COMMITTEE ON THE MEDICAL EFFECTS OF AIR POLLUTANTS

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

Contents

Executive summary

This document presents a collation of Committee on the Medical Effects of Air Pollutants' (COMEAP's) recommendations for quantifying the health effects of air pollutants 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.

This 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.

The document was originally 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. This updated document includes the latest recommendations made by COMEAP (up to September 2022).

Introduction

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

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 (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.

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 is not routinely available, and so cannot be adjusted for. In addition, there are difficulties in interpreting the results when pollutants are highly correlated.

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

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 2 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 4 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

Interventions reducing mixtures of pollutants: For some health impact assessments, it may be possible to use changes in either PM or NO_2 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 NO_2 coefficient or unadjusted $PM_{2.5}$ coefficient can be undertaken, and the higher of these used as an estimate of the impact of the intervention.

Nonetheless, we noted that either of these methods is likely to underestimate the total benefits of the reduction to some extent.

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 NO_x emissions. In this case, we recommended (COMEAP, 2018):

Health benefits of interventions targeted primarily at NO_x emissions: use a coefficient for NO_2 which has been reduced to take into account both an adjustment for associations with PM (using information from two-pollutant models) and also an assessment of the likely extent to which this adjusted coefficient represented NO_2 itself. We derived this reduced coefficient using expert judgement. [Note: COMEAP continues to consider the developments in the evidence regarding use of multi-pollutant model approaches.]

Overall, there is a larger evidence base reporting effects expressed as being associated with PM (particularly $PM_{2.5}$) than with NO_2, and more mechanistic evidence supporting a causal relationship for many health endpoints (including mortality) with PM than with NO_2. However, unadjusted associations with NO_2 may reflect the effects of traffic-related pollutants arising from local traffic sources better than associations reported with $PM_{2.5}$ concentrations do. This is because $PM_{2.5}$ concentrations are largely dominated by regional, rather than local, sources.

We have indicated that benefits will be over-estimated if the results of estimates of impacts predicted on the basis of reductions of both $PM_{2.5}$ and NO_2 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 (2020) 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 $PM_{2.5}$ (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). Defra (2020) 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

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 as 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 include the effect of fine 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

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, rather than 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 effect.

[1] Cross-sectional studies have been used in some cases.

(d) Cut-offs for quantification, and scale of concentration changes

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 concentration below which effects would not occur.

When quantifying the mortality burden attributable to particulate air pollution, COMEAP had previously chosen to undertake calculations using both a cut-off for quantification representing the lower end of the studied range and also by extrapolating to zero anthropogenic pollution (COMEAP, 2018). In COMEAP (2022a), the use of the anthropogenic fraction of $PM_{2.5}$ is no longer recommended; it is recommended to extrapolate down to very low or even zero $PM_{2.5}$ concentrations by assuming continuing linearity[2]. Thus, a cut-off value for quantification for core analysis is not recommended, but if a cut-off were to be selected, the range of concentrations which has been studied needs to be considered. The lowest value reported as a fifth percentile of population exposure from the studies included in the Chen and Hoek meta-analysis (Chen and Hoek, 2020) was 3 $\mu g/m^3$ from Pinault et al (2016) (the study contributed 3.40% of the weight to the meta-analysis). We acknowledge the considerable uncertainties involved in extrapolating above the range of studied concentrations. However, there is less uncertainty when extrapolating below studied concentrations when the concentrations studied have got very low: this can be regarded as interpolation between the studied effects and there being zero effects at zero exposure.

[2] Linearity on the log scale: log-linearity. Cohort studies of mortality typically relate the natural log of the hazard function to the concentration. In practice, for a small hazard ratio (as found in most air pollution studies) and over a small concentration range (as is usually the case in a health impact assessment) there is little difference between a linear and log-linear relationship. This might not be the case when larger concentration differences are being considered.

Defra/IGCB recommended methods for Health Impact Assessments

We are aware that the guidance published by Defra (2020) 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 (PHE), 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.

