QALY and health care costs-based damage cost toolkit for evaluating air quality measures

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GLOSSARY[1]

Health-related quality of life (HRQoL): A combination of a person's physical, mental and social well-being; not merely the absence of disease.

HRQoL weights: Measure of the preference or value that an individual or society places upon a particular health state. It is generally a number between zero (representing death) and 1 (perfect health).

National health services (NHS) costs/perspective: Costs and outcomes relating to hospital and primary care services and prescribed medications.

NO_2: Nitrogen dioxide.

NOx: Oxides of nitrogen, comprises Nitrogen dioxide (NO_2) and nitric oxide (NO)

Personal social services (PSS) costs/perspective: Care services for vulnerable people, including those with special needs because of old age or physical disability that fall outside the remit of the NHS. Examples include residential care homes for the elderly, home help and home care services, and social workers who provide help and support for a wide range of people.

PM2.5: Mass concentration of particles of generally less than 2.5 µm aerodynamic diameter. Often referred to as fine particles, this fraction can penetrate deep into the lungs.

PM10: Mass concentration of particles of generally less than 10 µm aerodynamic diameter. This fraction can enter the lungs. PM10 includes PM2.5.

Quality-adjusted life year (QALYs): A measure of the state of health of a person in which the benefits, in terms of length of life, are adjusted to reflect the quality of life. One QALY is equal to 1 year of life in perfect health.

Willingness to pay (WTP): The amount an individual is willing to pay to acquire some good or service. For non-traded goods, WTP may be elicited from stated or revealed preference approaches.

[1] Based on definitions provided by NICE; Department of Health and DEFRA.

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Executive summary

This toolkit is targeted at local authorities in the UK. It aims to generate estimates of QALY losses and health care costs (NHS and PSS) per tonne of emission of PM2.5 and NOx (or NO_2) emitted, in order to support the economic evaluation of air quality measures. As a number of inputs were derived from the Leeds-Bradford Low Emission Zone (LEZ) feasibility study, the toolkit is especially appropriate for the economic evaluation of air quality measures in this region, using information on the targeted population size and the expected annual reduction in pollutant load (spread-sheet "using emission data only"). Alternatively, in order to evaluate air quality measures outside of West Yorkshire or in areas with a substantial different population density than Leeds/Bradford, it is possible to enter area-specific estimates of avoided cases of adverse health endpoints as input data of the toolkit (via spread-sheet "Using emissions and HIA results").

The approach to toolkit development is detailed in sections 2 and 3 of the main report. In a first stage, the annual health effects per tonne emission reduction were computed based on information on estimated emissions changes and modeled reduction in concentrations for the Leeds-Bradford LEZ feasibility study. By contrasting the scope of analysis chosen for Leeds-Bradford LEZ study with HIA guidelines and evidence assessments from institutions in the UK, EU, and US and re-conducting the health impact assessment, the present work updated the study performed by Cooper at al. (2014). In a second stage, the QALY and health care costs impact associated with each endpoint was calculated by extending the work produced by Lomas et al. (2015) and modifying some structural assumptions.

All the explanations pertaining to the use of the toolkit including input data to enter (such as money value for a QALY, target population size, emission reduction) is provided in the spread-sheet "READ-ME" of the toolkit. A great strength of the toolkit is to allow a certain level of customization, so that it matches more closely with the end-user's needs and/or remains up-to date with the latest epidemiological evidence and guidelines. More specifically, the end-user can:

(i) Alter the selection of health endpoints constituting "low" and "high" ranges of impacts

(ii) Incorporate the latest epidemiological evidence for a particular endpoint, or guidance pertaining to adjustment for co-pollutant effect, by adjusting estimates of numbers of averted cases of this endpoint /tonne /year/ 100,000 individuals.
=>The formulas for adjustment are provided in section 4 of the report.

(iii) Enter area-specific estimates of avoided cases of adverse health endpoints as input data. The latter would need to be calculated separately by linking changes in emissions to change in concentrations and associated health effects.

1. Background

1.1. Toolkit objective and final users

The objective of this toolkit is to generate estimates of QALY losses and health care costs (NHS and PSS) per tonne of emission of PM2.5 and NOx (or NO_2) emitted, in order to support the economic evaluation of air quality measures. This toolkit is targeted at local authorities in the UK.

A number of inputs of the toolkit were derived from a study in Yorkshire (Leeds-Bradford Low Emission Zone "LEZ" feasibility study). Consequently the toolkit is especially appropriate for the economic evaluation of air quality measures in this region. However, in order to support the use of this toolkit across local authorities, two forms of analysis were allowed:
- One level: spread-sheet "using emission data only") is all automated and simply requires the size of the target population and the expected annual reduction in pollutant load (tonnes of PM2.5 and NOx or NO_2) as input data.
- Alternatively, for a more precise estimation of QALY gains and health care resource savings associated with air quality measures to be implemented outside of West Yorkshire or in areas with a substantial different population density than Leeds/Bradford, it is possible to enter area-specific estimates of avoided cases of adverse health endpoints in the spread-sheet "Using emissions and HIA results". These estimates are obtained by linking changes in emissions to changes in concentrations and associated health effects.

