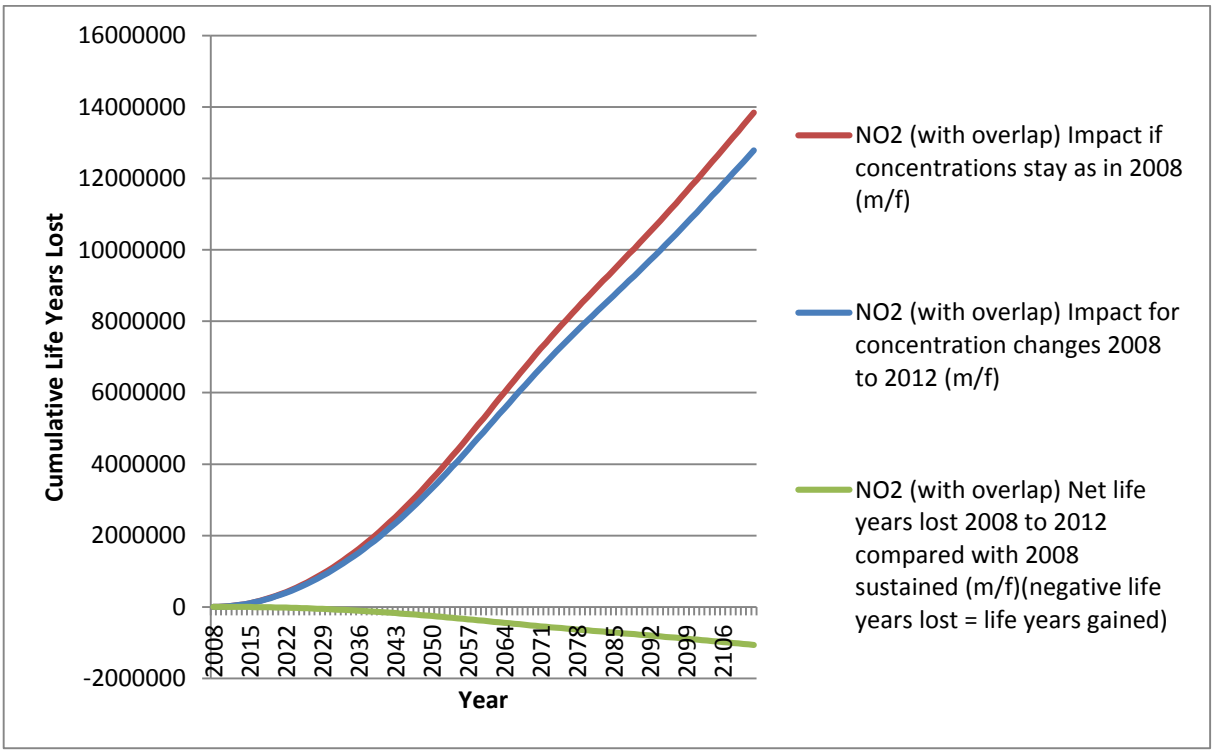


Figure 9 Impact of NO_2 concentration changes 2008-2012, compared with 2008 concentrations maintained over time (with overlap)

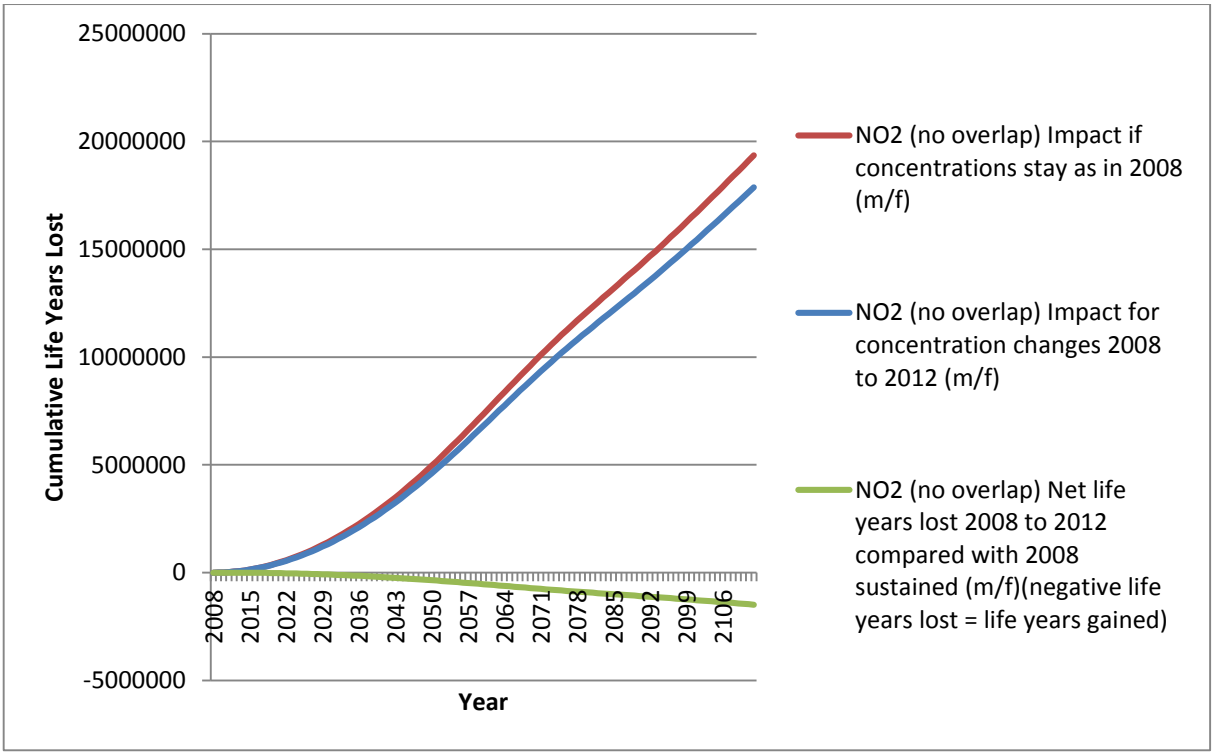


Figure 10 Impact of NO_2 concentration changes 2008-2012, compared with 2008 concentrations maintained over time (no overlap)

3.1.3.2 Effects of the changes in $PM_{2.5}$ and NO_2 from 2008 to 2012 on numbers of deaths in specific years (2008, 2010 and 2012)

The mortality burden in section 2.1 was expressed in terms of attributable deaths and it might be wondered why this has not been an output in this section so far. This is because life years are a more appropriate expression of the effect when considering effects over time. The numbers of deaths changes from year to year for a combination of reasons such as the lag between exposure and effect and changes in the size and age structure of the population and, in the long-term everyone in the population will die leaving no difference between scenarios. Figures for numbers of deaths in specific years can nonetheless be extracted from life tables and are shown for $PM_{2.5}$ in Table 12 below. This is to illustrate the issues with numbers of deaths.

Considering the third column first, this shows that even when the level of $PM_{2.5}$ is set to stay at the 2008 level the numbers of deaths in specific future years still change. There is an increase in 2010 and 2012 compared with 2008. The main driver for this is the lag between exposure and effect - about 80% of the effect from a change in 2008 has occurred by 2012, and the effect in 2012 also includes partial effects from lagged effect of the years between 2008 and 2012. After that there is a decline. This is because following an increase in numbers of deaths, the size of the population decreases and contains fewer older people (as they have already died). Smaller, younger populations have fewer deaths and this starts to cancel out the increased deaths due to the pollution.

In the fifth column, the effect of changes in $PM_{2.5}$ concentrations is superimposed on this effect. As the $PM_{2.5}$ concentrations are higher in 2010 and 2012 than 2008, the numbers of deaths are higher. Because of the lag between exposure and effect, the decline in concentration between 2010 and 2012 does not start to be apparent in the difference in the numbers of deaths between scenarios until 2020 (of the years chosen to present here).

These points illustrate why it is not appropriate to give a 'per year' figure for deaths as even in column 3 where the concentration is the same, the number of deaths is not the same from year to year. While the impact of the pollution on the hazard rate stabilizes once the lag has worked through, the resulting effect of the changes in the size and age distribution of the population continues for an extended period of time.

It will be noted that estimated numbers of deaths for 2010 is not the same as in the burden calculations in section 2.1. This is because (i) the burden calculations either assume no lag or assume pollution levels have been constant at 2010 levels previously, (ii) in this example 2010 mortality rates were projected forward in the life table from the 2007/8/9 mortality rates (iii) the life table approach takes into account ongoing changes in the size and age distribution of the population.

Table 12 Numbers of deaths in specific years as a result of the changes in $PM_{2.5}$ from 2008 to 2012

Year	$PM_{2.5}$ PWAC µg m^{-3} male/female with 2008 level of pollution maintained over time	Numbers of deaths in relevant year with 2008 level of pollution maintained over time	$PM_{2.5}$ PWAC µg m^{-3} male/female with 2008, 2010 then 2012 level of pollution maintained over time	Numbers of deaths in relevant year with 2008, 2010 then 2012 level of pollution maintained over time	Difference (column 3 subtracted from column 5)
2008	m 12.43/ f 12.37	967	m 12.43/ f 12.37	967	0
2010	m 12.43/ f 12.37	1698	m 13.75/ f 13.69	1804	106
2012	m 12.43/ f 12.37	2396	m 13.29/ f 13.23	2542	146
2015	m 12.43/ f 12.37	2226	m 13.29/ f 13.23	2399	174
2020	m 12.43/ f 12.37	2060	m 13.29/ f 13.23	2205	145

The same general comments apply to the numbers of deaths in specific years for NO_2. Again, where concentrations remain at 2008 levels (third column) the numbers of deaths in specific future years still change, increasing in 2010 and 2012 compared with 2008 before declining again. Again, the main driver for the initial increase is the lag between exposure and effect, followed by a decrease because the increase in numbers of deaths in the earlier years, decreases the size of the population and the number of older people (as they have already died). Smaller, younger populations have fewer deaths and this starts to cancel out the increased deaths due to the pollution.

In contrast to the results for $PM_{2.5}$ however, the decreases in NO_2 concentrations in 2010 and 2012 blunts the increase due to the lag (Table 13, column 5) so that the difference between the two scenarios shows a continuous decrease (column 6). Table 13 assumes a 30% overlap. Table 14 assumes no overlap – the numbers are different but the pattern is the same.

These results are also different to the burden results in section 2.1 for the same reasons as for $PM_{2.5}$.

Table 13 Numbers of deaths in specific years as a result of the changes in NO_2 from 2008 to 2012 (RR 1.039)

Year	NO_2 PWAC µg m^{-3} male/female with 2008 level of pollution maintained over time	Numbers of deaths in relevant year with 2008 level of pollution maintained over time RR 1.039	NO_2 PWAC µg m^{-3} male/female with 2008, 2010, then 2012 level of pollution maintained over time	Numbers of deaths in relevant year with 2008, 2010, then 2012 level of pollution maintained over time RR 1.039	Difference (column 3 subtracted from column 5)
2008	m 37.85/ f 37.4	1908	m 37.85/ f 37.4	1908	0
2010	m 37.85/ f 37.4	3331	m 36.63/ f 36.21	3269	-62
2012	m 37.85/ f 37.4	4680	m 34.87/ f 34.47	4482	-199
2015	m 37.85/ f 37.4	4380	m 32.84/ f 32.47	4033	-348
2020	m 37.85/ f 37.4	4084	m 28.38/ f 28.38	3732	-352

Table 14 Numbers of deaths in specific years as a result of the changes in NO_2 from 2008 to 2012 (RR 1.055)

Year	NO_2 PWAC µg m^{-3} male/female with 2008 level of pollution maintained over time	Numbers of deaths in relevant year with 2008 level of pollution maintained over time RR 1.055	NO_2 PWAC µg m^{-3} male/female with 2008 level of pollution maintained over time	Numbers of deaths in relevant year with 2008, 2010, then 2012 level of pollution maintained over time RR 1.055	Difference (column 3 subtracted from column 5)
2008	m 37.85/ f 37.4	2648	m 37.85/ f 37.4	2648	0
2010	m 37.85/ f 37.4	4603	m 36.63/ f 36.21	4519	-84
2012	m 37.85/ f 37.4	6447	m 34.87/ f 34.47	6178	-269
2015	m 37.85/ f 37.4	6067	m 32.84/ f 32.47	5593	-474
2020	m 37.85/ f 37.4	5689	m 28.38/ f 28.38	5203	-486

3.1.4 Effects of the changes in PM_{10}, $PM_{2.5}$ and NO_2 from 2008 to 2012 on hospital admissions and deaths brought forward

The methods described in section 2.2 for assessing the effects of short-term exposure to pollution were applied to the population-weighted average concentrations for anthropogenic $PM_{2.5}$ and NO_2 in Table 10 for the years 2008, 2010 and 2012. Input data such as the total population (not 30+)-weighted average concentration for all pollutants and all years as well as the total population (not 30+) and the baseline number of death brought forward and hospital admissions in London can be found in Annex 5. Population data and deaths brought forward for 2008, and hospital admissions data for 2008/9 were used for the year 2008. Population data and deaths brought forward for 2010, and hospital admissions data for 2010/11 were used for the year 2010 and for the subsequent years 2012, 2015, 2020.

Unlike for the effects of long-term exposure, no carry-over of effects from year to year needs to be considered.[54] As the effects are much smaller than for long-term exposure and are a tiny

[54] The deaths brought forward are assumed only to change the timing of deaths within one particular year. In practice, it is unknown whether more than one life year is lost for each death brought forward due to the seasonal adjustments used in time-series studies (this removes longer term trends to remove changes in deaths due to season in order to focus on

proportion of overall baseline rates, the effect of pollution within current baseline rates is ignored. Effects were thus calculated as increases.

For short-term exposure to anthropogenic $PM_{2.5}$, deaths brought forward should not be added to deaths from long-term exposure to $PM_{2.5}$ to avoid double-counting, so are not given here. The results are however given in Annex 5, as are results for total $PM_{2.5}$ and PM_{10}.

The results for anthropogenic $PM_{2.5}$ and hospital admissions are given in Table 15. The trend in respiratory hospital admissions in London has increased from 1,658 in 2008 to 1,992 in 2010 before declining slightly to 1,924 in 2012. Similarly, cardiovascular hospital admissions increased from 654 in 2008 to 740 in 2010 before declining slightly to 715 in 2012. There was an increase in population from 2008 to 2010.

Deaths brought forward, as a result of short term exposure to NO_2, declined from 499 in 2008, to 461 in 2010 to 439 in 2012 (Table 15). In this case, the larger concentration-response coefficient for NO_2 combined with the decline in baseline death rate between 2008 and 2010, and the decrease in concentration, meant that despite the increase in population there was a reduction in the impact of this pollutant on deaths brought forward.

NO_2 associated respiratory hospital admissions in London increased from 398 in 2008 to 419 in 2010 as the reduction in NO_2 concentrations was not sufficient to offset the increase in population and in the baseline rate for respiratory hospital admissions. The result did decline again to 398 respiratory hospital admissions in 2012.

short-term change). Hospital admissions may also be brought forward rather than additional, but again it is unknown whether and to what degree this is the case.

Table 15 Effects on hospital admissions and deaths brought forward for the year 2008, 2010 and 2012 for anthropogenic $PM_{2.5}$ and NO_2

Pollutant	Year	Central Relative Risk (RR) with lower and upper 95% confidence interval per 10 μg m^{-3}	Numbers of hospital admissions or deaths brought forward
Anthropogenic $PM_{2.5}$	2008	Respiratory Hospital Admissions	1658 (-157 – 3518)
	2010	(RR 1.019 (0.9982 – 1.0402))	1992 (-188 – 4232)
	2012		1924 (-182 – 4085)
Anthropogenic $PM_{2.5}$	2008	Cardiovascular Hospital Admissions	654 (122 – 1194)
	2010	(RR 1.0091 (1.0017 - 1.0166))	740 (138 – 1352)
	2012		715 (133 – 1306)
NO_2	2008	Deaths Brought Forward*	499 (295 – 704)
	2010	(RR 1.0027 (1.0016 - 1.0038))	461 (272 – 650)
	2012		439 (260 – 618)
NO_2	2008	Respiratory Hospital Admissions	399 (-212 – 1014)
	2010	(RR 1.0015 (0.9992 - 1.0038))	419 (-223 – 1064)
	2012		398 (-212 – 1012)

*Not to be added to life years gained from long-term exposure to NO_2 and mortality

3.2 Impacts of future trends in $PM_{2.5}$ and NO_2 in London

3.2.1 Input data and method of future impacts of $PM_{2.5}$ and NO_2 (2012, 2015 and 2020) on health and mortality in London

3.2.1.1 Processing of Input data

Modelled concentrations: PM_{10}, $PM_{2.5}$ and NO_2 annual mean concentrations were extracted from the LAEI2010 projections for the year 2015 and 2020 air quality results and processed as in section 2.1.2.1 to produce population-weighted average concentrations. 2013 and 2014 concentrations will be assumed to be as in 2012, derived as described previously; 2016-2019 concentrations as in 2015 and concentrations beyond 2020 as in 2020.

Population data: Provided as above and averaged for 2009/2010/2011 to represent 2010, then adjusted according to the life-table. The life table approach below includes new birth cohorts each year assuming the numbers of new births as in the 3 year average based 2010 life table - people born after the start of the pollution reductions will benefit from lower levels of pollution maintained into the future.

Mortality data: Life tables with mortality rates as above but averaged over the years 2009/2010/2011 and allocated as the starting point in 2010. These mortality rates were assumed to apply in future years.

Population-weighted average concentrations: As in section 3.1.2.1. The population-weighted average concentration used 2009/2010/2011 averaged population data for all years, meaning that the trends in population-weighted concentrations are driven by the changes in modelled concentrations.

3.2.1.2 Calculations

Our approach to the calculation of future health impacts is to start in 2010 and feed in changes in the size and age structure of the population from year to year, adjusting the mortality rates according to the projected concentrations of anthropogenic $PM_{2.5}$ (and NO_2) in 2012, 2015 and 2020. Essentially, this approach combines the health benefits of improvements in pollution between 2010 and 2020 but can still give outputs specific to 2012, 2015 and 2020.