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

In this document, the uncertainties relevant to our various assessments and recommendations are categorised into 5 groups:

- limited evidence base – limited evidence volume or size, old studies or limited geographical coverage of the studies
- inconsistent association – inconsistency as to whether an association is found (mixture of positive and negative associations), inconsistency in size of the relative risk (RR) (weak or strong positive associations), inconsistency in statistical significance of associations (for example marginally significant, non-statistically significant)
- 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
- uncertain quantification – adequate evidence base but uncertainty in baseline rates or wide confidence intervals or possibility that confounding from correlated pollutants may affect the size of the association
- lack of specificity to pollutant – the effect is consistently associated with air pollution, but the pollutant with which it is associated is not consistent (that is an association is not consistently found with the pollutant of interest)

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, for example uncertainties related to the model used for estimating pollutant concentrations and the 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, such as 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

Uncertainties regarding these issues or other inputs required for quantification of health effects or benefits are not addressed in this document.

Pollutant-outcome pairs included

The pollutant-outcome pairs included in the recommendations 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
- a summary of the recommendations is provided in Table 1 and additional details included in the ‘Further information’ section below it

$PM_{2.5}$, long-term – all-cause mortality (PM2.5_mortality)
$PM_{2.5}$, long-term – ischaemic (coronary) heart disease and cerebrovascular disease (stroke) (HealthEvidenceAdvice)
$PM_{2.5}$, short-term – respiratory and cardiovascular hospital admissions (Hospital admissions)
PM_{10}, long-term – chronic bronchitis symptoms (bronchitis)
PM_{10}, short-term – all-cause mortality (AP_health)
PM, long-term – cognitive decline and dementia (PM_dementia)
NO_2, long-term – all-cause mortality (NO2_mortality)
NO_2, long-term – respiratory morbidity in children (NO2_resp_morbidity)
NO_2, short-term – respiratory and cardiovascular hospital admissions (Hospital admissions)
PM and NO_2, long-term – asthma (asthma)

O_3, long-term – all-cause mortality (Ozone)
O_3, short-term – all-cause mortality (Ozone)
O_3, short-term – respiratory and cardiovascular hospital admissions (Ozone), (Hospital admissions)
SO_2, short-term – all-cause mortality (AP_health)
SO_2, short-term – respiratory hospital admissions (AP_health)
Air pollution, short-term – restricted activity days (RADs) and work days lost (workdays_lost)

Table 1. COMEAP recommendations on quantifying health effects associated with air pollutants

Pollutant, exposure[a] (long- or short-term)	Endpoint	Type of study – coefficients[b]	Further information
1. $PM_{2.5}$, long-term	All-cause mortality	Systematic review and a meta-analytical summary estimate published. Coefficient unadjusted for other pollutants (Chen and Hoek, 2020): RR 1.08 (95% CI: 1.06, 1.09) per 10 µg/m^3 annual average $PM_{2.5}$.	$PM_{2.5}$, long-term: all-cause mortality
2. $PM_{2.5}$, long-term	Ischaemic (coronary) heart disease (IHD) and cerebrovascular disease (CBD or stroke) – incidence	Meta-analytical summary estimates published. Coefficient unadjusted for other pollutants (COMEAP, 2021): – IHD: RR 1.07 (95% CI: 0.99, 1.16) – CBD: RR 1.11 (95% CI: 0.99, 1.25) per 10 µg/m^3 annual average $PM_{2.5}$.	$PM_{2.5}$, long-term: Ischaemic heart disease and cerebrovascular disease
3. $PM_{2.5}$, short-term	Respiratory and cardiovascular hospital admissions, all ages	Systematic review and meta-analysis of time-series studies. Coefficient unadjusted for other pollutants (Atkinson et al, 2014): – respiratory: 0.96% (-0.63, 2.58%) – cardiovascular: 0.90% (0.26, 1.53%) per 10 µg/m^3 increase in 24h mean.	$PM_{2.5}$, short-term