The present toolkit focuses on the health effects from chronic exposure to fine particulate matter (PM2.5) and nitrogen dioxide (NO_2). Whilst NO_2 concentrations will be the unit of reference when discussing epidemiological findings, since NOx emissions are the precursor to NO_2 concentrations, the computation of QALY and costs impacts will typically be based on the change in NOx emissions. In the toolkit, the end-user can enter a reduction (or an increase) in either NOx or NO_2 emissions by appropriately choosing the ratio of NO_2 / NOx in the "user input data" spreadsheet.

1.2. Differences with DEFRA's damage cost approach

This approach represents an alternative to DEFRA's damage costs approach, which is currently prevailing in the UK (DEFRA, 2013, 2015) for proposals of impacts below £50 million. The differences are threefold:

Firstly, this approach places a much stronger emphasis on the chronic morbidity effects of air pollution exposure and their associated quality of life loss, which thus far have been largely

ignored. Indeed, the morbidity effects currently considered by DEFRA are currently restricted to cardio-respiratory hospital admissions following acute exposure to particulate pollution (DEFRA, 2013, 2015). By contrast, the present approach considers the loss of quality of life from the development of chronic cardio-respiratory conditions and, in sensitivity analysis, prematurity.

Secondly, the identification of the endpoints to include in the scope of analysis was not solely based on current guidelines for the UK, but was also informed by recommendations and evidence assessments in the EU and US. Additionally, the choice of epidemiological evidence to inform the selected endpoints was informed by current guidelines as well as thorough searches on specialized databases.

Thirdly, whilst the DEFRA approach uses a social cost benefit perspective by monetizing health endpoints via willingness to pay (WTP), this approach instead focuses on the NHS perspective. It considers healthcare costs incurred by the NHS and personal social services (PSS) alongside health impacts. By using the QALY as health metric, this approach supports the comparison between the cost-effectiveness of air quality measures with health care interventions, which are commonly assessed in terms of cost/QALY gained. However, in contrast with DEFRA's off-the-shelf damage costs, the toolkit allows the end-user to enter the money value for a QALY. Therefore, depending on the perspective of analysis chosen, the value of a QALY to the NHS, or alternatively to the taxpayer, may be used. In practice, the value of £20,000/QALY is typically used as a reference for the value of health to the NHS (NICE, 2013) whereas, from the taxpayer's perspective, the Department of Health (Glover and Henderson, 2010) recommends to use a WTP value for a QALY of £60,000/QALY (in 2009 prices).

Fourth, instead of providing an overall damage cost result, a strength of this toolkit is to allow the end-user to: (i) understand the contribution of each health endpoint to the final result, (ii) customise the scope of analysis (beyond the choice of money value for a QALY) and (iii) easily update the toolkit input data as further epidemiological evidence is released or as guidelines are updated (see section 4).

1.3. Method outline

The present work builds upon:
(1) A health impact assessment (HIA) carried out as part of the Leeds-Bradford Low Emission Zone (LEZ) feasibility study (Cooper et al., 2014);
(2) Estimates of QALY gain and health care resource saving per adverse health outcome included in the above HIA from Lomas et al. (2015)

To ensure that the input parameters of the present toolkit were sound and most up-to date, these two components were re-analysed and sensitivity analysis were performed.

The first stage consisted of computing annual health effects per tonne emission reduction, based on information on estimated emissions changes and modeled reduction in concentrations for the Leeds-Bradford LEZ feasibility study. By contrasting the scope of analysis chosen for Leeds-Bradford LEZ HIA with HIA guidelines and evidence assessments from institutions in the UK, EU, and US and re-conducting the health impact assessment, the present work updated the study performed by Cooper at al. (2014). This will be discussed in section 2.

The second stage consisted of building on the work produced by Lomas et al. (2015) with regards to QALY loss and health care costs associated with each adverse health endpoints. To populate the toolkit, the analysis was extended and some of the structural assumptions underpinning QALY and costs computations were modified.

2. Computation of health effects per tonne emission reduction

This section outlines any updated methodology and input data compared to the HIA conducted by Cooper et al. (2014), in order to obtain estimates of health impacts per tonne emission reduction.

2.1. Health impact function

In addition to expanding the scope of analysis and using most up to date evidence, the present work applies alternative methods to generate health impacts.

The change in health effects associated with the expected change in pollutants concentrations was computed using the standard log-linear health impact function, as opposed to the formula of attributable fraction used in Duncan et al (2014), which is instead relevant for computing the burden of ill health attributable to a given level of exposure.

Indeed most epidemiological studies use a log-linear statistical model (e.g. Cox proportional hazard) to model the incidence rate of endpoint “y” for air pollution “x”, such that:
$\log(y) = \beta x + \log(\alpha)$, where:
- β is the slope coefficient of the concentration response function and

- α is the baseline incidence rate of endpoint under no pollution (α will itself be a function of many covariates impacting endpoint incidence).

The change in number of cases of health endpoint y (Δy) associated with a change in pollution concentration x (Δx) can therefore be expressed as:

$$\Delta y = y_0 [\exp(\beta \Delta x) - 1] * pop$$

where:

- $\exp(\beta \Delta x)$ equals to the risk estimate ($RE_{\Delta x}$) reported by epidemiological studies with Δx being typically equal to 10 or 5 μg/m^3 .
- y_0 is the baseline incidence rate of endpoint y.
- pop is the size of the population targeted by the change in exposure. Typically y_0 will be expressed per 1000 or 100,000 individuals so the parameter pop should be rescaled accordingly.

