# Lower Thames Crossing

## 9.67 Air Quality Quantitative Health Impact Assessment

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# 1 Executive summary

## Context

1.1.1 The Environmental Statement included a proportionate, appropriately scoped air quality assessment [APP-143] in the context of recognised AQS objectives and Limit Values protective of the environment and health, and concluded that the A122 Lower Thames Crossing (the Project) would not result in a significant effect on human health. While sufficient to determine compliance with the National Policy Statement for National Networks (Department for Transport, 2014), residual health concerns have been noted through wider engagement, and additional clarity was deemed of value to further respond to concerns in relation to non-threshold pollutants, and the perceived potential health risk from any changes in air quality as a result of the Project, regardless of meeting the regulatory air quality threshold objectives protective of health.

## Approach and methodology

1.1.2 The voluntary Air Quality Quantitative Health Impact Assessment (AQQHIA) was carried out to complement the submitted assessment by applying the approach and supporting evidence base collated by the Department of Health's Committee on the Medical Effects of Air Pollutants (COMEAP) and the Clean Air for Europe (CAFE) programme.

1.1.3 The AQQHIA builds upon the approach in the Design Manual for Roads and Bridges (DMRB) air quality assessment, by quantifying health effects associated with the absolute change in air pollutant concentrations (regardless of whether the total concentration is above or below a set threshold).

1.1.4 The quantification methodology is well-established, following approaches published by the COMEAP and the World Health Organization (WHO) Health risks of air pollution in Europe (HRAPIE) project. These organisations have reviewed the extensive international epidemiological literature on the subject to develop the methods for the quantification of health effects from air pollution.

1.1.5 The adopted methodology is appropriate to quantifying effects in large populations and has no relationship to individuals. As a result, the effect on health outcomes is observed across the population studied as a whole, and the final impact (be it mortality or morbidity) is one shared across a population.

In this context, care should always be taken when considering the calculated mortality and morbidity impact, as they are not individual impacts, but an aggregation of an impact shared across an entire population. As an example, one calculated additional hospital admission brought about by changes in an air pollutant is not one person experiencing a hospital admission, but potentially a (hypothetical) population of 100,000 experiencing an additional epidemiological risk of 0.00001 of an extra hospital admission per year.

## Assessment protocol

1.1.6 In order to quantify health effects, the operational air quality dispersion model outputs were applied to generate concentration exposure estimates within the air quality study area (i.e. the population residing within 200m of those roads which met the traffic change criteria set out in DMRB LA 105 (Highways

England, 2019)). A distance of 200 metres is generally used in road traffic air quality assessments as concentrations from vehicles decrease rapidly with distance from the source; beyond 200m the increase or decrease in the road source pollution contribution is not typically discernible from fluctuations in the background concentration. Therefore, the AQQHIA study area is limited to a 200m buffer around the ARN.

1.1.7 Annual average concentrations were then calculated using long term concentration response functions (CRFs), with and without the Project for 2030, and the number of people resident within small administrative geographical areas established (at Output Area (OA) level). Morbidity that could be caused by pollution changes over short-term periods such as cardiovascular and respiratory hospital admissions were assessed by utilising 24-hour CRFs, which are then aggregated over a year.

1.1.8 The change in concentration in each OA was used with the relevant CRF for each pollutant to calculate the attributable fraction. This was then multiplied by the existing burden of health (for each of morbidity and mortality) within the area to quantify the effect.

1.1.9 The baseline rate of mortality was derived at sub-local authority level from data published by the Office of National Statistics (ONS), and as such, the methodology takes into account varying rates of existing mortality and burdens of poor health across the entire study area. This method was repeated for hospital admissions, applying national scale data for England published by the National Health Service (NHS) on admission statistics to define the respective baseline rates for cardiovascular and respiratory hospital admissions. The appropriate COMEAP CRF was then applied.

## Results

1.1.10 The effect on mortality was quantified and concluded that there is no evidence for a measurable effect on population health (adverse or beneficial) from the Project. The effect on health from changes in nitrogen dioxide ($NO_{2)}$ associated with the Project was greater than those for particulate matter less than 2.5µm in aerodynamic diameter ($PM_{2.5}$), therefore in line with advice from COMEAP[1], the conclusions of the Project's effect on mortality are based on $NO_2$.

1.1.11 The effect on mortality from changes in $NO_2$ concentration exposure from the Project was an effect equivalent to approximately 0.3 of a death brought forward, shared across the population residing in the study area (c.148,000 people, in which the annual average number of deaths from all causes between 2017-2019 was 1,921 deaths). This represents an increase of <0.02% in relation to the annual average number of deaths from all causes recorded between 2017-2019 within the AQQHIA study area.

[1] According to COMEAP (2020), it is advised that the pollutant which leads to the greatest increase in a given health outcome should be used to represent the effect of the two pollutants in combination (i.e. either $NO_2$ or $PM_{2.5}$); as adding them together would represent a considerable overprediction. This is because the health effects of the two pollutants are correlated with each other according to the epidemiological studies which inform the CRFs. As a result, both the $NO_2$ and $PM_{2.5}$ CRFs may include effects of other pollutants and each other. For this reason, COMEAP advise on calculating the outcome for both pollutants, presenting the pollutant which leads to the greater health impact as the basis for informing effects.

1.1.12 The annual standard deviation in deaths from all causes in the population considered in the AQQHIA between 2017-2019 was 24 deaths per annum indicating that it would not be possible to separate the effect of the project on mortality in the opening year from natural variations.

1.1.13 The magnitude of the potential change in mortality is not of a level that would be measurable or detectable in annual public health statistics.

1.1.14 The effect of the Project on morbidity outcomes was quantified in terms of the effect on respiratory hospital admissions ($NO_2$), and cardiovascular hospital admissions ($PM_{2.5}$):

a. $NO_2$ impacts on respiratory hospital admissions were negligible with an additional 0.15 admissions per year in the population (all ages) residing within the AQQHIA study area. This represents an increase of 0.003% in relation to the annual average number of respiratory admissions recorded between 2017-2019 within the population residing in the AQQHIA study area.

b. $PM_{2.5}$ Impacts on cardiovascular hospital admissions were equally negligible with <0.1 additional admissions per year in the population residing within the AQQHIA study area. This represents an increase of 0.001% in relation to the annual average number of respiratory admissions recorded between 2017-2019 within the AQQHIA study area.

