Committee on the Medical Effects of Air Pollutants

Summary of COMEAP recommendations for the quantification of health effects associated with air pollutants

Executive summary

1. This document presents a collation of COMEAP’s recommendations for quantifying the health effects of air pollutants, as previously published, and it is intended to be helpful to policy makers. As well as recommendations for concentration-response functions for individual pollutant-outcome pairs, general principles when carrying out health impact assessments are also discussed, such as interpreting coefficients for individual pollutants, metrics for particulate matter, considering coefficients from cohort and time-series studies, scale of concentration changes and cut-offs for quantification and scale of concentration changes.

2. The summary document also includes information on the types of uncertainties relevant to the recommendations, which can be used to inform decisions regarding whether to include a pollutant-outcome pair in core health impact assessments or sensitivity analyses. Here, the types of uncertainties relevant to each of the various assessments and recommendations are categorised and explained, namely: limited evidence base, inconsistent association, uncertain underlying causality, uncertain quantification and lack of specificity to pollutant. These uncertainties relate to the development of recommendations for concentration-response functions. Other sources of uncertainty relevant to quantification of effects are not addressed in this document.

3. The document was developed through discussions in the Committee’s Subgroup on the quantification of air pollution risks in the UK (QUARK) and agreed by the full COMEAP Committee.
Introduction

4. This collation of COMEAP’s recommendations for quantifying the health effects of air pollutants is intended to be helpful to policy makers. It also includes information on the types of uncertainties relevant to our recommendations, which can be used to inform decisions regarding whether to include a pollutant-outcome pair in core health impact assessments or sensitivity analyses.

General principles

(a) Interpreting coefficients for individual pollutants

5. Because concentrations of pollutants are often correlated, associations reported in epidemiological studies between pollutants and health outcomes may reflect the effects of a mixture of pollutants. Therefore, a coefficient which has not been adjusted for effects associated with other pollutants (ie a coefficient from a single-pollutant model) likely also reflects, to some extent, effects associated with other correlated pollutants. If effects estimated using a single-pollutant coefficient are added to estimates of the same effect associated with other pollutants, this will likely give an overestimate of the effects of the pollution mix.

6. Application of two- or multi-pollutant models allows coefficients to be adjusted for effects associated with other pollutants to some extent. However, information on the concentrations of some potentially causal pollutants are not routinely available, and so cannot be adjusted for. In addition, there are difficulties in interpreting the results when pollutants are highly correlated.

7. Therefore, the specific assessment should be considered when selecting the coefficients to be used. Some examples are given below:

Burden estimates attributable to current levels of pollutants

8. Estimates of the mortality or health burden attributable to current levels of air pollution can be useful for communicating the scale of the effect on public health. Given this, and in view of the uncertainties in ascribing the extent of effects to individual pollutants, we have recommended (COMEAP, 2018) that it is preferable to estimate the burden attributable to the pollution mixture as a whole, rather than trying to apportion the burden to individual pollutants. For example:

- Mortality burden due to long-term exposure to air pollution: We undertook calculations using unadjusted coefficients (from single-pollutant models) for PM$_{2.5}$ or NO$_2$, regarding these as indicators of the pollution mixture. We compared the higher of these two estimates with burdens obtained using an exploratory method. This method involved producing mutually adjusted summary coefficients by applying paired reductions on mutual adjustment, taken from four individual studies, to the summary coefficients from single-pollutant models. The estimated burdens obtained using pairs of mutually adjusted summary coefficients were summed to give an estimated burden of the air pollution mixture. We presented the estimated burden as a range including the higher of the estimates generated using a single-pollutant coefficient and each of the estimates generated using the exploratory method.
Impact assessments of changes in pollutant concentrations

9. Interventions reducing mixtures of pollutants: For some health impact assessments, it may be possible to use changes in either PM or NO2 concentrations as an indicator for changes in all components of a pollutant mixture arising from a particular source, such as traffic. An example might be an intervention that reduces vehicle numbers, or pedestrianisation. In this case, we recommended (COMEAP, 2018):

- Health benefits of interventions which reduce all traffic-related emissions: that impact assessments based on either the unadjusted NO2 coefficient or unadjusted PM2.5 coefficient can be undertaken, and the higher of these used as an estimate of the impact of the intervention.

10. Nonetheless, we noted that both of these methods are likely to underestimate the total benefits of the reduction to some extent.

11. Interventions targeting specific components of the air pollution mix: In other cases, health impact assessments of the benefits expected from reductions in individual pollutants may be needed to support policy development. This would be the case for traffic interventions targeted specifically at NOx emissions. In this case, we recommended (COMEAP, 2018):

- Health benefits of interventions targeted primarily at NOx emissions: use a coefficient for NO2 which has been reduced to take into account both an adjustment for effects of PM (using information from two-pollutant models) and also an assessment of the likely extent to which this adjusted coefficient represented of NO2 itself. We derived this reduced coefficient using expert judgement. [Note: COMEAP will continue to consider the developments in the evidence regarding use of multi-pollutant model approaches.]