In line with the above - and in contrast with the approach used by Duncan et al (2014) which used linear scaling - risk estimates from epidemiological studies ($RE_{\Delta x}$) were scaled to the modeled change in concentrations associated with the intervention under assessment (Δm) using log multiplicative scaling, such that:

$RE_{\Delta m = x} = RE_{\Delta x}^{(\Delta m/\Delta x).}$

The health impact function used to compute the expected change in number of cases of endpoint *y (*Δy*),* attributable to the change in concentrations obtained from dispersion modelling (Δm) may therefore be expressed as:

$$\Delta y_{[\Delta m]} = y_0 [RE_{\Delta x}^{(\Delta m/\Delta x)} - 1] * pop \quad (1)$$

2.2. Identification of relevant health endpoints and epidemiological evidence

2.2.1. Analysis scope:

It should be underlined that the present scope of analysis only focuses on chronic health impacts associated with long-term exposure. In other words, acute effects associated with pollution episodes are not presently taken into account in the analysis. The reason for this is driven by the input data for the toolkit. It is populated by annual change in emissions and its impact on mean annual concentrations, as opposed to daily concentrations. The focus on chronic health effects associated with long-term exposure is unlikely to substantially

underestimate the benefit associated with emission reduction since: (i) the life expectancy effects of acute exposure are included in the excess mortality risk from long-term exposure (Kunzli et al., 2001) and (ii) acute effects are not expected to significantly contribute towards quality of life effects.

2.2.2. Comparative analysis of Cooper et al. (2014)'s HIA with current guidelines and past evidence assessments:

As explained in section 1.3, the HIA conducted for Leeds-Bradford low emission zone (LEZ) by Cooper et al. (2014) was the starting point of the current work. In populating the toolkit, this HIA was updated in order to ensure that this key input data would reflect the latest evidence and guidelines and used appropriate methodology.

More specifically, for each endpoint included in the HIA, two questions were evaluated:
Q1) Is there an adequate justification for endpoint inclusion in analysis?
Q2) Should the source of epidemiological evidence be updated and/or should the effect size be adjusted to account for pollutant overlap?

Question Q1 was informed by comparing the scope of Cooper et al. (2014)'s HIA against the latest guidelines for HIA of air pollution control interventions and evidence assessments from institutions in the UK, EU and the US, namely:
- COMEAP: Committee of Medical Effects of Air Pollutants (COMEAP, 2010, 2015),
- WHO - HRAPIE group: Health Risks of Air Pollution In Europe (WHO, 2013)
- US EPA. For the US EPA, both the approach followed in its latest Regulatory Impact Analysis for the Proposed Revisions to the National Ambient Air Quality Standards for Particulate Matter (US EPA, 2012a) and Integrated Science Assessments (ISA) for respectively PM (US EPA 2009, 2012b) and NO_2 (US EPA 2015) were used to inform the comparative analysis with Cooper et al. (2014).

Question Q2 was informed by reviewing the above institutions' guidelines and evidence assessments and also by performing thorough literature searches for latest epidemiological results for each endpoint using PubMed and Ludok databases. A summary of the evaluation undertaken to answer Q1 and Q2 is reported in Appendix A. In addition, the strength of evidence for widening the scope of endpoints, whilst accounting for the risk of double counting, was evaluated. The conclusions of this assessment are provided in Appendix B.
In comparing the HIA conducted for Leeds-Bradford LEZ against current guidelines and evidence assessments and results from literature searches in specialized databases, the following modifications were made to the HIA:

1- Epidemiological evidence was updated for the following endpoints:

- All cause mortality;
- Childhood asthma;
- Low birth weight of term birth.

2- Two endpoints were added to the scope of chronic adverse impacts for either main or sensitivity analysis:

(i) Incidence of chronic bronchitis in adults associated with PM exposure;

Parameterisation of the excess risk of developing chronic bronchitis in adults will rely on the risk estimate recommended by the HRAPIE group (WHO 2013) which results from the polling of two estimates from a study in the US (Abbey et al., 1995) and another in Switzerland (Schindler et al. 2009).

(ii) All cause mortality associated with NO_2 exposure.

Since the toolkit is to be used in assessments of air quality measures in the UK, the excess risk of mortality associated with chronic exposure to NO_2 will be informed by COMEAP's latest interim guidance (COMEAP, 2015). It is however, worth underlining that COMEAP's current recommended estimate to quantify the link between long-term exposure to NO_2 and all-cause mortality (1.025 with 95% CI = 1.01-1.04 for ΔNO_2 = 10 µg/m^3) appears conservative in comparison to past results from two meta-analyses studies. Indeed Hoek et al. (2013) and Faustini et al. (2014) reported pooled effect estimates of respectively 1.055 (95% CI = 1.031, 1.08) and 1.04 (95% CI =1.02–1.06) per 10 µg/m3 increment in annual average NO_2.

3- In line with COMEAP (2015) and WHO (2013) recommendations, risk estimates for health endpoints associated with both PM2.5 and NO_2 exposure were adjusted to account for effect overlap between both pollutants. For mortality effects, in line with HRAPIE guidelines (WHO, 2013)[2], COMEAP recommends to reduce NO_2 concentration-response coefficients by up to one third and, in order to obtain a central estimate, to reduce effect sizes by half this range, i.e. by 1/6th. Therefore, in the cases where the air quality measure under evaluation would reduce both NOx (NO_2) and PM emissions, a reduction by 1/6th was applied to the estimates linking NO_2 exposure with the excess risk of all-cause mortality and low birth weight term births[3] (see Table 4).