## Conclusion

1.1.15 The assessment concludes that the potential impacts of the Project on mortality, and respiratory and cardiovascular hospital admissions are neither measurable nor material in the context of public health. The impacts are orders of magnitude lower than is required to quantify any tangible adverse health outcome, and the net aggregate change was far less than 1% of the reported public health effects within the population studied.

1.1.16 The AQQHIA thereby concurs with the originally submitted air quality assessment within the Environmental Statement and reinforces the conclusion with respect to effects on human health, because:

a. The relative change in air pollution has been assessed to be not significant in terms of human health in the context of air quality thresholds in Chapter 5 – Air Quality of 6.1 Environmental Statement [APP-143].

b. the relative change in absolute concentration and exposure, factoring in existing burdens of poor health, remains orders of magnitude lower than is required to quantify any adverse health outcome.

# 2 Methodology

## 2.1 Scope of Assessment

2.1.1 The AQQHIA quantifies the health outcomes across the population residing within the operational phase local air quality study area as defined by DMRB LA 105 (Highways England, 2019). This study area comprises the area within 200m of roads where the following traffic change criteria (based on the outputs of the 2030 CS67 traffic model outputs) are met as a result of the operation of the Project:

a. Change in Annual Average Daily Traffic (AADT) of >=1,000 vehicles per day

b. Change in Heavy Duty Vehicles (HDV, >3.5 tonnes) flow of >=200 vehicles per day

c. Change in period speed band

d. Change in road alignment of >=5m

2.1.2 Those roads which meet any of the listed criteria are collectively referred to as the 'Affected Road Network' (ARN), and the area within 200m of these roads is referred to as the operational study area for assessing the impact on local air quality. A distance of 200 metres is generally used in road traffic air quality assessments as concentrations from vehicles decrease rapidly with distance from the source; beyond 200m the increase or decrease in the road source pollution contribution is not typically discernible from fluctuations in the background concentration. Therefore, the AQQHIA study area is limited to a 200m buffer around the ARN.

2.1.3 The population resident within this area is referred to as 'the population exposed' within the 'AQQHIA study area'. The operational phase ARN which details the extent of the study area is detailed in Environmental Statement Chapter 5: Air Quality [APP-143] and is displayed in maps on Environmental Statement Figure 5.3: Operational Study Area, parts (1 of 3) [APP-172], (2 of 3) [APP-173], and (3 of 3) [APP-174].

2.1.4 The ARN for assessment of local air quality also defines the geographical extent of the AQQHIA.

2.1.5 The following health outcomes have been calculated:

a. Mortality burden (all-causes mortality) ($NO_2$)

b. Mortality burden (all-cause mortality) ($PM_{2.5}$)

c. Respiratory hospital admissions ($NO_2$)

d. Respiratory hospital admissions ($PM_{2.5}$)

e. Cardiovascular hospital admissions ($PM_{2.5}$)

2.1.6 These outcomes have been chosen for calculation as $NO_2$ and $PM_{2.5}$ were assessed in Environmental Statement Chapter 5: Air Quality [APP-143] and are associated with road transport emissions. Additionally, the CRFs for the health outcomes selected are underpinned by a greater body of evidence in associating changes in $NO_2$ or $PM_{2.5}$ concentration with a change in the prevalence of the relevant health outcome. The COMEAP publications which inform the CRFs used and scope of the AQQHIA are detailed in the following section.

## 2.2 Outline Methodology

2.2.1 The effects of air pollution on acute health effects can be quantified using CRFs which can be used to provide an estimate of the change in risk associated with a given health effect as a consequence of a change in pollutant concentration. A range of CRFs are recommended by COMEAP based on ongoing reviews of evidence generated by epidemiological academic papers and research. The following COMEAP recommendation reports and statements have informed this assessment:

a. COMEAP (2020) Summary of COMEAP recommendations for quantification of health effects associated with air pollutants

b. COMEAP (2022a) COMEAP statement on quantifying mortality associated with long-term exposure to fine particulate matter

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

2.2.2 Risk coefficients are an epidemiological measure of exposure to a pollutant at a population level; these underpin air quality objective thresholds protective of health and can be further applied to set risk into context by establishing magnitude and distribution of effect. The CRF uses the exposure-response risk coefficient (or relative risk) to generate an outcome across a selected population where the change in pollution that the population would be exposed to is known. This calculation is summarised in the conceptual formula below:

**M** = *B* * *C* * *P* * *E*

**M = Change in rate of health outcome**

*B = Concentration Response Function*

*C = change in pollutant concentration*

*P = population exposed*

*E = existing baseline rate of health outcome*

2.2.3 The total population considered (*P*) varies depending on the outcome being calculated, as certain CRFs (*B*) are only applicable to certain cohorts of the population (i.e. adults >30yrs for all-cause mortality, all ages for hospital admissions etc). The existing baseline rate of health outcome (*E*) is a measure of the current rate of mortality or morbidity in a population and is typically calculated per 1000 population. The methodology utilised to quantify health

effects across a population due to a change in air pollutant concentrations (*C*) produces outcomes across the exposed population and not the effect on any individual living within that population. This is because the methodology is appropriate for the consideration of effects at the population level and impacts cannot be accurately ascribed to individuals given the variations of innate sensitivity, real-world exposure level, lifestyle etc.

2.2.4 The calculations were carried out using the Atmospheric Dispersion Modelling System (ADMS) Urban v5.0.1.3 software model setups which had provided the air quality predictions informing the operational phase local air quality assessment presented in Environmental Statement Chapter 5: Air Quality [APP-143] for the following scenarios:

a. Base year (2016)

b. Do Minimum (2030, future baseline)