12. Overall, there is a larger evidence base reporting effects expressed as being associated with PM (particularly PM2.5) than with NO2, and more mechanistic evidence supporting a causal relationship for many health endpoints (including mortality) with PM than with NO2. However, unadjusted associations with NO2 may reflect the effects of traffic-related pollutants arising from local traffic sources better than associations reported with PM2.5 concentrations do. This is because PM2.5 concentrations are largely dominated by regional, rather than local, sources.

13. We have indicated that benefits will be over-estimated if the results of estimates of impacts predicted on the basis of reductions of both PM2.5 and NO2 are added together, if one of the estimates is based on a single-pollutant coefficient. We have therefore suggested that this approach is not taken, to avoid overestimation of the effects of the combined reductions. However, we note that, when producing its revised guidance for economic analysis of impacts of changes to air quality, Defra (2019) has chosen to risk over-estimation of benefits associated with interventions, rather than risk under-estimating them. The current guidance therefore includes assessments based on coefficients for both PM2.5 (using an unadjusted coefficient) and NO2 (using a coefficient reduced by adjustment for PM2.5...
and also to reflect the likely extent for which the adjusted coefficient is causal). Defra (2019) recommends that the possibility of over-estimation is a limitation which should be clearly acknowledged in assessments, and the potential extent of the over-estimation on results examined through sensitivity analysis.

(b) **Metrics for particulate matter**
14. When making recommendations for quantification of effects using associations reported with a metric of particulate matter (PM$_{2.5}$ or PM$_{10}$) we have regarded these are indicating effects of particulate matter pollution more generally. Therefore, **coefficients for the same health effect associated with PM$_{2.5}$ and PM$_{10}$ should not be used together in the same assessment.** It should be noted that PM$_{2.5}$ is part of PM$_{10}$, and thus the exposure-response coefficients that have been derived by analysing PM$_{10}$ should be applicable for finer particles too and those coefficients from analysing PM$_{2.5}$ may have also included coarser particles.

(c) **Considering coefficients from cohort and time-series studies**
15. Two sorts of studies have been most used as the basis for recommendations for quantification of effects of air pollutants. Cohort studies exploit spatial variations in long-term average concentrations of pollutants. Their design means that they can detect effects such as the increased risk of induction of new disease, or of mortality. In contrast, time-series studies examine how routine medical statistics respond to day-to-day variations in pollutant concentrations$^1$. The extent to which the effects observed in time-series studies represent additional effects, or the bringing forward of effects, is not clear. Similarly, the extent to which associations reported in cohort studies may include the effects observed by time-series studies is not clear. Therefore, when applying the recommended coefficients, it is generally advised that associations for short- and long-term exposure for the same pollutant are not combined for the same health endpoint.

(d) **Cut-offs for quantification, and scale of concentration changes**
16. We have more confidence in the results of calculations of health impact when they are applied to small changes in pollutant concentrations. This is often the case when assessing the benefits of individual policy options. Large changes in pollutant concentrations in response to implementation of policies would likely also change the correlations between pollutants, introducing some uncertainties. Nonetheless, there is little in the epidemiological evidence to suggest that there is a threshold concentrations below which effects would not occur.

17. We have greater confidence in the portion of assessments which do not involve extrapolation beyond the range of available evidence. Therefore, for burden estimates, we regard the portion of the estimated burden obtained when cut-offs for quantification are implemented as being the portion in which these is greatest confidence. Extrapolating to zero (or zero anthropogenic) also includes the portion of the burden for which quantification is more uncertain.

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$^1$ Cross-sectional studies have been used in some cases.
Defra/IGCB recommended methods for Health Impact Assessments

18. We are aware that the guidance published by Defra (2019) (with the endorsement of the Interdepartmental Group on Costs and Benefits (IGCB) Air) draws on other recommendations for quantifying benefits associated with reductions in air pollution, as well as our own. The Defra guidance includes additional morbidity endpoints in the low, central and high scenarios. The additional pollutant-outcome pairs included are those used in a tool developed by UK Health Forum and Imperial College, in collaboration with Public Health England, to estimate costs to the NHS and social care due to the health impacts of air pollution (PHE, 2018). Effects on the economy, via air pollution affecting productivity, are also included. The approaches used are those developed by Ricardo-AEA (2014) for Defra.