Calculating the health impact of projected future trends in pollution was undertaken by comparing two scenarios in the same way as for the analysis of the recent trends. The first was a scenario in which 2010 levels of pollution were removed representing the effect of pollution remaining at 2010 levels for the next 105 years. The second was a scenario in which the effects of projected changes in pollution for 2012, 2015 and 2020 were calculated assuming a reduction equivalent to 2010 concentrations in 2010-11, 2012 concentrations in 2012-2014, 2015 concentrations in 2015-2019 and 2020 concentrations until 2114.

As before, each scenario consisted of a reduction equivalent to the relevant overall level of anthropogenic $PM_{2.5}$ or NO_2 from the baseline rate including effects of pollution. The difference between the two scenarios which were both decreases was then reversed to be a difference between two increases from a hypothetical baseline rate with no pollution.

This approach is illustrated in Figure 11 and Figure 12. (Note break in axis in Figure 12).

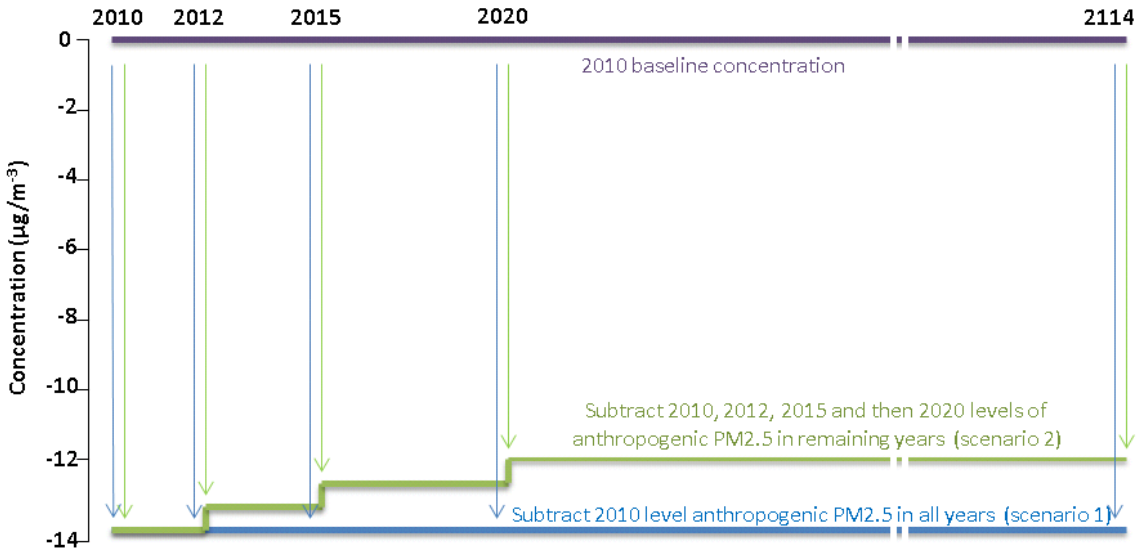


Figure 11 Future trends and counterfactual scenarios for population-weighted anthropogenic $PM_{2.5}$ (example for males, 30+)

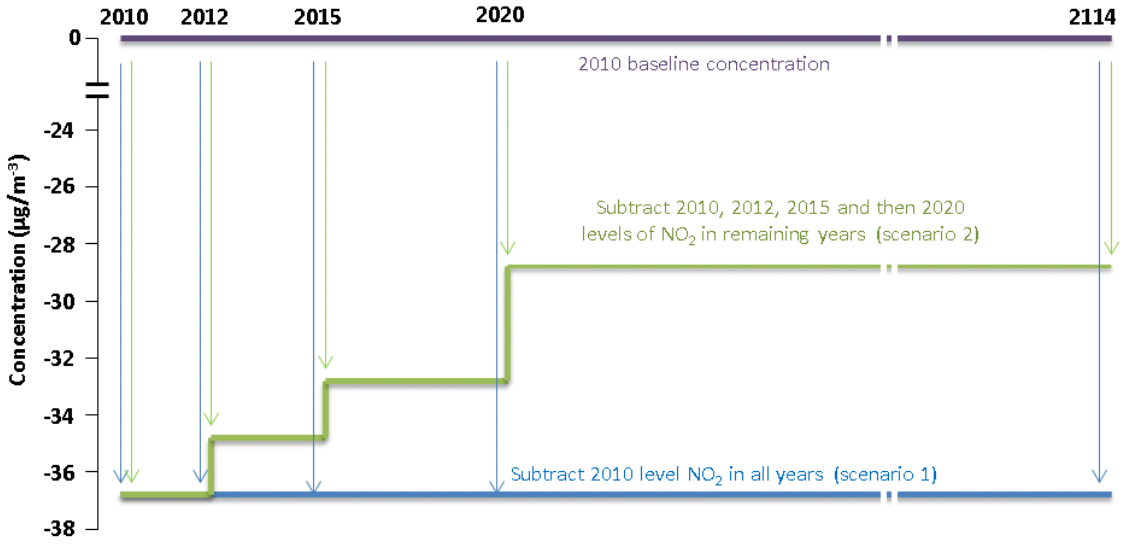


Figure 12 Future trends and counterfactual scenarios for population-weighted anthropogenic NO_2 (example for males, 30+)

Follow-up: Life tables were run through from 2010 to 2114 as discussed above.

Counterfactual: A baseline scenario in which 2010 concentrations were removed representing 2010 concentrations remaining unchanged over time.

Delay between exposure and effect: The approach allowed for a delay between exposure and effect using the recommended distribution of lags from COMEAP (2010) and recommended by the US EPA (see section 2.1.4.1). An analogous approach was used for the effects of long-term exposure to NO_2.

3.2.2 Results of future impacts of $PM_{2.5}$ and NO_2 (2012, 2015 and 2020) on health and mortality in London

3.2.2.1 Effects of the improvements in $PM_{2.5}$ and NO_2 from 2012 to 2020 on total life years

'Snapshots' of numbers of deaths in 2012, 2015 and 2020 will be provided later. This section first gives the total life years saved over time as a result of the improvements in pollution from 2012 to 2020 as this is the preferred metric.

The population-weighted average concentrations were projected to improve from 2010 to 2020 for both $PM_{2.5}$ and NO_2 (Table 16).

Table 16 Population-weighted average concentration (PWAC) for the population aged 30 and over of anthropogenic $PM_{2.5}$ and total NO_2 (μg m^{-3})

Year	Anthropogenic $PM_{2.5}$ PWAC (μg m^{-3}) male	Anthropogenic $PM_{2.5}$ PWAC (μg m^{-3}) female	Total NO_2 PWAC (μg m^{-3}) male	Total NO_2 PWAC (μg m^{-3}) female
2010-2011	13.75	13.69	36.63	36.21
2012-2014	13.29	13.23	34.87	34.47
2015-2019	12.81	12.75	32.84	32.47
2020-2114	12.09	12.05	28.38	28.08

For anthropogenic $PM_{2.5}$ it was estimated that these projected changes resulted in a gain of 901,466 life-years compared with levels remaining the same as in 2010 (estimated to lead to to 7,853,982 life years lost). For NO_2 the gains were substantially larger, from up to 2,919,741 life years assuming a 30% overlap with $PM_{2.5}$ up to about 4 million life years gained, assuming no overlap with $PM_{2.5}$ (Table 17). This compares with an estimate of up to 13,677,155 life years lost, assuming a 30% overlap with $PM_{2.5}$, if 2010 levels of NO_2 were not reduced for this time period. For context, the total life years lived for the whole population, followed up for 105 years, including new birth cohorts, is over a billion (1,019,644,053).

Table 17 Total life years saved over time as a result of the improvements in pollutant concentrations from 2012 to 2020, then sustained to 2114; with new birth cohorts; EPA lag compared with 2010 concentrations maintained over time

Gender	Life Years gained Anthropogenic $PM_{2.5}$	Life Years gained Total NO_2 (accounting for overlap)	Life Years gained Total NO_2 (assuming no overlap)
Female	422,576	1,364,421	1,904,560
Male	478,890	1,555,320	2,173,678
Total	901,466	2,919,741	4,078,237

The cumulative life years gained over time are given in Figure 13, Figure 14 and Figure 15.

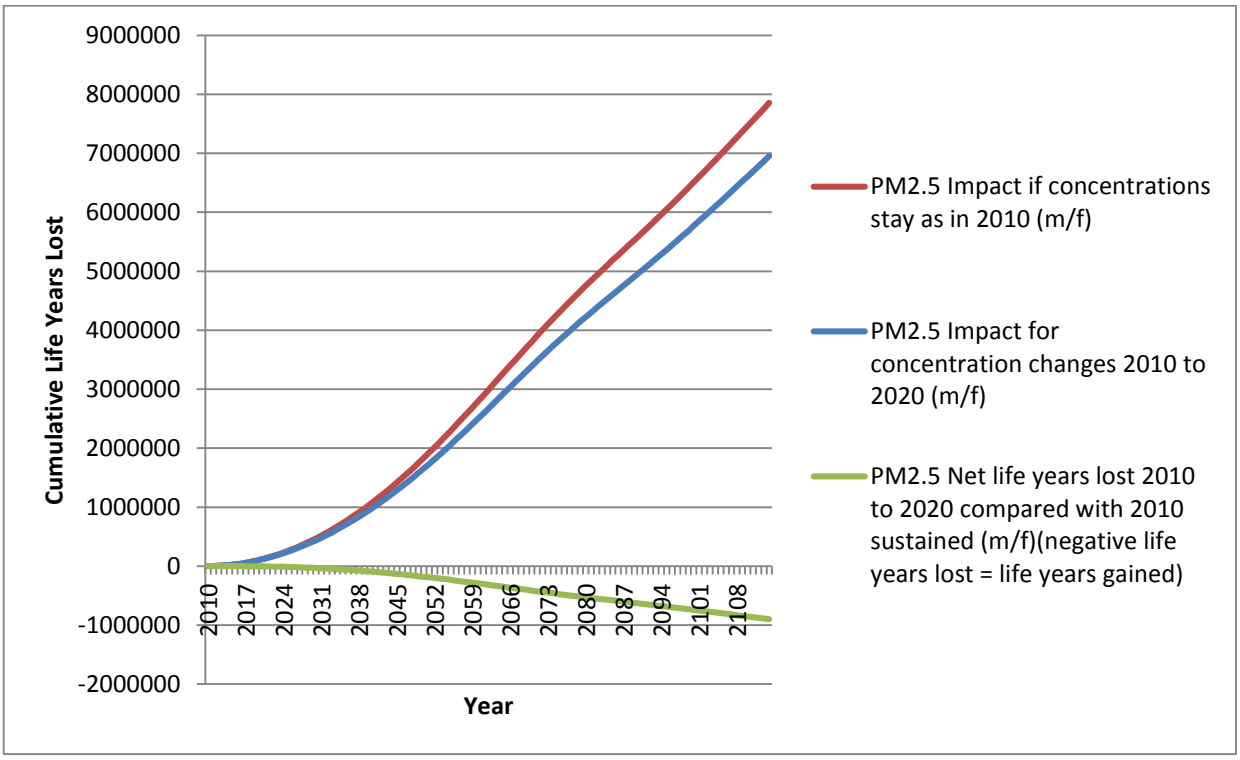


Figure 13 Impact of $PM_{2.5}$ concentration changes 2010-2020, compared with 2010 concentrations maintained over time

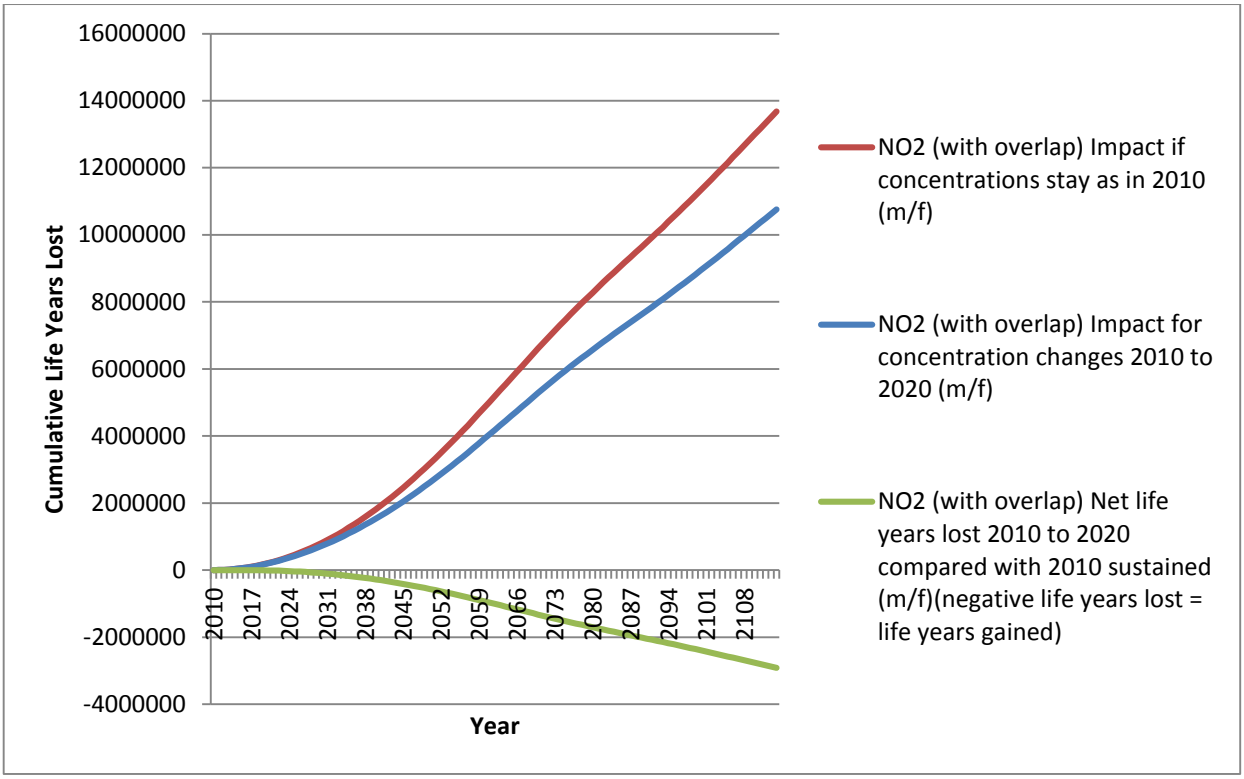


Figure 14 Impact of NO_2 concentration changes 2010-2020, compared with 2010 concentrations maintained over time (with overlap)

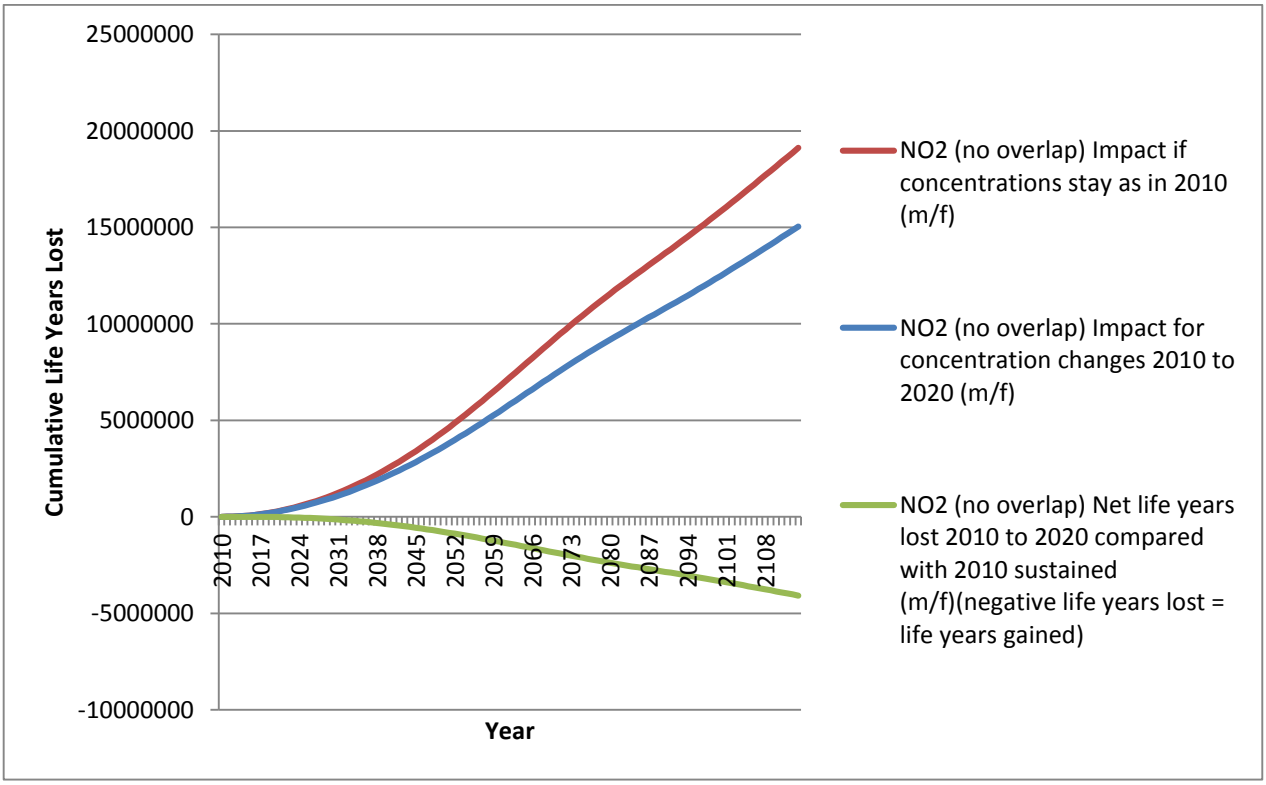


Figure 15 Impact of NO_2 concentration changes 2010-2020, compared with 2010 concentrations maintained over time (no overlap)

3.2.2.2 Effects of the changes in $PM_{2.5}$ and NO_2 from 2010 to 2020 on numbers of deaths in specific years (2010, 2012, 2015 and 2020)

As in the previous trends section, this section gives the numbers of deaths in specific years (Table 18, Table 19, Table 20). These change from year to year for a variety of reasons so are not as good a measure as total life years. As before, for the scenarios where 2010 levels remain unchanged going forward, the deaths build up as the lag between exposure and effect phases in and declines as the effect of previous increased deaths on the population and age distribution starts to counter the effects of the pollution (Third column of tables). The initial increase is blunted in column 5 of these tables where a decrease in pollutant concentrations is superimposed on keeping the concentration steady, resulting in fewer deaths in column 5 (future reductions) than column 3 and a greater reduction in deaths over time for these specific years.[55] NB As 2010 rather than 2008 mortality rates are used at the start, it is expected that the numbers of deaths are not the same in these tables as in the previous ones for recent trends.