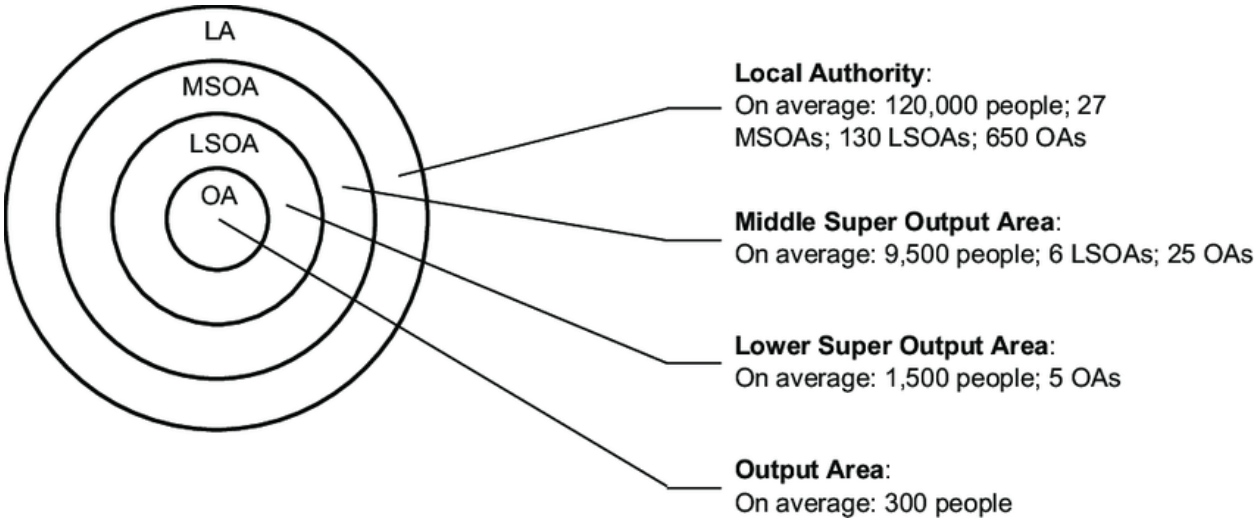
c. Do Something (2030, future baseline + the Project)

2.2.5 The approach to selecting receptor points to be included in the dispersion model is provided in Section 2.4.

## 2.3 Census geographies

2.3.1 The UK Government's population and health statistics are collated and reported at cascading geographical scales. This range of scales are referred to as census geographies. With reference to Plate 2.1 below, Output Areas (OAs) represent the smallest geographical areas where population statistics are counted and collated. Lower Super Output Areas (LSOAs) are geographically larger areas than OAs and represent the next level up in the hierarchy of census geographies, on average containing five OAs. Middle Super Output Areas (MSOAs) encompass both LSOAs and a larger number of OAs etc. This stepped agglomeration of smaller geographical areas with increasing scale applies all the way up to local authority level as per Plate 2.1.

**Plate 2.1 Graphical representation of census geographies (Laurence et al., 2019)**



2.3.2 The prevalence of mortality and morbidity are typically recorded and reported at coarser geographical levels (either MSOA, LA or national level). Therefore, to calculate local (smaller scale) estimates of the occurrence of existing health effects, the data should be population weighted. In terms of reporting, each OA has an identifying code for every geographical level of census reporting (as it sits within larger scale geographies). This pathway of identification means that it is possible to extrapolate local (OA level) estimates of mortality/morbidity derived from statistics recorded at a larger geographical scale through population weighting. Population-weighted baseline rate data at OA level is more representative the lower down the census hierarchy that the health effect is originally recorded (i.e. the death rate per 1,000 persons recorded in the MSOA that the OA lies within is likely to be more representative when analysing the population in the given OA than it would be if a coarser national rate were used as the basis for the extrapolation).

2.3.3 As cited previously, the most detailed and high-resolution estimates of population at the local level from census datasets are at OA level, where the typical population ranges between 100-620 persons. A total of 1,238 individual OAs bisected the AQQHIA study area.

## 2.4 Calculating average concentrations at OA level

2.4.1 Address Layer 2 datasets consisting of geographical points identifying land use were acquired from the Ordnance Survey as a means of identifying where sensitive receptors were located within the AQQHIA study area. There were approximately 91,000 sensitive receptor points located in the AQQHIA study area. To ensure a representative approach and to optimise processing, receptor locations representative of the distribution of the population were generated using the ‘integrate’ function in ESRI ArcGIS to a resolution of 25m which reduced the number of modelled points to 6,751 points representative of the geographical locations of population exposure within the OAs. The ‘integrate’ function simplifies receptor locations in a given area to a set resolution. This is similar to modelling a set grid, however the ‘integrate’ function only creates points in those areas where address layer 2 points were present and not in those uninhabited areas of OAs. An example of the actual address layer 2 receptor points and the representative modelling points generated using the ‘integrate’ function, is demonstrated on Plate 2.2.

**Plate 2.2 Indicative extract of geographical features informing the AQQHIA modelled points**


2.4.2 The representative points were inputted into the existing ADMS-Urban v5.0.1.3 models which had provided the air quality predictions informing the operational phase local air quality assessment presented in Environmental Statement Chapter 5: Air Quality [APP-143] for the Base Year 2016, Do Minimum 2030 and Do Something 2030 scenarios.

2.4.3 Each modelled point was assigned an OA ID, an MSOA ID and a local authority ID to aid in the calculation of baseline rates. Pollutant concentrations at modelled points within each OA were averaged in both the Do Minimum (2030, without the Project) and Do Something (2030 with the Project) scenarios. The average change in pollutant concentration in each OA was then inputted into the wider calculation of the relevant health outcome, and then aggregated to local authority, and Project-wide level.

## 2.5 Concentration response functions and health outcomes selected for assessment

2.5.1 The quantitative relationship between additional incidence or risk of a health outcome and long-term exposure to a pollutant is described by a CRF. For example, COMEAP (2020) states that a 10µg/m$^3$ increase in $PM_{2.5}$ concentration will increase the mortality risk by 8% - i.e., multiply existing risk by 1.08.

2.5.2 The CRF quantifies the ratio of a health outcome at one level of exposure to a pollutant at a set higher level (in this case per 10µg/$m^3$). A review of those CRFs recommended by COMEAP was carried out to determine the values inputted into the assessment. The selected CRFs have been reviewed and recommended by COMEAP in their most recent publications and therefore reflect the best estimates of the change in risk associated with increases in air pollutants. The long and short term CRF coefficients which were used in the assessment are detailed below in Table 2.1.