[2] This (0 – 33)% reduction range was suggested by the HRAPIE group (WHO 2013) since results from multi-pollutants models could not provide a precise estimation of the size of overlap, whereby some multi-pollutants models yielded effect size greater than results without adjustment for co-pollutants.

[3] In the toolkit, an adjustment factor of 1.20 is therefore applied to numbers of averted cases pertaining to these two endpoints if the policy under assessment is expected to only reduce NOx (NO_2) emissions. For these two endpoints, the value of 1.2 indeed represents a very close approximation of the true adjustment factor: $AF_{NO2} = (RE\Delta x - 1) / (RE\Delta x^{(1-1/6)} - 1)$, (see section 4.2.).

4- Adverse endpoints associated with PM2.5 exposure were classified in two ranges. The "low range" comprises endpoints for which current evidence strongly supports a causal link with exposure: namely all-cause mortality and coronary events. The "high range" complements these two endpoints with endpoints that are currently recommended to be considered in sensitivity analysis as the epidemiological evidence supporting them is slightly weaker - i.e. suggests, rather than indicates, a causal relationship – namely: adverse birth outcomes and incidence of chronic bronchitis in adults.

5- By contrast, the computation of a "high" versus "low" range of adverse effects associated with NOx emissions was not performed. Indeed, whilst the evidence of low birth weight associated with NOx is suggestive rather than indicative of a causal relationship, this endpoint was found to have a limited contribution to the overall net benefit from NOx emission reduction. Consequently, the creation of two ranges of impacts based solely on the inclusion/exclusion of this endpoint was not deemed necessary.

Table 1 presents the endpoints considered for each pollutant and each range of effect. It also highlights the difference in scope and epidemiological evidence used in comparison with the HIA conducted by Cooper et al. (2014).

			Evidence update (% diff in mean effect)	Source	Risk estimate ($RE_{\Delta x}$) and 95%CI Δx = 10 µg/m^3
Endpoint	**Low range**	**High range**	**PM2.5**		
All cause Death	x	x	Yes (0%) [1]	Hoek et al. 2013	1.06 (1.04 - 1.08)
CHD cases	x	x	No	Cesaroni et al. 2014 [2]	1.41 (1.00 - 2.01)
Chronic Bronchitis		Newly added	N.A	WHO 2013 (for PM10) [3]	1.12 (1.04 - 1.19)
Term LBW		x	Yes (-73%)	Dadvand et al. 2013	1.10 (1.03 - 1.18)
Pre Term Birth		x	No	Sapkota et al. 2012	1.15 (1.14 - 1.16)
Endpoint	**Base case**		**NO2**		
All cause Death	Newly added		N.A	COMEAP 2015 [4]	1.025 (1.01 - 1.04)
Childhood asthma (prev.)	x		Yes (-14%)	Favarato et al. 2014	1.06 (1.00 - 1.11)
Term LBW	x		No	Pedersen et al. 2013 [4]	1.09 (1.00 - 1.19)

Table 1: Health endpoints and risk estimates for health impact computations

[1] Hoek et al. (2013) pooled estimate has a smaller variance than the effect estimate from Pope et al (2002) but mean estimates are the same between both studies.

[2] Cesaroni et al. (2014) reported effect estimates for ΔPM2.5 = 5 µg/m3: 1.19 (1.00 - 1.42). Results have been converted here for ΔPM2.5 = 10 µg/m3 for presentational coherence.

[3] In line with WHO (2013), the effect size from this study will be multiplied by a factor of 1.54 to provide the equivalent impact per unit of PM2.5.

[4] As explained in section 2.2.2, these original estimates were reduced by 1/6th (middle value of the 0-33% range) when populating the toolkit, in order to account for overlap in effect between PM and NO_2.

2.3. Baseline health data

2.3.1. Mortality:

Directly age-standardised rates (DSR) that are obtained by combined age- specific event rates observed in the study population with the relative frequencies of age groups within a standard population (European Standard Population), were used for background mortality incidence. Whilst it may be argued that for health impact assessment, crude event rates - i.e. number of events per total at risk population - is preferable, this choice was driven by an effort to ensure data consistency between endpoints and districts. Indeed for Bradford and for hospitalizations data, only DSR for the total all age population were available. DSR were obtained based on the last 3 to 5 years of available crude data.

2.3.2. Low birth weight:

Baseline data on low birth weight was modified in order to account only for term births who were of low weight (<2500 kg), in order to avoid double counting with the health endpoint: pre-term birth which is also considered in the analysis. Data on term low birth weight, as of 2012, was obtained from the Public health outcome framework for each district. Considering only low weight births that are at term halved the baseline incidence rate used for health impact computations (4.5% and 3.1% of all births in Bradford and Leeds as opposed to respectively 9.1% and 7.2% of all births in Bradford and Leeds when considering all low birth weight births).

2.3.3. Pre-term birth:

As the source of data on prematurity used in the previous HIA could not be reliably reproduced, the latest available estimates on prematurity from England and Wales were used (7.3% of births as at 2012, ONS 2013).