Pollutant, exposure[a] (long- or short-term)	**Endpoint**	**Type of study – coefficients[b]**	**Further information**
4. PM_{10}, long-term	Chronic bronchitis symptoms – prevalence [Health endpoints considered: cough and phlegm on most days during at least 3 consecutive months for more than 2 years]	Cross-sectional and longitudinal studies. Coefficient unadjusted for other pollutants – OR 1.32 (95% CI 1.02, 1.71) per 10 µg/m^3 increase. Recommended for sensitivity analysis only.	PM_{10}, long-term
5. PM_{10}, short-term	All-cause mortality	Time-series studies. Coefficient unadjusted for other pollutants – 0.75% per 10 µg/m^3 increase in 24h mean.	PM_{10}, short-term
6. PM, long-term	Cognitive decline and dementia	No recommendation for quantification developed.	PM, long-term
7. NO_2, long-term	All-cause mortality	i. Meta-analysis – effects attributable to NO_2 and corresponding reductions in other traffic-related pollutant: coefficient unadjusted for other pollutants – RR 1.023 (95% CI: 1.008, 1.037) per 10 µg/m^3 annual average. ii. Meta-analytical estimate reduced by using expert judgement – effects attributable to NO_2 alone – RR 1.006 to 1.013 per 10 µg/m^3 annual average (not possible to derive CIs).	NO_2, long-term: all-cause mortality

Pollutant, exposure[a] (long- or short-term)	**Endpoint**	**Type of study – coefficients[b]**	**Further information**
8. NO_2, long-term	Respiratory morbidity in children [Health endpoints considered: respiratory symptoms (including bronchitic symptoms in asthmatic children), indices of lung function, asthma]	Recommendation against quantification.	NO_2, long-term: respiratory morbidity in children
9. NO_2, short-term	Respiratory and cardiovascular hospital admissions	Systematic review and meta-analysis of time-series studies. Coefficient unadjusted for other pollutants (Mills et al, 2015): – respiratory: 0.57% (0.33, 0.82%) – cardiovascular: 0.66% (0.32, 1.01%) per 10 µg/m^3 increase in 24h mean or – respiratory: 0.34% (-0.02, 0.70%) – cardiovascular: 0.36% (-0.16, 0.89%) per 10 µg/m^3 increase in 1h mean.	NO_2, short-term
10. PM and NO_2, long-term	Asthma	No recommendation for quantification developed.	PM and NO_2, long-term
11. O_3, long-term	Mortality	Recommendation against quantification.	O_3, long-term

Pollutant, exposure[a] (long- or short-term)	Endpoint	Type of study – coefficients[b]	Further information
12. O_3, short-term	All-cause mortality, all ages	Systematic review and meta-analysis of time-series studies. Coefficient unadjusted for other pollutants: 0.34% (0.12, 0.56%) per 10 µg/m^3 increase in daily maximum 8-hour running mean O_3.	O_3, short-term: all-cause mortality
13. O_3, short-term	Respiratory and cardiovascular hospital admissions, all ages	Systematic review and meta-analysis of time-series studies. Coefficient unadjusted for other pollutants (Walton et al, 2014): – respiratory: 0.75% (0.30, 1.20%) – cardiovascular: 0.11% (-0.06, 0.27%) per 10 µg/m^3 increase in daily maximum 8-hour running mean O_3.	O_3, short-term: respiratory and cardiovascular hospital admissions
14. SO_2, short-term	All-cause mortality	Time-series studies. Coefficient unadjusted for other pollutants: 0.6% per 10 µg/m^3 increase in 24h mean.	SO_2, short-term: all-cause mortality
15. SO_2, short-term	Respiratory hospital admissions	Time-series studies. Coefficient unadjusted for other pollutants: 0.5% per 10 µg/m^3 increase in 24h mean.	SO_2, short-term: respiratory hospital admissions
16. Air pollution, short-term	Restricted activity days (RADs) and work days lost	No recommended quantification method developed.	Air pollution, short-term

[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, while those derived by short-term exposure studies as percentage change.
[c] The committee has not reviewed the information listed here.

Further information

1. $PM_{2.5}$, long-term: all-cause mortality

Type of study – coefficients[b]

Systematic review and a meta-analytical summary estimate published. Coefficient unadjusted for other pollutants (Chen and Hoek, 2020) – RR 1.08 (95% CI: 1.06, 1.09) per 10 μg/m^3 annual average $PM_{2.5}$.

COMEAP comments and reference

This coefficient is not adjusted for effects of other pollutants, which means that:

- mortality estimates will likely include effects caused by other correlated pollutants (for instance 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 exposure to $PM_{2.5}$, 2022 (PM2.5_mortality).

Evidence base and uncertainties

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).

Potential applications

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)[3].

Benefits of reductions in particulate air pollution (possible overestimate) or an air pollution mixture for which $PM_{2.5}$ is an indicator (possible underestimate).