**Table 2.1 Summary of CRFs used in the AQQHIA**

| Pollutant | Health effect | Population cohort studied | CRF (per 10µg/$m^{3)}$ (95% confidence intervals in brackets) | Comments | Source |
|---|---|---|---|---|---|
| $NO_2$ | All-cause mortality | Adults ≥30 years | 1.023 (1.008, 1.037) | Unadjusted* for other pollutants, annual | COMEAP (2020) Summary of COMEAP recommendations for the quantification of health effects associated with air pollutants |
| | Respiratory hospital admissions | All ages | 1.0057 (1.0033, 1.0082) | Unadjusted* for other pollutants, 24-hour | COMEAP (2022b) Statement on update of recommendations for quantifying hospital admissions associated with short-term exposures to air pollutants |
| $PM_{2.5}$ | All-cause mortality | Adults ≥30 years | 1.08 (1.06, 1.09) | Unadjusted* for other pollutants, annual | COMEAP (2022a) COMEAP statement on quantifying mortality associated with long-term exposure to fine particulate matter |
| | Respiratory hospital admissions | All ages | 1.0096 (0.9937, 1.0258) | Unadjusted* for other pollutants, 24-hour | COMEAP (2022b) Statement on update of recommendations for quantifying hospital admissions associated with short-term exposures to air pollutants |
| | Cardiovascular hospital admissions | All ages | 1.009 (1.0026, 1.0153) | Unadjusted* for other pollutants, 24-hour | |

**According to COMEAP, it is advised that the pollutant which leads to the greatest increase in a given health outcome should be used to represent the effect of the two pollutants in combination (i.e. either $NO_2$ or $PM_{2.5}$) as adding them together would represent a considerable overprediction. This is because the health effects of the two pollutants are correlated with each other according to the epidemiological studies which inform the CRFs. As a result, both the '**unadjusted**' $NO_2$ and $PM_{2.5}$ CRFs may include effects of other pollutants and each other; therefore, summing them would represent an overestimate of the effect. For this reason, COMEAP advise on calculating the outcome for both pollutants, presenting the pollutant which leads to the greater health impact as the basis for informing scheme effects.*

2.5.3 The relative risk (RR) corresponding to an air pollution increment or difference other than the reference µg/$m^3$ value can be calculated by scaling according to the modelled concentrations. For example, if the increase in $NO_2$ was 5µg/$m^3$ and the metric being calculated was mortality then the calculation would appear as:

RR = 1 + (0.023*(5/10))

This gives an RR of 1.012.

2.5.4 The attributable fraction (AF) of mortality is calculated by the RR/OR by the formula:

AF = (RR-1)/RR

2.5.5 Therefore, an increased exposure of 5µg/$m^3$ would yield an AF of 0.012, or 1.2%. This value could then be multiplied by the population weighted baseline incidence of a given health outcome to quantify the impact on mortality. It should be noted that the AF is an epidemiological concept relating to effects at population level and is therefore an important part of calculating the change in mortality (or other health outcome) in a population as a result of changes in air pollution.

2.5.6 The adopted methodology initially produces estimates of the mortality burden from all causes without and with the Project in 2030. Any increase in the mortality burden is essentially expressed as 'deaths brought forward'; this term relates to the fact that this type of assessment cannot distinguish whether the deaths are brought forward by only a short amount of time or whether the deaths are brought forward by a longer time.

2.5.7 Care should be taken when considering the mortality burden expressed as deaths brought forward. This is because an additional 'death' is an aggregation of an impact across an entire population (in this case approximately 148,000 people) at population level and not on an individual.

2.5.8 Any measure of the increase in the mortality burden from air pollution is through deaths brought forward whereby the effect on life expectancy is caused over the long term and is akin to exposure to cigarette smoke or through a sustained unhealthy diet. COMEAP (2018) estimated the mortality burden from long-term exposure to air pollution to be equivalent to 28,000-36,000 deaths brought forward in the UK.

2.5.9 Short-term health impacts in terms of hospital admissions (for cardiovascular and respiratory diseases) have also been calculated for $NO_2$ and $PM_{2.5}$. Those short-term CRFs for hospital admissions published by COMEAP (2022b) which are typically applied to the 24-hour average pollutant concentration have been applied to annual mean concentrations generated as part of this assessment with the assumption that computing based on annual means will provide an approximation of the effect on hospital admissions; this follows other studies such as the Department for Environment, Food and Rural Affairs' (Defra's) (2023) Air Quality Damage Cost update report which provides justification for this approach and states:

'*The sum of short-term effects calculated on a daily basis from a combination of daily means over the period of a year and the CRF for daily effects is mathematically the same as a calculation based on an annual mean and a duration of a year. Hence it is more efficient and simpler to use annual mean for these calculations, whilst making no difference to the outturn results*.'

# 3 Results

## 3.1 Mortality – $NO_2$ & $PM_{2.5}$

### Baseline rate of mortality in AQQHIA study area

3.1.1 Information on deaths in the population aged 30 years or more was derived from the Office of National Statistics (ONS) via the Nomis online service (ONS, 2023) which publishes census data. Limiting the calculation of mortality to the population aged 30 or above is consistent with the American Cancer Society study populations (Pope et al., 2002), on which the original CRFs for fine particulates and mortality were based, and which is therefore typical of the approach used to calculate baseline mortality rates within other quantitative air quality Health Impact Assessments and quantitative exposure response assessments.

3.1.2 Mortality statistics were collected and recorded at MSOA level (also from the Nomis online service). This represents a good level of granularity which provides a more representative indication of existing burdens of poor health in and around the ARN than at local authority or national level. This approach also avoids the complications of reporting of health effects at lower census levels with smaller sample sizes (i.e. those covering a smaller or less populated geographical area), for example LSOA level. It was found that a number of LSOAs did not have any deaths across the 2017-2019 baseline years selected and therefore the calculation would return a zero value regardless of the concentration change.

3.1.3 In addition, to avoid the influence of the COVID-19 pandemic and individual year-on-year variations, MSOA death rates and population data were averaged across a three-year period encompassing mid-2017, mid-2018 and mid-2019 projected census datasets.

3.1.4 The MSOA level mortality data (i.e. number of deaths from all causes) was used to determine the existing MSOA baseline rate (per 1,000 people) which fed into the calculation of the number of deaths brought forward, attributable to the change in pollutants from the Project.

3.1.5 To calculate the MSOA baseline rate of mortality, census data on the population of the MSOA was also acquired from Nomis. The MSOA baseline rate was then population-weighted relative to the relevant +30yr cohort of the population residing within the AQQHIA study area within a given OA. An example of the application of this method is summarised in Table 3.1 for those OAs which intersect the AQQHIA study area within Uttlesford District Council. The concept of calculating a baseline rate recorded at a larger geographical area (in this case at MSOA level) and population weighting to a smaller geographical (OA) level was replicated for the other health outcomes generated across the relevant cohorts of the population.

