APPENDIX 7B: HUMAN HEALTH RISK ASSESSMENT

EP UK Investments

South Humber Bank Energy Centre Project

Planning Inspectorate Reference: EN010107

South Marsh Road, Stallingborough, DN41 8BZ

The South Humber Bank Energy Centre Order

Document Ref: 6.4 Environmental Statement – Volume III Appendix 7B: Human Health Risk Assessment

The Infrastructure Planning (Environmental Impact Assessment) Regulations 2017 (as amended)

The Infrastructure Planning (Applications: Prescribed Forms and Procedure)Regulations 2009 - Regulation 5(2)(a)

Applicant: EP Waste Management Ltd
Date: April 2020

DOCUMENT HISTORY

Document Ref	Appendix 7B Human Health Risk Assessment (HHRA)		
Revision	1.0		
Author	M. Hill		
Signed		**Date**	April 2020
Approved By	G. Gray		
Signed		**Date**	April 2020
Document Owner	AECOM		

CONTENTS

TABLES

ANNEXES

1.0 INTRODUCTION

1.1.1 This Appendix to the Air Quality Assessment (Chapter 7: Air Quality in ES Volume I, Document Ref. 6.2) is an assessment of the risk of effects on human health arising from changes in air quality from associated with the operation of the proposed South Humber Bank Energy Centre (the Proposed Development). For a full description of the Proposed Development refer to Chapter 4: The Proposed Development in ES Volume I (Document Ref. 6.2).

1.1.2 Full planning permission was granted by North East Lincolnshire Council (NELC) for an energy from waste power station with a gross electrical output of up to 49.9 MW and associated development (the Consented Development) on land at South Humber Bank Power Station (SHBPS) under the Town and Country Planning Act 1990 on 12 April 2019. A Human Health Risk Assessment (HHRA) was prepared in support of the Consented Development planning application. The Proposed Development air emissions will be the same as the Consented Development air emissions, so the conclusions of the HHRA for the Proposed Development are consistent with the previous conclusions of the HHRA for the Consented Development.

1.1.3 The study area for the HHRA extends 10 km from the Site and includes parts of North East Lincolnshire, North Lincolnshire, East Riding of Yorkshire and West Lindsey District. This area of interest is referred to in this report as the Study Area.

1.1.4 The potential health effects associated with emissions to air from the Proposed Development have been assessed assuming emissions at concentrations no greater than those included within the Industrial Emissions Directive (IED) 2010/75/EU (EU, 2010). The IED entered into force on 7th January 2011 and incorporated a number of directives, including the previous Waste Incineration Directive (WID) (EC, 2000), into a single overall directive. All European Union (EU) member states are required to transpose the directive into national legislation within two years, including the UK as a member at the time the Directive entered into force. The emission limit values and operating conditions specified within WID have been retained within the IED and will continue to be applied in respect of any installation in England from 6 January 2013 until revised Best Available Techniques Achievable Emission Levels (BAT-AELs) are published in the relevant EU BAT Reference document (BREF Note), which may be tighter than IED Emission Limits. In practice, the mitigation employed to ensure compliance with permitted emission rates will deliver lower levels of emissions over the many operational hours throughout the life time of a plant.

1.1.5 The European Joint Research Centre (JRC) has published a revised BREF for the Waste Incineration section (JRC, 2017) in draft form. The BREF considers the current best practice emission controls across the European Union and includes emission limits that are expected to be reached by modern waste incineration plants. The Environment Agency (EA) has also conducted a review of group 3 metals emissions from municipal waste incineration facilities in the UK (EA, 2016). The emission limits set out in the BREF and the EA guidance have been used in this assessment.

1.1.6 The health effects associated with exposure to air pollutants has been considered at the population level and in terms of the potential effect on hypothetical individuals experiencing maximum levels of exposure. These different elements of the assessment require the application of distinct assessment methods and are reported here as separate sections of the report.

1.1.7 Section 2 provides an overview of how the magnitude of the predicted concentrations of particulate matter, nitrogen dioxide, sulphur dioxide, metals and organic substances have been estimated using dispersion modelling techniques. The current health of the population of the area surrounding the Proposed Development is summarised in Section 3. The predicted annual mean pollutant concentration values for one of the inputs to the assessment of population level health effects are discussed in Section 4. Finally, the potential for the Proposed Development to affect the total risk for carcinogenic and non-carcinogenic effects occurring is considered in Section 5.

2.0 ESTIMATED POLLUTION CONCENTRATIONS

2.1 Overview

2.1.1 One of the required data inputs to the assessment is the predicted change in annual mean concentrations of particulate matter (PM_{10} and $PM_{2.5}$), sulphur dioxide (SO_2), nitrogen dioxide (NO_2), metals and organic substances across the assessment domain, due to the operation of the Proposed Development. In this instance, the dispersion model outputs have been taken from an ADMS model used to assess the air quality impacts of the Proposed Development as outlined in Chapter 7: Air Quality in ES Volume I (Document Ref. 6.2) and Appendix 7A in ES Volume III (Document Ref. 6.4), based on the design of the Proposed Development. The results have been provided as a spatial output for use with the Geographical Information System (GIS) and human health modelling software.

2.1.2 This section provides a summary of the inputs to the dispersion model.

Dispersion Model Setup

2.1.3 The assessment of emissions from the main stacks serving the Proposed Development has been undertaken using ADMS 5.2, supplied by Cambridge Environmental Research Consultants Limited. ADMS is a modern dispersion model that has an extensive published validation history for use in the UK (CERC, 2017). This model has been extensively used throughout the UK to demonstrate regulatory compliance.

2.1.4 The physical properties of the main stacks and the emissions data for input to the model were provided by EPWM Ltd. The modelled pollutant emission rates (in g/s) are equivalent to the emission limits set out within Annex VI of the Industrial Emissions Directive, amended for the BREF BAT-AELs as appropriate, and have been calculated by multiplying the relevant daily average emission limit concentrations by the volumetric flow rate. The data is based on 100% Maximum Continuous Rating (MCR) case when firing the design fuel.

2.1.5 The meteorological site that was selected for the assessment was Humberside Airport Meteorological station, located approximately 13 km west of the Site, in flat terrain (see Chapter 7 in ES Volume I, Document Ref. 6.2). The modelling for this assessment has utilised meteorological data for the period 2013 - 2017, with 2015 providing the worst-case results for long term impacts. The datasets were supplied by ADM Ltd, the UK agent for Trinity Consultants. The dispersion modelling output for each pollutant from this year were used as input for the GIS and health modelling software.

2.1.6 The Site is located to the north-west of Grimsby adjacent to the existing SHBPS on the south bank of the Humber Estuary. The area is a mix of open agricultural land and industrial developments. A surface roughness of 0.1 m, corresponding agricultural land, has been selected to represent the local terrain.

2.1.7 Emissions of NOx from the main stacks serving the Proposed Development will consist mainly of nitric oxide (NO) at the point of release, oxidising within the atmosphere to form NO_2 as it moves downwind. The modelling assessment has assumed a 70% NOx to NO_2 conversion rate at ground level in the

calculation of long-term annual mean calculations. Emissions have been modelled such that they are not subject to dry and wet deposition or depleted through chemical reactions. This results in an over-estimation of impacts at receptors.

Receptor Grid

2.1.8 The contribution of emissions from the main stacks serving the Proposed Development to ambient concentrations of pollutants have been modelled at points forming a Cartesian grid, in order to enable the generation of the spatial model output required for use with the GIS and health modelling software. A variable resolution grid was used in order to provide a higher resolution in the immediate area surrounding the Site. The receptor grid is centred on the main stacks, the details are presented in Table 2.1. The grid extends to 10 km from the stacks in all directions. The height of receptors within the grid was set at 1.5 m for impacts through respiration or at 0 m for impacts through other routes.

Table 2.1: Modelled Domain: Variable Receptor Grid

SPACING (M)	DIMENSIONS (M)	NATIONAL GRID REFERENCE OF SW CORNER OF RECEPTOR GRID
12.5	1,200 x 1,200	522571.7, 412870.9
50	4,800 x 4,800	520771.7, 411070.9
200	20,000 x 20,000	513171.7, 403470.9

3.0 BASELINE LOCAL HEALTH CONDITIONS

3.1.1 Health profiles are produced annually by the Association of Public Health Observatories (APHO), now part of Public Health England (PHE), and these provide a summary of the health of people within defined areas and a comparison of local health with average values for all areas of England. Health profiles have been obtained for the local authority areas of North East Lincolnshire (PHE, 2020a), North Lincolnshire (PHE, 2020b), West Lindsey District (PHE, 2020c), East Riding of Yorkshire (PHE, 2020d), and Lincolnshire County Council (PHE, 2020e) (as the non-metropolitan county authority for the area including West Lindsey District).

Table 3.1: Life Expectancy

COMMUNITY	FEMALE AVERAGE (YEARS[A])	MALE AVERAGE (YEARS[A])
England	83.2	79.6
North East Lincolnshire	82.2	77.6
North Lincolnshire	82.4	79.0
West Lindsey	83.5	79.6
East Riding of Yorkshire	83.8	80.1
Lincolnshire	82.9	79.2

[a] Values at birth (2016 - 2018) sourced from the Health Profile for the individual local authority

3.1.2 Local average life expectancy for people living within each local authority is similar to the national average (see Table 3.1), with North East Lincolnshire, North Lincolnshire and Lincolnshire all slightly below the national average, and West Lindsey and East Riding of Yorkshire slightly above the national average. There are various factors that may contribute to a lower or higher life expectancy, including life style, income, and behavioural related factors (such as smoking and diet) and environmental factors (such as air pollution).

3.1.3 There are well documented health inequalities between individual areas within each local authority. The most deprived areas within the NELC administrative area have an average life expectancy for men that is 13.1 years shorter than for men in the least deprived areas, and 9.1 years for women (based on the Inequality in life expectancy at birth (PHE, 2020a)). In the North Lincolnshire Council (NLC) administrative area, the gap in life expectancy between the most deprived and the least deprived for men is 9.7 years and for women it is 9.1 years. The Site is located in North East Lincolnshire, and NELC and NLC possess the greatest gaps in life expectancies within the Study Area.