Table 18 Numbers of deaths in specific years as a result of the changes in pollution in $PM_{2.5}$ from 2010 to 2020 then sustained to 2114; with new birth cohorts; EPA lag (RR = 1.06)

Year	$PM_{2.5}$ PWAC µg m^{-3} male/female with 2010 level of pollution maintained over time	Numbers of deaths in relevant year with 2010 level of pollution maintained over time	$PM_{2.5}$ PWAC µg m^{-3} male/female with 2010, 2012, 2015 then 2020 level of pollution maintained over time	Numbers of deaths in relevant year with 2010, 2012, 2015 then 2020 level of pollution maintained over time	Difference (column 3 subtracted from column 5)
2010	m 13.75/ f 13.69	1030	m 13.75/ f 13.69	1030	0
2012	m 13.75/ f 13.69	1825	m 13.29/ f 13.23	1790	-36
2015	m 13.75/ f 13.69	2543	m 12.81/ f 12.75	2427	-116
2020	m 13.75/ f 13.69	2360	m 12.09/ f 12.05	2110	-250

[55] However, as deaths cannot ultimately be 'saved' this greater reduction over time will reverse, another reason why total life years gained or lost is a better metric.

Table 19 Numbers of deaths in specific years as a result of the changes in NO_2 from 2010 to 2020 then sustained to 2114; with new birth cohorts; EPA lag (RR 1.039)

Year	NO_2 PWAC µg m^{-3} male/female with 2010 level of pollution maintained over time	Numbers of deaths in relevant year with 2010 level of pollution maintained over time RR 1.039	NO_2. PWAC µg m^{-3} male/female with 2010, 2012, 2015 then 2020 level of pollution maintained over time	Numbers of deaths in relevant year with 2010, 2012, 2015 then 2020 level of pollution maintained over time RR 1.039	Difference (column 3 subtracted from column 5)
2010	m 36.63/ f 36.21	1781	m 36.63/ f 36.21	1781	0
2012	m 36.63/ f 36.21	3141	m 34.87/ f 34.47	3054	-87
2015	m 36.63/ f 36.21	4369	m 32.84/ f 32.47	4077	-292
2020	m 36.63/ f 36.21	4087	m 28.38/ f 28.38	3342	-746

Table 20 Numbers of deaths in specific years as a result of the changes in NO_2 from 2010 to 2020 then sustained to 2114; with new birth cohorts; EPA lag (RR 1.055)

Year	NO_2 PWAC µg m^{-3} male/female with 2010 level of pollution maintained over time	Numbers of deaths in relevant year with 2010 level of pollution maintained over time RR 1.055	NO_2 PWAC µg m^{-3} male/female with 2010, 2012, 2015 then 2020 level of pollution maintained over time	Numbers of deaths in relevant year with 2010, 2012, 2015 then 2020 level of pollution maintained over time RR 1.055	Difference (column 3 subtracted from column 5)
2010	m 36.63/ f 36.21	2473	m 36.63/ f 36.21	2473	0
2012	m 36.63/ f 36.21	4343	m 34.87/ f 34.47	4224	-119
2015	m 36.63/ f 36.21	6031	m 32.84/ f 32.47	5634	-397
2020	m 36.63/ f 36.21	5683	m 28.38/ f 28.38	4660	-1023

3.2.3 Effects of the improvements in PM_{10}, $PM_{2.5}$ and NO_2 concentrations from 2012 to 2020 on hospital admissions and deaths brought forward

Effects of the improvements in PM_{10}, $PM_{2.5}$ and NO_2 concentrations from 2012 to 2020 on hospital admissions and deaths brought forward.

3.2.3.1 Effects on hospital admissions and deaths brought forward

Methods were as in section 3.1.4 except that population data and deaths brought forward for 2010, and hospital admissions data for 2010/11 were used for the year 2010 and for the subsequent years 2012, 2015, 2020[56]. Again, total population-weighted average concentration for all pollutants and all years as well as the total population and the baseline number of death brought forward and hospital admissions in London can be found in Annex 5, as can results for anthropogenic $PM_{2.5}$ and deaths brought forward and results for deaths brought forward and hospital admissions for total $PM_{2.5}$ and PM_{10}.

[56] The life tables used starting population and rates from 2010, with only pollutant concentrations contributing to future changes in population and rates. For this reason future population changes and baseline rates for reasons other than pollution were not included in the short-term exposure calculations.

Results are given in Table 21.

For $PM_{2.5}$, respiratory hospital admissions in London were projected to decrease from 1,924 in 2012, to 1,854 in 2015 to 1,749 in 2020. Similarly, cardiovascular hospital admissions were projected to decrease from 715 in 2012, to 689 in 2015, to 650 in 2020.

For NO_2, if an overall core summary is chosen that does not include life years gained from reductions in long-term exposure to NO_2 and mortality, then results for NO_2 and declines in deaths brought forward should be included. These declined from 439 estimated deaths brought forward in 2012, to 413 in 2015 to 355 in 2020. Respiratory hospital admissions in London were projected to decrease from 399 in 2012, to 375 in 2015, to 323 in 2020.

Table 21 Effects on hospital admissions and deaths brought forward for the years 2012, 2015 and 2020 for anthropogenic $PM_{2.5}$ and NO_2

Pollutant	Year	Central Relative Risk (RR) with lower and upper 95% confidence interval per 10 μg m^{-3}	Numbers of hospital admissions or deaths brought forward
Anthropogenic $PM_{2.5}$	2012	Respiratory Hospital Admissions	1924 (-182 – 4085)
	2015	(RR 1.019 (0.9982 – 1.0402))	1854 (-175 – 3934)
	2020		1749 (-165 – 3709)
Anthropogenic $PM_{2.5}$	2012	Cardiovascular Hospital Admissions	715 (133 – 1306)
	2015	(RR 1.0091 (1.0017 - 1.0166))	689 (129 – 1258)
	2020		650 (121 – 1187)
NO_2	2012	Deaths Brought Forward	439 (260 – 618)
	2015	(RR 1.0027 (1.0016 - 1.0038))*	413 (244 – 582)
	2020		355 (210 – 500)
NO_2	2012	Respiratory Hospital Admissions	398 (-212 – 1012)
	2015	(RR 1.0015 (0.9992 - 1.0038))	375 (-200 – 953)
	2020		323 (-172 – 819)

*Not to be added to life years gained from long-term exposure to NO_2 and mortality

4 Developing a robust economic understanding of the costs of air pollution in London (PM and NO_x emissions; PM_{10}, $PM_{2.5}$ and NO_2 concentrations)

4.1 London specific damage costs for PM and NO_x transport emissions

4.1.1 Background

This task focused on the economic valuation of the health impacts of air pollution in London. The economic valuation undertook two tasks. First, it developed new damage costs for transport emissions in London, i.e. new London specific damage costs (section 4.1), which were then used to produce the ready reckoner (section 4.3). Second, it valued the health impacts estimated in section 2.1, i.e. it estimated the current air quality costs of $PM_{2.5}$ and NO_2 in London (section 4.2). The approach for these two tasks built on previous work by the study team for Defra and the Inter-departmental Group on Costs and Benefits (IGCB), and the previous work of the study team for TfL valuing the health benefits of the Low Emission Zone.

4.1.2 The valuation of air pollution impacts

The impacts of air pollution have a number of important economic costs, even if these are not captured directly through market prices. These are known as external costs or externalities - as they are not included in the price of goods or services that lead to air pollution. In the health context, these costs include: resource costs i.e. medical treatment costs; opportunity costs, in terms of lost productivity; and dis-utility i.e. pain or suffering, concern and inconvenience to family and others. The first two components can be captured relatively easily and techniques are also available to capture the third component, by assessing for example the 'willingness to pay'. In the UK Government, there is a recognition that capturing and assessing these health-related economic costs is important, and that they can be used to compare the costs of a proposed air pollution policy against the economic health benefits. The UK Government has advanced the valuation of the health impacts of air pollution, and has provided recommended values for the various health end-points and their use in economic appraisal (e.g. IGCB, 2007; Defra, 2011b; HMT, 2013). These approaches have been used in section 4.2 to provide monetary valuation of the main health impacts of $PM_{2.5}$ and NO_2 concentrations in London.

4.1.3 Methods: Damage costs versus impact assessment

There are a number of possible ways to produce estimates of the economic costs of air pollution or the economic benefits of air pollution reductions from policy. In the UK, two main approaches[57] have been advanced (HMT, 2013 - Valuing impacts on air quality), reflecting a detailed and a simpler (quicker) approach.

The detailed approach is called the **'impact pathway'** approach. This approach involves detailed emission, air quality modelling and health impact assessment, followed by the valuation of the

[57] Note there is also a third approach, where if the proposal is expected to affect compliance with legal limits on air pollution, then the unit abatement cost approach for emission changes exceeding the limit should be used.

health endpoints estimated. This approach is recommended in analysis of national air quality policy, and for local air quality policy in cases where health effects (of air pollution) are greater than £50 million (in present value terms). This approach was applied in section 4.2, to value the burden/impacts estimated in section 2.

However, this approach is resource and time intensive. Therefore, for the appraisal of smaller (sub-national) policy, where total air quality impacts are estimated to be less than £50 million (in present value terms), and do not affect compliance with legal limits on air pollution, the '**damage cost'** approach can be used. This takes the form of simple look-up tables, which provide unit values for health costs (or benefits) of emissions. These are provided (Defra, 2011b: HMT, 2013) as the cost per tonne of pollutant, either as a national average value (for NO_X and SO_2), or split into values for different sources and locations (for PM). For this study, new London-specific transport emission damage costs were derived, in section 4.1, to provide the basis for a new ready reckoner for TfL. It is important to note, however, that these new damage costs were actually derived using the impact assessment approach, using an analysis that built on the section 2 methods and results. This followed the approach used for the existing Defra damage costs, which derives values for long-term mortality for an annual pollution pulse, calculated using the impact pathway method. The simplicity (and approximation) comes in applying these damage costs per tonne in new circumstances without doing further detailed modelling and health impact assessment.

4.1.4 Discounting and Uplifts

The estimation of the health costs of air pollution are complicated by an additional issue, in that time matters. This is because people/society prefer to receive goods and services now rather than later (called 'time preference'). The costs and / or benefits of health effects occurring in different future years therefore have to be adjusted, to make sure they are directly comparable in appraisal. This is undertaken using discounting. The UK Government (HMT, 2011) provides guidance on discounting, and also how this applies to air quality valuation (HMT, 2013), recommending the application of the declining discount rate scheme.

However, alongside this, the values of health costs/benefits in future years need to be uplifted, to reflect the assumption that willingness to pay for health will rise in line with economic growth. The UK Government (HMT, 2013) recommends that in air pollution appraisal, the health values in future years need to be uplifted by 2% per annum.

In the analysis of future air pollution – and this study – these issues have a number of important consequences in adjusting values in future years.

First, the unit cost of a life year lost (in £) varies with time, according to when it occurs in the future, due to the uplift, and after it is discounted back to the present day. These adjustments have to be taken into account when valuing the changes in life-expectancy and life years lost from air pollution. As an example, this means that a life year lost in 2075 has a higher unit value than one in 2015 (due to the uplift), but the value of a life year lost in 2075 is lower than the one in 2015 after both are discounted back to the present day. Following from this, when expressing the total changes in life expectancy, it is the sum of discounted values that is

presented. When this relates to long-term exposure, this can be expressed as an equivalent annual value, to provide a metric that is comparable to other health metrics, i.e. to provide an annual estimate for the year 2010.

Second, the use of damage costs in the ready reckoner has to take account of the changes in damage cost values over time, i.e. it has to build in uplifts and discounting. Thus while the damage costs have been produced in £2014 prices for 2014 emissions, the ready reckoner adjusts these values to provide appropriate values – and net present values - for emissions in different future years.

4.1.5 Damage Costs input data and method

4.1.5.1 Processing of input data

The production of new damage costs in the study used the impact-pathway approach, building on the methods of analysis in section 2. The aim was to produce new 'marginal' damage costs for **valuing road traffic emissions** in London. Note that the focus was on marginal values for the road transport sector – rather than average damage costs for total current air pollution levels in London – in order to produce specific values for TfL for transport scheme appraisal (in section 4.3). The analysis was also undertaken to provide area-specific values in terms of where emission reductions occur – but taking account of the improvement in air quality concentrations they achieve for London as a whole. This recognises that health impacts vary with population density. In line with the existing Defra damage costs, separate values for emission reductions in central, inner and outer London were produced, based on modelling the effects of emission changes in these areas on concentrations (and health impacts and economic costs) across London.

Modelled concentrations: To generate the damage costs, three new air quality model run scenarios were produced using both King's London Emission Toolkit (LET) and London Air Quality Toolkit (LAQT). Further details regarding the LET and LAQT models can be found in Annex 4. The three scenarios were as follow:

- 10% decrease in PM and NO_X emissions from road traffic only in the central London area.
- 10% decrease in PM and NO_X emissions from road traffic only in the inner London area.
- 10% decrease in PM and NO_X emissions from road traffic only in the outer London area.

PM_{10}, $PM_{2.5}$ and NO_2 annual mean concentrations were predicted based on the baseline in 2010 (extracted from the LAEI2010 as in section 2) to reflect the assumptions in the three scenarios above, whilst maintaining all other assumptions and non-vehicle emissions constant. Once complete, each scenario was compared with the baseline to establish the effectiveness of each in reducing air quality emissions and concentrations.

Population-weighted average concentration: The air quality scenario results from the above were further processed as in section 2 to produce London specific population-weighted average concentrations and health impact outcomes. The population-weighting was done by the

population aged 30+ for the effects of long-term exposure to $PM_{2.5}$ or NO_2 on mortality and by all ages for other health outcomes.

4.1.5.2 Calculations

The approach and analysis are consistent with the current Defra and HMT air quality appraisal guidance (Defra, 2011b; Defra, 2013; HMT, 2013), but derive London specific damage costs from new air quality modelling runs, health impact assessment and valuation of changes in transport emissions in London.

The method used to calculate these new damage costs is consistent with the Defra damage costs assessment (IGCB, 2007; Watkiss et al., 2007a; 2010), with updates as in section 2 to ensure consistency with the latest COMEAP recommendations (2010) and monetary values (Defra, 2013), updated to £2014 prices. The analysis applied the impact pathway approach to analyse the impact of marginal changes in emissions (10% changes in central, inner and outer London, in turn). The analysis then modelled the change in concentrations, health impacts and monetary values from these marginal emission changes, then adjusted the results by emission totals to derive unit damage costs, i.e. £ per tonne estimates.

There was one aspect that differed from the analysis of a 1 μg m^{-3} reduction in sections 2.1.4 and 2.1.5. This relates to the analysis of long-term life expectancy and life years lost. The analyses in sections 2.1.4/2.1.5 assessed changes from sustained air pollution levels, i.e. over the next 105 years. However, damage costs are annual values that are applied to assess changes in annual emissions. To address this, new life table results were produced for a 1 year pollution pulse using the COMEAP/Interdepartmental Group on Costs and Benefits impact methodology, based on the original methods of Miller and Hurley (2003). This approach was used to generate the previous damage costs (IGCB, 2007: 2011b). This required a complex economic analysis that assessed the value of life years in every future year (from a one year pulse), for the next 105 years, uplifting these values at the recommended rate of 2% per year, then discounting these future values using declining discount rates as recommended by the Green Book (HMT, 2011).

This approach produced damage costs for air quality changes in London, for the direct local health impacts. However, transport emissions in London also have an impact at the regional and national level. Two issues are important here and were accounted for.

First, primary PM pollution emitted in London leads to health impacts outside London, from the contribution to non-London background pollution levels. While these are small (relative to inside London impacts) they were considered in the previous analysis with TfL (on LEZ1). Therefore they were included in the PM damage costs. This used the central rural and unit damage cost values from the Defra damage cost values (2011), updated to current prices (noting these are based on the same hazard rate and lag profile [EPA lag] for long-term exposure to $PM_{2.5}$ and mortality and thus consistent with the analysis here).

Second, NO_X emissions in London lead to the formation of secondary particulates (PM as nitrates) and these lead to PM related health impacts (which are assumed to be the same as primary PM health effects) at the London and national scale. These secondary pollution effects are included in the existing Defra damage costs for NO_X, and indeed they are larger in monetary terms than the contribution of NO_X morbidity and short-term exposure and mortality. For this reason, the secondary PM component from the Defra NO_X damage costs (Defra, 2011b) was included in the new London damage costs estimates, updated to 2014 prices (again these are based on the same hazard rate and lag, but the estimates are based on formation of secondary PM_{10} rather than $PM_{2.5}$ although much of the secondary particulates would be in the fine fraction).

The general caveats with the use of damage costs - as outlined in Defra (2011b) and HMT (2013) guidance apply: they should not be used for policies where air pollution changes last for longer than around 20 years (IGCB, 2007; Defra, 2011b), where values are likely to be larger than £50 million in present value terms, and their use for large concentration increments may be inaccurate (see Annex 6). It is stressed that not all of these damage costs are additive, and thus information on how overlap between pollutants is dealt with is discussed in Annex 6.

The damage costs were produced for a "core" set of values, with a central value and a low/high range, and also a low/high sensitivity range, in line with the underlying health impacts based on COMEAP recommendations, with some updating such as using $PM_{2.5}$ rather than PM_{10} for effects of short-term exposure, and the health valuation based on IGCB and Defra recommendations (Defra, 2011b: HMT, 2013). An "extended set" was also produced – as a set of adders - with more outcomes but more uncertainty based on WHO/HRAPIE recommendations. This latter includes the important recommendation regarding mortality impacts of NO_2.