[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 NO_2. See COMEAP (2018) for details.

2. $PM_{2.5}$, long-term: Ischaemic (coronary) heart disease (IHD) and cerebrovascular disease (CBD or stroke) – incidence

Type of study – coefficients[b]

Meta-analytical summary estimates published. Coefficient unadjusted for other pollutants (COMEAP, 2021):

- IHD – RR 1.07 (95% CI: 0.99, 1.16)
- CBD – RR 1.11 (95% CI: 0.99, 1.25)

per 10 $\mu g/m^3$ annual average $PM_{2.5}$.

COMEAP comments and reference

Advice on health evidence relevant to setting PM2.5 targets (HealthEvidenceAdvice).

Forthcoming report on air pollution and cardiovascular disease will be published on Committee on the Medical Effects of Air Pollutants in due course.

COMEAP's approach integrated mortality and morbidity and demonstrated complex patterns in prevalence.

Evidence base and uncertainties

We note that the 95% confidence intervals for summary effects estimates linking PM2.5 concentrations with incidence of IHD and CBD marginally fail the usual criteria for statistical significance (inconsistent association).

There are few studies available for case fatality, so the case fatality coefficient (used in assessments which integrate mortality and morbidity) is uncertain.

Potential applications

HIA and/or burden estimates related to incidence of ischaemic (coronary) heart disease (IHD) and incidence of stroke (CBD), ideally integrating with mortality assessments (COMEAP, in preparation).

Integration with mortality assessments is not likely to be practical in routine cost-benefit assessments at this stage.

3. $PM_{2.5}$, short-term: respiratory and cardiovascular hospital admissions, all ages

Type of study – coefficients[b]

Systematic review and meta-analysis of time-series studies. Coefficient unadjusted for other pollutants (Atkinson et al, 2014):

- respiratory – 0.96% (-0.63, 2.58%)
- cardiovascular – 0.90% (0.26, 1.53%)

per 10 μg/m^3 increase in 24h mean.

COMEAP comments and reference

Recent meta-analyses of studies evaluating the associations between (total, all-cause) respiratory and cardiovascular hospital admissions and short-term exposures to $PM_{2.5}$ were examined by the committee.

Statement on update of recommendations for quantifying hospital admissions associated with short-term exposures to air pollutants, 2022 (Hospital admissions).

Note: These recommendations are for quantification of effects associated with PM on the basis of $PM_{2.5}$ concentrations. We previously recommended coefficients from studies on PM_{10} for quantifying hospital admissions associated with PM. These coefficients for PM_{10} (AP_health), (PM_CVmorbidity) should be used only when the coefficients for $PM_{2.5}$ are not able to be used (see paragraph 12 above).

Evidence base and uncertainties

PM is considered to be causally related to the respiratory and cardiovascular effects associated with it in epidemiological studies. Nonetheless, the comparison of effects estimates from single- and two-pollutant models demonstrated considerable attenuation on adjustment for effects associated with NO_2 (uncertain quantification).

Potential applications

Number of hospital admissions associated with days of higher air pollution, for example HIA and/or burden estimates during episodes.

Likely to reflect effects in patients who already have severe, pre-existing disease.

4. PM_{10}, long-term: chronic bronchitis symptoms

Prevalence (health endpoints considered: cough and phlegm on most days during at least 3 consecutive months for more than 2 years).

Type of study – coefficients[b]

Cross-sectional and longitudinal studies.

Coefficient unadjusted for other pollutants – OR 1.32 (95% CI 1.02, 1.71) per 10 μg/m^3 increase.

Recommended for sensitivity analysis only.

COMEAP comments and reference

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.

Long-term Exposure to Air Pollution and Chronic Bronchitis, 2016 (bronchitis).

Evidence base and uncertainties

Large number of studies available but inconsistent association.

Potential applications

Burden estimates and impact assessments (number of cases) of particulate pollution or a mixture for which PM_{10} is an indicator.

Some recent studies reported since COMEAP recommendation[c]

- US Environmental Protection Agency (EPA) Integrated Science Assessment (ISA) (2019; 2022)
- Doiron et al (2019)

5. PM_{10}, short-term: all-cause mortality

Type of study – coefficients[b]

Time-series studies.

Coefficient unadjusted for other pollutants – 0.75% per 10 µg/m^3 increase in 24h mean.