Table 3.2: Baseline Mortality Rates

COMMUNITY	HEALTH OUTCOME PER 100,000 POPULATION[A]				INFANT DEATHS[D]
	DEATHS: ALL CAUSES[B]	EARLY DEATHS: HEART DISEASE AND STROKE[B]	EARLY DEATHS: CANCER[B]	ROAD INJURIES AND DEATHS[C]	
England	330.5	71.7	132.3	42.6	3.9
North East Lincolnshire	399.4	88.2	162.6	53.6	4.85
North Lincolnshire	257.2	72.3	144.1	64.0	3.72
West Lindsey	310.9	70.4	128.8	94.7	3.4
East Riding of Yorkshire	297.3	64.9	122.4	63.0	2.0
Lincolnshire	339.6	78.2	132.5	97.4	3.0

[a] sourced from the Health Profile for the individual local authority.
[b] values expressed as directly age standardised rate per 100,000 population under 75, 2016-2018.
[c] values expressed as rate per 100,000 population 2016-2018.
[d] rate per 1,000 live births 2016-2018 sourced from the Health Profile for the individual local authority.

3.1.4 Similar differences in the average male life expectancy were found between the most and least deprived areas of the local authority areas of North East Lincolnshire (13.1 years), North Lincolnshire (9.7 years), West Lindsey (7.7 years), East Riding of Yorkshire (6.3 years), and Lincolnshire (8.2 years) (based on the Inequality in life expectancy at birth published in the Health Profile for each administrative area). Both the male and female average life expectancy values for all the local authority areas shown in Table 3.1 are within approximately 2.5 years of the average life expectancy for males and females in England as a whole.

3.1.5 The health outcomes for people living in the different local authority areas of the region set out in Table 3.2 are contrasted against the England average and considered for each administrative area in turn in the following sections for each administrative area.

3.1.6 An annual report on the health of the local population is undertaken on each administrative area in combination with the local National Health Service (NHS). This used to take the form of an annual report by the director of public health for the area but these are being gradually replaced by a Joint Strategic Needs Assessment (JSNA) report on the health and well-being of the local population. The health of the local population living within each local authority area in the region is discussed in the following sections.

3.2 Administrative Area of North East Lincolnshire Council

3.2.1 Performance against various indicators of health for people living in North East Lincolnshire (NEL) is generally worse that the England average (PHE, 2018a).

3.2.2 The JSNA (NELC, 2018) and Joint Health and Wellbeing Strategy (JHWS) (NELC, 2016) have been prepared by NELC and the North East Lincolnshire Clinical Commissioning Group (NELCCG), and covers North East Lincolnshire. North East Lincolnshire has higher levels of deprivation compared to the national average, with over 38% of the population living in the bottom quintile (fifths) of the Index of Multiple Deprivation 2019 (PHE, 2020a). North East Lincolnshire is one of the 20% most deprived areas in England, and about 7,815 children under the age of 16 (26%) live in low income families. The areas of highest deprivation (those within the lowest quintile) within NELC's administrative area are in the East Marsh, South, Sidney Sussex, Freshney, Heneage, West Marsh, Immingham, Croft Baker, Yarborough and Park wards, predominantly in Grimsby.

3.2.3 The Public Health Outcomes Framework (PHE, 2020f) gives the fraction of mortality attributable to particulate air pollution is 5.0% of all mortality in the authority in persons aged 30 years or over, slightly lower than the national average of 5.2%, and higher than the regional (Yorkshire and Humber) average of 4.5%.

3.2.4 Early deaths from all causes have decreased for both men and women over the past 15 years, however it still remains higher than the England average. Many indicators are significantly worse than the England average, including child poverty, teenage pregnancy, smoking prevalence and obesity in both adults and children.

3.2.5 Many of the indices used in the health profile for the area are worse than the national average, and the JHWS identifies priorities to tackle many of the health inequalities within the area. These priorities include reducing smoking, improving screening and early detection of illness, tackling drug and alcohol misuse, and developing healthy habits and lifestyles.

3.2.6 The Director of Public Health for North East Lincolnshire has published an Annual Report (NEL, 2019) which provides an overview of the issues contributing to health inequalities in the district. The report outlines issues such as better access to green spaces and nature, sustainable design, and establishing healthy lifestyle such as reducing smoking and more active travel. The report mentions air quality, and the need for improvement, and encourages the local authority to maintain a focus on improving air quality.

3.3 Administrative Area of North Lincolnshire Council

3.3.1 Performance against various indicators of health for people living in North Lincolnshire is generally mixed when compared to the England average (PHE, 2019b).

3.3.2 The JSNA (NLC, 2013) and Joint Health and Wellbeing Strategy (JHWS) (NLC, 2016) have been prepared by NLC and the North Lincolnshire Clinical Commissioning Group (NLCCG), and covers North Lincolnshire. North

Lincolnshire has similar levels of deprivation compared to the England average, with a slightly lower proportion of the population living in the top two quintiles of the Index of Multiple Deprivation 2019 (PHE, 2020b), although a lower proportion of the population live in the top quintile, while the bottom quintile is similar to the England average. However, about 5,655 children under the age of 16 (18.7%) are from low income families. The areas of highest deprivation (those within the lowest quintile) within NLC's administrative area are in the Brumby, Crosby and Park, Frodingham, Town, Ashby, Kingsway with Lincoln Gardens, Burthon upon Stather and Winterton, and Barton wards, predominantly in Scunthorpe.

3.3.3 The Public Health Outcomes Framework (PHE, 2020f) gives the fraction of mortality attributable to particulate air pollution as 4.9% of all mortality in the authority in persons aged 30 years or over, slightly lower than the national average of 5.2%, and higher than the regional (Yorkshire and Humber) average of 4.5%.

3.3.4 Early deaths from all causes have decreased for both men and women over the past 15 years, however it still remains slightly higher than the England average. Many indicators are significantly worse than the England average, including prevalence of smoking (as adults, at time of delivery/ birth and in routine and manual occupations), physically active adults and excess weight in adults.

3.3.5 The JHWS focuses on 5 priority actions, including a focus on the 'best start' from conception to 2 years of age, addressing poverty and reducing the impact on people, improving literacy (including health literacy) and numeracy skills, improving the safety and vibrancy of the night time economy and advocating and modelling behaviour change. NLC also published an Annual Public Health Report (NLC, 2018). These reports focus on the main health priorities in the area, including smoking, mental health, and healthy lifestyles.

3.4 Administrative Area of West Lindsey District Council

3.4.1 Performance against various indicators of health for people living in West Lindsey District is varied when compared to the England average (PHE, 2020c).

3.4.2 West Lindsey District Council (WLDC) works with the Lincolnshire West Clinical Commissioning Group (LWCCG) through the Health and Wellbeing Board of Lincolnshire County Council (LCC), as the unitary authority, to produce the JSNA (LCC, 2017) and the JHWS (LCC, 2018), which cover the county of Lincolnshire. West Lindsey District has similar levels of deprivation compared to the England average, with a lower proportion of the population living in the lowest quintile of the Index of Multiple Deprivation 2019 (PHE, 2020c). However about 2,945 children under the age of 16 (19.4%) live in low income families. The areas of highest deprivation (those within the lowest quintile) within WLDC's administrative area in the Gainsborough South-West, Gainsborough East, and Gainsborough North wards, all in Gainsborough.

3.4.3 The Public Health Outcomes Framework (PHE, 2020f) gives the fraction of mortality attributable to particulate air pollution as 4.9% of all mortality in the authority in persons aged 30 years or over, slightly lower than the national

average of 5.2%, and the same as the regional (East Midlands) average of 4.9%.

3.4.4 Early deaths from all causes have decreased for both men and women over the past 15 years, and are slightly lower than the England average. There are a few indicators that are significantly worse than the England average: killed and seriously injured on roads, smoking status at time of delivery, and children in low income families. The majority of indicators are similar to the England average, or are not significantly different.

3.4.5 Based on the outcomes of the JSNA, the JHWS has 7 priorities for improving the health of the residents of Lincolnshire: mental health and emotional wellbeing (children and young people), mental health (adults), carers, physical activity, housing and health, obesity and dementia.

3.5 Administrative Area of East Riding of Yorkshire Council (ERYC)

3.5.1 Performance against various indicators of health for people living the East Riding of Yorkshire is generally better than the England average (PHE, 2020d).

3.5.2 The JSNA (ERYC, 2020a) and the JHWS (ERYC, 2020b) have been prepared by ERYC and the East Riding of Yorkshire Clinical Commissioning Group (ERYCCCG) and covers the East Riding. The East Riding has significantly lower levels of deprivation compared to the England average, with over 50% of the population living in the top two quintiles of the Index of Multiple Deprivation 2019 (PHE, 2020d). Less than 25% of the population live in the bottom two quintiles. However about 6,370 children under the age of 16 (12.2%) live in low income families. The areas of highest deprivation (those within the lowest quintile) within ERYC's administrative authority area are in the Bridlington South, South East Holderness, Bridlington Central and Old Town, Goole South, Minster and Woodmansey, and East Wolds and Coastal. These areas are within Bridlington, Beverley, Withersea and Goole.

3.5.3 The Public Health Outcomes Framework (PHE, 2020f) gives the fraction of mortality attributable to particulate air pollution as 4.5% of all mortality in the authority in persons aged 30 years or over, lower than the national average of 5.3%, and the same as the regional (Yorkshire and Humber) average of 4.5%.

3.5.4 Early deaths from all causes have decreased for both men and women over the past 15 years, and are slightly lower than the England average. There are two indicators that are significantly worse than the England average: killed and seriously injured on roads, and smoking status at time of delivery. The majority of indicators are better than the England average, or are not significantly different.

3.5.5 The JSNA and JHWS provide a guide to the health of the population in the East Riding of Yorkshire, and the areas they seek to improve. These documents focus on mental health, dementia support, tackling child poverty, and helping people develop healthy lifestyles from childhood through adulthood to the elderly through exercise, healthy eating and mental health. The Director of Public Health had published an Annual Report (ERYC, 2018) which provides an outline on health issues and a direction for improvement. This report highlights a number of topics including physical activity, social involvement, smoking

during pregnancy, obesity, and sexual health, but also addresses the issue of air pollution and its effect on health. The report encourages partner bodies to consider the health effects of air pollution in development plans and policies, while acknowledging that air pollution is not a major concern within the area.