Note that the damage cost values exclude the effects of NO_X emissions on ozone (both local and regional). They also exclude non-health impacts (damage to materials, crops) from PM and NO_X. However, both these omissions would not have material impacts on the size of the damage costs, as evidenced by their contribution to the total existing Defra damage costs (see Watkiss, 2010). The damage cost sets are described below.

4.1.5.3 Core damage costs (central estimate) calculations

The approach estimates long-term exposure and mortality and short-term exposure and morbidity for $PM_{2.5}$ (rather than PM_{10}). It used a methodology consistent with that recommended by COMEAP (COMEAP, 1998; COMEAP, 2010).

PM damage costs (core) include:

- Long-term exposure and mortality (from modelled $PM_{2.5}$ concentrations) using COMEAP impact methodology for life-years lost (COMEAP, 2010) with updates to the input data as outlined in section 2.1. The central estimate used a relative risk of 1.06 (6%) as used to derive damage costs before. However, the COMEAP recommended lag profile for life years lost was used to provide this central estimate, rather than the previous approach

using the average of a 0 and 40 year lag (Watkiss et al., 2007a) or the Monte Carlo analysis (Watkiss et al., 2007b: 2010). The life years lost were estimated using London specific life tables based on averaging death and population data from 2009/2010/2011 by single year of age (ONS, 2013) and followed through the impact of an annual pollution pulse over time (see IGCB, 2007). Life years lost were valued using the recommended IGCB value in the Defra guidance (Defra, 2013), updated to 2014 prices (Annex 7). Consistent with this guidance, values for future life years lost were increased at 2% per annum, then discounted using the declining discount rate scheme in the HMT Green Book (2011).

- Short-term exposure and hospital admissions (from modelled $PM_{2.5}$ concentrations) using concentration-response functions from the Department of Health (DH) commissioned systematic review report (Atkinson et al. 2014), which were also used for the HRAPIE recommendations, limited set (WHO, 2013b). The valuation of respiratory and cardiovascular hospital admissions was undertaken using the IGCB values in the Defra impact assessment guidance (Defra, 2013), updated to 2014 prices.
- For $PM_{2.5}$, an adder was included in the damage costs to take account of outside London impacts, i.e. the effects of London emissions on regional (UK) pollution. This is based on the rural damage cost values in the Defra damage cost values (2011), updated to 2014 prices.

NO_x damage costs (core) include:

- Short-term exposure and NO_2 (1 hour average) for deaths brought forward and hospital admissions used the HRAPIE recommendations, limited set, (WHO, 2013b) which is closely related to the results from the DH commissioned systematic review (Mills et al. 2015*)*. The valuation of hospital admissions and deaths brought forward was undertaken using the IGCB values in the Defra guidance (Defra, 2013), updated to 2014 prices (Annex 7).
- The impacts of secondary pollution (NO_2 contribution to nitrate as $PM_{2.5}$) was included, to take account of the effects of London emissions on regional (UK) secondary particulates, using the secondary PM component of the Defra NO_X damage costs (Defra, 2011b), updated to 2014 prices.

4.1.5.4 Core damage costs (low/high and low/high sensitivity) calculations

The current Defra damage costs (Defra, 2011b: HMT, 2013) have a central value, a low and high range, and also a low and high sensitivity set (i.e. 5 values are provided). The low and high values relate to different choices on the lag phase for long-term exposure and mortality, and the full sensitivity set also includes the low and high hazard rates for long-term mortality. A similar set of low/high and also a low/high sensitivity damage cost set were produced for the study. These are similar to the approach above, with the following differences:

- The low and high values used the single recommended distribution of lags from COMEAP (2010) – rather than the previous range, thus lag uncertainty is removed.
- The low and high values used the CORE health impact set, but then use the low and high values for mortality and morbidity, using the recommended low and high estimates in

the Defra impact assessment guidance (Defra, 2013), updated to 2014 prices (see Annex 7).

- For the low and high sensitivity values, long-term exposure and mortality life years lost for $PM_{2.5}$ were estimated using the lower and higher (sensitivity) hazard rate from IGCB (2007) and COMEAP (2010), i.e. 1% and 12%. They also adopt the low and high monetisation values for all end-points.
- For the high sensitivity value, short-term exposure of $PM_{2.5}$ deaths brought forward was also included.

4.1.5.5 Extended damage costs (core plus HRAPIE extended set) calculations

Alongside the CORE damage costs, a new set of sensitivity damage cost values was produced. These were produced as a set of 'adders' i.e. additional damage costs that could be assessed and added to the CORE consistent damage costs. This allows the addition of some functions that use PM_{10} and different functions that use $PM_{2.5}$, thus the PM_{10} and $PM_{2.5}$ adders are additive.

The extended set was based on the HRAPIE EXTENDED method and included long-term mortality for NO_2, with a 30% reduction (see earlier section) to reduce double counting. They also included an extended morbidity set for NO_2 and PM_{10}/$PM_{2.5}$. The extended method is based on recommendations from HRAPIE (WHO, 2013b) but using the more precise method of using odds ratios directly and scaling on the log odds scale rather than assuming the odds ratio is a relative risk and scaling on a linear basis (Annex 10). (HRAPIE suggests more complex analysis might be required for large baseline prevalences or large concentration increments.)
Valuation of endpoints was based on new valuation estimates (Watkiss and Hunt, forthcoming), which updated and extended previous values from CAFE (Hurley et al., 2005), adjusted to £2014 (see Annex 7).

The level and type of uncertainty varies for the different extended outcomes. This is fine for the use of damage costs for screening proposals but full analysis of the proposals would need to include a full discussion of these uncertainties and the plausibility of the results.

PM damage costs (extended set) include:
As core above for CORE $PM_{2.5}$ plus from $PM_{2.5}$ concentrations:

- Restricted activity days (avoiding overlap with restricted activity days due to hospital admissions, and bronchitis in children as above); London specific data was used for baseline postneonatal mortality (ONS, 2012) and for the percentage of children with severe asthma or asthma ever in North and South Thames from Lai et al. (2009). Otherwise, baseline rates were as recommended in WHO (2013b) scaled to the London population.

Plus from PM_{10} concentrations:

- Infant mortality;
- Asthmatic symptoms in asthmatic children;
- Prevalence of bronchitis in children;
- Incidence of adult bronchitis.

NO_x damage costs (extended set) include:
As core above for CORE NO_2 but

- Without NO_2 deaths brought forward (to avoid double counting of mortality from short-term and long-term exposure).
- With long-term exposure and mortality from NO_2 directly with 30% reduction to reduce double counting with $PM_{2.5}$. The maximum overlap of 30% was recommended in WHO (2013a) but was regarded as too uncertain for the main recommendation in WHO (2013b). It is argued in the current project that some overlap is likely even if the numerical size 'up to 30%' is uncertain, and it is better to include some overlap rather than none.
- Long-term exposure to NO_2 and prevalence of bronchitic symptoms in asthmatic children based on recommendations in WHO (2013b).

For implementation of the long-term exposure to NO_2 and mortality functions above, various counter factuals were discussed in Annex 1, including a sensitivity analysis with a counter factual at 20 $\mu g\ m^{-3}$. Allowing for this did not arise in the derivation of the damage costs using a small decrease from 2010 baseline concentrations, as all output areas were above 20 $\mu g\ m^{-3}$ with, and without, the emissions reduction. However, when analyzing future policies involving decreases in NO_X emissions, this may no longer be the case. The damage cost approach could then be an overestimate for some choices of counter factual. NO_2 damage costs could also be an overestimate due to contributions from other traffic pollutants rather than NO_2 itself. This is one reason why the effect of long-term exposure to NO_2 and mortality is in the extended rather than the limited set.

4.1.6 Damage Costs Estimates

The method above was used to produce new London-specific population-weighted average concentrations and health impact results and health costs. The change in health costs for each pollutant, for the baseline and the additional marginal run was produced, then divided by the change in emissions (tonnes) to derive new London specific damage costs. As outlined above, this was undertaken separately for each major London area to provide differentiated values for emissions changes in central, inner and outer London.

The new London specific damage cost estimates – for the central CORE method - are shown in Table 22 below, along with the adders for the extended set. They provide unit values for assessing the health costs or benefits of marginal changes in emissions from road transport in London. The low and high values, and the low and high sensitivity values, are included in Annex 8.

Table 22 London Transport Emissions Damage Costs values (£ per tonne in 2014 prices) – CORE and Extended Adder

London zone	Core $PM_{2.5}$	Core NO_X	Extended PM_{10}	Extended $PM_{2.5}$	Extended NO_X
Central	£125,329	£884	£22,395	£118,360	£39,442
Inner	£157,794	£910	£27,598	£152,884	£52,344
Outer	£90,466	£861	£14,224	£79,540	£27,948

Note. For use, the ADDERS are additional damage costs, which should be added to the CORE set. Note that the extended set includes separate PM_{10} and $PM_{2.5}$ damage costs. These relate to different health endpoints and thus they are additive, thus the total damages from PM for an extended analysis would be the sum of CORE $PM_{2.5}$+ EXTENDED PM_{10} + EXTENDED $PM_{2.5}$. The CORE values in Table 22 can be compared to the current Defra damage costs, which are shown in Table 23 below.

Table 23 DEFRA London Damage Costs values (£ per tonne in 2010 prices).

London zone	Central Estimate (1)	Low Central Range (2)	High Central Range (3)
NO_X	£955	£744	£1,085
PM transport central	£221,726	£173,601	£251,961
PM transport inner	£228,033	£178,540	£259,129
PM transport outer	£148,949	£116,621	£169,261

The values for NO_X are fairly similar to the existing Defra values (HMT, 2013), once updated to 2014 values. This is due to the dominant impact of secondary particulate formation on long-term exposure and mortality. These secondary impacts are responsible for around two-thirds of the total NO_X damage costs for London, and thus dominate when compared to direct local impacts from short-term NO_2 exposure in London (deaths brought forward and hospital admissions).

The difference between the Defra and new London specific values for PM are much greater, for all three areas. The London values are significantly lower than the Defra values, e.g. £90,500 per tonne of PM_{10} in outer London (new London) versus £149,000 (Defra). There are a number of reasons for this, associated with the approaches used for air quality modelling, and the impacts/valuation analysis.

- The main difference is likely to be due to the air quality modelling approach used in the two assessments. The Defra damage costs used an aggregated national air quality analysis (PCM, Pollution Climate Mapping model), as compared to the detailed local air quality model used by King's for the London-specific new analysis. Both models have been submitted to the UK Model Inter-comparison Exercise (MIE), run by King's on behalf of DEFRA (http://uk-air.defra.gov.uk/library/reports?report_id=777). The MIE showed that the PCM model has much higher PM concentrations in London than the model used by King's (see figure 3.8 from MIE report).
- There are differences in relative size of values between the three different zones. This could be due to air quality modelling effects, but could also be influenced by the population-weighted exposure, i.e. between the more aggregated Defra damage costs, and higher resolution London specific analysis.
- For the CORE $PM_{2.5}$ set, long-term exposure and mortality dominates the numbers (it is around 99% of total PM CORE damage costs, whereas morbidity is only 1% of damage costs). Any changes in the HIA method or valuation for this single endpoint are therefore extremely important in the overall damage costs. There are some small differences with the new London specific values, with the use of the COMEAP recommended lag profile. There is also the use of London specific life tables (in this analysis) rather than the national life tables used previously. These may also explain some of the differences seen.
- There is also a large difference in the implementation of the PM damage costs, because the Defra damage costs values are for PM_{10}, while the new London values are for $PM_{2.5}$. This is particularly important for long-term exposure – noting that the previous damage costs used $PM_{2.5}$ functions directly for long-term exposure but applied to PM_{10}, thus overestimating likely impacts. Therefore the use of $PM_{2.5}$ for estimating long-term exposure (rather than PM_{10}) leads to a significant reduction in health benefits (or costs) when actually applying the damage costs, when compared to the use of the older Defra damage costs, as the new London specific values are applied to the smaller $PM_{2.5}$ fraction, rather than the larger PM_{10} fraction.

Finally, the CORE values can be compared to the EXTENDED set. These show there are large additional costs/benefits associated with the new functions. For PM, the EXTENDED set effectively doubles the damage costs. This is due to the inclusion of restricted activity days – which is responsible for the entire $PM_{2.5}$ adder. However, there are important additions from the other morbidity functions in the PM_{10} adder, from adult bronchitis (71% of the PM_{10} adder), but also contributions from asthmatic symptoms in asthmatic children; (13% of the PM_{10} adder), prevalence of bronchitis in children (13% of the PM_{10} adder) and infant mortality (12% of the PM_{10} adder).

The impact of the EXTENDED set is even more important for NO_X. This is because of the introduction of the mortality effects of long-term exposure to NO_2, which increases the damage costs by between 1 – 2 orders of magnitude (and is responsible for 87% of the NO_X damage cost adder, with the rest from morbidity [bronchitic symptoms in asthmatic children]). While the EXTENDED damage costs for NO_X are still lower than for PM, the much higher concentrations of NO_2– and the generally higher levels from transport - means that in implementation, NO_X damage costs will often dominate the health valuation results. For example, the total emissions

in 2010 from road transport in the greater London area were 23,657 and 1,343 tonnes per annum from the LAEI2010, for NO_X and $PM_{2.5}$ respectively. The average annual mean concentration in 2010 in the greater London area was 33.4 and 13.8 $\mu g\ m^{-3}$ from the LAEI2010, for NO_2 and $PM_{2.5}$ respectively.

Further work to explore the differences between the Defra and London values, and some discussion between TfL and Defra, is recommended.

4.2 Estimates of the current costs of $PM_{2.5}$ and NO_2 in London

4.2.1 Background

The results of section 2 were also used to estimate the health costs of current air pollution in London, i.e. to look at the costs in 2010. It is stressed that it is not appropriate to use damage costs to estimate this total economic cost of current pollution, because the costs are large, and because it involves multiple sources (rather than just road transport emissions).

4.2.2 Method

The analysis used the outputs of section 2 directly (see earlier section), in terms of numbers of health impacts, then applied the health valuation estimates from Defra (2013), updated to 2014 prices (see Annex 7). This allows the direct valuation of health costs from overall air pollution in 2010 in London (presented in current 2014 prices).

The valuation of the hospital admissions and deaths brought forward simply multiplied the estimated numbers of hospital admissions and deaths brought forward by the monetary values for these outcomes (Annex 7).

The valuation of the attributable deaths has had to consider an additional step, in order to account for the fact that the life years lost will arise over future time periods. To account for this, the profile of remaining life years lived for each five year age group (separately for men and women) was taken, and this was used to create a profile of baseline life years over time. This was then multiplied by the appropriate future monetary values for a life year (VOLY) lost, i.e. the value after uplift/discounting for each future 5 year time period. The resulting weighted VOLY was then multiplied by the life years lost for the relevant gender and 5 year age group. The results therefore provide the annual costs of 2010 pollution on mortality in London.

Finally, as these values are focused on London, they only include direct impacts associated with section 2, i.e. they do not include the outside London effects (from primary PM or secondary particulates from NO_X) in the results.

4.2.3 Estimates of the economic costs of the mortality burden of current air pollution in London ($PM_{2.5}$ and NO_2)

The estimated annual cost of air pollution related mortality for $PM_{2.5}$ (2010) for London is £1,358 million (in 2014 prices). Using the same approach, the analysis has also estimated the

potential air pollution related mortality for NO_2, using the HRAPIE recommendation (with NO_2 RR 1.039). The estimated annual costs of air pollution related mortality for NO_2 (2010) for London is up to £2,273 million (in 2014 prices).

4.2.4 Estimate of the economic costs of hospital admissions and deaths brought forward from current air pollution in London ($PM_{2.5}$ and NO_2)

Table 24 below show the short-term economic impacts of air pollution on London, capturing the respiratory (RHA) and cardiovascular hospital admissions (CHA), and a sensitivity analysis on deaths brought forward (DBF).

Table 24 Estimate of DBF, RHA and CHA costs of 2010 current air pollution in London for anthropogenic $PM_{2.5}$/PM_{10} and NO_2 for the central estimate

Pollutants	Sensitivity estimate	DBF economic impact	RHA economic impact	CHA economic impact
Anthropogenic $PM_{2.5}$	Central	£4,906,994	£13,770,028	£4,960,165
Anthropogenic PM_{10}	Central	£4,675,021	£9,050,355	£6,819,300
NO_2	Central	£2,875,454	£2,893,801	

Estimates of the economic costs of hospital admissions and deaths brought forward from current air pollution for total (i.e. including the non-anthropogenic part) $PM_{2.5}$ and PM_{10} can be found in Annex 9.

4.2.5 Combined economic costs of current air pollution in London ($PM_{2.5}$ and NO_2)

The estimated annual costs across both pollutants ranges from a core result of £1,383 million (including all the hospital admission effects of $PM_{2.5}$, plus respiratory hospital admissions and deaths brought forward from short-term exposure to NO_2) to an 'extended' result of £3,653 million[58], including all core results except deaths brought forward from short-term exposure to NO_2 as the effect of long-term exposure to NO_2 on mortality is now added.

These estimates exclude the extended morbidity outcomes from HRAPIE for PM_{10}/$_{2.5}$ and NO_2. These were not included in the 2010 analysis in section 2, which is what we have valued here. However, the economic costs of current levels of air pollution cited above would be higher if they were included. This includes additional impacts for PM_{10} (post-neonatal mortality, chronic bronchitis, asthmatic symptoms in asthmatic children, bronchitis in children), $PM_{2.5}$ (restricted activity days), and NO_2 (bronchitic symptoms in asthmatic children). These outcomes have been assessed in other parts of the study for the extended set damage costs for use in screening

[58] This does not include the possible further outcomes recommended in the extended set from HRAPIE.

proposals. However, this would be expected to be followed by a fuller analysis in which all the uncertainties could be spelt out and the results cross-checked for plausibility. The level and type of uncertainty varies for the different extended outcomes so it might be appropriate to include some but not all of them. This would require further work that was not part of this project.