19. The guidance also includes assessments based on coefficients for both PM$_{2.5}$ (using an unadjusted coefficient) and NO$_2$ (using a coefficient reduced by adjustment for PM$_{2.5}$ and also to reflect the likely extent for which the adjusted coefficient is causal). This means that it risks over-estimation of benefits associated with interventions. Defra recommends that the possibility of over-estimation is a limitation which should be clearly acknowledged in assessments, and the potential extent of the over-estimation on results examined through sensitivity analysis.

Types of uncertainties

20. In the summary table, the uncertainties relevant to our various assessments and recommendations are categorised into five groups, reflecting:

a) **limited evidence base**: limited evidence volume/size, old studies, or limited geographical coverage of the studies;

b) **inconsistent association**: inconsistency as to whether an association is found (mixture of positive and negative associations), inconsistency in size of the RR (weak or strong positive associations), inconsistency in statistical significance of associations (eg marginally significant, non-statistically significant);

c) **uncertain underlying causality**: other strands of evidence, such as animal or chamber studies, other epidemiological study designs, do not confirm causality, likelihood of major confounding from correlated pollutants;

d) **uncertain quantification**: available evidence base, availability of baseline rates, confirmed clinical significance, uncertain confidence intervals, likelihood of minor confounding from correlated pollutants but the influence of confounding is not clear; and

e) **lack of specificity to pollutant**: the effect is associated with many pollutants consistently or there is consistently an effect but it is associated with different pollutants in different studies.

21. The uncertainties listed are concerned with the development of recommendations for concentration-response functions. We are aware of other sources of uncertainty, such as:
- exposure ascertainment: there are uncertainties in the exposure estimation methods used eg related to the model used for estimating pollutant concentrations and spatial resolution of the estimates or use of concentration measurements from a local monitor, as well as uncertainties in use of pollutant concentrations as a proxy for exposure, eg use of concentrations at place of residence or consideration of time-activity patterns;
- variation in timeframes of when the exposure to air pollution is measured and the health outcomes are recorded;
- differences in quantifications in terms of population projections and background rates.

22. Uncertainties regarding these issues or other inputs required for quantification of health effects/benefits are not addressed in this document.
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23. The pollutant-outcome pairs included in the table are listed below, using the following approach:

- By pollutant in this order: PM$_{2.5}$, PM$_{10}$, NO$_2$, O$_3$, SO$_2$
- Within the section on each pollutant, associations with long-term average concentrations are included first, followed by associations with short-term variations in concentration
- Within the sections on long- and short-term concentrations, coefficients reflecting mortality risk are included first, and then those for morbidity endpoints

PM$_{2.5}$, long-term – all-cause mortality (PM$_{mortality}$)
PM$_{10}$, long-term – chronic bronchitis symptoms (bronchitis)
PM$_{10}$, short-term – all-cause mortality (AP$_{health}$)
PM$_{10}$, short-term – cardiovascular hospital admissions (PM$_{CVMorbidity}$)
NO$_2$, long-term – all-cause mortality (NO$_2$$_{mortality}$)
NO$_2$, long-term – respiratory morbidity in children (NO$_2$$_{resp_morbidity}$)
NO$_2$, short-term – respiratory hospital admissions (AP$_{health}$)
PM and NO$_2$, long-term – cardiovascular morbidity (to be published)
PM and NO$_2$, long-term – asthma (asthma)
O$_3$, long-term – all-cause mortality (Ozone)
O$_3$, short-term – all-cause mortality (Ozone)
SO$_2$, short-term – respiratory and cardiovascular hospital admissions (Ozone)
SO$_2$, short-term – all-cause mortality (AP$_{health}$)
Air pollution, short-term – Restricted activity days (RADs) and work days lost (workdays_lost)

[Note: COMEAP intends to review recommendations for some of the presented coefficients as part of the advice provided to Defra for the development of targets under the Environment Bill. Initial discussions are planned for November 2020.]
Table 1. COMEAP recommendations on quantifying health effects associated with air pollutants.