3.6 Summary

3.6.1 The predicted health effects in the assessment of exposure to particulate matter, nitrogen dioxide and sulphur dioxide is considered in the context of observed rates of disease and observed life expectancies on a national level. The methods used in this assessment could make use of either national statistics or local level statistics if such data exists. In this assessment national level statistics have been used, as there are benefits to determining baseline population disease rates on statistics that represent larger numbers of people. There may be differences in the values for the statistical parameters used between the local and national level datasets, but the associated difference in the calculated health effects under consideration would be small.

3.6.2 The assessment of health effects arising from the exposure to metals and organic substances associated with emissions to air from the Proposed Development calculates the additional risk of developing carcinogenic and non-carcinogenic health effects for individual receptors within the potentially exposed population.

3.6.3 The priority action areas for improving the health of people within each local authority area focus on bringing forward changes to the policies on the social determinants of health namely, mental health, smoking and obesity. The four local authorities within the region (NELC, NLC, WLDC and ERYC) have no specific priority policies for improving the health of the local population by targeting a reduction in air pollution.

4.0 POTENTIAL FOR HEALTH EFFECTS FROM EXPOSURE TO PARTICULATE MATTER, NITROGEN DIOXIDE AND SULPHUR DIOXIDE

4.1.1 An assessment of the potential effects on human health due to the operation of the Proposed Development has been carried out with respect to the predicted change in population exposure to particulate matter, sulphur dioxide and nitrogen dioxide (refer to Annex 7B.1 of this report). This report applies approaches to the quantification of health effects from predicted pollutant concentrations published by the Department of Health's Committee on the Medical Effect of Air Pollutants (COMEAP) and the Clean Air for Europe (CAFE) programme.

4.1.2 The total population of an area extending 10 km from the location of the Proposed Development was considered in the assessment of acute effects associated with exposure to particulate matter, nitrogen dioxide and sulphur dioxide. The same total population was also used in the assessment of mortality effects associated with chronic exposure to fine particulate matter.

4.1.3 The assessment concluded that, for each pollutant under consideration, the effect of the Proposed Development emissions of particulate matter (PM_{10} and $PM_{2.5}$), nitrogen dioxide and sulphur dioxide on human health would be relatively small.

4.1.4 The main outcomes of the study are as follows:

- For cardiovascular and respiratory health effects, the effect of each pollutant considered is:
  - an average of 35 minutes of life lost as a result of the Proposed Development for the male population and 11 minutes for the female population for nitrogen dioxide alone;
  - an average of 16 minutes of life lost as a result of the Proposed Development for the male population and 5 minutes for the female population for particulate matter alone; and
  - an average of 31 minutes of life lost as a result of the Proposed Development for the male population and 15 minutes for the female population for the combination of nitrogen dioxide and particulate matter using mutually adjusted coefficients.
- In comparison, the results published by COMEAP (2018) predict the mortality effects of long term exposure to air pollution to be equivalent to 28,000 – 36,000 deaths in the UK associated with a loss of total population life of 328,000 – 416,000 years. RCPCH and RCP reported that the total mortality burden due to outdoor air pollution is of the order of 40,000 early deaths per year, with a cost to the economy of over £20 billion per year.
- The estimated number of extra chronic bronchitis events, associated with the predicted change in concentration of particulate matter in the study area, is 0.034 per annum, which represents an increase of 0.0029% on the corresponding baseline rates for the entire exposed population. Additional

cases of hospital admissions for cardiovascular and respiratory symptoms are predicted to rise by 0.005 per annum each. The estimated increase in the occurrence of lower respiratory symptoms in children is 0.008 per annum, which represents a 0.00002% increase on baseline rates. This can be considered as an insignificant effect on the health of the exposed population and the health care system as a whole.

- Rates of hospital admissions for cardiovascular symptoms, associated with the predicted change in concentration of nitrogen dioxide in the Study Area, are estimated to increase by 0.102 per annum which represents an increase of 0.005% on the corresponding baseline rates for the entire exposed population. This is considered insignificant when compared to the total incidence of heart disease in the entire population of England, attributable to factors such as diet and lifestyle. Hospital admissions for respiratory symptoms are predicted to increase by 0.001% on a baseline rate of 1,156 admissions per year. The predicted impact for the measure 'death brought forward' is an increase of 0.001% on a baseline rate of 1,140 deaths brought forward per annum.
- The predicted impact for measured deaths brought forward, associated with the predicted change in concentrations of sulphur dioxide in the Study Area, is an increase of 0.0015% on a baseline rate of 1,140 deaths brought forward per annum. Rates of hospital admissions for respiratory symptoms are estimated to increase by 0.015 per annum, which represents a 0.000013% increase on baseline rates. This can be considered as an insignificant effect on the health of the exposed population and the health care system as a whole.

5.0 HEALTH EFFECTS ARISING FROM EMISSIONS OF METALS AND ORGANIC COMPOUNDS.

5.1.1 An assessment of the potential effects on human health due to the operation of the Proposed Development has been carried out with respect to the predicted change in population exposure to Chemicals of Potential Concern (COPCs), which include metals and organic substances (refer to Annex 7B.2 of this report). This report applies approaches to the quantification of health effects from predicted pollutant concentrations published by the United States Environmental Protection Agency (US EPA) Human Health Risk Assessment Protocol (HHRAP). Tolerable Daily Intake (TDI) values published by the UK Committee on Toxicity (COT) have also been used where appropriate for the quantification of health effects at selected receptors.

5.1.2 The method used to quantify potential health effects associated with the Proposed Development is presented in detail within Annex 7B.2. Relevant receptor locations are shown on Figure 7B.2.1 within this annex.

5.1.3 The assessment of health effects from exposure to metals and organic substances associated with the operation of the Proposed Development are reported in turn.

5.1.4 The contribution of emissions from the Proposed Development to soil concentrations of each metal and the total dioxins/furans and dioxin-like PCBs are low. The impacts represent an additional contribution of less than 0.025% of the respective soil guideline concentration values for metals and less than 0.06% of the soil guideline concentration values for total dioxins/ furans and dioxin-like PCBs.

5.1.5 A relatively low additional dietary intake of metals and dioxins/ furans and dioxin-like PCBs, when compared to the typical dietary intake values, is predicted to be associated with the operation of the Proposed Development. The predicted additional dietary intake of total mercury in the hypothetical resident G&C_2 and resident R3 receptor scenarios of less than 1.8 $x10^{-5}$ µg kg-$BW^{-1}d^{-1}$ is markedly less than the equivalent typical UK dietary value of $9.0x10^{-2}$ – $1.0x10^{-1}$ µg kg-$BW^{-1}d^{-1}$. The additional dietary intake of total dioxins/ furans and dioxin-like PCBs at resident receptors is predicted to be approximately 0.03% of typical background UK dietary values, with the daily intake predicted to be approximately 5.6% of the COT TDI value at the farmer receptor location with the highest predicted impact, NELN_C_3.

5.1.6 A low additional exposure to total dioxins/ furans and dioxin-like PCBs of infants via their mother's breast milk is predicted. Additional daily intake values at resident receptors are predicted to be 0.001% of the US EPA criteria and approximately 0.04% of the UK COT TDI value. At farmer receptors, the highest concentrations represent approximately 1.6% of the US EPA criteria, and 47.28% of the UK COT TDI at NELN_C_3.

5.1.7 The maximum predicted non-carcinogenic impact within an urban area would occur at the hypothetical receptor called G&C_2 and the maximum predicted impact in a rural area would occur at the hypothetical receptor called NELN_C_1. The maximum predicted non-carcinogenic impact at any resident

receptor would occur at receptor R3. The location of these three receptors and other receptors predicted to experience smaller impacts are illustrated on Figure 7B.2.1 within Annex 7B.2.

5.1.8 A range of chemicals of potential concern have been assessed and of these arsenic, nickel, inorganic mercury and thallium are predicted as having the largest contribution to non-carcinogenic health effects via the inhalation and ingestion pathway. The exposure pathways predicted to contain the largest risk to non-carcinogenic health effects is by inhalation for the hypothetical resident receptor and the hypothetical farmer receptor. The total hazard indices for these hypothetical receptors locations are predicted to be approximately a factor of 15 - 100 below the reference dose at which there is an appreciable risk of health effects occurring over a 70 year lifetime.

5.1.9 The maximum predicted carcinogenic impact within an urban area would occur at the hypothetical receptor called G&C_2 and the maximum predicted impact in a rural area would occur at the hypothetical receptor called NELN_C_3. The maximum predicted carcinogenic impact at any resident receptor would occur at receptor R3. These receptors represent locations with larger risks to carcinogenic health effects predicted to be associated with the Proposed Development than at any other of the other resident and farmer receptor scenarios.

5.1.10 A range of chemicals of potential concern have been assessed and of these arsenic and cadmium are predicted as having the largest contribution to carcinogenic health effects via the ingestion pathway for resident type receptors, while benzo[a]pyrene and total dioxins/ furans and dioxin-like PCBs are predicted as having the largest contribution to carcinogenic health effects via the ingestion pathway for farmer type receptors.

5.1.11 The largest risk of carcinogenic health effects is predicted to occur for arsenic via the inhalation exposure pathway in the hypothetical resident and farmer receptor scenarios. The ingestion of milk and inhalation are predicted to be the exposure pathways with the largest risk of carcinogenic health effects in the hypothetical farmer and resident receptor scenarios respectively. The total lifetime risk at these locations is a 1 in 6,599,379 for receptor G&C_2, 1 in 4,382,214 for R3, 1 in 51,287 for NELN_C_3 and 1 in 607,940 for NELN_C_2 risk of developing cancer over the entire lifetime of an individual receptor, which translates into an annual risk of 1 in 461,956,558, 1 in 306,754,972, 1 in 3,590,088 and 1 in 42,555,778 respectively. This is well within the acceptable annual risk of 1 in 1,000,000 for UK industrial operations (CIWEM, 2001).