The difference between the core and the extended damage costs for PM gives a very rough indication that the economic costs could be considerably higher if these were included but, in addition to the points noted above, the health results do not scale exactly with emissions/concentrations, due to non-linearities, and the PM damage costs include both $PM_{2.5}$ and PM_{10} so will vary with the exact $PM_{2.5:10}$ ratio. For NO_2, the effects of long-term exposure to NO_2 on mortality are already included and the size of the effect of long-term exposure to NO_2 on bronchitic symptoms in asthmatic children is smaller. However, the non-linearities in converting NO_x emissions into NO_2 concentrations mean scaling the damage costs across large concentration changes is unwise. This again emphasises that extending the current costs to cover these additional extended outcomes requires further work.

4.3 Developing a "ready reckoner" to help estimates of health impacts of future policies

4.3.1 Background

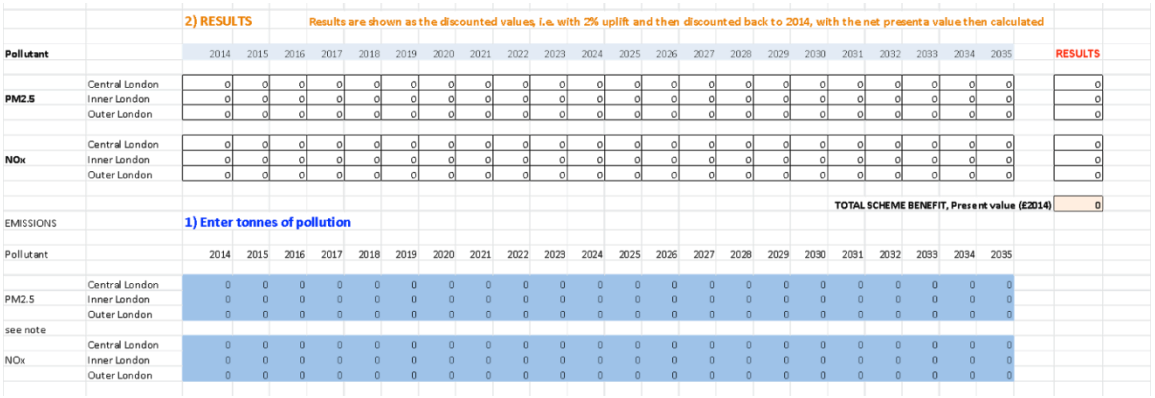
The final task used the new damage costs (from section 4.1) to build a 'ready reckoner' for TfL. The aim was to produce an emission-based damage cost calculator, with the new London-specific damage costs included, to allow the assessment of the economic health benefits of transport proposals. This tool is appropriate for use in screening policies (large numbers of alternative options), and/or for policies that produce a small or temporary reduction in emissions over time, i.e. consistent with the Defra (2011b, 2013) and HMT guidance (2013) on air quality valuation. It is not appropriate for use in other applications.

4.3.2 Approach

The damage costs from section 4.1 were used and incorporated in a set of simple spreadsheet tools. This followed the request from TfL to provide simple tools (avoiding macros). The spreadsheets were designed with a simple single input sheet for emissions, allowing the input of proposed emission reductions over time, for different pollutants, in the three areas of London (central, inner and outer). A key part of the tool was to ensure that the £2014 damage costs were adjusted to estimate the future damage costs in future years (applying the uplift and then discounting) and summing to produce a net present value (the sum of the discounted values, i.e. the total scheme benefit in £).

The 'ready reckoner' was produced as a series of three spreadsheets 1) for the CORE analysis of $PM_{2.5}$ and NO_X emissions, 2) a CORE sensitivity sheet, and 3) an additional spreadsheet for the analysis of the EXTENDED set for 'adders'. The simple central CORE calculator is shown below in Figure 16. This has a simple input box for emissions, which the user enters, and the ready

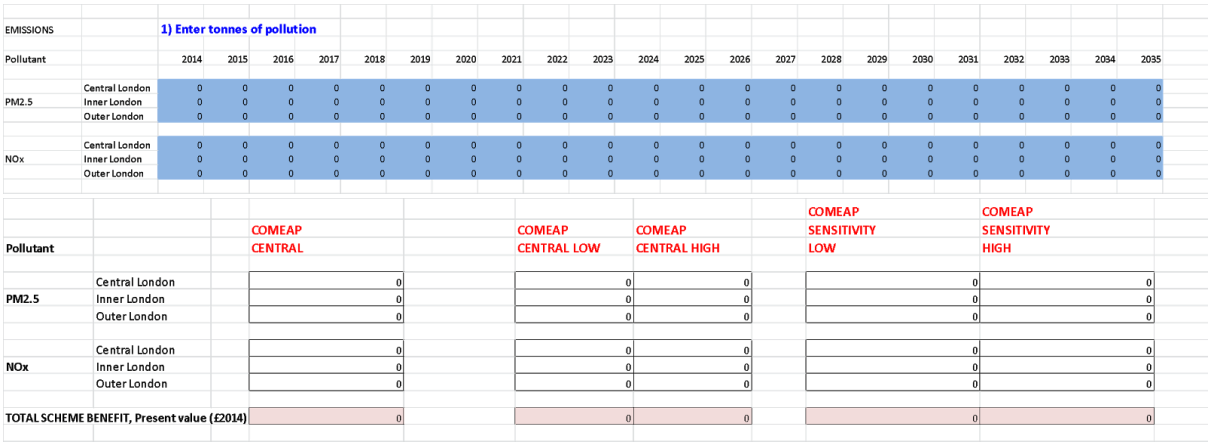
reckoner then calculates the scheme benefits – in every year – and as a total present value, in 2014 prices.



		2) RESULTS	Results are shown as the discounted values, i.e. with 2% uplift and then discounted back to 2014, with the net presenta value then calculated																			
Pollutant		2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	RESULTS
PM2.5	Central London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Inner London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Outer London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
NOx	Central London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Inner London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Outer London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
																							TOTAL SCHEME BENEFIT, Present value (£2014)	0
EMISSIONS		1) Enter tonnes of pollution																						
Pollutant		2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035	
PM2.5	Central London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
	Inner London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
	Outer London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
see note																								
NOx	Central London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
	Inner London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	
	Outer London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	

Figure 16 Illustration of the 'ready reckoner' spreadsheets - simple central CORE calculator

An additional calculator (Figure 17) was produced to allow the analysis of the core low and high values, and the low and high sensitivity. Again, this has a simple 'emissions' entry sheet, and then estimates the different monetary values and scheme benefits on a separate 'results' page.



EMISSIONS		1) Enter tonnes of pollution																					
Pollutant		2014	2015	2016	2017	2018	2019	2020	2021	2022	2023	2024	2025	2026	2027	2028	2029	2030	2031	2032	2033	2034	2035
PM2.5	Central London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Inner London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Outer London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
NOx	Central London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Inner London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	Outer London	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Pollutant		COMEAP CENTRAL	COMEAP CENTRAL LOW	COMEAP CENTRAL HIGH	COMEAP SENSITIVITY LOW	COMEAP SENSITIVITY HIGH
PM2.5	Central London	0	0	0	0	0
	Inner London	0	0	0	0	0
	Outer London	0	0	0	0	0
NOx	Central London	0	0	0	0	0
	Inner London	0	0	0	0	0
	Outer London	0	0	0	0	0
TOTAL SCHEME BENEFIT, Present value (£2014)		0	0	0	0	0

Figure 17 Illustration of the 'ready reckoner' spreadsheets - EXTENDED set for 'adders'

Finally, a calculator was produced to allow the analysis of the EXTENDED set, to be used in conjunction with the CORE analysis. This took the same form as above, with an emissions sheet and an output 'results' page, noting that separate emission inputs for PM_{10}, $PM_{2.5}$ and NO_X are included. The results from this sheet can then be added to the CORE values.

The ready reckoner was successfully tested in an analysis of the health benefits of two possible emission reduction schemes.

The ready reckoner includes text to alert the user to the circumstances in which it is, or is not, appropriate to use the damage costs. The full reasoning for these caveats are given in Annex 6 of this report, which is referenced in the calculator.

5 Discussion

This report outlines a large body of work characterising the health impacts of air pollution in London, the economic analysis of these impacts and new tools to help assess policies to reduce air pollution. This discussion picks up some broader issues.

The number of health outcomes and types of analysis is considerably expanded from the previous work by Miller (2010) that was limited to quantifying the impacts of long-term exposure to $PM_{2.5}$ on mortality in London. As a first step, the effects of long-term exposure to $PM_{2.5}$ on mortality were a good place to start. It was the largest health impact at the time and remains the largest impact when considering only the most established evidence. However, it is clear that, while the additional outcomes may be more uncertain (e.g. long-term exposure to NO_2 and mortality) or much smaller (e.g. effects on hospital admissions), the overall health impacts are likely to be higher than that from long-term exposure to $PM_{2.5}$ and mortality alone.

One question that arises is whether the mortality effect of long-term exposure to NO_2 is plausible. This is discussed, in general terms, in WHO (2013a) by considering evidence from the original epidemiological and toxicological studies rather than the results of health impact quantification. REVIHAAP (WHO, 2013a) considered the evidence for hazard (is there an effect on mortality?); HRAPIE (WHO, 2013b) considered the size of the risk (recommending functions relating concentration change to change in mortality risk) whereas this report works through the effects of the change in mortality risk on numbers of deaths and life years using the London population and baseline mortality risk. REVIHAAP concluded that, while NO_2 might, at least in part, represent the mixture of traffic pollutants rather than just NO_2 itself, the mechanistic evidence, particularly on respiratory effects, and the weight of evidence on short-term associations are suggestive of a causal relationship for the effects of long-term exposure to NO_2 on mortality. Nonetheless, it is important to regard this report as providing a range for the possible effects of NO_2 itself. For burden calculations, the results may still be valid as an expression of the effects of traffic pollution, provided NO_2 correlations with other traffic pollutants are similar to those in the original studies.

It should be remembered that the original studies meta-analysed in Hoek et al. (2013) indicate that mortality over a specific time period is higher in areas where NO_2 is higher. This does not prove that NO_2 (or in fact another pollutant if it acts as an indicator) is the **sole** cause of these deaths. It probably acts in combination with a variety of other risk factors.

Determining priorities for public health action by translating epidemiological evidence into quantified health impacts is not a simple process and the recommendations from the WHO HRAPIE project are only a first step. A detailed methodology needs to be developed for using newly recommended concentration-response functions, as illustrated by the differing interpretations of potential counter factuals for burden calculations for the effects of long-term exposure to NO_2 on mortality. This report contributes to that process, particularly for the pollutant-health outcomes pairs that have only recently been recommended for quantification.

More consideration should be given to how to deal with some of the options for counter factuals in a life-table context as the size of the exposed population changes as concentrations fall below the cut-off in different areas. This did not apply to NO_2 in London as all output areas were projected to remain above 20 µg m^{-3} beyond 2020.

This report is new in terms of implementing WHO recommendations (from 2013), combined with following earlier COMEAP and PHE recommendations for $PM_{2.5}$ and general methodology (from 2010/2014). However, it should be noted that new studies continue to be published and these may lead to a need to update past recommendations.

The present work responded to a particular specification, but this report can be used as a basis for discussion of what future outputs would be useful. For example, the projected benefits of reductions from 2010 to 2020 could be calculated and updated in future years, to take into account actual rather than projected reductions and updates to emissions inventories. This would be a 'rolling' progress report to complement the 'snapshot' burden calculations. Further analysis using air quality modelling with different metereology assumptions might also be useful to illustrate how much of a difference between actual and projected reductions is due to emissions changes and how much to year- to-year variations in the weather.

This report has taken advantage of more detailed information that is available in London in the burden calculations and also explored some alternative methodology. The Public Health Outcome Indicator for the fraction of mortality attributable to $PM_{2.5}$ remains the official one as it is important to have a nationally comparable indicator and not all areas of the country have the detailed information that is available for London. The analyses reported here may help for future developments of the indicator if data availability, such as local age-specific mortality rate information by 5 year age group, improves elsewhere.

Whilst the report quantified the burden of many key health outcomes in London in 2010, it only included the additional mortality burden of long-term exposure to NO_2 from the extended set of HRAPIE recommended outcomes. However, for comparison, the extended set of outcomes were included in the 'extended set' damage costs. The uncertainties around outcomes in this extended set differ in nature. For long-term exposure to NO_2 and mortality, meta-analyses of several studies are available with uncertainties in allocation of the effect to NO_2 rather than in the existence of an effect. Other outcomes are based on only one or two studies, on situations where most studies do not show an effect but one or two do, or where the appropriate baseline rates or definition of sub-populations is unclear (WHO, 2013b). The wider range of outcomes could be considered for burden calculations in the future, although uncertainties need to be borne in mind.

The report has concentrated mainly on $PM_{2.5}$, NO_2 and sometimes PM_{10}. It has not covered PAHs (although the association between $PM_{2.5}$ and lung cancer may act through particle-bound PAHs), CO, SO_2 (concentrations of which are small) or ozone (which is more complicated to model). Effects of short-term exposure to ozone are well established, and there are suggestions now of an effect on long-term exposure, although the evidence is somewhat contradictory (WHO, 2013 a,b). Ozone levels increase as NO levels decrease. If the increased concentration is

still below the threshold (if there is one), then this concentration increase may not result in increased health impacts (ozone concentrations are still low in central urban areas). However, the evidence on whether or not a threshold exists is not clear cut. Quantifying the health effects of ozone in London is an area for further work.

There are benefits from this project that go beyond the air pollution-related health impact outputs. This includes calculation of baseline expected remaining life expectancy by 5 year age groups by borough (available on request) and a new system of producing weighted values of a life year to link to the burden calculations.

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7 Annexes

7.1 Annex 1. Burden calculations for NO_2 and possible counter-factuals

The text in the main report explained that the HRAPIE recommendation regarding quantification above 20 μg m^{-3} applied to small concentration changes. Discussion of counter-factuals for burden calculations is still needed. This Annex discusses this in more detail.

Extracts from the HRAPIE report (WHO, 2013b) as regards a 'cut-off' at 20 μg m^{-3} are given below. We use the term 'cut-off' here to mean the point at which the slope of a concentration-response relationship becomes uncertain, such that quantification is not recommended below that point.

Table 25 Extract* from 'CRFs recommended by the HRAPIE project' Table 1 of the HRAPIE report

Pollutant metric	Health outcome	RR (95% CI) per 10 μg m^{-3}	Range of concentration	Comments
NO_2, annual mean	Mortality, all (natural) causes, age 30+ years	1.055 (1.031 –1.080)	>20 μg m^{-3}	Some of the long-term NO_2 effects may overlap with effects from long-term $PM_{2.5}$ (up to 33%); this is therefore recommended for quantification under Group B to avoid double counting in Group A analysis.

*Only rows and columns relevant to this discussion have been extracted.

'The HRAPIE experts recommended applying to adult populations (age 30+ years) a linear CRF for all-cause (natural) mortality, corresponding to an RR of 1.055 (95% CI = 1.031, 1.08) per 10 μg m^{-3} annual average NO_2. The impact should be calculated for levels of NO_2 above 20 μg m^{-3}.'

'The possible threshold above which the NO_2 effect can be estimated has been the focus of few studies. The study by Naess et al. (2007) investigated the CRF between NO_2 and mortality. The study included all inhabitants of Oslo, Norway, aged 51 – 90 years on 1 January 1992 (n = 143 842) with follow-up of deaths from 1992 to 1998. In the youngest age group (51 – 70 years) risk of death from all causes started to increase at the level of 40 μg m^{-3}. In the oldest age group (71 – 90 years) this increase in risk was linear in the interval 20 - 60 μg m^{-3}. In the study by Cesaroni et al. (2013), investigating the general population of Rome, Italy, (n = 1 265 058) with a follow-up from 2001 to 2010, a statistically significant linear CRF of NO_2 and natural mortality was detected above 20 μg m^{-3}. On the basis of these observations, therefore, it is recommended that the NO_2 impact should be calculated for levels above 20 μg m^{-3}.'

It is clear from the above that a calculation of the health impact of a policy that changes concentrations from, say, an annual average concentration of 35 to 34 μg m^{-3} would be

recommended whereas a calculation of the health impact of a policy that changes concentrations from, say, an annual average concentration of 19 to 18 μg m^{-3} would not. However, it is less clear what is intended if doing a burden calculation of an absolute concentration that is above 20 μg m^{-3}. Discussions amongst people in the field ranged all the way from there should not be a cut-off at all to calculations should be done as if the same slope reaches a threshold of zero at 20 μg m^{-3}. The latter involves setting concentrations below 20 μg m^{-3} to zero and also subtracting 20 from concentrations above this and only applying the concentration-response relationship to the incremental difference between the specified concentration and 20 μg m^{-3}. In other words, the burden of a concentration of 21 μg m^{-3} would be regarded as equivalent to a 21-20 = 1 μg m^{-3} decrease down to 20 μg m^{-3}. We were concerned that this interpretation would underestimate the effect compared with the original data.

It is important to emphasise that:
(i) Most studies that contributed to the meta-analysis used to set the concentration-response relationship did not examine whether there was a threshold or not but the concentration-response relationships are fitted using all the data and an assumption of linearity with no threshold.
(ii) No studies have proved that there is a zero effect at 20 μg m^{-3} (although this is difficult to do).
(iii) There is not an absence of data below 20 μg m^{-3}, although the data is more sparse.
(iv) There are no observational data points at a zero concentration of NO_2.

The studies examining the shape of the concentration-response relationship do this by expressing the effect relative to the mortality rate at the average concentration of NO_2 so that a zero change in mortality rate (hazard ratio of 1) is in the centre of the graphs. Consideration of a threshold is in terms of whether the slope is flat and then turns to increase more steeply. This was suggested to be the case for all cause mortality in the youngest age group (51-70 years) in the study by Naess et al. (2007), although the curve was not formally tested for non-linearity. However, the curve for the older age group (71-90 years) was more or less straight with tight confidence intervals around the slope in the range 20 – 60 μg m^{-3}. It should be noted that the older age group is likely to dominate the overall concentration-response relationship for all ages as there are more deaths in the older age group.