<table>
<thead>
<tr>
<th>Pollutant exposure (long- or short-term)</th>
<th>Endpoint</th>
<th>Type of study – Coefficients</th>
<th>COMEAP comments &amp; reference</th>
<th>Evidence base and uncertainties</th>
<th>Potential applications</th>
<th>Some recent studies reported since COMEAP recommendation</th>
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| PM$_{2.5}$, long-term                   | All-cause mortality | Systematic review and a meta-analytical summary estimate published. Coefficient unadjusted for other pollutants (Hoek et al, 2013) – RR 1.06 (95% CI: 1.04, 1.08) per 10 μg/m$^3$ annual average PM$_{2.5}$ | This coefficient is not adjusted for effects of other pollutants, which means that:  
  - Mortality estimates will likely include effects caused by other correlated pollutants (eg NO$_2$) to some extent;  
  - If mortality effects estimated using this coefficient are added to estimates of mortality effects associated with other pollutants, this will likely give an overestimate of the effects of the pollution mix  
  Statement on quantifying mortality associated with long-term average concentrations of fine particulate matter (PM$_{2.5}$), 2018 (PM$_{2.5}$, mortality) | There are many cohort studies and meta-analyses and there is good mechanistic evidence for a causal role of PM$_{2.5}$ in shortening life. Nonetheless, because of the close correlations between pollutants, it is likely that the recommended coefficient reflects the effect of PM$_{2.5}$ and also, to some extent, of other pollutants such as other fractions of PM, NO$_2$ and other components of the air pollution mixture (uncertain quantification associated with other pollutants). | Mortality burden of particulate air pollution (acknowledging that this may be an overestimate, and may include effects of other correlated pollutants to some extent)  
  Mortality burden of an air pollution mixture for which PM$_{2.5}$ is an indicator (acknowledging that this may be an underestimate)$^2$  
  Benefits of reductions in particulate air pollution (possible overestimate) or an air pollution mixture for which PM$_{2.5}$ is an indicator (possible underestimate) | U.S. EPA, ISA (2018)  
 ESCAPE Beelen et al. (2014)  
 Pope et al. (2020) meta-analysis  
 Chen and Hoek (2020) meta-analysis |