6.0 CONCLUSIONS

6.1.1 The change in annual mean concentrations of particulate matter, nitrogen dioxide and sulphur dioxide, experienced by the population located within the Study Area (being within 10 km of the Proposed Development) has been used to estimate effects on the health of the population as a whole. The assessment considers a total population of 148,000 within the Study Area (which includes both urban and rural areas). The assessment concluded that predicted impacts associated with emissions of particulate matter, nitrogen dioxide and sulphur dioxide do not represent a significant effect when compared to the local baseline health of the population in each local authority area.

6.1.2 The HHRA assessment protocol has been widely applied to quantify the carcinogenic and non-carcinogenic risk to human health from exposure of the local community to emissions of metals (elemental Sb, As, Cd, Cr, Hg, Pb and Ni) and organic substances (PCDD/F congeners and PAHs (B[a]P, B[a]A, B[b]F and Chrysene) chemicals of potential concern. The assessment concluded that the maximally exposed individuals within North East Lincolnshire and surrounding areas, would not be subject to a significant additional carcinogenic risk or non-carcinogenic hazard as a consequence of being exposed to metals and organic substances emitted to air from the Proposed Development.

6.1.3 In order to deliver improvements to the quality of life and overall life expectancy of the local population the local health authorities have identified a number of priority areas to target. The areas identified as being able to deliver the greatest benefit to public health do not specifically relate to exposure to pollutants in ambient air but instead focus on wider social and economic determinants of health. The magnitude of the impacts predicted from the operation of the Proposed Development is so small that the Proposed Development is not considered to represent a significant risk to the health of the population in the Study Area.

7.0 REFERENCES

Cambridge Environmental Research Consultants (CERC) CERC (2017) *ADMS Roads Validation Papers, Cambridge Environmental Research Consultants,* from: http://www.cerc.co.uk/environmental-software/model-validation.html

Environment Agency (2016) *Releases from waste incinerators, Guidance on assessing group 3 metal stack emissions from incinerators, July 2016*

European Council (2000) *Directive on the Incineration of Waste, 2000/76/EC*

European Union (2010) *Directive 2010/75/EU on Industrial Emissions (Integrated Pollution Prevention and Control) (recast)*

East Riding of Yorkshire Council (2020a) *Joint Strategic Needs Assessment,* Accessed 12/02/2020 (URL: http://https://intel-hub.eastriding.gov.uk/jsna-home/)

East Riding of Yorkshire (2020b) *Joint Health & Wellbeing Strategy 2019-2022*

East Riding of Yorkshire (2018) *Annual Report of the Director of Public Health, 2018/19*

Joint Research Centre (2017) *Best Available Techniques (BAT) Reference Document on Waste Incineration, Draft 1, May 2017*

Lincolnshire County Council (2017) *Lincolnshire Joint Strategic Needs Assessment, June 2017* (URL: http://www.research-lincs.org.uk/Joint-Strategic-Needs-Assessment.aspx)

Lincolnshire County Council (2018) *Joint Health and Wellbeing Strategy for Lincolnshire, June 2018*

North East Lincolnshire Council (2019) *Director of Public Health Annual Report 2019*

North East Lincolnshire Council (2018) *Joint Strategic Needs Assessment, State of the Borough 2018*

North East Lincolnshire Council (2016) *Joint Health and Wellbeing Strategy for North East Lincolnshire 2013-2016*

North Lincolnshire Council (2013) *Securing the future together, North Lincolnshire's Joint Strategic Needs Assessment 2012/13, January 2013*

North Lincolnshire Council (2016) *North Lincolnshire Joint Health and Wellbeing Strategy 2013-2018*

North Lincolnshire Council (2020) *Annual Public Health Reports, Accessed: 12/02/2020* (URL: http://nldo.northlincs.gov.uk/IAS_Live/sa/jsna/public-health-reports)

Public Health England (2020a) *Health Profile 2019 North East Lincolnshire, Published 03/03/2020* (URL: http://fingertips.phe.org.uk/profile/health-profiles)

Public Health England (2020b) *Health Profile 2019 North Lincolnshire, Published 03/03/2020* (URL: http://fingertips.phe.org.uk/profile/health-profiles)

Public Health England (2020c) *Health Profile 2019 West Lindsey District, Published 03/03/2020* (URL: http://fingertips.phe.org.uk/profile/health-profiles)

Public Health England (2020d) *Health Profile 2019 East Riding of Yorkshire, Published 03/03/2020* (URL: http://fingertips.phe.org.uk/profile/health-profiles)

Public Health England (2020e) *Health Profile 2019 Lincolnshire, Published 03/03/2020* (URL: http://fingertips.phe.org.uk/profile/health-profiles)

Public Health England (2020f) *Public Health Outcomes Framework, Accessed 10/02/2020* (URL: fingertips.phe.org.uk)

ANNEX 7B.1: ASSESSMENT OF HEALTH EFFECTS FROM EXPOSURE TO PARTICULATE MATTER, NITROGEN DIOXIDE AND SUPLHUR DIOXIDE

EP UK Investments

South Humber Bank Energy Centre Project

Planning Inspectorate Reference: EN010107

South Marsh Road, Stallingborough, DN41 8BZ

The South Humber Bank Energy Centre Order

Document Ref. 6.4 Appendix 7B: Human Health Risk Assessment Annex 7B.1: Assessment of Health Effects from Exposure to Particulate Matter, Nitrogen Dioxide and Sulphur Dioxide

The Infrastructure Planning (Environmental Impact Assessment) Regulations 2017 (as amended)

The Infrastructure Planning (Applications: Prescribed Forms and Procedure) Regulations 2009 - Regulation 5(2)(a)

Applicant: EP Waste Management Ltd
Date: April 2020

DOCUMENT HISTORY

Document Ref	HHRA Annex 1		
Revision	1.0		
Author	M. Hill		
Signed		**Date**	April 2020
Approved By	G. Gray		
Signed		**Date**	April 2020
Document Owner	AECOM		

GLOSSARY

Term	Description
Acute effect	An effect that occurs within a short time after exposure.
Air pollutant	A substance present in the atmosphere at concentrations that are elevated, usually by human activities. Most air pollutants occur naturally in the atmosphere at low concentrations.
Ambient concentrations	Concentrations of airborne substances in outdoor air.
Chronic effect	An effect that occurs over a long time period or following a long period of exposure.
Chronic bronchitis	A daily cough with production of sputum for 3 months, two years in a row.
Cohort study	A study in which a particular health effect, is compared using groups of people who are alike in most ways but differ by a defined characteristic, such as exposure to a source of pollution for example.
Concentration – response function	An equation that represents, for example, the relationship between the predicted concentration of a pollutant in the air and the exposed population response.
Deaths brought forward	This does not constitute new/additional deaths but represents a reduction in life expectancy for those whose health is already seriously compromised, where one death brought forward represents a cumulative two to six month loss of life expectancy for the population exposed.
Emissions	The substances or mass of a substance emitted into the atmosphere.
Epidemiology	The study of populations in order to determine the frequency and distribution of disease and to measure risks.
Exposed population	The population exposed to a meaningful change in air pollutant concentrations.
Exposure	Inhalation of air containing substances at predicted concentrations.
Fine particulate matter	Size fractions of particulate matter smaller than PM_{10}. In this report represented by $PM_{2.5}$.
Hazard	Something (e.g. an object, a property of a substance, a phenomenon or an activity) that can cause adverse effects.

Term	Description
Life table	A way of summarising mortality rates for the age classes within a population.
Lower respiratory system	The human respiratory system below the larynx.
Morbidity	The incidence or prevalence of disease/ill health in a population.
Mortality	The incidence of death or the number of deaths in a population.
Nitrogen dioxide	A molecule composed of one nitrogen atom and two oxygen atoms, present in outdoor air as a gas.
Oxides of nitrogen	A collective term for all gases composed of nitrogen and oxygen, including nitrogen dioxide.
Particulate Matter	A solid or liquid particle (a droplet) that in the context of this report is small enough to be suspended in air.
PM_{10}	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 10 micrometres.
$PM_{2.5}$	Mass per cubic metre of particles passing through the inlet of a size selective sampler with a transmission efficiency of 50% at an aerodynamic diameter of 2.5 micrometres.
PM_{1}	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 1 micrometre.
Population	All people living in a defined area.
Predicted concentrations	Mass of pollutant per volume of air. Normally expressed as mean values over a defined time period, as calculated using dispersion models.
Relative risk	The likelihood of the event in an exposed group relative to those who have not been exposed.
Risk	The likelihood that a hazard will actually cause its adverse effects, together with a measure of the effect.
Sensitivity analysis	A procedure by which numerical estimates are tested to aid the interpretation of predicted values.
Years of life lost	A statistical measure of mortality effects at the population level.

ABBREVIATIONS

Abbreviation	Description
ACS	American Cancer Society
CAFE	Clean Air For Europe programme
COMEAP	Committee on the Medical Effects of Air Pollution
EC	European Commission
EU	European Union
GP	General Practitioner
IOM	Institute of Occupational Medicine
LRS	Lower Respiratory Symptoms
ONS	Office of National Statistics
WHO	World Health Organisation

CONTENTS

TABLES

FIGURES

ANNEX 7B.1 - HEALTH EFFECTS FROM EXPOSURE TO PARTICULATE MATTER, NITROGEN DIOXIDE AND SULPHUR DIOXIDE

7B.1.1 Introduction

7B.1.1.1 This Annex to Appendix 7B of the ES Volume III (Document Ref. 6.4) quantifies the human health effects associated with the exposure of the local community to the predicted change in the atmospheric concentrations of particulate matter, nitrogen dioxide and sulphur dioxide within 10 km of the Site arising from the Proposed Development (the Study Area). The emissions have been calculated using the methodology set out in the air quality dispersion modelling report (Appendix 7A in ES Volume III).