**Table 3.1 Indicative example of the application of baseline rate calculated at MSOA level to the AQQHIA Study Area; Uttlesford District Council**

| OAs which intersect AQQHIA study area | MSOA code | Baseline rate per 1,000 | Total population in OA | Deaths in OA per annum | Population within AQQHIA study area | Extrapolated deaths in OA within AQQHIA study area |
|---|---|---|---|---|---|---|
| E00169976 | Uttlesford 005 | 12.2 | 247 | 3.02 | 5 | 0.06 |
| E00112088 | Uttlesford 009 | 10.7 | 167 | 1.78 | 167 | 1.78 |
| E00112084 | | 10.7 | 216 | 2.30 | 19 | 0.20 |
| E00112086 | | 10.7 | 245 | 2.61 | 13 | 0.13 |
| E00112079 | | 10.7 | 267 | 2.84 | 267 | 2.84 |
| *MSOA Uttlesford 005: 9,314 population, 114 deaths per year.* | | | | | | |
| ***Baseline rate of mortality in Uttlesford 005: 12.2 per 1,000 people*** | | | | | | |
| *MSOA Uttlesford 009: 5,283 population, 56 deaths per year.* | | | | | | |
| ***Baseline rate of mortality in Uttlesford 009: 10.7 per 1,000 people*** | | | | | | |

3.1.6 As stated previously, this approach ensures a greater level of detail and variability within a given local authority than using a baseline rate calculated at the local authority or national level.

3.1.7 Table 3.2 below summarises the population-weighted baseline rate of mortality within the AQQHIA study area by local authority. This demonstrates that there are significant geographical variations in the death rate per 1,000 within the AQQHIA study area. For example, the death rate across the AQQHIA study area within Harlow is 7.0 per 1,000 people, whereas in Chelmsford the death rate is 18.9 per 1,000 people. There are a number of reasons which may explain the geographical variations in the death rate such as the age and socio-economic status of the residents.

3.1.8 Presenting the data in this manner demonstrates the value of using baseline rates calculated on a more localised level where possible.

**Table 3.2 Summary of study area baseline rates derived from MSOA level**

| Local Authority | Population within AQQHIA study area (+30yrs, 2017-2019 annual average) | Annual AQQHIA study area deaths (+30yrs, 2017-2019 average) | AQQHIA study area baseline rate of mortality per 1,000 people | Number of MSOAs intersecting AQQHIA study area | Number of OAs intersecting AQQHIA study area |
|---|---|---|---|---|---|
| **Barking & Dagenham** | 3,899 | 29 | 7.6 | 6 | 19 |
| **Basildon** | 6,960 | 95 | 13.6 | 11 | 60 |
| **Bexley** | 117 | 1 | 11.3 | 1 | 3 |
| **Brentwood** | 3,217 | 46 | 14.2 | 6 | 28 |
| **Broxbourne** | 788 | 8 | 10.7 | 1 | 10 |
| **Canterbury** | 1,394 | 25 | 17.7 | 5 | 19 |
| **Castle Point** | 737 | 10 | 13.4 | 4 | 15 |
| **Chelmsford** | 3,617 | 68 | 18.9 | 6 | 24 |
| **Dartford** | 11,925 | 151 | 12.6 | 12 | 103 |
| **Enfield** | 976 | 11 | 11.0 | 3 | 9 |
| **Epping Forest** | 2,450 | 34 | 13.9 | 8 | 26 |
| **Gravesham** | 15,140 | 215 | 14.2 | 12 | 136 |
| **Greenwich** | 4,623 | 37 | 8.0 | 7 | 42 |
| **Harlow** | 934 | 7 | 7.0 | 3 | 11 |
| **Havering** | 13,237 | 219 | 16.5 | 14 | 112 |
| **Maidstone** | 2,749 | 31 | 11.3 | 4 | 28 |
| **Medway** | 13,158 | 163 | 12.4 | 12 | 101 |
| **Rochford** | 6,150 | 79 | 12.8 | 4 | 47 |
| **Sevenoaks** | 1,444 | 19 | 13.5 | 9 | 33 |
| **Southend-on-Sea** | 3,151 | 55 | 17.6 | 4 | 30 |
| **Swale** | 6,405 | 79 | 12.4 | 8 | 39 |
| **Tandridge** | 708 | 11 | 15.9 | 3 | 4 |
| **Thurrock** | 31,069 | 360 | 11.6 | 19 | 231 |
| **Tonbridge and Malling** | 12,751 | 163 | 12.8 | 6 | 103 |
| **Uttlesford** | 471 | 5 | 10.7 | 2 | 5 |
| **TOTAL** | **148,070** | **1,921** | **13.0** | **170** | **1,238** |

## Mortality burden attributed to changes in $NO_2$ and $PM_{2.5}$

3.1.9 Total modelled pollutant concentrations within the OAs in the AQQHIA study area were modelled in order to generate estimates of the existing and future mortality burden attributable to $NO_2$ and $PM_{2.5}$ in the AQQHIA study area, for the following scenarios:

a. Base year (2016)

b. Do Minimum (2030, future baseline)

c. Do Something (2030, future baseline + the Project)

3.1.10 Effects of the change in $NO_2$ and $PM_{2.5}$ concentrations on mortality as a result of the operation of the Project in the AQQHIA study area were quantified.

3.1.11 As detailed in Table 2.1, the unadjusted coefficient of 1.023 per 10µg/$m^3$ was used to calculate the mortality burden for $NO_2$. For $PM_{2.5}$, an unadjusted coefficient of 1.08 per 10µg/$m^3$ was used. The difference in the mortality burden between the Do Something and the Do Minimum was then used to calculate the potential change in population health as a result of the Project in the AQQHIA study area. The results are summarised in Table 3.3.

**Table 3.3 Impact on mortality due to change in $NO_2$/$PM_{2.5}$ as a result of the operation of the Project**

| **Pollutant** | **$NO_2$** | | | **$PM_{2.5}$** | | |
|---|---|---|---|---|---|---|
| **Scenario** | **Do Minimum 2030** | **Do Something 2030** | **Change** | **Do Minimum 2030** | **Do Something 2030** | **Change** |
| **Mortality burden from air pollutant (deaths brought forward annually)** | 84.01 | 84.33 | +0.32 | 166.59 | 166.84 | +0.25 |
| **Change in mortality as a percentage of total existing deaths from all causes (three year annual average, 2017-2019)** | 0.017% | | | 0.013% | | |

3.1.12 Table 3.3 demonstrates the impact of the Project on mortality across the population residing within the AQQHIA study area. Changes in $NO_2$ concentrations are expected to lead to an effect equivalent to less than one (0.32) death brought forward across the population studied (approximately 148,000 people). Similarly, changes in $PM_{2.5}$ concentration also led to less than one death brought forward (0.25) per year. Therefore, in line with the recommendations from COMEAP discussed previously, the change in mortality associated with changes in $NO_2$ should be reported as proxy to represent the effect of the two pollutants in combination.