$^2$ COMEAP (2018) also proposed exploratory methods to calculate the burden attributable to the air pollution mixture, based on mutual adjustment of single-pollutant coefficients for PM$_{2.5}$ and NO$_2$. See COMEAP (2018) for details.
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<th>Pollutant, Exposure (long- or short-term)</th>
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<td>PM10, long-term</td>
<td>Chronic bronchitis symptoms – Prevalence (Health endpoints considered: cough and phlegm on most days during at least three consecutive months for more than two years)</td>
<td>Cross-sectional and longitudinal studies. Coefficient unadjusted for other pollutants – OR 1.32 (95% CI 1.02, 1.71) per 10 µg/m³ increase</td>
<td>The evidence considered does not sufficiently establish causality to justify inclusion of this outcome in core HIA regarding long-term exposure to air pollution. We recommend instead that only sensitivity calculations are undertaken. These may be used to define a range of estimates of the size of the possible effect of long-term exposure to ambient air pollutants on chronic bronchitis in the UK, on the assumption that the relationship is a causal one. If the relationship is not causal, the best estimate is of no effect.</td>
<td>Large number of studies available but inconsistent association and lack of specificity to pollutant.</td>
<td>Burden estimates and impact assessments (number of cases) of particulate pollution or a mixture for which PM10 is an indicator</td>
<td>U.S. EPA, ISA (2018) Doiron et al. (2019)</td>
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<td>PM$_{10}$, short-term</td>
<td>All-cause mortality</td>
<td>Time-series studies. Coefficient unadjusted for other pollutants – 0.75% per 10 µg/m$^3$ increase in 24h mean</td>
<td>Co-variation of pollutants means that in some instances we do not know which individual pollutant or mixtures of pollutants has caused the recorded effects or whether some additive or synergistic effects have taken place. A reduction in the concentration of a single pollutant may produce different benefits than predicted by exposure-response relationships based on observational studies. Quantification of the effects of air pollution on health in the United Kingdom, 1998 (AP_health)</td>
<td>There was a decent evidence base but COMEAP has agreed that this need updating. A number of meta-analyses are now available in grey and peer-reviewed literature and WHO have also funded updated meta-analyses. The uncertainties are related to lack of specificity to pollutant. COMEAP/2017/MIN/2, para 81-102. <a href="https://app.box.com/s/qv2xjsp6g6fzp1zhm72igzjwrt7uf7/file/379307255019">link</a> COMEAP/2017/MIN/3, para 80-82. <a href="https://app.box.com/s/qv2xjsp6g6fzp1zhm72igzjwrt7uf7/file/371330762273">link</a></td>
<td>Number of deaths associated with days of higher air pollution Not to be included in assessments which also include mortality associated with long-term average concentrations of PM$_{2.5}$ Likely to reflect effects on patients who already have severe, pre-existing disease</td>
<td>PM$<em>{2.5}$ – WHO HRAPIE (2013) U.S. EPA. ISA (2018) PM$</em>{2.5}$ – Atkinson et al. (2014) Orellano et al. (2020)</td>
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<td>PM$_{10}$, short-term</td>
<td>Cardiovascular hospital admissions, all ages</td>
<td>Meta-analysis. Coefficient unadjusted for other pollutants – 0.8% (95% CI 0.6%, 0.9%) per 10 µg/m$^3$ increase in 24h mean</td>
<td>This may misrepresent the overall benefits of pollution control because part of the observed association with PM$<em>{10}$ may be due to correlations with other pollutants, or other confounding factors. COMEAP statement on short-term associations between ambient particles and admissions to hospital for cardiovascular disorders, 2001 (PM$</em>{CVmorbidity}$)</td>
<td>There was a decent evidence base but COMEAP has agreed that this need updating. COMEAP/2017/MIN/2, para 81-102.</td>
<td>Number of CV hospital admissions, associated with days of higher air pollution. Burden estimates and impact assessments. Not to be included in assessments which also include hospital admissions arising from CV morbidity associated with long-term average concentrations of PM$_{2.5}$. Likely to reflect effects on patients who already have severe, pre-existing disease</td>
<td>WHO HRAPIE (2013) – PM$<em>{2.5}$ Hospitalization for respiratory and cardiac diseases in APHEKOM (2004-2006), APHEIS 3 Medina et al. (2004), WHO EBD (2004) U.S. EPA. ISA (2018) PM$</em>{2.5}$ – Atkinson et al. (2014)</td>
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<td>PM(_{10}), short-term</td>
<td>Respiratory hospital admissions</td>
<td>Time-series studies. Coefficient unadjusted for other pollutants – 0.8% per 10 (\mu g/m^3) increase in 24h mean</td>
<td>Co-variation of pollutants means that in some instances we do not know which individual pollutant or mixtures of pollutants has caused the recorded effects or whether some additive or synergistic effects have taken place. A reduction in the concentration of a single pollutant may produce different benefits than predicted by exposure-response relationships based on observational studies. Quantification of the effects of air pollution on health in the United Kingdom, 1998 (AP(_{\text{health}}))</td>
<td>There was a decent evidence base but COMEAP has agreed that this need updating. A number of meta-analyses are now available in grey and peer-reviewed literature and WHO have also funded updated meta-analyses. The uncertainties are related to lack of specificity to pollutant. COMEAP/2017/MIN/2, para 81-102. [link](<a href="https://app.box.com/s/gv2xjsp6g6f">https://app.box.com/s/gv2xjsp6g6f</a> fp1zhm72igzjlwtrt7uf7/file/379307255019) COMEAP/2017/MIN/3, para 80-82. [link](<a href="https://app.box.com/s/gv2xjsp6g6f">https://app.box.com/s/gv2xjsp6g6f</a> fp1zhm72igzjlwtrt7uf7/file/371330762273)</td>
<td>Number of respiratory hospital admissions associated with days of higher air pollution Not to be included in assessments which also include hospital admissions arising from respiratory morbidity associated with long-term average concentrations of PM(_{2.5}). Likely to reflect effects on patients who already have severe, pre-existing disease</td>
<td>Hospitalization for respiratory and cardiac diseases in APHEKOM (2004-2006), APHEIS 3 Medina et al. (2004), WHO EBD (2004) PM(_{2.5}) – Atkinson et al. (2014) DH funded meta-analysis</td>
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<td>NO₂, long-term</td>
<td>All-cause mortality</td>
<td>i. Meta-analysis – Effects attributable to NO₂ and corresponding reductions in other traffic-related pollutants. Coefficient unadjusted for other pollutants – RR 1.023 (95% CI: 1.008, 1.037) per 10 µg/m³ annual average</td>
<td>Recommendations for various purposes were provided, ie a) assessment of the health benefits of interventions that primarily target emissions of oxides of nitrogen (NOx), b) assessment of the health benefits of interventions that reduce traffic-related pollutants, c) assessment of the mortality burden of air pollution in the UK based on long-term average concentrations of NO₂ and PM₂.₅. The uncertainty is greater in recommendation for quantifying effects of NO₂ itself than for NO₂ as an indicator of traffic air pollution. Three Committee Members didn’t agree to the recommendations a) and c). The areas which caused disagreement were those relating to: 1) the causality of NO₂ associations with mortality; 2) the interpretation of results from multi-pollutant models in cohort studies; and 3) the calculation of mortality burden.</td>
<td>Several cohort studies and meta-analyses for single-pollutant coefficient are available but there are few studies with two-pollutant results. There are some issues in regard to interpreting two-pollutant model results (uncertain quantification associated with the pollutant itself) and the mechanistic evidence of NO₂ being causal is limited with respect to long-term exposure and all-cause mortality (uncertain underlying causality).</td>
<td>i. Unadjusted coefficient: - Health benefits of interventions that reduce all traffic-related pollutants - Mortality burden of an air pollution mixture for which NO₂ is an indicator (acknowledging that this may be an underestimate)³</td>
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<td>ii. Meta-analytical estimate reduced by using expert judgement – Effects attributable to NO₂ alone – RR 1.006 to 1.013 per 10 µg/m³ annual average (not possible to derive CIs)</td>