2.3.4. Coronary event:

The background data used to model the change in numbers of coronary events was restricted to CHD hospitalizations (I20:I25) only, as opposed to hospitalizations for all CVD causes (ICD10: I00- I199) as was done in Cooper et al. (2014). This choice is justified by the need to be coherent with the final endpoint chosen (i.e. cases of coronary events) as well as the epidemiological evidence used in the health impact function (Cesaroni et al. 2014). Like for mortality data, DSR of hospitalizations for the entire population was used.

The obtained results in terms of avoided CHD cases is obviously lower when using CHD hospitalizations, as opposed to CVD hospitalizations, to inform the baseline incidence rate of coronary events. Indeed in 2011, CHD hospitalizations represented about 29% of all CVD hospitalizations in England (Towsend et al., 2012).

In addition, the analysis was extended by including the change in incidence of coronary event in Leeds since the past HIA (Cooper et al., 2014) only included computations for Bradford. CHD hospitalizations rate in Leeds are publicly available at MSOA level from the West Yorkshire Observatory.

2.3.5. Chronic bronchitis:

Since chronic bronchitis is part of the disease group: chronic obstructive pulmonary disease (COPD), there is sparse population statistics collected on the incidence of chronic bronchitis only. In addition it was not possible to get hold of data on COPD incidence (only prevalence data was available in Leeds). In the absence of adequate information on the background incidence rate of chronic bronchitis, WHO (2013) recommends to use the incidence rate measured in the SAPALDIA study (Schindler et al., 2009)[4]. Therefore, the background incidence rate used for the present computation is: 3.9 cases / 1,000 individuals aged 19 and above.

2.3.6. Childhood asthma:

For this endpoint, the same background data was used as in Cooper et al. (2014). Data on childhood (0-18 yrs old) asthma prevalence was collected in Leeds in 2014, as part of a regular health audit of the population. For Bradford, estimates of asthma prevalence in children were available at district level only.

3.3.7. Summary of spatial resolution of baseline health data for each endpoint:

Table 2 shows the spatial resolution of the baseline health data corresponding to each endpoint included in the analysis. District level signifies Leeds or Bradford overall.

[4] This study was also used to inform the excess risk of developing chronic bronchitis due to PM exposure see section 2.2.2.

Endpoint	Spatial resolution	Source
All cause mortality	- Bradford: LSOA - Leeds: LSOA	- Bradford city council - Leeds city council
CHD incidence	- Bradford: LSOA - Leeds: MSOA	- Bradford city council - Leeds: West Yorkshire observatory (http://observatory.leeds.gov.uk/dataviews/)
Chronic bronchitis	Bradford & Leeds: district level (using estimates for Switzerland as proxy)	WHO (2013) (using estimates from Schindler et al., 2009)
Term birth low birth weight	- Bradford: District level - Leeds: District level	Public health outcome framework (http:// http://www.phoutcomes.info/)
Pre-term birth	Bradford & Leeds: district level (using national estimates for England and Wales as proxy)	Office for National Statistics (2013)
Asthma prevalence (0-18 yrs old)	- Bradford: district level - Leeds: LSOA	- Bradford city council - Leeds asthma audit 2014.

Table 2: Spatial resolution of baseline health data for each endpoint.

2.4. Pollution concentration data

Pollution concentrations data by LSOA was provided by Leeds city council. A previous mismatch between concentrations and emission data for Bradford was corrected by using the most up to date figures.

2.5. Computation of avoided cases per tonne of pollutant emission reduction

Computation of estimates of health effects per tonne emission reduction require health impacts associated with a given decrement in concentrations to be linked with the level of emissions reduction that is expected to drive such a concentration change. This was done using the scenario ``All pre EURO 4 buses and HGVs upgraded to EURO 6 by 2016" to be implemented in Outer Ring Roads (ORR) of Leeds Bradford.

Table 5 reports the expected change in annual emissions and the corresponding decrement in population-weighted concentrations for respectively Leeds and Bradford Outer Ring Roads areas associated with this scenario. Emissions data was reported in Appendix 1 - Tables A6, A7, A14, A15 of Cooper et al. (2014). It is worth noting that in line with Cooper et al. (2014), it was assumed that only 50% of NOx concentrations is of NO_2 (the pollutant for which epidemiological evidence is based).

	Leeds (ORR area)	Bradford (ORR area)
Population (all ages)	375,082	79,124
Δ PM2.5 emissions/year	11.6	1.9
Δ pop-weighted PM.2.5 conc. in µg/m^3	0.57	0.08
Δ NOx emissions/year	290	62.3
Δ pop-weighted NOx conc. in µg/m^3	5.23	0.62
Δ pop-weighted NO_2 conc. in µg/m^3 [1]	2.62	0.31

[1] Assuming 50% of NOx concentrations is NO2.

Conc. = concentration (in µg/m^3); Δ = "change in".

Table 3: Reduction in annual PM2.5 and NOx emissions, and corresponding decrements in population-weighted concentrations in Leeds and Bradford Outer Ring Roads areas, associated with the scenario ``All pre EURO 4 buses and HGVs upgraded to EURO 6 by 2016".

Health impact assessment was computed separately for Leeds and Bradford. The health impact function (equation 1, section 2.1.) was applied to compute the numbers of avoided cases of each selected endpoint associated with PM2.5 and/or NOx exposure, whilst adjusting for overlap in effect between both pollutants (see section 2.2.2).