3.1.13 The increase in mortality from $NO_2$ is an effect equivalent to 0.32 additional deaths per year and represents a 0.017% increase in the baseline number of deaths that occurred in the population residing within the AQQHIA study area (1,921 baseline deaths per year in a population cohort aged =>30yrs of approximately 148,000 people). Therefore, as a fraction of the existing total deaths from all causes, a change equivalent to less than one death brought forward would not be measurable in terms of impact on public health amongst the annual natural variations in death rate from all causes (this is discussed further in Paragraph 3.1.14). The effect on mortality within the AQQHIA study area by local authority is detailed in Annex A.

3.1.14 The magnitude of the calculated impact on mortality can be put into context by analysing the historic death statistics which were utilised to inform the calculated baseline rates used in the assessment. The actual number of deaths from all causes recorded within the population residing in the AQQHIA study area in each of 2017, 2018, and 2019 shows that the standard deviation in deaths is 24 deaths per year (a standard variation of 1.25%) in this period (see Table 3.4); therefore the increase of 0.32 additional deaths from operation of the Project would not be discernible amongst the expected natural variation in deaths from all causes amongst the population studied.

**Table 3.4 Annual Standard Deviation in the number of deaths from all causes within the population resident in the AQQHIA study area (2017-2019)**

| Year | Deaths in population residing in AQQHIA study area |
|---|---|
| 2017 | 1,909 |
| 2018 | 1,954 |
| 2019 | 1,899 |
| **Standard deviation** | **24** |

## 3.2 Respiratory hospital admissions and cardiovascular hospital admissions – $NO_2$ & $PM_{2.5}$

3.2.1 Respiratory admissions (those admissions with a diagnosis categorised between International Classification of Disease (ICD) codes ICD J00-J99) have been calculated for $NO_2$ and $PM_{2.5}$. Cardiovascular admissions (ICD I00-I99) have been calculated for $PM_{2.5}$ only. COMEAP (2022b) advises that the relationship between cardiovascular admissions and $NO_2$ is subject to a higher level of uncertainty than those associated with particulate matter. In addition, the evidence available for plausible biological mechanisms for cardiovascular effects is greater for $PM_{2.5}$ than for $NO_2$.

3.2.2 Baseline rates for both respiratory and cardiovascular disease hospital admissions in England were derived from data recorded in the NHS Hospital Episode Statistics data warehouse (for each of 2017, 2018 and 2019) and consequently population-weighted to the population resident within the AQQHIA study area. It should be noted that all hospital admissions for respiratory admissions and cardiovascular admissions were used as the basis for defining the baseline rates, and not just emergency admissions; this is considered to be a conservative assumption.

3.2.3 It should be reiterated that the population studied for the effect on hospital admissions is the total population of all ages and not a particular age cohort. This is because the studies informing the CRFs for hospital admissions are based on studies encompassing effects at all ages. The baseline rates for England were population-weighted to the population within the AQQHIA study area and are presented in Table 3.5.

**Table 3.5 Population studied and baseline rates for respiratory and cardiovascular hospital admissions**

| | Respiratory admissions (2017-2019 annual average) | Cardiovascular admissions (2017-2019 annual average) |
|---|---|---|
| **AQQHIA study area population (all ages, 2017-2019 average)** | 235,290 | |
| **Baseline rate of health outcome per 1,000 population (England), per annum** | 19.0 | 16.7 |
| **Approximated annual hospital admissions in population included in the AQQHIA study area** | 4,473 | 3,931 |

3.2.4 Table 3.6 below summarises the impact of the Project on respiratory hospital admissions and highlights that impacts from $NO_2$ are greater than impacts induced by increases in $PM_{2.5}$; therefore, conclusions have been made by analysing the increase in respiratory admissions from $NO_2$.

**Table 3.6 Impact on respiratory hospital admissions due to change in $NO_2$/$PM_{2.5}$ as a result of the operation of the Project**

| Pollutant | $NO_2$ | | | $PM_{2.5}$ | | |
|---|---|---|---|---|---|---|
| **Scenario** | **Do Minimum 2030** | **Do Something 2030** | **Change** | **Do Minimum 2030** | **Do Something 2030** | **Change** |
| **Respiratory hospital admissions attributable to air pollutant in AQQHIA study area** | 52.05 | 52.20 | +0.15 | 51.29 | 51.35 | +0.06 |
| **Change in respiratory admissions as a percentage of total existing respiratory admissions (three-year annual average, 2017-2019)** | 0.003% | | | 0.001% | | |

3.2.5 Changes in $NO_2$ concentrations are expected to lead to an increase of 0.15 of a respiratory admission per year. This represents a 0.003% increase in the total number of respiratory admissions that occurred in the population residing within the AQQHIA study area (4,473 admissions across a population of approximately 235,000 people).

3.2.6 Table 3.7 below summarises the impact of the Project on cardiovascular hospital admissions from changes in $PM_{2.5}$ concentration. Changes in $PM_{2.5}$ concentrations are expected to lead to an increase of <0.1 of a cardiovascular admission per year. This represents a 0.001% increase in the total number of cardiovascular admissions that occurred in the population residing within the AQQHIA study area (3,931 admissions across a population of approximately 235,000 people).

**Table 3.7 Impact on cardiovascular hospital admissions due to the change in $PM_{2.5}$ as a result of the operation of the Project**

| **Pollutant** | **$PM_{2.5}$** | | |
|---|---|---|---|
| **Scenario** | **Do Minimum 2030** | **Do Something 2030** | **Change** |
| Cardiovascular hospital admissions attributable to air pollutant | 42.30 | 42.35 | +0.05 |
| Change in cardiovascular admissions as a percentage of total existing cardiovascular admissions (three-year annual average, 2017-2019) | 0.001% | | |

# 4 Conclusions

4.1.1 An AQQHIA has been carried out to establish the potential effect of the Project on mortality and cardiovascular and respiratory admissions within the population residing in the air quality study area. The assessment has been undertaken in accordance with the methodological principles published by COMEAP. The effects of the Project on mortality and hospital admissions were chosen for quantification as the epidemiological evidence base underpinning the CRFs informing these health outcomes is considered to be sufficiently reliable for quantification by COMEAP. Changes in both $NO_2$ and $PM_{2.5}$ concentration were modelled to explore the potential health outcomes directly attributable to the Project, and to completement the findings of Chapter 5 – Air Quality of 6.1 Environmental Statement [APP-143].