7B.1.1.2 The Clean Air for Europe (CAFE) programme (CAFE, 2013) revisited the management of air quality within the EU and resulted in The Ambient Air Quality and Cleaner Air for Europe Directive (EC, 2008). This Directive defines Limit Values for ambient concentrations of specified air pollutants, including sulphur dioxide, nitrogen dioxide and particulate matter (as PM_{10} and as $PM_{2.5}$). These limit values represent a minimum standard of ambient air quality that all member states of the EU are obliged to achieve, everywhere except for a small number of prescribed locations. At the present time, the limit values have been transposed into national legislation through the Air Quality Standards Regulations 2010.

7B.1.1.3 The National Air Quality Strategy (Defra, 2007) brought forward Air Quality Objectives to assist National and Local Government in achieving the Limit Values to prescribed timetables. The setting of national air quality Objective Values and EU Limit Values, for the protection of human health, was based on a substantial body of scientific evidence. The need for the EC and for National Governments to consider the costs and benefits of proposed Limit Values resulted in the development of robust methodologies for the quantification of health effects associated with exposure to air pollution outside of the workplace.

7B.1.1.4 This report applies approaches to the quantification of health effects from predicted pollutant concentrations published by the Department of Health's Committee on the Medical Effect of Air Pollutants (COMEAP) and the Clean Air for Europe (CAFE) programme. These methods are as set out in COMEAP's reports on the quantification of the effects of air pollution on health (COMEAP, 1998), the effect of long term exposure to air pollution (COMEAP, 2009a), the mortality effects of long term exposure to particulate air pollution (COMEAP, 2010) and a cost benefit analysis methodology for CAFE (AEA, 2005). COMEAP and CAFE both reviewed the scientific literature and took full account of this knowledge in the development of their methods for quantifying the health effects of air pollution. No further consideration of the scientific literature, on the epidemiology of exposure to air pollution that underpins these methods, has been undertaken in support of this report.

7B.1.1.5 The relationship between exposure to air pollutants, either singly or in combination, and the resulting effects on health remains a topic of active research. Exposure to increased concentrations of pollutants such as particulate matter and sulphur dioxide are associated with effects on the respiratory and cardiovascular system, leading to increased morbidity and such exposure may contribute to individual deaths through mechanisms that are not yet fully understood. The methods used are based on current understanding of the effect of exposure on health as reported in the cited publications.

7B.1.1.6 The methodologies employed to quantify the health effect associated with the exposure of populations to predicted concentrations of air pollutants consider the effect on the affected population and not the effect on each individual living within that population. The health effects are reported as population statistics that should be considered appropriately[1] and in the context of the methods used to calculate them.

7B.1.1.7 It is likely that exposure to airborne pollutants can cause acute effects on human health in the short term and chronic effects over the longer term. The vulnerability of individuals to short term effects of air pollution can vary depending on their general health at the time of exposure, their lifestyle and on the presence of specific medical conditions. Exposure to air pollutants over the longer term may have a marginal effect that contributes to the progression of chronic diseases that have other causes.

7B.1.1.8 In this report the terminology used is of necessity technical and the meaning of the terms may differ from their use in conversational English. A glossary of the terms used is provided within this report.

7B.1.2 Methodology

Overview of the Approach

7B.1.2.1 The approach to quantifying acute health effects is based on the use of a concentration-response function. The functions used by COMEAP and CAFE and the exposure-response coefficients used within them, are derived from reviews of the empirical evidence generated by epidemiological studies. This body of evidence is such that the World Health Organisation and national bodies, with responsibility for public health, are convinced that the associations between exposure to polluted air and specific health outcomes (events) should be considered as causal.

7B.1.2.2 The concentration-response function (Equation 7B.1.1 – see below) combines the use of an exposure-response coefficient with, details of the

[1] (COMEAP specifically highlight the need for appropriate consideration of predicted effects on health in their report Statement on the Applicability of time-series coefficients to areas affected by emissions of air pollutants from industrial sources, September 2000 (COMEAP, 2000))

specific population affected and the predicted change in ambient pollutant concentrations that the population would be exposed to.

Equation 7B1.1 $$\Delta E = \beta \times \Delta C \times P \times E$$

Where:

ΔE = (change in) background rate of events;

β = exposure-response coefficient;

ΔC = change in concentration of pollutant;

P = population exposed.

7B.1.2.3 The effect of exposure on health is described as a change in the rate of occurrence of specified events. For example an event might be a hospital emission. For each pollutant considered in this study, the specific events used to represent health effects are considered in turn in the following sections of this report.

7B.1.2.4 The total population considered is the same for assessment of acute effects associated with exposure to particulate matter, nitrogen dioxide and sulphur dioxide. The same total population is also used in the assessment of mortality effects associated with chronic exposure to fine particulate matter.

7B.1.2.5 The CAFE methodology adopts the relationship between mortality and long-term exposure to fine particulate matter ($PM_{2.5}$) based on a cohort study by the American Cancer Society (Pope et al, 2002) and expresses the results of the calculations in terms of life years lost by the population, rather than the numbers of deaths within the population. This approach has been adopted in this assessment as it is consistent with the current consensus view of the subject. It requires an alternative spreadsheet method to be employed based on life tables, instead of using Equation 7B.1.1. This approach was employed by COMEAP in its most recent report on the quantification of the long term effects on mortality (COMEAP, 2001).

Approach to Quantifying the Health Effects of Process Emissions

Assessment of Mortality Effects

7B.1.2.6 The Institute of Occupational Medicine (IOM) (Miller et al, 2006), (Miller, 2013) have developed a series of spreadsheets to predict the change in mortality based on the life table approach. This approach has the advantage of addressing the complicating issue of considering the link between death rates and surviving populations (Miller et al, 2003) when calculating impacts on chronic mortality.

7B.1.2.7 In 2009, COMEAP recommended (COMEAP, 2009a) coefficients which, when used in conjunction with methods developed for the Department of Health and the European Commission by the IOM, allow the calculation of the potential impact on mortality and life expectancy of specified changes in concentrations of air pollutants presented in the 2010 COMEAP report

(COMEAP, 2010). This quantification used the coefficients in the 2009 report to calculate that decreasing $PM_{2.5}$ by 1 μgm^{-3} would save 4 million life years and increase life expectancy at birth by 20 days. The coefficients recommended by COMEAP in 2009 remain unchanged from those identified in the previous 2001 report (COMEAP, 2001), however COMEAP report that the evidence base relating to the effects of long-term exposure to air pollutants had strengthened since the publication of the 2001 report.

7B.1.2.8 The dispersion model predictions of particulate matter concentrations can be treated as being either PM_{10} or $PM_{2.5}$. In practice, almost all of the particulate matter emitted from the proposed plant will be in the size fraction 2.5 µm and less, because the fabric filter used will remove almost all of the particles with a larger diameter, whilst being less efficient at around 1 µm. For the assessment of mortality associated with long term exposure to particulate matter the predicted particulate matter concentrations are considered to relate to particles within the size fraction $PM_{2.5}$.

7B.1.2.9 The population located within the Study Area is determined from census data using GIS methods. A Study Area boundary of 10 km from the pollution source is greater than necessary for a study of this type, and it is used here for consistency with other elements of the air quality assessment, demonstrating a precautionary approach. Baseline life expectancies for the whole population are calculated based on data for male and female life expectancies. For a given change in the ambient concentration of $PM_{2.5}$ that the population are exposed to there is an associated change in the risk that the exposure will result in a decrease in life expectancy, or loss of life. The risk is expressed as an estimate of life years lost for the total population exposed from cardiovascular and respiratory health effects.

7B.1.2.10 In 2015, COMEAP published an interim report on the association of long term concentrations of nitrogen dioxide and mortality (COMEAP, 2015). The conclusion of this statement was to support earlier publications on the effect of nitrogen dioxide on mortality, and concluded that coefficients in these works should be used until further evidence has been gathered. The report also considered the combined effect on mortality due to nitrogen dioxide and particulate matter. COMEAP recommend that the nitrogen dioxide coefficient should be reduced by 33% when assessed in combination with $PM_{2.5}$. COMEAP also noted that they were not in a position to provide a comparative reduction in the particulate matter coefficient, and noted that this would produce an over estimate of the mortality effects of $PM_{2.5}$ when assessed in combination with nitrogen dioxide.

7B.1.2.11 In 2018, COMEAP published its final report on the associations of long term average concentrations of nitrogen dioxide with mortality (COMEAP, 2018). This report confirms the nitrogen dioxide and particulate matter coefficients previously reported, but do change their recommendation of how the mortality effects of nitrogen dioxide are quantified. The report separates the quantification of effects into two: those that change emissions of oxides of nitrogen (NOx), and those that change traffic-related pollutants. It should be noted that the COMEAP committee were divided in regards to how the health impacts of nitrogen dioxide should be quantified, and the values used in this

report therefore reflect the view of the majority of committee members. The full details and justifications for the dissention are given in the 2018 report.

7B.1.2.12 When assessing the health impacts of changes in oxides of nitrogen, COMEAP recommends that the unadjusted coefficient of 1.023 (95% confidence interval (CI) of 1.008 to 1.037 per 10 µg/m^3) for nitrogen dioxide should be should be reduced by 20%. This is due to confounding effects of $PM_{2.5}$ and other pollutants closely correlated to $PM_{2.5}$.

7B.1.2.13 When considering the effects of nitrogen dioxide alone, COMEAP recommends the nitrogen dioxide coefficient should be further reduced by 30-70%. This results in reducing the nitrogen dioxide coefficient by 25-55% that is a range of 1.006 to 1.013 per 10 µg/m^3.

7B.1.2.14 As nitrogen dioxide is not assessed in isolation, this method has not been considered further in this report. The emissions from the Proposed Development are a complex mixture of substances, and the impacts to mortality due to individual substances is equally complex.

7B.1.2.15 When assessing changes in a pollutant mixture, such as traffic related pollutants, COMEAP recommends using the unadjusted nitrogen dioxide coefficient (1.023, 95% CI of 1.008 to 1.037 per 10 µg/m^3). An additional calculation for $PM_{2.5}$ (using a coefficient of 1.06, 95% CI of 1.04 to 1.08 per 10 µg/m^3) can also be done. These results should not be added, in order to capture the effects of the mixture of air pollutants as a whole.