Whilst concentration data was available at LSOA level, as underlined in Table 2, the background rate of incidence of adverse endpoints considered in the analysis was available at three different administrative levels, namely: LSOA, MSOA or district. The change in avoided cases was therefore computed using population-weighted concentration data corresponding to the spatial scale for which baseline health data was available. Results computed at a finer spatial scale than district-level were then aggregated to provide an estimate for Leeds and Bradford respectively.

Table 6 reports the total annual numbers of avoided cases of each endpoint for Leeds and Bradford ORR areas. Importantly with regards to children asthma, the health impacts pertain to a change in prevalence as opposed to incidence and the intervention effect is therefore not annual. Figures in brackets represent the lower and upper bound values based on the 95% confidence intervals of epidemiological risk estimates.

Pollutant	**Endpoint**	**Leeds (ORR area)**	**Bradford (ORR area)**
PM	All-cause Death	8.4 (5.7 - 11.1)	0.3 (0.2 - 0.35)
	CHD cases	20.5 (0 - 41.7)	2.8 (0.0 - 5.56)
	Chronic bronchitis	10.9 (3.9 - 17.1)	0.26 (0.09 - 0.40)
	Term LBW	0.9 (0.3 - 1.5)	0.04 (0.01 - 0.07)
	Pre Term Birth	3.0 (2.8 - 3.2)	0.10 (0.09 - 0.10)
NO_2	All-cause Death	13.6 (5.5 - 21.7)	0.4 (0.15 - 0.60)
	Childhood asthma (prev.) [1]	117.7 (0 - 212.0)	3.7 (0 - 6.60)
	Term LBW	3.0 (0 - 6.1)	0.12 (0 - 0.25)

[1] One-off impact.

Table 4: Expected change in numbers of adverse cases per year associated with ``All pre EURO 4 buses and HGVs are upgraded to EURO 6 by 2016".

These results were combined with data on emission and targeted population size presented in Table 3 to generate the total number of cases averted /tonne emission reduction /year/ 100,000 inds (all ages).

Results for Leeds and Bradford were then combined using a population-weighted approach (Leeds $_{\text{pop-weight}}$ = 83%; Bradford $_{\text{pop-weight}}$ = 17%, see population numbers in Table3) in order to populate the toolkit with a single set of estimates for each endpoint. For childhood asthma, only results for Leeds were used since the baseline health data was available at a much more refined scale than for Bradford (see Table 2).

3. Linking averted cases with QALY and NHS and PSS cost impacts

This step relied on the work produced by Lomas et al. (2015) but implemented some modifications with regards to the structural assumptions underpinning QALY and health care resource use computations. In addition, since Lomas et al (2015)'s work was performed for the endpoints considered in the HIA conducted by Cooper et al. (2014), i.e. not including chronic bronchitis, QALY losses and health care costs associated with this endpoint were calculated. A summary of the differences between the present computations and those of Lomas et al. (2015) may be found in Appendix C.

3.1. General framework

A QALY is computed by multiplying the time spent in a given health condition with the health-related quality of life (HRQoL) associated with that state of health (Gold et al., 2001). It follows that the QALY loss associated with each case of chronic disease *x* may be expressed as the sum of two elements A+ B where:
A represents the quality of life loss occurring over the individual's remaining life expectancy conditional on having disease *x*;
B represents the life year loss associated with disease, scaled to the counterfactual HRQoL of "healthy" individuals (i.e. without disease *x*).

Denoting:
- DLE$_{i,x}$ and DLE$_{i,h}$ the discounted remaining life expectancy of individuals of age *i* if respectively with disease *x* or if healthy;
- HRQoL$_{i,x}$ and HRQoL$_{i,h}$ the HRQoL scores for individuals of age *i* if respectively with disease or healthy

We can express A and B as:
A= DLE$_{i,x}$ (HRQoL$_{i,h}$ - HRQoL$_{i,x}$)
B= HRQoL$_{i,h}$ (DLE$_{i,h}$ - DLE$_{i,x}$)

It is worth underlining that element B is expected to be captured in the overall loss of life expectancy that is informed by the risk estimates of excess mortality from all causes. It follows that, *when all-cause mortality is also considered as a health endpoint*, to avoid double-counting of life expectancy gains, the QALY loss from each case of disease *x* should be based on element A only.

3.2. Method

Computation of QALY gain - and by extension health care resource savings - associated with a reduction in cases of morbidity and mortality following air pollution decrement required to characterise four components. Each component will be described in the sections 3.2.1. to 2.2.4 and summarised in Table 5.

3.2.1. Timing of disease development / adverse event occurrence

Since age-specific morbidity and mortality data was unavailable, the numbers of (avoided) cases of each endpoint were computed for the total all-age population targeted by the air quality measure (see section 2). Characterisation of the timing of adverse event was therefore based on the average age of the population subgroup assumed to be at risk of experiencing adverse health events[5].

At-risk subgroups were defined as: 45 years old and above for CHD and chronic bronchitis; 0-18 years old for asthma and 75 years old and above for premature death. Data on age-specific life expectancy and statistics on age and gender population distribution required to compute the average age of individuals in at-risk subgroups was based on the population of England and Wales, to support the generalizability of the toolkit outside of West Yorkshire. Importantly, the use of population statistics for England and Wales, as opposed to Leeds and Bradford, had a minimal impact on final results.