In the study by Cesaroni et al. (2013), the relationship was again straight but the confidence intervals around the slope widen as the NO_2 concentrations decrease. When only data above 20 μg m^{-3} was analysed, the central estimate concentration-response function was the same as that using all data and it was statistically significant (G. Cesaroni; F. Forastiere, personal communication). Analysing only data below 20 μg m^{-3}, the slope was steeper but the confidence intervals were very wide, including the possibility of no effect. The results across the whole range of concentrations showed no evidence of deviance from linearity.

The HRAPIE report discussed the above two studies when considering the issue of potential thresholds, but not a further study (Raaschou-Nielsen et al., 2012) that only studied NO_2 and not $PM_{2.5}$ as well. This study found an almost linear relationship (although again confidence intervals

around the slope widen at lower concentrations). The median NO_2 concentration at the participant's residence was 16.6 µg m^{-3} and the 5th percentile was 11.5 µg m^{-3} i.e. in this particular study, data points below 20 µg m^{-3} were not sparse.

It seems from this that it is not so much that there is a clear threshold of no effect at 20 µg m^{-3} but that the shape of the relationship becomes less clear below 20 µg m^{-3}. If there were a clear threshold, this would be an obvious choice for a baseline with which to compare the burden of current concentrations. However, when this is not the case, there are a variety of options to use to choose a counter factual. Confidence intervals widening at 20 µg m^{-3} is one option but, as discussed above, there is evidence of effects below 20 µg m^{-3}. We therefore did not choose this option, although we performed a sensitivity analysis on this basis. The Global Burden of Disease study suggested using the 5th percentile of the exposure distribution of the key epidemiological study or studies as the counter-factual, with the minimum concentration as the lower bound of the counter-factual (Lim et al., 2012; Burnett et al., 2014). Although this was for $PM_{2.5}$, similar principles could be applied to NO_2. The WHO recommended concentration-response coefficient came from a meta-analysis of several studies (Hoek et al., 2013*)* and it was outside the scope of this project to review the concentration ranges in each study. Many of the cohort locations from the meta-analysis were also included in the ESCAPE study for which a paper on NO_2 concentrations has been published (Cyrys et al., 2012). The lowest NO_2 concentrations were from Umeå in Sweden (minimum 1.5 µg m^{-3}). The minimum in the study in Oslo mentioned above (Naess et al., 2007) was 2 µg m^{-3}. The minimum in an England wide study, published since the Hoek et al. (2013) meta-analysis, was 4.5 µg m^{-3} (Carey et al., 2013).

Given that, where performed, tests for non-linearity over the whole data range were not statistically significant, a reasonable counter factual would be considerably below 20 µg m^{-3}. As this was a small project, we did not do multiple sensitivity analyses but considered that doing calculations with a counter-factual at 20 µg m^{-3} and at zero would encompass the range of possible counter factuals. We consider the most appropriate counter-factual would be much closer to zero than to 20 µg m^{-3}.

We recommend further discussion of this issue so that guidance can be provided as to how to deal with choosing a counter-factual. Discussion with colleagues in the field (see acknowledgements) after the main analysis was complete suggested support for sensitivity analyses at 15, 10 and 5 µg m^{-3}, based on evidence from the studies discussed above. Further work to explore the effects of these assumptions on the results would be helpful.

7.2 ANNEX 2. Sensitivity analysis results for the mortality burden of $PM_{2.5}$

Table 26 Estimated burden of effects on annual mortality in 2010 of 2010 levels of anthropogenic $PM_{2.5}$, using COMEAP's upper and lower plausibility interval of 1 and 12% increase in mortality per 10 µg m^{-3} $PM_{2.5}$ to inform sensitivity analysis

Borough	Attributable fraction (%) 1%	Attributable fraction (%) 12%	Attributable deaths*** 1%	Attributable deaths*** 12%	Life years lost 1%	Life years lost 12%
City of London	1.8	18.4	1	7	11	111
Barking and Dagenham	1.3	13.9	16	173	223	2379
Barnet	1.3	13.9	31	326	416	4429
Bexley	1.3	13.7	23	248	312	3328
Brent	1.4	14.3	20	211	327	3478
Bromley	1.3	13.6	32	343	419	4468
Camden	1.5	15.7	16	170	278	2929
Croydon	1.3	13.9	31	325	427	4548
Ealing	1.3	14.2	25	265	384	4078
Enfield	1.3	13.7	24	259	342	3651
Greenwich	1.3	14.3	21	226	293	3111
Hackney	1.4	15.2	15	154	253	2673
Hammersmith and Fulham	1.4	15.1	13	134	206	2181
Haringey	1.4	14.4	15	158	260	2760
Harrow	1.3	13.5	18	187	272	2901
Havering	1.2	13.3	26	282	346	3700
Hillingdon	1.3	13.4	22	235	315	3359
Hounslow	1.3	14.1	18	192	276	2934
Islington	1.5	15.9	16	165	247	2601
Kensington and	1.5	15.7	12	126	198	2089

Chelsea						
Kingston upon Thames	1.3	13.8	13	138	178	1893
Lambeth	1.4	15.1	19	204	318	3361
Lewisham	1.4	14.5	21	225	313	3322
Merton	1.3	14.2	15	161	222	2362
Newham	1.4	14.7	17	179	278	2944
Redbridge	1.3	14.0	22	239	317	3376
Richmond upon Thames	1.3	13.9	15	157	218	2323
Southwark	1.5	15.5	20	207	317	3352
Sutton	1.3	13.7	18	191	241	2567
Tower Hamlets	1.5	16.2	15	158	233	2451
Waltham Forest	1.3	14.2	18	191	273	2899
Wandsworth	1.4	14.8	21	223	298	3157
Westminster	1.5	16.1	16	172	278	2930
Total			624	6632	9287	98648

***Attributable deaths and associated life years lost, age 30+ and calculated by 5 year age groups and gender.

Table 27 Estimated burden of effects on annual mortality in 2010 of NO_2, using upper and lower confidence intervals for the concentration-response coefficient of 2.2 and 5.6% increase in mortality per 10 µg m^{-3} NO_2 to inform sensitivity analysis (30% overlap with $PM_{2.5}$)

Borough	Attributable fraction (%) 2.2%	Attributable fraction (%) 5.6%	Attributable deaths*** 2.2%	Attributable deaths*** 5.6%	Life years lost 2.2%	Life years lost 5.6%
City of London	11.9	27.2	5	11	71	163
Barking and Dagenham	6.7	16.0	83	197	1140	2714
Barnet	6.9	16.5	163	387	2211	5251
Bexley	6.4	15.4	117	280	1570	3743

Brent	7.8	18.4	113	267	1872	4421
Bromley	6.3	15.1	158	377	2058	4914
Camden	9.5	22.0	102	238	1760	4100
Croydon	6.8	16.2	158	376	2217	5272
Ealing	7.7	18.2	143	339	2203	5202
Enfield	6.6	15.7	124	295	1750	4168
Greenwich	7.4	17.6	117	277	1618	3827
Hackney	8.6	20.2	87	204	1513	3547
Hammersmith and Fulham	8.8	20.7	78	182	1273	2978
Haringey	7.7	18.1	84	199	1470	3473
Harrow	6.4	15.2	88	209	1358	3241
Havering	5.7	13.7	120	289	1583	3795
Hillingdon	6.4	15.2	110	262	1579	3768
Hounslow	7.5	17.6	102	241	1555	3679
Islington	9.4	21.8	97	226	1528	3560
Kensington and Chelsea	9.8	22.8	78	182	1302	3025
Kingston upon Thames	6.9	16.3	69	163	939	2232
Lambeth	8.7	20.3	117	274	1925	4512
Lewisham	7.8	18.4	120	283	1775	4188
Merton	7.3	17.3	83	196	1212	2871
Newham	8.0	18.8	97	229	1593	3754
Redbridge	6.8	16.2	117	278	1646	3912
Richmond upon Thames	7.1	16.8	80	189	1176	2791
Southwark	9.1	21.4	122	284	1972	4605
Sutton	6.6	15.7	92	218	1231	2931

Tower Hamlets	9.6	22.4	93	217	1454	3384
Waltham Forest	7.3	17.2	96	228	1471	3486
Wandsworth	8.2	19.3	123	290	1747	4109
Westminster	10.2	23.6	109	253	1858	4301
Total			3444	8138	51629	121918

***Attributable deaths and associated life years lost, age 30+ and calculated by 5 year age groups and gender.

Table 28 Estimated burden of effects on annual mortality in 2010 of NO_2, using upper and lower confidence intervals for the concentration-response coefficient of 3.1 and 8% increase in mortality per 10 µg m^{-3} NO_2 to inform sensitivity analysis (no overlap with $PM_{2.5}$)

Borough	Attributable fraction (%) 3.1%	Attributable fraction (%) 8%	Attributable deaths*** 3.1%	Attributable deaths*** 8%	Life years lost 3.1%	Life years lost 8%
City of London	16.3	36.1	6	14	97	216
Barking and Dagenham	9.3	21.8	115	269	1578	3703
Barnet	9.6	22.5	225	527	3058	7157
Bexley	8.9	21.0	162	382	2174	5114
Brent	10.8	24.9	156	362	2585	6001
Bromley	8.7	20.6	219	515	2851	6720
Camden	13.0	29.6	140	320	2422	5517
Croydon	9.4	22.1	219	513	3067	7192
Ealing	10.6	24.7	198	460	3042	7062
Enfield	9.1	21.4	172	403	2422	5691
Greenwich	10.3	23.9	162	377	2235	5203
Hackney	11.9	27.3	120	275	2085	4793
Hammersmith and Fulham	12.2	27.9	107	246	1753	4019
Haringey	10.6	24.6	116	270	2030	4716
Harrow	8.8	20.8	121	286	1881	4431

Havering	7.9	18.7	167	396	2194	5206
Hillingdon	8.8	20.8	152	358	2187	5152
Hounslow	10.3	24.0	141	327	2149	5000
Islington	12.9	29.4	133	304	2102	4792
Kensington and Chelsea	13.5	30.6	108	245	1790	4062
Kingston upon Thames	9.5	22.2	95	222	1300	3044
Lambeth	11.9	27.4	161	370	2653	6096
Lewisham	10.8	25.0	165	384	2450	5684
Merton	10.1	23.5	114	267	1675	3906
Newham	11.0	25.5	134	310	2199	5090
Redbridge	9.4	22.1	162	379	2277	5336
Richmond upon Thames	9.8	22.9	110	258	1627	3802
Southwark	12.6	28.8	167	383	2715	6206
Sutton	9.1	21.5	127	298	1704	4001
Tower Hamlets	13.2	30.1	128	292	2000	4550
Waltham Forest	10.0	23.4	133	311	2033	4746
Wandsworth	11.3	26.1	170	393	2410	5565
Westminster	14.0	31.7	150	339	2551	5763
Total			4756	11054	71294	165536

***Attributable deaths and associated life years lost, age 30+ and calculated by 5 year age groups and gender.

7.3 ANNEX 3. Concentration-response functions and baseline rates used in calculation of the effects of short-term exposure to PM and NO_2

The concentration-response functions used to calculate deaths brought forward and hospital admissions as a result of short-term exposure to PM_{10}, $PM_{2.5}$ and NO_2 are given in Table 29 below. The baseline annual numbers of all-cause deaths all ages (excluding external causes (ICD10 V01 - Y89 and U509)) in London (2009/2010/2011) or baseline annual numbers of emergency hospital admissions all ages in London (first episode, finished consultant episode, London residents) (2010/11) for respiratory disease ICD 10 J00-J99 and cardiovascular disease ICD 10 I00-I99 are also given.

Table 29 Concentration-response functions and baseline rates used in calculation of the effects of short-term exposure NO_2, $PM_{2.5}$ and PM_{10}

Pollutant	Outcome	% increase per 10 µg m^{-3}	Lower 95% confidence interval	Upper 95% confidence interval	Baseline numbers of health outcome
NO_2	Deaths brought forward[a]	0.27	0.16	0.38	46,397
NO_2	Respiratory hospital admissions[a]	0.15	-0.08	0.38	75,953
$PM_{2.5}$	Deaths brought forward[b]	1.23	0.45	2.01	46,397
$PM_{2.5}$	Respiratory hospital admissions[b]	1.90	-0.18	4.02	75,953
$PM_{2.5}$	Cardiovascular hospital admissions[b]	0.91	0.17	1.66	59,005
PM_{10}	Deaths brought forward[c]	0.75	0.62	0.86	46,397
PM_{10}	Respiratory hospital admissions[c]	0.8	0.48	1.12	75,953
PM_{10}	Cardiovascular hospital admissions[d]	0.8	0.6	0.9	59,005

[a] From WHO (2014) for daily max 1 hour average NO_2.

[b] From WHO (2014) and Atkinson et al. (2014) for daily 24-hour average.

[c] Central estimate from COMEAP (1998), itself derived from WHO (2000) which gives the confidence intervals (available in draft form at the time of the COMEAP (1998) report). PM_{10} 24-hour average concentration response functions used as an alternative to $PM_{2.5}$.

[d] From COMEAP (2001).

7.4 ANNEX 4. The London Emissions Toolkit and the London Air Quality Toolkit

This section provides a summary of the London Air Quality Toolkit (LAQT). For those readers requiring further information, a complete description of the model is available from a Health Effects Institute report[59].

In brief, the LAQT model used a kernel modelling technique, based upon the ADMS 4 and ADMS-roads models[60] , to describe the initial dispersion from each emissions source. The contribution from each source was summed onto a fixed 20 m x 20 m grid across London assuming that one can calculate the contribution of any source to total air pollution concentrations by applying each kernel and adjusting for the source strength. The kernels have been produced using an emissions source of unity, either 1 g s^{-1} (point and jet sources), 1 g m^{-3} s^{-1} (volume sources) or a 1 g km^{-1} s^{-1} (road and railway sources) and have been created using hourly meteorological measurements from the UK Meteorological Office site at Heathrow. Data from the Heathrow site is recorded at a height of 10 metres and includes measurements of temperature, wind speed, wind direction, precipitation, relative humidity and cloud cover.

The LAQT model is computationally efficient and provides annual mean concentrations of NO_X, NO_2, PM_{10} and $PM_{2.5}$ as well as the number of exceedences of the daily PM_{10} 50 µg m^{-3} objective, all at a resolution of 20 m x 20 m. Predictions in London were based upon the most recent version of the LAEI (LAEI2010), available from the London Datastore[61].

King's highly flexible emissions model, the 'London Emissions Toolkit (LET)', and air quality model, the London Air Quality Toolkit (LAQT), have both been an integral part of the quantification of transport policies such as:

- Congestion Charging Scheme (CCS) and Western Extension Zone (WEZ) (http://www.tfl.gov.uk/roadusers/congestioncharging/6722.aspx);
- Low Emissions Zone (LEZ): King's has provided emissions (and concentrations) at every stage of the LEZ from the feasibility study of LEZ options during the design of the scheme (http://www.tfl.gov.uk/assets/downloads/roadusers/lez/phase-2-feasibility-summary.pdf), to the most recent monitoring report (http://www.tfl.gov.uk/assets/downloads/roadusers/lez/lez-impacts-monitoring-baseline-report-2008-07.pdf);
- Mayor's Air Quality Strategy (MAQS): King's has provided the emissions (and concentrations) results for all elements of the MAQS (http://www.london.gov.uk/publication/mayors-air-quality-strategy);

[59] Health Effects Institute, 2011. The Impact of the Congestion Charging Scheme on Air Quality in London. Available from: <http://pubs.healtheffects.org/getfile.php?u=638> Accessed on 22/06/2013.
[60] CERC, 2013, ADMS 5 and ADMS-roads User Guides. Available from: <http://www.cerc.co.uk/environmental-software/model-documentation.html> Accessed 22/06/2013.
[61] The London Datastore, 2013. Available from: <http://data.london.gov.uk/> Accessed 22/06/2013

- Olympic Route Network (ORN): The emissions (and concentrations) impacts of traffic changes on the ORN (http://www.tfl.gov.uk/assets/downloads/corporate/tfl-orn-air-quality-report-march-2012.pdf);
- Ultra Low Emission Zone (ULEZ): The LET has provided the emissions (and concentrations) calculations for the ULEZ policy analysis during the early design of the scheme (ULEZ briefing – http://www.london.gov.uk/sites/default/files/ULEZ%20scrutiny%20briefing%20-%20July%202013.pdf).
- As part of the ULEZ public consultation, King's has also just produced the latest emissions (and concentrations) calculations. https://consultations.tfl.gov.uk/environment/ultra-low-emission-zone

7.5 ANNEX 5. Additional effects on hospital admissions and deaths brought forward, population-weighted average concentration, total population and baseline number of death brought forward and hospital admissions for the year 2008, 2010, 2012, 2015 and 2020

Table 30 Additional analyses on hospital admissions and deaths brought forward for the year 2008, 2010, 2012, 2015 and 2020 for $PM_{2.5}$ and PM_{10}

Pollutant	Year	Central Relative Risk (RR) with lower and upper 95% confidence interval per 10 µg m^{-3}	Numbers of hospital admissions or deaths brought forward
Anthropogenic $PM_{2.5}$	2008	Deaths brought forward	745 (272 – 1218)
	2010	(RR 1.0123 (1.0045– 1.0201))	787 (287 – 1288)
	2012		760 (278 – 1244)
	2015		732 (268 – 1198)
	2020		691 (253 – 1131)
$PM_{2.5}$	2008	Deaths brought forward	817 (299 – 1337)
	2010	(RR 1.0123 (1.0045– 1.0201))	818 (299 – 1340)
	2012		792 (289 – 1296)
	2015		764 (279 – 1250)
	2020		723 (264 – 1182)
$PM_{2.5}$	2008	Respiratory Hospital Admissions	1821 (-172 – 3867)
	2010	(RR 1.019 (0.9982 – 1.0402))	2072 (-195 – 4405)
	2012		2004 (-189 – 4258)
	2015		1934 (-183 – 4106)
	2020		1829 (-173 – 3881)
$PM_{2.5}$	2008	Cardiovascular Hospital Admissions	717 (134 – 1311)
	2010	(RR 1.0091 (1.0017 - 1.0166))	769 (144 – 1406)
	2012		744 (139 – 1360)
	2015		718 (134 – 1312)
	2020		680 (127 – 1241)