<td>Associations of long-term average concentrations of nitrogen dioxide with mortality, 2018 (NO₂_mortality)</td>
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³ Several studies have estimated the health benefits of interventions that reduce NO₂ as an indicator of traffic-related air pollution. However, the uncertainty associated with this approach is high, and the results should be interpreted with caution.
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<td>NO2, long-term</td>
<td>Respiratory morbidity in children [Health endpoints considered: respiratory symptoms (including bronchitic symptoms in asthmatic children), indices of lung function, asthma]</td>
<td>Recommendation against quantification</td>
<td>Members agreed that the available studies were unable to provide estimates of the size of a direct effect of NO2, ie disentangled from the effects of other pollutants in the mixture. An effect of NO2 was unlikely to be dominant amongst those of the pollutants in the mixture. In addition, the epidemiological studies were unlikely to provide other than weak evidence for a direct effect of NO2 on health due to the fact that none of the epidemiological studies reviewed by the Secretariat was able to disentangle the possible adverse effects of NO2 from those of the other pollutants in the urban mixture which includes particulate matter (PM). Furthermore, a lack of control for ultra-fine particles, an important component of the traffic-related pollution mixture, could mean that some of the reported effects of NO2 could be confounded by exposure to ultra-fine particles. Although it is possible that NO2 might play some small part in respiratory effects in children, it is difficult, on the basis of the epidemiological studies considered, to find a numerical expression of these possible effects.</td>
<td>Lack of specificity to pollutant, uncertain quantification</td>
<td>Bronchitic symptoms in asthmatic children – WHO HRAPIE (2013)</td>
<td>Lung function – Gehring et al. (2013)</td>
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<td>Mölter et al. (2015)</td>
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3 COMEAP (2018) also proposed exploratory methods to calculate the burden attributable to the air pollution mixture, based on mutual adjustment of single-pollutant coefficients for PM_{2.5} and NO2. See COMEAP (2018) for details.
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<td>NO₂, short-term</td>
<td>Respiratory hospital admissions</td>
<td>Time-series studies. Coefficient unadjusted for other pollutants – 2.5% per 50 µg/m³ increase</td>
<td>Co-variation of pollutants means that in some instances we do not know which individual pollutant or mixtures of pollutants has caused the recorded effects or whether some additive or synergistic effects have taken place. A reduction in the concentration of a single pollutant may produce different benefits than predicted by exposure-response relationships based on observational studies. Quantification of the effects of air pollution on health in the United Kingdom, 1998 (AP_health)</td>
<td>There was a decent evidence base but COMEAP has agreed that this need updating. A number of meta-analyses are now available in grey and peer-reviewed literature and WHO have also funded updated meta-analyses. The uncertainties are related to lack of specificity to pollutant. COMEAP/2017/MIN/2, para 81-102. <a href="https://app.box.com/s/qv2xjisp6g6fjp1zhm72igzjlwrt7u7/file/379307255019">https://app.box.com/s/qv2xjisp6g6fjp1zhm72igzjlwrt7u7/file/379307255019</a> COMEAP/2017/MIN/3, para 80-82. <a href="https://app.box.com/s/qv2xjisp6g6fjp1zhm72igzjlwrt7u7/file/371330762273">https://app.box.com/s/qv2xjisp6g6fjp1zhm72igzjlwrt7u7/file/371330762273</a></td>
<td>Number of hospital admissions associated with days of higher air pollution Not to be included in assessments which also include hospital admissions arising from respiratory morbidity associated with long-term average concentrations of NO₂. Likely to reflect effects in patients who already have severe, pre-existing disease</td>
<td>WHO HRAPIE (2013) Mills et al. (2015, 2016) DH funded meta-analysis U.S. EPA. ISA (2016)</td>
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<tr>
<td>PM₂.₅, PM₁₀, and NO₂, long-term</td>
<td>Cardiovascular morbidity</td>
<td>To be published</td>
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<th>Type of study – Coefficients [a]</th>
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<th>Potential applications</th>
<th>Some recent studies reported since COMEAP recommendation [b]</th>
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<tr>
<td>PM and NO₂, long-term</td>
<td>Asthma</td>
<td>No recommendation for quantification developed</td>
<td>i. Evidence from studies comparing communities (ie at a city or administrative area level) suggests that the induction of asthma does not appear to be associated, at a population level, with levels of air pollutants. ii. Evidence from studies on traffic-related air pollution suggests that it is possible that air pollution plays a part in the induction of asthma in some individuals who live near busy roads, particularly roads carrying high numbers of heavy goods vehicles. iii. Our examination of the mechanistic evidence bearing on the possible interaction between exposure to air pollutants and the induction of asthma leads us to think that a causal explanation for conclusion ii. above is plausible. iv. The contribution of exposure to air pollutants to the induction of asthma in those in whom it plays a part is likely to be small in comparison with those from other contributory factors. The proportion of the population so affected is also likely to be small. Does Outdoor Air Pollution Cause Asthma?, 2010 (asthma)</td>
<td></td>
<td>NO₂ and asthma (children) – CAPTOR tool (2016) PM₂.₅, NO₂ and asthma (children), NO₂ and asthma (adults) – PHE NHS and Social Care tool (2018) APHEKOM (2004-2006) WHO HRAPIE (2013) ESCAPE Gehring et al. (2015) - children ESCAPE Jacquemin et al. (2015) - adults ESCAPE Mölter et al. (2015) – prevalence in children U.S. EPA. ISA (2016) U.S. EPA. ISA (2018)</td>
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<tr>
<td>O₃, long-term</td>
<td>Mortality</td>
<td>Recommendat</td>
<td>The evidence from all-year</td>
<td>Limited evidence base</td>
<td>Health effects of day-to-day variations in ambient O₃ concentrations</td>
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<td></td>
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<td>ion against</td>
<td>associations between long-term exposure to ozone and mortality is not convincing. There is limited evidence for an association between ozone concentrations during the warmer months of the year. Quantification of Mortality and Hospital Admissions Associated with Ground-level Ozone, 2015 (Ozone)</td>
<td></td>
<td>Likely to reflect effects in patients who already have severe, pre-existing disease</td>
<td></td>
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<td></td>
<td></td>
<td>quantification</td>
<td></td>
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<tr>
<td>O₃, short-term</td>
<td>All-cause mortality, all ages</td>
<td>Systematic review and meta-analysis of time-series studies. Coefficient unadjusted for other pollutants – 0.34% (0.12, 0.56%) per 10 µg/m³ increase in daily maximum 8-hour running mean O₃</td>
<td>These recommendations are for the purpose of planned health impact assessment for current and future scenarios that do not cover other pollutants. This was in the context of knowing that the Climate Change Risk Assessment was only assessing ozone and not other pollutants. Therefore, use of single pollutant models for recommendations was appropriate. It is likely that correlations with other pollutants may continue to be similar in the future. Quantification of Mortality and Hospital Admissions Associated with Ground-level Ozone, 2015 (Ozone)</td>
<td>There is a substantial number of studies, but there is less evaluation of two-pollutant models. There is good mechanistic evidence for respiratory and limited for cardiovascular effects, but there is consistency across the studies (uncertain quantification associated with the pollutant itself). Temporal correlations may be negative and vary with season.</td>
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Some recent studies reported since COMEAP recommendation:

- HEI Frampton et al. (2017)
- Orellano et al. (2020)
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| O₃, short-term                         | Respiratory and cardiovascular hospital admissions, all ages | Systematic review and meta-analysis of time-series studies. Coefficient unadjusted for other pollutants –  
  *Respiratory:* 0.75% (0.30, 1.20%)  
  *Cardiovascular:* 0.11% (–0.06, 0.27%)  
  per 10 µg/m³ increase in daily maximum 8-hour running mean O₃ | These recommendations are for the purpose of planned health impact assessment for current and future scenarios that do not cover other pollutants. This was in the context of knowing that the Climate Change Risk Assessment was only assessing ozone and not other pollutants. Therefore, use of single pollutant models for recommendations was appropriate. It is likely that correlations with other pollutants may continue to be similar in the future.  
  *Cardiovascular:* There is large evidence base, but the size of the association is small and marginally not statistically significant (inconsistent association). Quantification is supported by statistically significant associations with cardiovascular mortality.  
  *Respiratory:* Good evidence base | Health impacts of day-to-day variations in ambient O₃ concentrations  
  Likely to reflect effects in patients who already have severe, pre-existing disease |
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<tr>
<td>SO₂, short-term</td>
<td>All-cause mortality</td>
<td>Time-series studies. Coefficient unadjusted for other pollutants – 0.6% per 10 µg/m³ increase in 24h mean</td>
<td>Co-variation of pollutants means that in some instances we do not know which individual pollutant or mixtures of pollutants has caused the recorded effects or whether some additive or synergistic effects have taken place. A reduction in the concentration of a single pollutant may produce different benefits than predicted by exposure-response relationships based on observational studies.</td>
<td>There was a decent evidence base but COMEAP has agreed that this needs updating. A number of meta-analyses are now available in grey and peer-reviewed literature and WHO have also funded updated meta-analyses. The uncertainties are related to <strong>lack of specificity to pollutant</strong>.</td>
<td>Number of deaths associated with days of higher air pollution</td>
<td>Likely to reflect effects in patients who already have severe, pre-existing disease</td>
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Quantification of the effects of air pollution on health in the United Kingdom, 1998 ([AP_health](https://app.box.com/s/gv2xjsp6g6ffp1zhm72igzljwtrt7uf7/file/379307255019)).