7B.1.2.16 The report also provides a separate methodology for the assessment of nitrogen dioxide and $PM_{2.5}$ in combination, by adjusting both coefficients. An average value for the coefficients for nitrogen dioxide (1.0165, CI of 1.011 and 1.02) and particulate matter (1.0335, CI 1.019 and 1.053) were used as listed in Table 9.3 in the COMEAP report (COMEAP, 2018). In each of these cases the CI represents the minimum and maximum coefficients determined by the studies as referenced in the COMEAP document. This method allows the changes in mortality to be summed, providing a single statistic for the assessment of changes in mortality due to changes in pollutant concentrations containing both nitrogen dioxide and $PM_{2.5}$.

7B.1.2.17 The assessment of mortality effects has therefore used two of the methodologies set out above: the use of unadjusted coefficients for nitrogen dioxide and $PM_{2.5}$ to assess the impacts of both pollutants separately (including respective confidence intervals), and the use of adjusted coefficients for both nitrogen dioxide and $PM_{2.5}$ (including the reported range of coefficients). The coefficients used in this assessment are summarised in Table 7B.1.1.

7B.1.2.18 The combined period of life lost by the local community calculated for the change in concentration of pollutants has been reported as a value due to particulate matter, a value due to nitrogen dioxide, and a value due to the combined impacts of particulate matter and nitrogen dioxide.

Assessment of Acute Health Effects

7B.1.2.19 Acute health effects associated with exposure to airborne particulate matter are quantified using the concentration-response function presented as Equation 7B.1.1. The health effects associated with exposure to particulate matter (as PM_{10}) considered in this assessment as specific events are:

- chronic bronchitis (adults);
- respiratory hospital admissions;
- cardiac hospital admissions;
- lower respiratory system symptom days (children); and
- lower respiratory system symptom days (adults).

7B.1.2.20 The respective concentration-response coefficients applied for each of the event classes are summarised in paragraph 7B.1.2.29.

Approach to Quantifying the Health Effects of Nitrogen Dioxide

7B.1.2.21 The health effects associated with exposure to nitrogen dioxide that are considered in this assessment as specific events are:

- respiratory hospital admissions;
- cardiac hospital admissions; and
- mortality.

7B.1.2.22 The impact of exposure to nitrogen dioxide for respiratory hospital admissions are considered through the use of the relationship cited by COMEAP (Department of Health, 2006), of a 0.038% increase in the rate of the health event for every 1 µg m^{-3} rise in NO_2 concentrations.

7B.1.2.23 In earlier publications, COMEAP have outlined the fact that acute mortality and respiratory hospital admissions from NO_2 should be considered as an alternative to that used for particulate matter and not in addition. This is because NO_2 may be acting as a marker for the effect of locally emitted particulate matter (COMEAP, 2009b) and there is therefore a risk of double counting the impact of local emissions on health. While subsequent documents have postulated the use of a mutually adjusted coefficient for use of nitrogen dioxide and particulate matter (as referred to in paragraph 7B.1.2.16), mortality and respiratory hospital admissions associated with SO_2 should not be added, as there may be some synergistic effects, i.e. the observed associations are not independent of each other.

7B.1.2.24 The respective concentration-response coefficients applied for each of the event classes are summarised in paragraph 7B.1.2.29.

7B.1.2.25 In this assessment it has been assumed that 70% of the predicted oxides of nitrogen concentrations contributed to total annual mean concentrations are in the chemical form of nitrogen dioxide. In practice this is likely to be a very robust approach especially at receptors predicted to experience the greatest change in annual mean concentrations of oxides of nitrogen. The use of a

70% conversion rate for long term average concentrations is in line with Environment Agency recommendations (EA, 2013).

Approach to Quantifying the Health Effects of Sulphur Dioxide

7B.1.2.26 Health effects associated with exposure to sulphur dioxide that are considered in this assessment as specific events are:

- respiratory hospital admissions; and
- mortality.

7B.1.2.27 The respective concentration-response coefficients applied for each of the event classes are summarised in paragraph 7B.1.2.29.

7B.1.2.28 The impact of exposure to sulphur dioxide for respiratory hospital admissions are considered through the use of the relationship cited by COMEAP (COMEAP, 1998), of a 0.05% increase in the rate of the health event for every 1 µg m^{-3} rise in sulphur dioxide concentrations. The corresponding value of 0.06 % has been used for the change in the rate of mortality per 1 µgm^{-3}.

Summary of Concentration-Response Coefficients

7B.1.2.29 Concentration-response coefficients for health events used in this study and applied to the increased exposure to air pollution are shown in Table 7B.1.1 and Table 7B.1.2.

Table 7B.1.1: Increases in Mortality Rates from Exposure to $PM_{2.5}$ and Nitrogen Dioxide

CHANGE IN MORTALITY HAZARDS	CONCENTRATION-RESPONSE FUNCTION	95 % CONFIDENCE LIMITS
$PM_{2.5}$	1.06	1.04 – 1.08
NO_2	1.023	1.008 – 1.037
NO_2 (when assessed with $PM_{2.5}$)	1.0165	1.011 – 1.02
$PM_{2.5}$ (when assessed with NO_2)	1.0335	1.019 – 1.053

Table 7B.1.2: Increases in Health Effects from Exposure to an Additional 1 µgm^{-3} of Air Pollutant

HEALTH EVENT	INCREASE (BASED ON RELATIVE RISK)[A]	POLLUTANT
Particulate Matter – CAFE (CAFE, 2013)		
Chronic bronchitis (attack rates)	0.7%	PM_{10}
Cardiovascular hospital admissions	0.06%	PM_{10}
Respiratory hospital admissions	0.114%	PM_{10}
Consultation with GPs (asthma, April – Sept, 15 – 64 years age)	0.25%	PM_{10}
Lower respiratory symptoms (wheeze, shortness of breath, phlegm production) (in children)	0.0004%	PM_{10}
Lower respiratory symptoms (in adults)	0.0017%	PM_{10}
Nitrogen Dioxide – COMEAP (COMEAP, 1998, 2009)		
Cardiovascular hospital admissions	0.13%	NO_2
Respiratory hospital admissions	0.038%	NO_2
Deaths brought forward	0.035%	NO_2
Sulphur Dioxide – COMEAP (COMEAP, 1998)		
Deaths brought forward	0.06%	SO_2
Respiratory hospital admissions	0.05%	SO_2

a - Relative risk is defined as the ratio of the incidence of disease in the exposed group divided by the corresponding incidence of disease in the non-exposed group.

Summary of Input Information

7B.1.2.30 The calculation of health effects is based on the following information specific to the Proposed Development:

7B.1.2.31 Predicted changes in annual mean pollutant concentrations for sulphur dioxide, nitrogen dioxide and particulate matter, expressed as µg/m^3. The values are made available to this assessment as a variable Cartesian grid of receptor points, covering a model domain of 20 km by 20 km. The grid is centred on the location of the source of emissions under consideration (Appendix 7A in ES Volume III, Document Ref. 6.4):

- population data, at the 'super output area level', based on the 2011 census (ONS, 2018);
- background data on the rates of all relevant health outcomes (national and local). This input is considered in paragraph 7B.1.3 of this report.

7B.1.2.32 The exposed population within the Study Area has been defined as that within 10 km of the source of emissions. This circular boundary for the exposed population sits within the boundary of the dispersion model domain. The exposed population boundary encompasses an area that is large enough to capture the incremental reduction in meaningful effects. In setting this boundary there is a need to balance the requirement to provide adequate spatial coverage to capture the events under consideration, whilst avoiding the generation of values that are artefacts of the method, caused by the inclusion of an unnecessarily large population in the model.

7B.1.2.33 The pollutant concentrations are plotted as isopleths (lines of equal value) that form a pattern of decreasing magnitude and this is overlaid onto the population data using GIS software. The total population is then subdivided into 'bands' on the basis of the magnitude of the change in concentrations of pollution that they are predicted to experience. The process is repeated for each pollutant. The pollutant concentration used to represent each band is taken as the highest isopleth bounding the band, or in the case of the worst case bands the highest value at any receptor is used.

7B.1.2.34 The population of each band is then calculated, from the population density of the wards that make up the area within the band. This technique assumes that there is an equal distribution of people within each super output area and the number of people in each area is determined on a pro rata basis.

7B.1.2.35 This input information is illustrated in Figures 7B.1.1 to 7B.1.3 within Attachment A of this report.

Summary of Output Information

7B.1.2.36 This assessment reports numerical information for each of the health events at the total population level per annum. The numerical estimates for morbidity events for the total population are the sum of the values for each band as summarised in Attachment B of this report.

7B.1.2.37 Results are expressed as numerical estimates for the morbidity outcomes described above over a 30-year period and this same information is also expressed as an estimate of the number of years operation that would give rise to a single new event.

7B.1.2.38 Numerical estimates of life years lost are reported for the whole population for the effect on mortality.

Approach to Consideration of Additive Effects

7B.1.2.39 The results for each pollutant are presented independently. In practice it is highly likely that the health effects estimated for each pollutant are not independent of each other. The approach taken to the calculation of the numerical estimates for the effect of exposure to each pollutant have taken a

robust approach that already incorporates conservative values at several points in the calculation process. Adding the health effect estimates for separate pollutants together will result in an unreasonable over estimate of any health effects.

7B.1.3 Baseline Conditions

The context

7B.1.3.1 The predicted health effects are considered in the context of observed rates of disease and observed life expectancies in the UK. The method used could make use of either national statistics or local level statistics if such data exists. In this assessment national level statistics have been used, as there are benefits to determining baseline population disease rates on statistics that represent larger numbers of people. There may be differences in the values for the statistical parameters used between the local and national level datasets, but the associated difference in the calculated health effects under consideration, would be so small as to be insignificant.

7B.1.3.2 National statistics for disease rates and life expectancy have been used for this assessment, as presented in Table 7B.1.3.

7B.1.3.3 Use has been made of episode statistics sourced from surveys published by the Office of Population Censuses and Surveys (OPCS, predecessor to the Office for National Statistics) (OPCS, 1995). Life expectancy at birth figures for England has been sourced from the Office of National Statistics (ONS) (ONS, 2017).