3.2.2. The reduction in life expectancy associated with each adverse morbid event

The reduction in life expectancy associated with CHD was computed by applying hazard ratios of excess death provided by Whiteley et al. (2005) to the baseline mortality rates of individuals of the general population who do not suffer from CHD, that were obtained from life-table computation using ONS data (ONS, 2014). CHD was found to reduce life expectancy by 3.4 years for males at age 62 and 2.6 years for females at age 63. When compared with individuals of the general population (which include both individuals with and without CHD), suffering from CHD was associated with a 2-year reduction in life expectancy for both genders.

For prematurity, the reduction in life expectancy was computed based on the excess risk of suffering from a disability (Mangham et al. 2009). By contrast, chronic bronchitis and asthma were assumed to have no impact on life expectancy and the reduction.

[5] Importantly, this is not necessarily the same as the population subgroup for which the change in incidence was computed. For instance, whilst the change in numbers of CHD cases was computed using directly age-standardised rates for the total all-age population, it is well known that young individuals are very unlikely to suffer from coronary events.

3.2.3. The duration of the disease

As shown in equation A, if the disease is chronic, as is the case of CHD, chronic bronchitis, the duration is simply the remaining life expectancy at time of event. For childhood asthma, it was assumed that the disease would continue into adulthood with a 58% probability (Oswald et al., 1994).

3.2.4. The health-related quality of life (HRQoL) weight associated with each condition.

For mortality, the HRQoL of individuals of the general population of England (Kind et al., 2009) aged above 75 were used as counterfactual for quality of life. For morbid conditions, age-constant decrement in *HRQoL (i.e.* HRQoL*i,h* - HRQoL*i,x*) estimated by Sullivan et al. (2011) were used.

For CHD, decrements were provided for specific heart conditions (i.e. acute myocardial infarction, old myocardial infarction, angina pectoris and other chronic ischemic heart disease). To obtain an overall result for this disease, a weighted average of these decrements was computed based on hospitalizations statistics by causes of heart disease for England and Wales (Townsend et al., 2012). Additionally, in order to account for the fact that the loss of quality of life after a myocardial infarction is expected to be partially regained in the following months after the event, two CHD-related HRQoL decrements were computed for respectively (i) the year of event onset and (ii) subsequent years.

3.2.5. Summary of assumptions underpinning each four components

The assumptions pertaining to each four components underpinning QALY and health care costs impacts computations for each adverse health endpoint are presented in Table 5.

Endpoint	1 - Timing of event	2- Life expectancy impact/person	3- Duration	4- HRQoL
All-cause mortality	After age 75	LE loss = 2 years [2]	N.A.	Used counterfactual HRQoL score for the general population of England aged 75+ from Kind et al. (1999): HRQoL*75,h* =0.73
CHD incidence	After age 45 (mean age: 62 for males, 63 for females)	See section 2.3.2.2. DLE*i,x* = DLE*i,h - 2*	From onset until death (brought forward)	Weighted HRQoL decrement (see section 2.3.2.2.) Yr1: HRQoL*i,h* - HRQoL*i,x* = -0.0688 Yr2: HRQoL*i,h* - HRQoL*i, x* = -0.0627
Chronic bronchitis	After age 45 (mean age: 62 for males, 63 for females)	No life-shortening, i.e. DLE*i,x* = DLE*i,h*	From onset until death	HRQoL decrement from Sullivan et al (2011): HRQoL*i,h* - HRQoL*i,x* = -0.0444
Term birth low birth weight	N.A. Only health care costs were attributed to this endpoint			
Pre-term birth (PTB)	At birth	Depending on level of disability:11 years of life loss for moderate disability and 53 life years loss for severe disability	For those suffering from a disability due to PTB, a lifetime impact was assumed	HRQoL associated with each disability level from Colbourn et al. (2007) [1]
	N.B. Computation of QALY loss (and health care costs) associated with PTB, were underpinned by differences in risk of being born with a disability using the disability rates of term birth as counterfactual. Data was obtained from Mangham et al (2009) [1]			
Asthma prevalence (0-18 yrs old)	At 10 years old which is the mean age of England and Wales population aged 0-18	No life-shortening, i.e. DLE*i,x* = DLE*i,h*	From onset until 18 years old with a 58% probability (Oswald et al., 1994) [1] that the disease will continue in adulthood until death	HRQoL decrement from Sullivan et al (2011) HRQoL*i,h* - HRQoL*i,x* = -0.0463

[1] Study sourced from Lomas et al (2015).
[2] Sensitivity checks assuming that individuals dying prematurely from air pollution exposure would loose on average 1 year (as opposed to 2 years) of life expectancy barely impacted on results (8.6 QALY loss as opposed to 8.4 in base case).
i represents the average age at which event are assumed to occur; *h* represent individuals of the general population of age *i*; *x* stands for the morbid endpoints considered: chronic bronchitis, CHD, asthma.

Table 5: Assumptions underpinning QALY and health care cost impacts computation.

3.3. Costs data sources.

Costs were computed based on the same data sources as Lomas et al (2015). For chronic bronchitis, which was added to the scope of relevant endpoints, the costs was based on available evidence for COPD.