Anthropogenic PM_{10}	2008	Deaths brought forward	699 (577 – 802)
	2010	(RR 1.0075 (1.0062– 1.0086))	750 (619 – 860)
	2012		735 (608 – 844)
	2015		723 (597 – 829)
	2020		706 (583 – 810)
Anthropogenic PM_{10}	2008	Respiratory Hospital Admissions	1074 (643 – 1506)
	2010	(RR 1.008 (1.0048– 1.0112))	1309 (784 – 1836)
	2012		1285 (769 – 1802)
	2015		1263 (756 – 1771)
	2020		1233 (739 – 1729)
Anthropogenic PM_{10}	2008	Cardiovascular Hospital Admissions	885 (663 – 996)
	2010	(RR 1.008 (1.006– 1.009))	1017 (762 – 1145)
	2012		998 (748 – 1123)
	2015		981 (735 – 1104)
	2020		958 (718 – 1078)
PM_{10}	2008	Deaths brought forward	820 (678 – 941)
	2010	(RR 1.0075 (1.0062– 1.0086))	803 (663 – 921)
	2012		788 (651 – 905)
	2015		776 (641 – 890)
	2020		759 (627 – 871)
PM_{10}	2008	Respiratory Hospital Admissions	1261 (755 – 1769)
	2010	(RR 1.008 (1.0048– 1.0112))	1402 (839 – 1967)
	2012		1377 (825 – 1932)
	2015		1355 (811 – 1901)
	2020		1326 (794 – 1859)
PM_{10}	2008	Cardiovascular Hospital Admissions	1039 (779 – 1170)
	2010	(RR 1.008 (1.006– 1.009))	1089 (816 – 1226)

2012	1070 (801 – 1204)
2015	1053 (789 – 1185)
2020	1030 (771 – 1159)

Table 31 Total Population-weighted average concentration (PWAC) (μg m^{-3}) of $PM_{2.5}$, PM_{10} and NO_2

Year	Anthropogenic $PM_{2.5}$ PWC (μg m^{-3})	Total $PM_{2.5}$ PWC (μg m^{-3})	Anthropogenic PM_{10} PWC (μg m^{-3})	Total PM_{10} PWC (μg m^{-3})	Total NO_2 PWC (μg m^{-3})
2008	12.44	13.65	19.11	22.41	37.92
2010	13.76	14.30	21.45	22.95	36.67
2012	13.29	13.84	21.05	22.55	34.91
2015	12.81	13.36	20.69	22.19	32.87
2020	12.10	12.64	20.21	21.71	28.29

Table 32 Total Population and baseline number of death brought forward and hospital admissions in London (2008/09 applied to 2008 and 2010/11 applied to 2010 onwards)

Year	Population	Baseline Number DBF (excluding external causes) in London	Baseline number of emergency RHA in London	Baseline number of emergency CHA in London
2008	7816076	48581	69990	57682
2010	8069499	46397	75953	59005
2012	8069499	46397	75953	59005
2015	8069499	46397	75953	59005
2020	8069499	46397	75953	59005

7.6 ANNEX 6. Caveats and uncertainties in using damage costs

7.6.1 Short and long-term policies

The impact pathway approach follows up effects on mortality for 105 years from the start of the pollution change, as does the derivation of the damage costs per tonne. Damage costs per tonne use an annual pulse i.e. the impacts of a 1 year change in pollution on the subsequent 105 years. However, when applying the damage costs for sequential years, the impact extends for a further year each time. The longer the pollution change applies, the more it will overestimate the results of the impact pathway approach. For this reason, Defra recommends that the use of damage costs is inappropriate for policies lasting longer than about 20 years (IGCB, 2007; Defra, 2011b).

The damage costs treat each year completely independently. This is fine for short-term effects and for outcomes expressed as effects of annual average pollution exposure on annual incidence or prevalence of a health outcome. This assumption of independence of one year from the next is, however, an inaccuracy for the effects of long-term exposure to pollution on mortality. This is because a change in the numbers surviving from one year to the next, as a result of pollution, changes the size and age of the population in the following year. This change in population size and age distribution affects the baseline death rate in the following year and thus affects the calculation of the impact of pollution in that year. Damage costs applied over several years ignore this effect. Omitting this effect is a tolerable approximation for short-lived policies but the effects of this year by year change in the population size and age distribution leads to increasing differences from the full impact pathway approach the longer pollution changes persist. This is another reason for limiting analysis to policies lasting less than 20 years.

7.6.2 Non-linearities

The studies used to derive the concentration-response functions use logistic or Poisson regression, or Cox proportional hazard modelling in their analysis, all of which involve logarithmic relationships. This has been taken into account in the original derivation of the damage costs per tonne but subsequent analysis using the damage cost calculator to scale by tonnes of emissions assumes linearity. This is a reasonable approximation for small concentration (emission) changes but differences from the impact pathway approach will increase with the size of the concentration/emission increment. Logarithmic relationships also imply that the impacts derived from the slope of the relationships will not be the same when the same size concentration increment is calculated as an increase or a decrease. Damage cost analysis will give the same answer for an increase or decrease for the same number of tonnes of emissions.

7.6.3 Non-linearities of NO_2 concentration changes

We would not advise calculating annual mean NO_2 concentration changes based solely on NO_X emissions changes.

NO_2 concentrations include contributions from both directly emitted primary NO_2 and secondary NO_2 formed in the atmosphere by the oxidation of NO. There is no simple linear relationship between NO_2 concentrations and NO_X emissions or concentrations.
The magnitude of reduction in NO_2 concentration associated with a reduction in NO_X emissions depends on the reduction of primary NO_2 and the initial NO_X concentration. Thus, the same reduction in NO_X emissions will have a different impact on NO_2 concentrations where both the primary NO_2 reduction and initial NO_X concentration are different.

The damage cost calculator was built using a reduction in NO_X and primary NO_2 emissions on an initial NO_X concentration in 2010. The same reduction in NO_X emissions will have a higher impact on NO_2 concentrations in future predictions if the initial NO_X concentration is lower (for example 2020/2025 *versus* 2010 NO_X levels). This is important given the very high benefit of the extended set associated with long term exposure to NO_2 and mortality.

The paragraphs above demonstrate that while use of damage costs is convenient, it can involve significant approximations. It is for this reason that Defra advises that damage costs should not be used for policies involving benefits above about £50 million (Defra, 2011b).

7.6.4 NO_2 counter factuals

Various counter factuals were discussed in Annex 1, including a sensitivity analysis with a counter factual at 20 $\mu g\ m^{-3}$. Allowing for this did not arise in the derivation of the damage costs using a small decrease from 2010 baseline concentrations. However, when analyzing future policies involving decreases in NO_X emissions, this may no longer be the case. The damage cost approach using this sensitivity analysis could then be an overestimate. With the full impact pathway approach this would be taken into account by calculating the population-weighted average and population sizes for only those output areas above the relevant counter factual.

7.6.5 Double-counting

Double counting between the effects of short-term and long-term exposure on mortality is taken into account by the following:

- Core PM damage costs include long-term exposure to $PM_{2.5}$ and mortality but not short-term exposure to $PM_{2.5}$ and mortality
- The possibility that there is not a full overlap in these effects is covered in the high sensitivity damage costs, where both are included
- Core NO_2 damage costs include the effects of short-term exposure on mortality but not the effects of long-term exposure
- Extended NO_2 damage costs include the effects of long-term exposure to NO_2 on mortality but exclude the effects of short-term exposure to NO_2 and mortality.

Double counting between pollutants is taken into account in the following way:

- The coefficient for NO_2 and deaths brought forward is adjusted for PM_{10} (WHO, 2013b)
- The coefficient for NO_2 and respiratory hospital admissions is not adjusted but is supported by evidence that the coefficient is robust to adjustment for other particle metrics (WHO, 2013b)

- The coefficient for NO_2 and bronchitic symptoms in asthmatic children is adjusted for organic carbon (the smallest adjusted coefficient of those adjusted for a wide variety of pollutants), (WHO, 2013b)
- The coefficient for long-term exposure to NO_2 and mortality is reduced by 30% to allow for overlap with the effects of $PM_{2.5}$. The maximum overlap of 30% was recommended in WHO (2013a) but was regarded as too uncertain in WHO (2013b). It is argued in the current project that some overlap is likely even if the numerical size 'up to 30%' is uncertain, and it is better to include some overlap rather than none.

7.6.6 Caveats for adding PM and NO_2 damage costs effects together

Adding together the values for the PM and NO_2 extended sets results in large numbers. These need to be accompanied by the following caveat (with a reference to the King's report):
It is likely that inclusion of only the core PM and NO_2 damage costs underestimates the effect of long-term exposure to pollution on mortality. A significant group of studies, with fine spatial scale modelling to pick up traffic pollution contrasts, indicate an effect of long-term exposure to NO_2 and mortality. A subset of these studies showed that this effect was independent of $PM_{2.5}$ and quantification of the effects of long-term exposure was recommended in the extended set of outcomes by WHO (2013b). WHO (2013a) stated that, 'as with the short-term effects, NO_2 in these studies may represent other constituents. Despite this, the mechanistic evidence, particularly on respiratory effects, and the weight of evidence on short-term associations are suggestive of a causal relationship'. So, there is likely to be an effect of long-term exposure to NO_2. Nonetheless, the use of the extended set damage costs for NO_2, which includes the effects of long-term exposure to NO_2 on mortality, may overestimate the effects of NO_2 to some extent, as part of the effect may be due to other traffic pollutants.

7.7 ANNEX 7. Monetary values used for damage costs

The monetary values for the health endpoints estimated in the analysis are shown below. All values are in £2014 prices. Note that the values in the CORE IGCB/DEFRA set are the recommended values from Defra (2013) updated to current prices. The extended set values are based on an update of Hurley et al. (2005).

Table 33 Unit values for Health Valuation

CORE IGCB/DEFRA		**£2014 prices**	
	Low	**Central**	**High**
Acute mortality (death brought forward)*	£3,118	£6,236	£9,355
Year of Life Lost (chronic) **	£27,336	£36,379	£45,526
Respiratory hospital admissions	£2,702	£6,912	£11,122
Cardiovascular hospital admissions	£3,118	£6,704	£10,290
*Value £18709 (2014) for life lost in poor health, then assuming 2-6 months loss of life expectancy for every death brought forward. Low and high reports range of 2 and 6 months			
** note calculation needs to be based on life tables			
Extended set - values	**Low**	**Central**	**High**
Chronic bronchitis (case)	£38,354	£53,519	£89,191
Severe COPD (case)	£62,437	£107,027	£231,901
Restricted activity day (RADs)	£173	£173	£173
Minor Restricted activity day (MRAD)			
Adults	£51	£51	£51
Children	£51	£77	£102
Lower respiratory symptoms (LRS)			
Adults	£51	£51	£51
Children	£51	£77	£102
Asthma attack			
Adults	£66	£71	£86
Children	£67	£108	£170
Lung cancer	£62,437	£642,183	£3,786,070
Acute bronchitis in children (6 - 12 yrs)	£1,984	£3,217	£5,147
Bronchitic symptoms in asthmatic	£3,967	£6,433	£10,293

children (2 months)			
Post neonatal mortality (1 - 12 months)	£2,066,373	£3,099,559	£4,132,745

7.8 ANNEX 8. Full Set of Damage Cost Values

The full set of CORE Damage costs are presented below.

Table 34 $PM_{2.5}$ Damage Costs, 2014 prices

	£ per tonne, 2014 prices				
$PM_{2.5}$	CORE	Low	High	Low sensitivity	High sensitivity
Central London	**125,329**	93,699	157,314	16,667	304,414
Inner London	**157,794**	117,950	198,083	21,001	383,235
Outer London	**90,466**	67,656	113,531	12,000	219,757

Table 35 NO_X Damage Costs, 2014 prices

	£ per tonne, 2014 prices				
NO_X	CORE	Low	High	Low sensitivity	High sensitivity
Central London	**884**	641	1,129	138	2,085
Inner London	**910**	653	1,169	149	2,125
Outer London	**861**	631	1,093	127	2,049

Notes:

- The low and high values used the CORE health impact set, but then use the low and high values for mortality and morbidity, using the recommended low and high estimates in the Defra impact assessment guidance (Defra, 2013), updated to 2014 prices (see Annex 7).
- For the low and high sensitivity values, long-term exposure and mortality life years lost for $PM_{2.5}$ was estimated using the lower and higher (sensitivity) hazard rate from IGCB (2007) and COMEAP (2010), i.e. 1% and 12%. They also adopt the low and high monetisation values for all end-points. For the high sensitivity value, short-term exposure of $PM_{2.5}$ deaths brought forward was also included.

The full set of extended adders is presented below.

Table 36 EXTENDED SET - ADDER: $PM_{2.5}$ Damage Costs, 2014 prices

	£ per tonne, 2014 prices				
$PM_{2.5}$	EXTENDED	Low	High	Low sensitivity	High sensitivity
Central London	**118,360**	118,360	118,360	112,499	126,635
Inner London	**152,884**	152,884	152,884	146,131	162,759
Outer London	**79,540**	79,540	79,540	76,018	84,686

Includes: Restricted activity days (avoiding overlap with restricted activity days due to hospital admissions, and bronchitis in children). Notes:

- The low and high values reflect the valuation range (see Annex 7). In this case, there is no range for valuation of restricted activity days so the figures do not differ.
- For the low and high sensitivity values include the valuation range and the impact function range.

Table 37 EXTENDED SET - ADDER: PM_{10} Damage Costs, 2014 prices

	£ per tonne, 2014 prices				
PM_{10}	EXTENDED	Low	High	Low sensitivity	High sensitivity
Central London	**22,395**	15,397	36,071	4,888	60,949
Inner London	**27,598**	18,974	44,452	6,026	75,090
Outer London	**14,224**	9,779	22,910	3,106	38,697

Includes:

- Infant mortality;
- Asthmatic symptoms in asthmatic children;
- Prevalence of bronchitis in children;
- Incidence of adult bronchitis.

Notes:

- The low and high values reflect the valuation range (see Annex 7).
- For the low and high sensitivity values include the valuation and the impact function range.

Note that the extended set includes separate PM_{10} and $PM_{2.5}$ damage costs. These relate to different health endpoints and thus they are additive, thus the total damages from PM for an extended analysis would be the sum of CORE $PM_{2.5}$+ EXTENDED PM_{10} + EXTENDED $PM_{2.5}$.

Table 38 EXTENDED SET - ADDER: NO_X Damage Costs, 2014 prices

	£ per tonne, 2014 prices				
NO_X	EXTENDED	Low	High	Low sensitivity	High sensitivity
Central London	**39,442**	28,917	51,222	14,574	84,739
Inner London	**52,344**	38,367	68,003	19,311	112,598
Outer London	**27,948**	20,500	36,272	10,356	59,833

Includes:

- Chronic Mortality with 30% adjustment downwards.
- Bronchitic symptoms in asthmatic children.

Notes:

- The low and high values reflect the valuation range (see Annex 7).
- For the low and high sensitivity values include the valuation and the impact function range.

7.9 ANNEX 9. Total (i.e. including the non-anthropogenic part) $PM_{2.5}$ and PM_{10} estimates of the costs of current air pollution for hospital admissions and deaths brought forward

Table 39 Estimate of DBF, RHA and CHA costs of 2010 current air pollution in London for total $PM_{2.5}$/PM_{10} and NO_2 for the lower/central/upper sensitivity interval

Pollutants	Sensitivity estimate	DBF economic impact	RHA economic impact	CHA economic impact
$PM_{2.5}$	lower	£932,070	-	£447,506
$PM_{2.5}$	central	£5,103,829	£14,324,991	£5,158,684
$PM_{2.5}$	upper	£12,531,418	£48,988,334	£14,467,133
PM_{10}	lower	£2,066,893	£2,267,858	£2,543,298
PM_{10}	central	£5,004,747	£9,689,032	£7,300,533
PM_{10}	upper	£8,614,276	£21,871,754	£12,614,458

7.10 ANNEX 10. Method for calculating health outcomes from studies based on logistic regression – a worked example for PM_{10} and chronic bronchitis in adults

This method is also the basis for the calculation for PM_{10} and postneonatal mortality, asthma symptoms and acute bronchitis in children and to NO_2 and bronchitic symptoms in asthmatic children.

The studies examining the effect of air pollution on, for example, chronic bronchitis get results in terms of a binary outcome i.e. subjects answer yes or no to a question regarding having or not having symptoms of cough and/or phlegm for 3 months of the year for 2 years in a row. The relationship between a binary outcome (presence or absence of symptoms) and a variable (pollutant concentration) is analysed using logistic rather than linear regression. Logistic regression plots the natural logarithm of the odds against the pollutant concentration as this gives a linear relationship. Hence, if we want to scale our health impact by concentration, this should ideally be done on the log odds scale.

The HRAPIE project made recommendations assuming that odds ratios could be assumed to be the same as relative risks (the risk is the numbers with symptoms divided by the total number of subjects rather than by the number of subjects without symptoms as for the odds). This is a reasonable approximation when the prevalence is low (Davies et al. 1998), as it is in the chronic bronchitis example we are using here. However, it is not the case for some of the other outcomes for which we have done calculations and it is easier to use the same method throughout. The main HRAPIE recommendations also assume linearity but while logarithmic relationships can be assumed to be linear for small concentration changes, this is not the case for larger concentration changes. The concentration changes used to derive damage costs are small but it is worth noting that further use of damage costs assumes linearity in scaling by tonnes of emissions. This means that damage costs become more inaccurate for large concentration changes such as in burden calculations, which should be done directly rather than via damage costs.

Bearing in mind the points above, we have taken the view that where we can improve the accuracy of the calculation by a small amount we should do so. Otherwise there is the potential for small approximations to add up over the full chain of calculations used to derive damage costs. We have therefore used the method that scales by concentration on the log odds scale, as set out below.