Table 7B.1.3: Background Rates of Disease

DISEASE	BASELINE RATE PER 1,000 POPULATION
Chronic Bronchitis	8
Cardiovascular hospital Admissions	14
Respiratory hospital admissions	7.8
GP Consultation Asthma	64.13
LRS Children	325
LRS Adults	204.44
Mortality – Deaths (non traumatic) brought forward	7.69
Life Expectancy for 2012 to 2014 (Men - England)	79.5 years
Life Expectancy for 2012 to 2014 (Women - England)	83.1 years

7B.1.4 Results

Mortality Effects Through Exposure to Nitrogen Dioxide and Particulate Matter

7B.1.4.1 The assessment using the method described in Section 7B.1.2 has calculated the average period of life lost by the local community as a whole due to the maximum concentration of pollutants considered alone:

- an average of 35 minutes and 16 minutes lost due to the effects of exposure to a maximum concentration of 2.1 µg/m^3 of nitrogen dioxide for the male and female population, respectively; and
- an average of 11 minutes and 5 minutes lost due to the effects of exposure to a maximum concentration of 0.25 µg/m^3 of particulate matter for the male and female population, respectively.

7B.1.4.2 Using the combination method as described in Section 7B.1.2, the average period of life lost by the local community as a whole due to the maximum concentration of pollutants are as follows:

- a combined average of 31 minutes and 15 minutes lost due to the effects of exposure to a maximum concentration of nitrogen dioxide (2.1 µg/m^3) and particulate matter (0.25 µg/m^3) for the male and female population, respectively.

7B.1.4.3 However these results would not be consistent over the entire exposed population as this only considers the cohort of society with the greatest exposure.

7B.1.4.4 The latest Air Quality Strategy (Defra, 2007) produced by Defra estimates that the average loss of life expectancy at 2005 levels of exposure to all anthropogenic $PM_{2.5}$ is 8 months for each person in the UK. The most recent report published by COMEAP in 2018 calculates the mortality effects of long term exposure to particulate air pollution to be equivalent to 28,000 – 36,000 deaths in the UK associated with a loss of total population life of 328,000 – 416,000 years and a loss of life expectancy from birth of approximately 6 months per person. The Royal College of Paediatrics and Child Health (RCPCH) and the Royal College of Physicians (RCP) jointly produced a report in February 2016, calculating the mortality burden of outdoor air pollution to be in the order of 40,000 early deaths (RCPCH and RCP, 2016), with a cost to the economy of over £20 Billion per year. The greatest predicted additional life years lost due to the Proposed Development of 26 minutes per person in the male population, and 12 minutes for the female population due to cardiovascular and respiratory health effects, can be considered as very low when taken in context with the background figure for air pollution.

7B.1.4.5 A sensitivity analysis for the number of potential life years lost was performed based on the upper and lower confidence levels for the concentrations-response coefficient for mortality due to nitrogen dioxide exposure alone, particulate matter exposure alone and the sum of the two based on mutually adjusted coefficients. The results of the sensitivity analysis are:

- for nitrogen dioxide, a range from 12 to 55 minutes of life lost across the male population, and 6 to 26 minutes of life lost for the female population;
- for particulate matter a range of 7 to 14 minutes of life lost across the male population, and 3 to 7 minutes of life lost across the female population; and
- for the combination of pollutants using the maximum and minimum mutually adjusted coefficients as reported by COMEAP (2018), a range of 20 to 40 minutes of life lost across the male population, and 10 to 19 minutes of life lost across the female population.

Morbidity Effects Associated with Exposure to Nitrogen Dioxide

7B.1.4.6 Figure 7B1.1, Figure 7B.1.2 and Figure 7B.1.3 show the exposed population affected by the predicted change in concentration of nitrogen dioxide, particulate matter and sulphur dioxide due to the Proposed Development. Table 7B.1.4 shows the predicted change in the number of health events due changes in nitrogen dioxide concentrations with the full set of results shown in Table 7B.1.4b in Attachment B.

Table 7B.1.4: Predicted Change in the Number of Health Events due to the Additional Nitrogen Dioxide from the Proposed Development

DISEASE	BASELINE RATE FOR TOTAL EXPOSED POPULATION PER ANNUM	EXTRA CASES PER ANNUM	EXTRA CASES IN 30 YEAR PERIOD	YEARS OF OPERATION NEEDED FOR ONE EXTRA CASE	EXTRA CASES PER ANNUM AS A % OF BASELINE
Cardio-vascular hospital admissions	2,075	0.11	3.1	10	0.005
Respirator-y hospital admissions	1,156	0.04	<1.0	60	0.001
Deaths bought forward	1,140	0.03	<1.0	67	0.001

7B.1.4.7 The number of cardiovascular hospital admissions for the population as a whole is predicted to increase from a baseline rate of 2,075 admissions per year by 0.005%. An alternative way of expressing this population statistic is as a single additional admission within a time period of 10 years. This means that this very small impact is likely to occur during the operational life time of the Proposed Development.

7B.1.4.8 Additional hospital admissions for respiratory symptoms are predicted to increase by 0.001% on a baseline rate of 1,156 admissions per year. The population statistic of deaths brought forward is an abstract concept where one death brought forward represents a cumulative two to six month loss of life expectancy for the population exposed. The predicted impact for the measure death brought forward is an increase of 0.001% on a baseline rate of 1,140 deaths brought forward per annum.

7B.1.4.9 These figures can be compared to the total number of Ischemic Heart Disease (Coronary Heart Disease) primary diagnoses obtained from Hospital Episode Statistics. In the year 2017-2018, 397,639 diagnoses, attributed to diet/ lifestyle etc., were made in England (The Health and Social Care Information Centre, 2015). The extra cases of cardiovascular hospital admissions estimated from the operation of the Proposed Development are 0.102 per annum, which represents 0.000028% of the total cases in England. In comparison with the baseline rate for the entire exposed population, the Proposed Development will cause a 0.005% change in the number of cardiovascular hospital admissions.

7B.1.4.10 In the context of baseline rate, the additional burden to human health and the health care system as a result of operation of the Proposed Development can be considered very small, and is considered to be an insignificant effect.

Morbidity Effects Associated with Exposure to Particulate Matter

7B.1.4.11 Figure 7B.1.2 shows the exposed population affected by the change in concentration of particulate matter due to the Proposed Development.

7B.1.4.12 Table 7B.1.5 shows the predicted change in the number of health events due to the change in concentration attributed to the Proposed Development with the full set of results shown in Table 7B.1.6b in Attachment B.

7B.1.4.13 The change in concentration of PM_{10} due to the Proposed Development is predicted to produce a slight increase in the number of cases of all the acute health events per annum.

7B.1.4.14 The baseline rate for each of the health events has been calculated for the entire exposed population (approximately 148,000 people) in this study. The extra number of health events generated due to the change in concentration of particulate matter from the Proposed Development can be considered as less than 0.003% when compared to the baseline rate for the entire exposed population.

7B.1.4.15 The largest potential impact on the number of health events is predicted to occur in GP consultation rates for Asthma. Approximately 4 new cases would be expected in the estimated 30 year operating period of the Proposed Development with the first extra case anticipated to occur after approximately 8 years. This represents an estimated increase in the rates of GP consultation for asthma of 0.0013% on baseline rates. The lowest change is predicted to occur in lower respiratory symptoms for adults where the Proposed Development would need to be operated for over 470 years for a single extra case to be observed.

7B.1.4.16 In the context of baseline rates, this can be considered as a relatively small additional burden on the health of the exposed population and the health care system as a whole, and is considered to be an insignificant effect.

Table 7B.1.5: Predicted Change in the Number of Health Events due to the Additional Particulate Matter from the Proposed Development

DISEASE	BASELINE RATE FOR TOTAL EXPOSED POPULATION PER ANNUM	EXTRA CASES PER ANNUM	EXTRA CASES IN 30 YEAR PERIOD	YEARS OF OPERATION NEEDED FOR ONE EXTRA CASE	EXTRA CASES PER ANNUM AS A % OF BASELINE
Chronic Bronchitis	1,186	0.034	1	29	0.0029
Cardio-vascular Hospital admissions	2,075	0.005	<1	196	0.00025
Respiratory hospital admissions	1,156	0.005	<1	185	0.00047
GP Consultation Asthma	9,507	0.121	4	8	0.0013
LRS Children	48,178	0.008	<1	127	0.00002
LRS adults	30,306	0.002	<1	474	0.00001

Morbidity Effects Associated with Exposure to Sulphur Dioxide

7B.1.4.17 Figure 7B.1.3 shows the exposed population within the Study Area affected by the change in concentration of sulphur dioxide due to the Proposed Development.

7B.1.4.18 Table 7B.1.6 shows the predicted change in the number of health events due to the change in concentration of sulphur dioxide attributed to the Proposed Development with the full set of results shown in Table 7B.1.5b in Attachment B.

Table 7B.1.6: Predicted Change in the Number of Health Events due to the Additional Sulphur Dioxide from the Proposed Development

DISEASE	BASELINE RATE FOR TOTAL EXPOSED POPULATION PER ANNUM	EXTRA CASES PER ANNUM	EXTRA CASES IN 30 YEAR PERIOD	YEARS OF OPERATION NEEDED FOR ONE EXTRA CASE	EXTRA CASES PER ANNUM AS A % OF BASELINE
Deaths (non-traumatic brought forward)	1,140	0.017	1	58	0.0015
Respiratory hospital admissions	1,156	0.015	<1	68	0.000013

7B.1.4.19 The number of respiratory hospital admissions for the population as a whole is predicted to increase from a baseline rate of 1,156 admissions per year by 0.000013%. An alternative way of expressing this population statistic is as an additional admission within a time period of 68 years. The predicted impact for the measure death brought forward is an increase of 0.0015% on a baseline rate of 1,140 deaths brought forward per annum.

7B.1.4.20 Throughout the estimated operating time period of the Proposed Development approximately one additional case of the above health events is predicted to occur.