COMEAP comments and reference

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).

Evidence base and uncertainties

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 World Health Organization (WHO) have also funded updated meta-analyses.

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

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

Potential applications

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.

Some recent studies reported since COMEAP recommendation[c]

- $PM_{2.5}$ – World Health Organization (WHO) Health Risks of Air Pollution in Europe (HRAPIE) (2013)
- US EPA ISA (2018)
- $PM_{2.5}$ – Atkinson et al (2014)
- Orellano et al (2020)

6. PM, long-term: cognitive decline and dementia

Type of study – coefficients[b]

No recommendation for quantification developed.

COMEAP comments and reference

The committee regarded the current evidence base as inadequate for direct quantification of the effects of air pollutants on cognitive decline or dementia, partly because the available epidemiological studies are too heterogeneous to be suitable to allow meta-analysis to be used to derive a summary effects estimate. Nonetheless, they regarded the association between air pollutants and effects on cognition as likely to be causal, because of the evidence base indicating effects on the cardiovascular system. (PM_dementia).

7. NO_2, long-term: all-cause mortality

Type of study – coefficients[b]

Meta-analysis:

- effects attributable to NO_2 and corresponding reductions in other traffic-related pollutant
  - coefficient unadjusted for other pollutants – RR 1.023 (95% CI: 1.008, 1.037) per 10 µg/m^3 annual average

Meta-analytical estimate reduced by using expert judgement:

- effects attributable to NO_2 alone – RR 1.006 to 1.013 per 10 µg/m^3 annual average (not possible to derive CIs)

COMEAP comments and reference

Recommendations for various purposes were provided, that is:

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 NO_2 and $PM_{2.5}$.

The uncertainty is greater for the recommendation for quantifying effects of NO_2 itself than for NO_2 as an indicator of traffic air pollution. Three committee members did not agree to the recommendations a) and c). The areas which caused disagreement were those relating to:

- the causality of NO_2 associations with mortality
- the interpretation of results from multi-pollutant models in cohort studies
- the calculation of mortality burden

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

Note: The burden method using CRFs for NO_2 and $PM_{2.5}$ jointly was illustrated in COMEAP (2018) with the previously recommended $PM_{2.5}$ coefficient of 1.06 per 10 µg/m^3. The method is unchanged but the newly recommended $PM_{2.5}$ coefficient of 1.08 (COMEAP, 2022a) should be used instead.

Evidence base and uncertainties

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 NO_2 being causal is limited with respect to long-term exposure and all-cause mortality (uncertain underlying causality).

Potential applications

Unadjusted coefficient:

- health benefits of interventions that reduce all traffic-related pollutants
- mortality burden of an air pollution mixture for which NO_2 is an indicator (acknowledging that this may be an underestimate)[4]

Reduced coefficient:

[4] 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.

- health benefits of interventions that primarily target emissions of oxides of nitrogen (NOx)

Some recent studies reported since COMEAP recommendation[c]

- Huangfu and Atkinson (2020)

8. NO_2, long-term: respiratory morbidity in children

Health endpoints considered:

- respiratory symptoms (including bronchitic symptoms in asthmatic children)
- indices of lung function
- asthma

Type of study – coefficients[b]

Recommendation against quantification.

COMEAP comments and reference

Members agreed that the available studies were unable to provide estimates of the size of a direct effect of NO_2, that is disentangled from the effects of other pollutants in the mixture. An effect of NO_2 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 NO_2 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 NO_2 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 NO_2 could be confounded by exposure to ultra-fine particles. Although it is possible that NO_2 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.

Statement on the quantification of the effects of long-term exposure to nitrogen dioxide on respiratory morbidity in children, 2009 (NO2_resp_morbidity).

Evidence base and uncertainties

Uncertain underlying causality.

Some recent studies reported since COMEAP recommendation[c]

- Bronchitic symptoms in asthmatic children – WHO HRAPIE (2013)
- Lung function – Gehring et al (2013)
- Lung function – Gauderman et al (2015)
- Gehring et al (2015)
- Jacquemin et al (2015)

- Mölter et al (2015)

9. NO_2, short-term: respiratory and cardiovascular hospital admissions

Type of study – coefficients[b]

Systematic review and meta-analysis of time-series studies. Coefficient unadjusted for other pollutants (Mills et al 2015):

- respiratory – 0.57% (0.33, 0.82%)
- cardiovascular – 0.66% (0.32, 1.01%)

per 10 µg/m^3 increase in 24h mean or

- respiratory – 0.34% (-0.02, 0.70%)
- cardiovascular – 0.36% (-0.16, 0.89%)

per 10 µg/m^3 increase in 1h mean.