In the UK, the department of health produced an estimate of annual cost of COPD of £ 810 million for about 800,000 patients as at 2009 (DH, 2010), which is equivalent to an annual cost of £1,102 per COPD patient in 2014 prices. However, COPD includes emphysema which is a more severe condition and the health care costs associated with the disease strongly depends on the level of advancement of the disease, i.e. degree of airflow obstruction. Therefore, the costs of stage 2 COPD estimated for Sweden by Jansson et al. (2013) was instead selected as a proxy for the cost of chronic bronchitis in the present analysis. When converted into GBP and inflated to 2014 prices, stage 2 was associated with an annual cost of £955/patient.

3.4. QALY and health care costs impacts estimates

Table 6 presents the estimates of QALY and health care (NHS and PSS) costs impacts used to populate the toolkit

Endpoint	Health care cost (£)	QALY loss
All-cause mortality	0	8.4 [1]
CHD incidence	31,000	0.93
Chronic bronchitis	15,443	0.70
Term birth low birth weight	2,374	N.A.
Pre-term birth (PTB)	24,071	1.26
Children asthma prevalence	2,439	0.87

[1] It should be reminded that this QALY impact pertains to each attributable death, which is simply a statistical number obtained by multiplying background mortality rate by the risk estimate of excess death and does not reflect the "true" numbers of death occurring in the population. In other words, it should not be not interpreted as: "each person dying prematurely will lose on average 8.4 QALY".

Table 6: Health care costs and QALY impacts associated with each adverse health endpoint.

4. Toolkit customisation and input data update

All the explanations pertaining to the use of the toolkit including input data to enter (such as money value for a QALY, target population size, emission reduction) is provided in the spread-sheet "READ-ME" of the toolkit.

This section aims to underline possibilities to customize the toolkit, so that it matches more closely with the end-user's needs and/or to update the toolkit input data so that it remains up-to date with the latest epidemiological evidence and guidelines.

All results provided in Table 3, 4 and 6 that were used to populate the toolkit are accessible in the spreadsheet "using emissions and HIA results". Therefore, instead of providing an overall damage cost result, a strength of this toolkit is to allow the end-user to understand the contribution of each health endpoint to the final result and if desired, to adjust all or a selection of input data.

Note that adjustments can only be done is the spread-sheet "using emissions and HIA results".

4.1. Alter selection of health endpoints constituting "low" and "high" ranges

The selection of endpoints that constitutes the "low" and "high" range of impacts (described in Table 3) may be modified by adjusting Table 2 of the spread-sheet (Cells O12 to T17).

4.2. Incorporate new pieces of epidemiological evidence / change in guidelines

As further epidemiological results become available, the end-user may easily incorporate the latest evidence for a particular endpoint, by multiplying estimates of numbers of averted cases of this endpoint /tonne /year/ 100,000 individuals (all ages) provided in cells E9 to G18 with an adjustment factor AF that derives from the formula for health impact function provided in equation (1):

$$AF_{Endpoint} = (\text{New } RE_{\Delta x\text{new}}{}^{(\Delta m/\Delta x\text{new})} - 1) / (\text{Current } RE_{\Delta x}{}^{(\Delta m/\Delta x)} - 1) \quad (2)$$

Where "Current $RE_{\Delta x}$" and "Δx" are defined in Table 1 and "Δm" represents the population-weighted change in concentration modelled for the Leeds-Bradford LEZ scenario used to populate the toolkit i.e: Δm = 0.5 for PM2.5 and 2.2 for NO_2 (see Table 3 with Leeds $_{pop\text{-}weight}$ = 83%; Bradford $_{pop\text{-}weight}$ = 17%).

For instance if, in the future, additional evidence were to suggest that the mean effect size for the excess risk of mortality for PM is 1.07 per 10 µg/m^3 as opposed to the currently used value of 1.06 per 10 ug/m3 (see Table 4), then:
$AF_{Death} = (1.07^{(0.5/10)} - 1) / (1.06^{(0.5/10)} - 1) = 1.16$ and
Updated mean number of deaths /tonne /100,000 individuals = 0.19 * 1.16 = 0.22

Alternatively, if new evidence were to suggest an effect size of 1.07 per 5 µg/m^3 :
$AF_{Death} = (1.07^{(0.5/5)} - 1) / 1.06^{(0.5/10)} - 1) = 2.33$ and updated mean number of deaths /tonne /100,000 individuals = 0.19 * 2.33 = 0.44

Similarly, as mentioned in section 2.2., there is currently great uncertainty with regards to the overlap in effects between PM and NO_2. Currently, in line with current recommendations (COMEAP, 2015), the risk estimates linking NO_2 exposure with all-cause of death and low birth weight are reduced by 1/6th. However, if the guidelines were to change, the $AF_{Overlap}$ to apply to the estimates of numbers of averted cases of all-cause of death and low birth weight / tonnes of NO_2 reduction /year/ 100,000 provided in cells E9 to G18 would be:

$$AF_{Overlap} = (RE_{\Delta x}{}^{(1 - \% \text{ reduction new})} - 1) / (RE_{\Delta x}{}^{(1 - 1/6)} - 1)$$

4.3. Enter estimates from a full impact pathway approach

Finally, as mentioned in section 1.2, for greater accuracy when assessing air quality measures outside of West Yorkshire, area-specific estimates of avoided cases of adverse health endpoints obtained via the "full impact pathway approach" (i.e. by linking changes in emissions to change in concentrations and associated health effects) may be entered as input data of the toolkit (Cells E9 to G18 of in spread-sheet "using emissions and HIA results").

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