COMEAP/2017/MIN/2, para 81-102.

COMEAP/2017/MIN/3, para 80-82.

https://app.box.com/s/gv2xjsp6g6ffp1zhm72igzljwtrt7uf7/file/371330762273 |
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<tr>
<td>SO₂, short-term</td>
<td>Respiratory hospital admissions</td>
<td>Time-series studies. Coefficient unadjusted for other pollutants – 0.5% per 10 (\mu g/m^3) increase in 24h mean</td>
<td>Co-variation of pollutants means that in some instances we do not know which individual pollutant or mixtures of pollutants has caused the recorded effects or whether some additive or synergistic effects have taken place. A reduction in the concentration of a single pollutant may produce different benefits than predicted by exposure-response relationships based on observational studies.</td>
<td>There was a decent evidence base but COMEAP has agreed that this need updating. A number of meta-analyses are now available in grey and peer-reviewed literature and WHO have also funded updated meta-analyses. The uncertainties are related to lack of specificity to pollutant.</td>
<td>Number of hospital admissions associated with days of higher air pollution</td>
<td>Health Canada AQBAT (2012)</td>
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Quantification of the effects of air pollution on health in the United Kingdom, 1998 (AP_health)

COMEAP/2017/MIN/2, para 81-102. [Link](https://app.box.com/s/gv2xjsp6g6f fp1zhm72igzjlwr7utf7/file/379307 255019)
COMEAP/2017/MIN/3, para 80-82. [Link](https://app.box.com/s/gv2xjsp6g6f fp1zhm72igzjlwr7utf7/file/371330 762273)
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<tbody>
<tr>
<td>Air pollution, short-term</td>
<td>Restricted activity days (RADs) and work days lost</td>
<td>No recommended quantification method developed</td>
<td>Members agreed that it was likely that elevated levels of air pollution affected the health of some people in a way that impacted on their ability to undertake their normal daily activities. This might include attendance at work. An approach to quantification of restricted activity days and days of work lost by inferring from other, more studied, endpoints could be developed. This would involve constructing a “health triangle” or “health pyramid” using data on health endpoints of varying severity (e.g., the number of hospital admissions, attendances at A&amp;E departments and primary care attendances for lower respiratory tract infections, together with data on sickness absence) to understand the quantitative relationship between them. The likely burden of air pollution from each of these could be estimated by calibration against an effect for which methods for quantification have already been developed (for example, hospital admissions).</td>
<td></td>
<td></td>
<td>PM$<em>{2.5}$ and RADs, PM$</em>{2.5}$ and work days lost, O$_3$ and minor RADs (mRADs) – WHO HRAPIE (2013) PM and mRADs – US EPA BenMAP (2017) Summer O$<em>3$ and mRADs, PM$</em>{2.5}$ and RADs – Health Canada AQBAT (2012) Ricardo AEA (2014)</td>
</tr>
</tbody>
</table>

[a] Short-term: studies of temporal variation of exposure, long-term: studies of spatial variation of exposure.

[b] Coefficients derived by long-term exposure studies are expressed as RR or OR, while those derived by short-term exposure studies as percentage change.

[c] The Committee has not reviewed the information listed here.
References


COMEAP (2018) Associations of long-term average concentrations of nitrogen dioxide with mortality (NO2_mortality)


Orellano P, Reynoso J, Quaranta N, Bardach A, Ciapponi A (2020) Short-term exposure to particulate matter (PM10 and PM2.5), nitrogen dioxide (NO2), and ozone (O3) and all-cause and cause-specific mortality: Systematic review and meta-analysis, Environmental Research 142: 105876.


Abbreviations

PM$_{2.5}$, PM$_{10}$: suspended particles with diameter not greater than 2.5, 10 µm respectively
PM: suspended particles
NO$_2$: nitrogen dioxide
O$_3$: ozone
SO$_2$: sulphur dioxide
CV: cardiovascular
RR: relative risk
OR: odds ratio
COMEAP Sub-group on the quantification of air pollution risks in the UK
(QUARK)

Chair Dr Heather Walton (Imperial College London)
Members Professor Paul Wilkinson (London School of Hygiene and Tropical Medicine)
Professor Gavin Shaddick (University of Exeter)
Professor Debbie Jarvis (Imperial College London)
Professor Anna Hansell (University of Leicester)
Mr John Stedman (Ricardo Energy and Environment)
Dr Mike Holland (EMRC and Imperial College, London)
Professor Duncan Lee (University of Glasgow)

Secretariat Dr Christina Mitsakou (Public Health England)
Ms Alison Gowers (Public Health England)

COMEAP Chair: Professor Frank Kelly (Imperial College London)