COMEAP comments and reference

Recent meta-analyses of studies evaluating the associations between respiratory and cardiovascular hospital admissions and short-term exposures to NO_2 were examined by the Committee. It is recommended that the 24-hour effect estimates are used in HIA of interventions to improve air quality.
If effects of hospital admissions estimated using the recommendations for coefficients from single pollutant models for PM and NO_2 are added to each other, this would give an overestimate of the effects of the pollution mixture as a whole.

Regarding associations of health effects with short-term variations of O_3, these can be considered as independent of the associations with NO_2.

Statement on update of recommendations for quantifying hospital admissions associated with short-term exposures to air pollutants (Hospital admissions).

Evidence base and uncertainties

The evidence suggesting a causal role for NO_2 in both respiratory and cardiovascular effects has strengthened in recent years. It is, however, stronger for respiratory effects than for cardiovascular effects, for which there remains a higher level of uncertainty. In addition, the evidence available for plausible biological mechanisms for cardiovascular effects is greater for PM than for NO_2 (COMEAP, 2018).

Respiratory: uncertain quantification.

Cardiovascular: uncertain underlying causality, uncertain quantification.

Potential applications

Number of hospital admissions associated with days of higher air pollution, for example HIA and/or burden estimates during episodes.

Not to be included in assessments which also include respiratory or cardiovascular morbidity associated with long-term average concentrations of NO_2.

Likely to reflect effects in patients who already have severe, pre-existing disease.

10. PM and NO_2, long-term: asthma

Type of study – coefficients[b]

No recommendation for quantification developed.

COMEAP comments and reference

i. Evidence from studies comparing communities (for example 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)

Some recent studies reported since COMEAP recommendation[c]

- NO_2 and asthma (children) – CAPTOR tool (2016)
- $PM_{2.5}$, NO_2 and asthma (children), NO_2 and asthma (adults) – PHE NHS and Social Care tool (2018)
- APHEKOM (2008 to 2011)
- WHO HRAPIE (2013)
- ESCAPE Gehring et al (2015) – children
- ESCAPE Jacquemin et al (2015) – adults
- ESCAPE Mölter et al (2015) – prevalence in children
- US EPA ISA (2016)
- US EPA ISA (2018)

11. O_3, long-term: mortality

Type of study – coefficients[b]

Recommendation against quantification.

COMEAP comments and reference

The evidence from all-year 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).

Evidence base and uncertainties

Limited evidence base.

Some recent studies reported since COMEAP recommendation[c]

- US EPA ISA (2020)
- Huangfu and Atkinson (2020)

12. O_3, short-term: all-cause mortality, all ages

Type of study – coefficients[b]

Systematic review and meta-analysis of time-series studies. Coefficient unadjusted for other pollutants – 0.34% (0.12, 0.56%) per 10 μg/m^3 increase in daily maximum eight-hour running mean O_3.

COMEAP comments and reference

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. As discussed for hospital admissions, these correlations can vary with season and/or temperature; however, on balance, we consider that the coefficients for all-year O_3 are likely to be independent of those for either $PM_{2.5}$ or NO_2 (COMEAP, 2022b).

Quantification of Mortality and Hospital Admissions Associated with Ground-level Ozone, 2015 (Ozone).

Evidence base and uncertainties

There is a substantial number of studies, but there is less evaluation of two-pollutant models. Temporal correlations may be negative and vary with season.

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).

Potential applications

Health effects of day-to-day variations in ambient O_3 concentrations, for example HIA and/or burden estimates in a period of time.

Likely to reflect effects in patients who already have severe, pre-existing disease.

Some recent studies reported since COMEAP recommendation[c]

- HEI Frampton et al (2017)
- Orellano et al (2020)
- US EPA ISA (2020)

13. O_3, short-term: respiratory and cardiovascular hospital admissions, all ages

Type of study – coefficients[b]

Systematic review and meta-analysis of time-series studies. Coefficient unadjusted for other pollutants (Walton et al, 2014):

- respiratory – 0.75% (0.30, 1.20%)
- cardiovascular – 0.11% (-0.06, 0.27%)

per 10 µg/m^3 increase in daily maximum eight-hour running mean O_3.