4.1.2 The impact from $NO_2$ was found be greater than $PM_{2.5}$ in terms of mortality (deaths brought forward) and respiratory hospital admissions as a result of larger changes in concentration and exposure. The magnitude of impact from $PM_{2.5}$ was less than $NO_2$ for mortality and respiratory admissions despite the higher CRF associated with $PM_{2.5}$; this was due to lower concentration changes and lower levels of exposure to $PM_{2.5}$. Impacts on cardiovascular hospital admissions were calculated based on the $PM_{2.5}$ concentrations.

4.1.3 The assessment concludes the impact of the Project on mortality, and respiratory and cardiovascular hospital admissions is negligible and can be considered neither measurable nor material in the context of existing levels of the health outcomes analysed. The impacts are orders of magnitude lower than is required to quantify any tangible adverse health outcome, and the net aggregate change was far less than 1% of the reported public health measures. There was no evidence for a measurable adverse or beneficial effect on population health. This is supported by the following points:

a. There is an increase of approximately 0.3 in the mortality burden as a result of the changes in $NO_2$ concentration caused by the operation of the Project. This represents an increase of <0.02% in relation to the annual average number of deaths from all causes recorded between 2017-2019 within the AQQHIA study area.

b. The annual standard deviation in deaths from all causes in the population considered in the AQQHIA between 2017-2019 was 24 deaths per annum (a standard variation of 1.25%) indicating that it would not be possible to separate the effect of the project on mortality in the opening year from natural variations.

4.1.4 The effect of the Project on morbidity outcomes was quantified in terms of the effect on respiratory hospital admissions ($NO_2$), and cardiovascular hospital admissions ($PM_{2.5}$). The effect is considered to be neither measurable nor material in the context of existing levels of the morbidity outcomes analysed:

a. $NO_2$ impacts on respiratory hospital admissions were negligible with an additional 0.15 admissions per year in the population residing within the AQQHIA study area. This represents an increase of 0.003% in relation to the annual average number of respiratory admissions recorded between 2017-2019 within the AQQHIA study area.

b. $PM_{2.5}$ impacts on cardiovascular hospital admissions were negligible with <0.1 additional admissions per year in the population residing within the AQQHIA study area. This represents an increase of 0.001% in relation to the annual average number of cardiovascular admissions recorded between 2017-2019 within the AQQHIA study area.

# Annex A Mortality Burden by Local Authority ($NO_2$)

| Local authority | Population studied (those residing within AQQHIA study area) | Unique OAs | Mortality Burden attributable to $NO_2$ | | | |
|---|---|---|---|---|---|---|
| | | | Base Year 2016 | Do Minimum 2030 | Do Something 2030 | Change (Do Something minus Do Minimum) |
| Barking and Dagenham | 3,899 | 19 | 2.39 | 1.79 | 1.79 | 0.00 |
| Basildon | 6,960 | 60 | 5.72 | 4.30 | 4.32 | 0.02 |
| Bexley | 117 | 3 | 0.09 | 0.07 | 0.07 | 0.00 |
| Brentwood | 3,217 | 28 | 2.23 | 1.66 | 1.66 | 0.00 |
| Broxbourne | 788 | 10 | 0.52 | 0.39 | 0.39 | 0.00 |
| Canterbury | 1,394 | 19 | 0.84 | 0.62 | 0.62 | 0.00 |
| Castle Point | 737 | 15 | 0.55 | 0.41 | 0.41 | 0.00 |
| Chelmsford | 3,617 | 24 | 2.92 | 2.14 | 2.14 | 0.00 |
| Dartford | 11,925 | 103 | 9.42 | 7.15 | 7.08 | -0.07* |
| Enfield | 976 | 9 | 0.67 | 0.51 | 0.50 | 0.00 |
| Epping Forest | 2,450 | 26 | 1.71 | 1.27 | 1.28 | 0.00 |
| Gravesham | 15,140 | 136 | 12.96 | 9.78 | 9.82 | 0.04 |
| Greenwich | 4,623 | 42 | 3.45 | 2.56 | 2.56 | 0.00 |
| Harlow | 934 | 11 | 0.27 | 0.20 | 0.20 | 0.00 |
| Havering | 13,237 | 112 | 13.52 | 10.14 | 10.19 | 0.05 |
| Maidstone | 2,749 | 28 | 1.71 | 1.28 | 1.29 | 0.00 |
| Medway | 13,158 | 101 | 9.66 | 7.31 | 7.42 | 0.12 |

| Local authority | Population studied (those residing within AQQHIA study area) | Unique OAs | Mortality Burden attributable to $NO_2$ | | | |
|---|---|---|---|---|---|---|
| | | | Base Year 2016 | Do Minimum 2030 | Do Something 2030 | Change (Do Something minus Do Minimum) |
| Rochford | 6,150 | 47 | 4.48 | 3.34 | 3.35 | 0.02 |
| Sevenoaks | 1,444 | 33 | 0.85 | 0.64 | 0.63 | 0.00 |
| Southend-on-Sea | 3,151 | 30 | 2.92 | 2.15 | 2.15 | 0.00 |
| Swale | 6,405 | 39 | 3.16 | 2.33 | 2.33 | 0.01 |
| Tandridge | 708 | 4 | 0.53 | 0.40 | 0.40 | 0.00 |
| Thurrock | 31,069 | 231 | 21.95 | 16.54 | 16.66 | 0.12 |
| Tonbridge and Malling | 12,751 | 103 | 9.11 | 6.89 | 6.90 | 0.01 |
| Uttlesford | 471 | 5 | 0.22 | 0.16 | 0.16 | 0.00 |

**Minus values represent a reduction in the mortality burden as a result of decreases in pollutants concentrations within the specified locality.*

*Note:*

*Changes in the mortality burden have been calculated based on the unrounded absolute changes. The table above presents the changes rounded to two decimal places and therefore rounding inconsistencies may occur.*

*Slight inconsistencies occur if disaggregated local authority results are summed – this is due to the vales in the table above being rounded to two decimal places. The calculations presented in the assessment were based on the absolute unrounded changes.*