We start from the HRAPIE recommendation to use a baseline incidence of 3.9 new cases of chronic bronchitis[62] per 1000 adults who previously had no symptoms. This is equivalent to a probability of 0.0039, with a probability of (1000 – 3.9)/1000 = 0.9961 for people not having

[62] There is some uncertainty as to what chronic bronchitis, as defined by studies using questionnaires, actually means in terms of severity. It probably does not mean quite the same thing as a clinical diagnosis of chronic bronchitis.

new persistent symptoms. The odds is the ratio of the number of people having symptoms to the number of people not having symptoms i.e. 3.9/996.1 or, equivalently 0.0039/0.9961. Thus, in steps:

1) Baseline probability of chronic bronchitis (P_b) = 0.0039
2) Baseline odds of chronic phlegm (O_b) = $P_b/(1- P_b)$ = 0.0039/0.9961 = 0.00392
3) Baseline log odds of chronic phlegm = ln ($P_b/(1- P_b)$) =ln 0.00392 = -5.5429 (for use later)
4) Turning to the effect of pollution, we start with the odds ratio for a 10 μg m^{-3} increase in PM_{10} of 1.117, as recommended by HRAPIE. The odds ratio is the ratio between the odds of chronic bronchitis at a PM_{10} concentration 10 μg m^{-3} higher than the baseline (O_{10}) and the odds at the baseline (O_b). Knowing the odds ratio and the baseline odds, we can derive the odds at the concentration 10 μg m^{-3} above the baseline.
5) Odds ratio (OR) = O_{10}/O_b = 1.117
6) Rearranging, O_{10} = OR x O_b = 1.117 x 0.00392 = 0.0044
7) Log odds at a 10 μg m^{-3} increased concentration = Ln 0.0044 = -5.432
8) We now have both the log odds at the baseline (step 3) and the log odds at a 10 μg m^{-3} increased PM_{10} concentration from step 6. This allows us to derive the change in log odds for a 10 μg m^{-3} increase and hence the slope of the logistic regression.
9) Change in log odds for a 10 μg m^{-3} increase[63] = ln O_{10} – ln O_b = -5.432 – (-5.5429) = 0.111
10) Change in log odds per μg m^{-3} increase (slope of the logistic regression) = 0.111/10 = 0.0111
11) We are now in a position to derive the change in log odds for a new concentration change. In this example, this is the population-weighted[64] average concentration change across the whole of London as a result of a 10% reduction in transport emissions in central, inner or outer London. Here, we use the example for central London of a PM_{10} population-weighted average concentration change of -0.0188 μg m^{-3} (this is a very small change as central London is a small area for a change in emissions and the impact on concentration is averaged across the whole of London).
12) The baseline log odds already includes the effect of current levels of pollution. Therefore, we need the change (decrease) in log odds that relates to the decrease in population-weighted average anthropogenic PM_{10} from the baseline. In other words, we scale by concentration on the log odds scale because the analysis in the original studies is based on plotting the log odds against the concentration. To find this change in log odds we multiply the slope from step 8 by the new concentration change (-0.0188 μg m^{-3}) with a negative sign as it is a decrease. This negative sign is important – while the log odds scale is linear, the calculations subsequently come out of the log scale and as, for example, a plot of odds against concentration is curved, an increase and a decrease of the same amount will give different answers (i.e. the slope is different at different absolute concentrations).
13) New change in log odds for a 0.0188 μg m^{-3} decrease in PM_{10} = 0.0111 x -0.0188 = -0.000208

[63] The change in log odds for a 10 μg m^{-3} increase is the same as the log of the odds ratio per 10 μg m^{-3} increase as dividing two numbers is the same as subtracting their logs.

[64] Population weighting the average concentration across London allows us to do one calculation for London, rather than separate calculations in each output area. It assumes that the background incidence is the same across London.

14) This, in turn, gives us the log odds at the new lower concentration i.e. the concentration 0.0188 μg m^{-3} below the baseline. This is the baseline log odds plus the change in log odds (which gives a smaller log odds because the change is negative)
15) Log odds at new lower concentration of PM_{10} = ln O_b + (-0.000208) = -5.5429 + (-0.000208) = -5.543
16) Reversing the previous steps by taking the antilog of the figure from step 10 and then converting the resulting odds back to a probability by reversing step 2, gives the proportion of subjects with new chronic bronchitis at the new lower concentration of PM_{10}.
17) Odds of new chronic bronchitis at new lower concentration of PM_{10} ($O_{-0.02}$) = exp(-5.543) = 0.003915
18) Probability of chronic phlegm at new lower concentration of PM_{10} = $O_{-0.02}$/1+ $O_{-0.02}$ = 0.003899
19) In other words, the new incidence of chronic bronchitis in London after a 10% reduction in emissions in central London is predicted to be 3.8992 per 1000 rather than 3.9 per 1000, a difference of 0.0008 per 1000.
20) Change in incidence = 0.0038992 – 0.0039 = - 0.0000008
21) Expressing the health outcome in terms of numbers of people with new chronic bronchitis requires multiplying the incidence by the size of the relevant population at risk, in this case the London adult population age 18+ without symptoms (Pop_{risk}). The population of all adults is 6,280,596. The SAPALDIA study by Schindler et al. (2009) (which formed part of the HRAPIE recommendations) suggested that 91% of their study population were asymptomatic. The asymptomatic population at risk is therefore 6,280,596 x 0.91 = 5,715,343.
22) Change in numbers of people in London with new chronic bronchitis for a 10% reduction in emissions in central London = -0.0000008 x 5,715,343 = -4.57
23) NB – there has been some rounding for the purposes of the above explanation but we left rounding until the end in the actual calculations. The result without rounding in previous steps was -4.618 (prior to rounding at the valuation stage).

Related calculations

The above method was also used for several other calculations (all for London). The inputs were as in Table 40. Results are included within the extended set damage costs (available on request).

Table 40 Input data and health impact results for EXTENDED SET outcomes using logistic regression in the original studies

Input	Step	Chronic bronchitis in adults	Acute bronchitis in children	Asthma symptoms in asthmatic children	Postneonatal mortality	Bronchitic symptoms in asthmatic children
Pollutant		PM_{10}	PM_{10}	PM_{10}	PM_{10}	NO_2
Baseline rate	1-3	Incidence of new persistent bronchitic symptoms 3.9 per 1000 adults without symptoms (Schindler et al. 2009)	Background prevalence acute bronchitis in children 6-12 in last 12 months 18.6% (Hoek et al. 2012)	Background daily incidence of asthma symptoms in asthmatic children 17% (WHO, 2013b)	Baseline rate of postneonatal mortality 1.4 per 1000 live births (ONS)*	Background rate bronchitic symptoms in children with 'ever asthma' 38.7% (McConnell et al. 2003) sensitivity 21.1%
Odds ratio	4-8	1.117 (1.04 – 1.189)	1.08 (0.98 – 1.19)	1.028 (1.006 – 1.051)	1.04 (1.02 – 1.07)	1.021 (0.99 – 1.06)
Odds ratio increment μg m^{-3})	4-8	10	10	10	10	1
Population-weighted average concentration increments across Greater London from 10% reduction in emissions in central, inner or outer London (μg m^{-3})	9-13	Central -0.0188, Inner -0.1268, Outer -0.1525	Central -0.0188, Inner -0.1268, Outer -0.1525	Central -0.0188, Inner -0.1268, Outer -0.1525	Central -0.0188, Inner -0.1268, Outer -0.1525	Central -0.0745 Inner -0.4498 Outer -0.5009
Population basis (London)	14	5715342.663 adults 18+ without persistent bronchitic symptoms	645034.3 children aged 6-12	160041asthmatic children aged 5-19	131111live births	232267 children aged 5-14 with 'ever asthma'
Derivation of population basis	14	6280596.333 adults 18+ times 91% without persistent bronchitic symptoms from Schindler *et al.* (2009)		1397738 children aged 5-19 times 11.45% with severe asthma in North and South Thames from Lai et al. (2009)*		929067 children aged 5-14 times 25% with 'ever asthma' in North and South Thames from Lai et al. (2009)* Sensitivity 11.45%*

*Local data rather than general recommendation for Europe from HRAPIE.

7.11 ANNEX 11. GLOSSARY

Several definitions have been derived from Gowers et al. (2014).

30Plus: Population aged 30 and above (as this was the population used in the original studies used to derive the coefficient for long-term exposure to PM and mortality)

ADMS: Atmospheric Dispersion Modelling Software.

Anthropogenic average concentration: $PM_{2.5}$ concentration without the natural component of $PM_{2.5}$ (in this case sea salt from $PM_{2.5}$ is equal to 0.55 μg m^{-3} in 2010); consistent with EU guidance (European Commission, 2011).

Total NO_2 concentration was used as the state of knowledge (European Commission, 2011) does not allow for a natural part of NO_2 to be measured or quantified.

Attributable fraction: The proportion of deaths estimated as due to long-term exposure to anthropogenic particulate air pollution or to nitrogen dioxide.

Attributable deaths: Long-term exposure to anthropogenic particulate air pollution or to nitrogen dioxide is estimated to have an effect on mortality risks equivalent to the number of attributable deaths. Air pollution is likely to contribute a small amount to the deaths of a larger number of exposed individuals rather than being solely responsible for the number of deaths equivalent to the calculated figure of attributable deaths.

BoroughID: Nine character code for local authority district as defined by ONS in 2011.

Bronchitic symptoms: Symptoms of cough and phlegm.

Bronchitis: Inflammation of the main airways (bronchi) in the lung.

Burden: The total effect on health or a specific health outcome resulting from the total amount or majority of air pollution. Given as a 'snapshot' in a specified year. See COMEAP (2010).

CAFE: Clean Air for Europe.

Cardiovascular: Relating to the heart and circulation. Includes stroke and problems with arteries or veins in other parts of the body not just the heart.

CHA: Abbreviation for cardiovascular hospital admissions used in this report.

Chronic: Long-term e.g. a disease is chronic if it is established over the long-term rather than transient.

Cohort: A group of people that have shared experience over a particular time period e.g. a birth cohort is the group of people born in a particular year followed over time.

Concentration-response coefficient: In this report, a number representing the slope of a graph between air pollutant concentrations and a health effect.

COMEAP: Committee on the Medical Effects of Air Pollutants.

CRF: Abbreviation for concentration-response coefficient.

95% Confidence interval: A statistical measure of uncertainty calculated in such a way that, in the absence of bias, 95% of such intervals will include the parameter being estimated.

COPD: Chronic obstructive pulmonary disease.

Counter factual: A pollutant concentration used for comparison with the total effect of pollution. This can be a concentration of zero, the lowest concentration found in the environment, the lowest concentration in studies of the effects of air pollution on health or other options (see Annex 1).

Cox proportional hazard model: A form of statistical analysis used to investigate time to events (e.g. time until death) that assumes that the hazard rates in different groups (e.g. high and low pollution exposure) are related in a constant proportion.

Damage costs: Damage costs reflect the health impact of a tonne of emissions of a particular pollutant, expressed in monetary terms. They value impacts from the perspective of social welfare, and capture the wider costs to society as a whole (the environmental, social and economic impacts). For health impacts, this includes analysis of resource costs, opportunity costs and dis-utility.

Deaths 30+ average 2009/2010/2011: The total of the deaths aged 30 and above for each year 2009, 2010 and 2011 averaged to give a more stable estimate for 2010 (avoiding the random variability as a result of small numbers of deaths).

Deaths brought forward (DBF): Term used in COMEAP (1998) to denote the fact that the increased deaths detected in time-series studies of daily exposure to air pollutants are not additional deaths but deaths occurring at an earlier time than expected, perhaps by only a short time. Subsequent work has indicated that at least some of the deaths are brought forward by months or more.

Defra: Department for the Environment, Food and Rural Affairs.

EPA: United States Environmental Protection Agency.

ERG: Environmental Research Group.

GLA: Greater London Authority.

Hazard rate: The mortality rate in a specific age group conditional on reaching that age.

HMT: Her Majesty's Treasury.

Hospital admissions: Self-explanatory but the quantified hospital admissions are either additional or brought forward; the original studies do not distinguish between these possibilities (see COMEAP (1998)). Air pollution related hospital admissions cannot be identified directly in individuals but hospital admissions increase on high air pollution days after other possible explanations have been taken into account.

HRAPIE: Health Risks of Air Pollution in Europe. A project co-funded by WHO and the European Commission to provide concentration-response coefficients for use in cost benefit analysis of policy measures by the European Commission.

IGCB: Inter-department Group on Costs and Benefits (a group of officials, mainly economists, from different Government Departments, co-ordinating cost benefit analysis of policies to reduce air pollution).

Impact: The effect on health or a specific health outcome of a change in air pollution.

Incidence: the number of new cases of a disease over a period of time.

IOM: Institute for Occupational Medicine, Edinburgh.

IOMLIFET: System for calculating changes in life tables using Excel spreadsheets separating age and calendar year. Developed by IOM. See http://www.iom-world.org/research/research-expertise/statistical-services/iomlifet/

ICD: International Classification of Disease. ICD codes are numerical codes defined by WHO to ensure a common understanding of exact disease definitions. ICD10 is the 10th revision of the ICD codes.

KCL: King's College, London.

Kernel: kernel methods are a class of algorithms for pattern analysis.

LAEI: London Atmospheric Emissions Inventory. An inventory of the amount of pollutants emitted from particular sources in London.

LAQT: London Air Quality Toolkit.

Life expectancy: The average number of years lived in a defined population, often a birth cohort (all those born in the starting year).**Life tables**: Tables which show, for each age, the probability that a person will die before their next birthday (if given by 1 year age groups).
Life time: Time period encompassing the life years lived of the vast majority of people. In this report, 105 years has been used following IOMLIFET. The most recent ONS statistics on deaths by single year of age go up to age 105+ http://www.ons.gov.uk/ons/publications/re-reference-tables.html?edition=tcm%3A77-317522
Life year: One year lived for one person. Usually added up over the population and a specific duration. Allows quantification of changes in timing of deaths.
Logistic regression: A form of regression used for outcomes defined in categories e.g. with or without symptoms. It plots the natural logarithm of the odds against, for example, the pollutant concentration.
Meta-analysis: A statistical method used to combine the results of a number of individual studies.
Monte Carlo analysis: A problem solving technique used to approximate the probability of certain outcomes by running multiple trial runs, called simulations, using random variables.
Mortality: Relating to death.
m^{-3}: per cubic metre.
µg: microgramme. One millionth of a gramme.
NAEI: National Atmospheric Emissions Inventory.
NO_2: Nitrogen dioxide.
Odds ratio (OR): the odds that an outcome will occur given a particular exposure, compared to the odds of the outcome occurring in the absence of that exposure.
ONS: Office for National Statistics.
OA: Output area. The lowest geographical area at which Census estimates are provided. A minimum of 40 resident households or 100 resident people.
PCM model: pollution climate mapping model. http://uk-air.defra.gov.uk/research/air-quality-modelling?view=modelling
PHE: Abbreviation for Public Health England.
Plausibility interval: A term used by COMEAP to express uncertainty around the concentration-response coefficient for long term exposure to $PM_{2.5}$ and mortality including both sampling uncertainty (as represented by the confidence interval) and wider uncertainties. See COMEAP (2009)
https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/304667/COMEAP_long_term_exposure_to_air_pollution.pdf
$PM_{2.5}$: Mass per cubic metre of particles passing through the inlet of a size selective sampler with a transmission efficiency of 50% at an aerodynamic diameter of 2.5 micrometres
PM_{10}: As for $PM_{2.5}$ but for an aerodynamic diameter of 10 micrometres.
Poisson regression: A form of regression applying to rare events in a given time period (such as deaths per day).
Population 30+ average 2009/2010/2011: The population aged 30 and above for each year 2009, 2010 and 2011 averaged to give a more stable estimate for 2010 (to match the approach for deaths).
Population-weighted annual average anthropogenic $PM_{2.5}$: Population-weighted average concentration can be defined as the average concentration in each OA multiplied by the total

population within each individual OA furthermore summed in each borough and divided by the total population in each individual borough. In the $PM_{2.5}$ case, we used the anthropogenic concentration and only the population over 30 years of age.

Population-weighted annual average NO_2: Same as above but it was decided to use full NO_2 concentrations instead of anthropogenic NO_2. This was based on the 2011 EU report (European Commission, 2011) which says that 'the state of knowledge does not allow a natural part of NO_2 to be measured or quantified'. A counter factual of 20 $\mu g\ m^{-3}$ was used in a sensitivity analysis where only the average concentration of NO_2 above or equal to 20 $\mu g\ m^{-3}$ was used in the calculation but it made no difference in the year 2010 as the average concentration in every OA in all of London exceeded the threshold of 20 $\mu g\ m^{-3}$.

Postneonatal mortality: Deaths occurring in the first year of life excuding the neonatal period (the first month after birth).

Prevalence: the number of cases of a disease or disease state that are present in a particular population at a given time.

PWAC: Population-weighted average concentration.

Regression: A statistical process for examining the relationship between different variables (such as health outcomes and pollutant concentrations).

RR Relative risk: The ratio of the probability of the event occurring in the exposed group compared with the non-exposed group. In the case of air pollution, where exposure is ubiquitous, it is the ratio of the probability of the event occurring in groups with higher and lower exposure (actually the concentrations studied are continuous). The relative risks here are expressed, conceptually, in terms of a 10 $\mu g\ m^{-3}$ greater concentration in the higher compared with the lower exposure group. For the results of long-term exposure (mortality burden) the ratio refers to the ratio of the age-specific death rates (assuming other factors are equal). It is derived from a Cox proportional hazard model that assumes the risks (hazards) change by the same proportion at all ages.

Respiratory: Relating to the lungs.

REVIHAAP: Review of the Health Aspects of Air Pollution, a project co-funded by WHO and the European Commission to provide advice to the European Commission.

RHA: Abbreviation for respiratory hospital admissions used in this report.

Secondary particulates: particles created by chemical reactions in the atmosphere (for e.g. the oxidation of sulphur dioxide into sulphuric acid/ammonium sulphate and and nitrogen oxides into nitric acid/ammonium or sodium nitrate).

Sensitivity analysis: An analysis used to test how sensitive an overall results is to changes in assumptions. Often used to see how much difference the addition of less certain health outcomes would make to the overall result.

SEPHO: South East Public Health Observatory.

TfL: Transport for London.

VOLY Value of a life year: The monetary value of a year of life lost, based on studies that assess the willingness to pay for reducing mortality risks associated with air pollution.

WHO: World Health Organisation.