COMEAP comments and reference

Correlations between O_3 and other pollutants ($PM_{2.5}$ or NO_2) can vary with season and/or temperature; these effects may be independent of each other and depend upon the climate of the location.

However, on balance, we consider that the coefficients for all-year O_3 are likely to be independent of those for either $PM_{2.5}$ or NO_2, meaning that that there is less concern about possible over-estimation when using them in a combined assessment. In addition, policy-makers should be aware that localised interventions designed to reduce NO_2 may have the unintended consequence of increasing localised concentrations of O_3.

Quantification of Mortality and Hospital Admissions Associated with Ground-level Ozone, 2015 (Ozone); Statement on update of recommendations for quantifying hospital admissions associated with short-term exposures to air pollutants (Hospital admissions).

Evidence base and uncertainties

Cardiovascular: there is a 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.

Potential applications

Health impacts of day-to-day variations in ambient O_3 concentrations, for example HIA and/or burden estimates in a period of time.

Likely to reflect effects in patients who already have severe, pre-existing disease.

14. SO_2, short-term: all-cause mortality

Type of study – coefficients[b]

Time-series studies. Coefficient unadjusted for other pollutants – 0.6% per 10 µg/m^3 increase in 24h mean.

COMEAP comments and reference

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).

Evidence base and uncertainties

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.

COMEAP/2017/MIN/2, para 81-102
COMEAP/2017/MIN/3, para 80-82

Potential applications

Number of deaths associated with days of higher air pollution.

Likely to reflect effects in patients who already have severe, pre-existing disease.

Some recent studies reported since COMEAP recommendation[c]

- Orellano et al (2021)

15. SO_2, short-term: respiratory hospital admissions

Type of study – coefficients[b]

Time-series studies. Coefficient unadjusted for other pollutants – 0.5% per 10 μg/m^3 increase in 24h mean.

COMEAP comments and reference

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).

Evidence base and uncertainties

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.

COMEAP/2017/MIN/2, para 81-102
COMEAP/2017/MIN/3, para 80-82

Potential applications

Number of hospital admissions associated with days of higher air pollution.

Likely to reflect effects in patients who already have severe, pre-existing disease.

Some recent studies reported since COMEAP recommendation[c]

- Health Canada Air Quality Benefits Assessment Tool (AQBAT) (2012)

16. Air pollution, short-term: restricted activity days (RADs) and work days lost

Type of study – coefficients[b]

No recommended quantification method developed.

COMEAP comments and reference

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 heath endpoints

of varying severity (for example the number of hospital admissions, attendances at A&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).

Minutes – November 2013 (workdays_lost).

Some recent studies reported since COMEAP recommendation[c]

- $PM_{2.5}$ and RADs, $PM_{2.5}$ and work days lost, O_3 and minor RADs (mRADs) – WHO HRAPIE (2013)
- PM and mRADs – US EPA BenMAP (2017)
- Summer O_3 and mRADs, $PM_{2.5}$ and RADs – Health Canada AQBAT (2012)
- Ricardo AEA (2014)

[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, while those derived by short-term exposure studies as percentage change.
[c] The committee has not reviewed the information listed here.

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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

IHD: ischaemic heart disease

CBD: cerebrovascular disease

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) (until September 2022)
	Professor Gavin Shaddick (University of Exeter)
	Professor Klea Katsouyanni (Imperial College London)
	Professor Duncan Lee (University of Glasgow)
	Mr John Stedman (Ricardo Energy and Environment)
	Dr Mike Holland (EMRC and Imperial College, London)
	Dr Dimitris Evangelopoulos (Imperial College London) – co-opted
Secretariat:	Dr Christina Mitsakou (UK Health Security Agency)
	Ms Alison Gowers (UK Health Security Agency)
	Dr Naomi Earl (UK Health Security Agency)
	Dr Artemis Doutsi (UK Health Security Agency)
	Dr Karen Exley (UK Health Security Agency)

(COMEAP Chair: Professor Anna Hansell (University of Leicester))