# Annex B AQQHIA Data Sources

## B.1 Population data at output area

| Name of Dataset | Link to Data source |
|---|---|
| Table SAPE20DT10h: Mid-2017 Population Estimates for Census Output Areas in the East region of England by Single Year of Age and Sex, Persons - Supporting Information | Census Output Area population estimates – England East of England |
| Table SAPE21DT10h: Mid-2018 Population Estimates for Census Output Areas in the East region of England by Single Year of Age and Sex, Persons - Supporting Information | |
| Table SAPE22DT10h: Mid-2019 Population Estimates for Census Output Areas in the East region of England by Single Year of Age and Sex, Persons - Supporting Information | |
| Table SAPE20DT10a: Mid-2017 Population Estimates for Census Output Areas in the London region of England by Single Year of Age and Sex, Persons - Supporting Information | Census Output Area population estimates – London, England |
| Table SAPE21DT10a: Mid-2018 Population Estimates for Census Output Areas in the London region of England by Single Year of Age and Sex, Persons - Supporting Information | |
| Table SAPE22DT10a: Mid-2019 Population Estimates for Census Output Areas in the London region of England by Single Year of Age and Sex, Persons - Supporting Information | |
| Table SAPE20DT10i: Mid-2017 Population Estimates for Census Output Areas in the South East region of England by Single Year of Age and Sex, Persons - Supporting Information | Census Output Area population estimates – South East, England |
| Table SAPE21DT10i: Mid-2018 Population Estimates for Census Output Areas in the South East region of England by Single Year of Age and Sex, Persons - Supporting Information | |
| Table SAPE22DT10i: Mid-2019 Population Estimates for Census Output Areas in the South East region of England by Single Year of Age and Sex, Persons - Supporting Information | |

## B.2 MSOA level mortality data

B.2.1 Nomis (ONS, 2023) was used to ascertain MSOA mortality data 2017-2019 for all causes, ages 30 and above.

## B.3 Hospital admissions data (England)

| Year | Source |
|---|---|
| 2016/2017 | NHS Digital: Hospital Admitted Patient Care Activity 2016-2017: Diagnosis |
| 2017/2018 | NHS Digital: Hospital Admitted Patient Care Activity 2017-2018: Diagnosis |
| 2018/2019 | NHS Digital: Hospital Admitted Patient Care Activity 2018-2019: Diagnosis |

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Pope, C.A. III, Burnett, R.T., Thun, M.J., Calle, E.E., Krewski, D., Ito, K. and Thurston, G.D (2002). Lung Cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA, 287(9), pp. 1132-1141

# Glossary

| Term | Abbreviation | Explanation |
|---|---|---|
| **Atmospheric Dispersion Modelling System** | **ADMS** | A comprehensive software-based modelling tool for air quality developed by Cambridge Environmental Research Consultants (CERC) |
| **Air Quality Quantitative Health Impact Assessment** | **AQQHIA** | The assessment which quantifies mortality and hospital admissions in the operational phase of the Project as a result of changes in air pollution. |
| **Application Document** | | In the context of the Project, a document submitted to the Planning Inspectorate as part of the application for development consent. |
| **Attributable deaths** | | Long-term exposure to anthropogenic particulate air pollution or to nitrogen dioxide is estimated to have an effect on mortality risks equivalent to the number of attributable deaths. Air pollution is likely to contribute a small amount to the deaths of a larger number of exposed individuals rather than being solely responsible for the number of deaths equivalent to the calculated figure of attributable deaths. |
| **Attributable fraction** | **AF** | The proportion of deaths estimated as due to long-term exposure to anthropogenic particulate air pollution or to nitrogen dioxide. |
| **Cardiovascular** | | Relating to the heart and circulation. Includes stroke and problems with arteries or veins in other parts of the body not just the heart. |
| **Construction** | | Activity on and/or offsite required to implement the Project. The construction phase is considered to commence with the first activity on site (e.g. creation of site access), and ends with demobilisation. |
| **Committee on the Medical Effects of Air Pollutants** | **COMEAP** | An expert group providing independent advice to government departments and agencies on how air pollution impacts on health. |
| **Deaths brought forward** | | Term used by COMEAP to denote the fact that the increased deaths detected in time-series studies of daily exposure to air pollutants are not additional deaths but deaths occurring at an earlier time than expected, perhaps by only a short time. |
| **Design Manual for Roads and Bridges** | **DMRB** | A comprehensive manual containing requirements, advice and other published documents relating to works on motorway and all-purpose trunk roads for which one of the Overseeing Organisations (National Highways, Transport Scotland, the Welsh Government or the Department for Regional Development (Northern Ireland)) is highway authority. For the A122 Lower Thames Crossing the Overseeing Organisation is National Highways. |
| **Development Consent Order** | **DCO** | Means of obtaining permission for developments categorised as Nationally Significant Infrastructure Projects (NSIP) under the Planning Act 2008. |
| **Development Consent Order application** | **DCO application** | The Project Application Documents, collectively known as the 'DCO application'. |
| **Environmental Statement** | **ES** | A document produced to support an application for development consent that is subject to Environmental Impact Assessment (EIA), which sets out the likely impacts on the environment arising from the proposed development. |

| Term | Abbreviation | Explanation |
|---|---|---|
| **Highways England** | | Former name of National Highways. |
| **International Classification of Disease** | **ICD** | ICD codes are numerical codes defined by WHO to ensure a common understanding of exact disease definitions. ICD11 is the 11th revision of the ICD codes. |
| **National Highways** | | A UK government-owned company with responsibility for managing the motorways and major roads in England. Formerly known as Highways England. |
| **Morbidity** | | The condition of suffering from a disease or medical condition |
| **Mortality** | | Relating to death. |
| **Nitrogen dioxide** | **$NO_2$** | An air pollutant created by combustion |
| **Operation** | | Describes the operational phase of a completed development and is considered to commence at the end of the construction phase, after demobilisation. |
| **Particulate matter <2.5µm** | **$PM_{2.5}$** | Mass per cubic metre of particles passing through the inlet of a size selective sampler with a transmission efficiency of 50% at an aerodynamic diameter of 2.5 micrometres |
| **Respiratory** | | Relating to the lungs |
| **Relative risk** | **RR** | The ratio of the probability of the event occurring in the exposed group compared with the non-exposed group. In the case of air pollution, where exposure is ubiquitous, it is the ratio of the probability of the event occurring in groups with higher and lower exposure. The relative risks here are expressed, conceptually, in terms of a 10 µg/$m^3$ greater concentration in the higher compared with the lower exposure group. For the results of long-term exposure (mortality burden) the ratio refers to the ratio of the age-specific death rates (assuming other factors are equal) |
| **World Health Organization** | **WHO** | A specialised agency of the United Nations that is concerned with international public health. |

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