7B.1.4.21 In the context of baseline rates of morbidity, these changes can be considered as a relatively small additional burden on the health of exposed population and health care system as a whole, and is considered to be an insignificant effect.

7B.1.5 Conclusion

7B.1.5.1 An assessment of the potential effects on human health due to the operation of the Proposed Development has been carried out with respect to the predicted change in population exposure to nitrogen dioxide, particulate matter and sulphur dioxide. This report applies approaches to the quantification of health effects from predicted pollutant concentrations published by the Department of Health's COMEAP and the CAFE programme.

7B.1.5.2 The total population of the Study Area (being an area extending 10 km from the location of the Proposed Development) was considered in the assessment of potential effects associated with exposure to nitrogen dioxide, particulate matter and sulphur dioxide. The same total population was also

used in the assessment of mortality effects associated with chronic exposure to fine particulate matter.

7B.1.5.3 The assessment concluded that, for each pollutant under consideration, the effect of the Proposed Development emissions of particulate matter (PM_{10} and $PM_{2.5}$), nitrogen dioxide and sulphur dioxide on human health would be insignificant.

7B.1.5.4 The main outcomes of the study are summarised below.

- For cardiovascular and respiratory health effects, the potential effect of each pollutant considered is:
  - an average of 35 minutes of life lost as a result of the Proposed Development for the male population and 11 minutes for the female population for nitrogen dioxide alone;
  - an average of 16 minutes of life lost as a result of the Proposed Development for the male population and 5 minutes for the female population for particulate matter alone; and
  - an average of 31 minutes of life lost as a result of the Proposed Development for the male population and 15 minutes for the female population for the combination of nitrogen dioxide and particulate matter using mutually adjusted coefficients.
- In comparison, the results published by COMEAP (2018) predict the mortality effects of long term exposure to air pollution to be equivalent to 28,000 – 36,000 deaths in the UK associated with a loss of total population life of 328,000 – 416,000 years. RCPCH and RCP reported that the total mortality burden due to outdoor air pollution is of the order of 40,000 early deaths per year, with a cost to the economy of over £20 billion per year
- The estimated number of extra chronic bronchitis events, associated with the predicted change in concentration of particulate matter in the study area, is 0.034 per annum, which represents an increase of 0.0029% on the corresponding baseline rates for the entire exposed population. Additional cases of hospital admissions for cardiovascular and respiratory symptoms are predicted to rise by 0.005 per annum each. The estimated increase in the occurrence of lower respiratory symptoms in children is 0.008 per annum, which represents a 0.00002% increase on baseline rates. This can be considered as an insignificant effect on the health of the exposed population and the health care system as a whole.
- Rates of hospital admissions for cardiovascular symptoms, associated with the predicted change in concentration of nitrogen dioxide in the Study Area, are estimated to increase by 0.102 per annum which represents an increase of 0.005% on the corresponding baseline rates for the entire exposed population. This is considered insignificant when compared to the total incidence of heart disease in the entire population of England, attributable to factors such as diet and lifestyle. The estimated increase in hospital admissions for respiratory symptoms is predicted to be by 0.001%

on a baseline rate of 1,156 admissions per year. The predicted impact for the measure death brought forward is an increase of 0.001% on a baseline rate of 1,140 deaths brought forward per annum.

- The predicted impact for the measure death brought forward, associated with the predicted change in concentrations of sulphur dioxide in the Study Area, is an increase of 0.0015% on a baseline rate of 1,140 deaths brought forward per annum. Rates of hospital admissions for respiratory symptoms are estimated to increase by 0.015 per annum, which represents a 0.000013% increase on baseline rates. This can be considered as a insignificant effect on the health of the exposed population and the health care system as a whole.

7B.1.6 References

AEA Technology (2005) *Methodology for the Cost Benefit Analysis for CAFE Volume 2: Health Impact Assessment, Accessed via* URL http://ec.europa.eu/environment/archives/cafe/pdf/cba¬_methodology_vol2.pdf, date accessed 09/07/2013

CAFE Programme, Accessed via URL http://ec.europa.eu/atoz_en.htm, date accessed 09/07/2013.

Committee on the Medical Effects of Air Pollutants (COMEAP) (1998) *Quantification of the Effects of Air Pollution on Health in the United Kingdom, Department of Health, The Stationery Office, London.*

COMEAP (2000) *Statement on the Applicability of time-series coefficients to areas affected by emissions of air pollutants from industrial sources, September 2000.*

COMEAP (2001) *Statement and Report on the Long Term Effects of Particles on Mortality*, Accessed via URL: http://www.comeap.org.uk/documents/statements/39-page-linking.page-linking/15-long-term-effects-of-paricles-on-mortality-march-2001, Date accessed 09/07/2013.

COMEAP (2009a) *Long term Exposure to Air Pollution: Effect on Mortality, June 2009.*

COMEAP (2009b*) Statement on the Quantification of The Effects of Long-Term Exposure to Nitrogen Dioxide on Respiratory Morbidity in Children*

COMEAP (2010) *The Mortality Effects of Long-Term Exposure to Particulate Air Pollution in the United Kingdom, 2010*

COMEAP (2015) *Interim Statement on Quantifying the Association of Long-Term Average Concentrations of Nitrogen Dioxide and Mortality*

COMEAP (2018) *Association of Long-Term Average Concentrations of Nitrogen Dioxide with Mortality, August 2018*

Department of Health (2006) *Cardiovascular Disease and Air Pollution, A Report by the Committee on Medical Effects of Air Pollutants' cardiovascular sub-group*

Defra (2007) *The Air Quality Strategy for England, Scotland, Wales and Northern Ireland*

Environment Agency, *Conversion Ratios for NO_X and NO_2, Air Quality Modelling and Assessment Unit (AQMAU).* Obtained from the Environment Agency website available at: http://www.environment-agency.co.uk/static/documents/Conversion_ratios_for__NOx_and_NO2_.pdf Date accessed 09/07/2013

European Commission (2008) *Directive 2008/50/EC on Ambient Air Quality and Cleaner Air for Europe, Journal of the European Union*

H.M. Government (2010) *The Air Quality Standards Regulations SI 1001, the Stationary Office*

Miller B. and Hurley J., (2006) *Comparing estimated risks for air pollutants with risks for other health effects, Research Report TM/06/01, Institute of Occupational Medicine.*

Miller B, and Hurley J (2003) *Life table methods for quantitative impact assessments in chronic mortality Journal of Epidemiology and Community Health*.2003; 57: 200-206

Miller B., (2013) *IOMLIFET version 2013, Spreadsheets for life-table calculations, Institute of Occupational Medicine.*

Office for National Statistics (2018) URL: http://www.ons.gov.uk/ons/guide-method/method-quality/specific/index.html, Date accessed (20/08/2018)

Office for National Statistics (2017) *Health state life expectancies, UK: 2014 to 2016,* Accessed at URL: www.ons.gov.uk/ons/, Date accessed 13/09/2018

Office of Population Censuses and Surveys (1995) *Morbidity Statistics from General Practice, Fourth National Study 1991-1992.*

Pope CA, Burnett RT, Thun MJ, Calle EE, Kreswki D, Ito K, Thurston GD (2002) *Lung cancer, cardiopulmonary mortality and long-term exposure to fine particulate pollution. Journal of the American Medical Association 287 1132-1141*

Royal College of Paediatrics and Child Health and the Royal College of Physicians (2016) *Every Breath We Take: The Lifelong Impact of Air Pollution, Report of a Working Party, February 2016*

The Health and Social Care Information Centre (2018*) Hospital Episode Statistics: Hospital admitted patient care activity, England 2017-2018*, available at https://digital.nhs.uk/data-and-information/publications/statistical/hospital-admitted-patient-care-activity/2017-18https://files.digital.nhs.uk/publicationimport/pub16xxx/pub16719/hosp-epis-stat-admi-summ-rep-2013-14-rep.pdf - last accessed 06/11/2018

ATTACHMENT A

Figure 7B.1.1 – Nitrogen Dioxide Impacts and Population Density

THIS DRAWING IS TO BE USED ONLY FOR THE PURPOSE OF ISSUE THAT IT WAS ISSUED FOR AND IS SUBJECT TO AMENDMENT

LEGEND

Population Density

(People per Hectare)

- 0.2 - 10
- 10- 50
- 50 - 100
- 100 - 150
- 150 - 200
- Emission Sources
- Modelled Domain
- NO_2 Process Contribution

Zone	Max Conc. µg/m³	Total Peop.
1	0.015	2,297
2	0.025	64,235
3	0.035	54,006
4	0.05	18,550
5	0.075	6,077
6	0.100	1,690
7	0.2	1,269
8	2.1	117

Copyright
Reproduced from Ordnance Survey digital map data © Crown copyright 2020. All rights reserved. Licence number 0100031673.

Contains National Statistics data © Crown copyright and database right 2020. Adapted from the Office for National Statistics licenced under the Open Government Licence v.1.0

Contains Ordanance Surevey data © Crown copyright and database right 2020

Purpose of Issue
ENVIRONMENTAL STATEMENT

Client
EP WASTE MANAGEMENT LTD

Project Title
SOUTH HUMBER BANK ENERGY CENTRE DCO

Application Document Ref
NITROGEN DIOXIDE IMPACTS AND POPULATION DENSITY

Drawn	Checked	Approved	Date
AB	MH	GG	05/03/2020

AECOM Internal Project No.	Scale @ A3
60580855	1:80,000

THIS DOCUMENT HAS BEEN PREPARED PURSUANT TO AND SUBJECT TO THE TERMS OF AECOM'S APPOINTMENT BY ITS CLIENT. AECOM ACCEPTS NO LIABILITY FOR ANY USE OF THIS DOCUMENT OTHER THAN BY ITS ORIGINAL CLIENT OR FOLLOWING AECOM'S EXPRESS AGREEMENT TO SUCH USE, AND ONLY FOR THE PURPOSES FOR WHICH IT WAS PREPARED AND PROVIDED.

AECOM
One Trinity Gardens
Quayside
Newcastle upon Tyne
NE1 2HF
T +44 (0)191 224 6500
www.aecom.com

Drawing Ref
FIGURE 7B.1.1

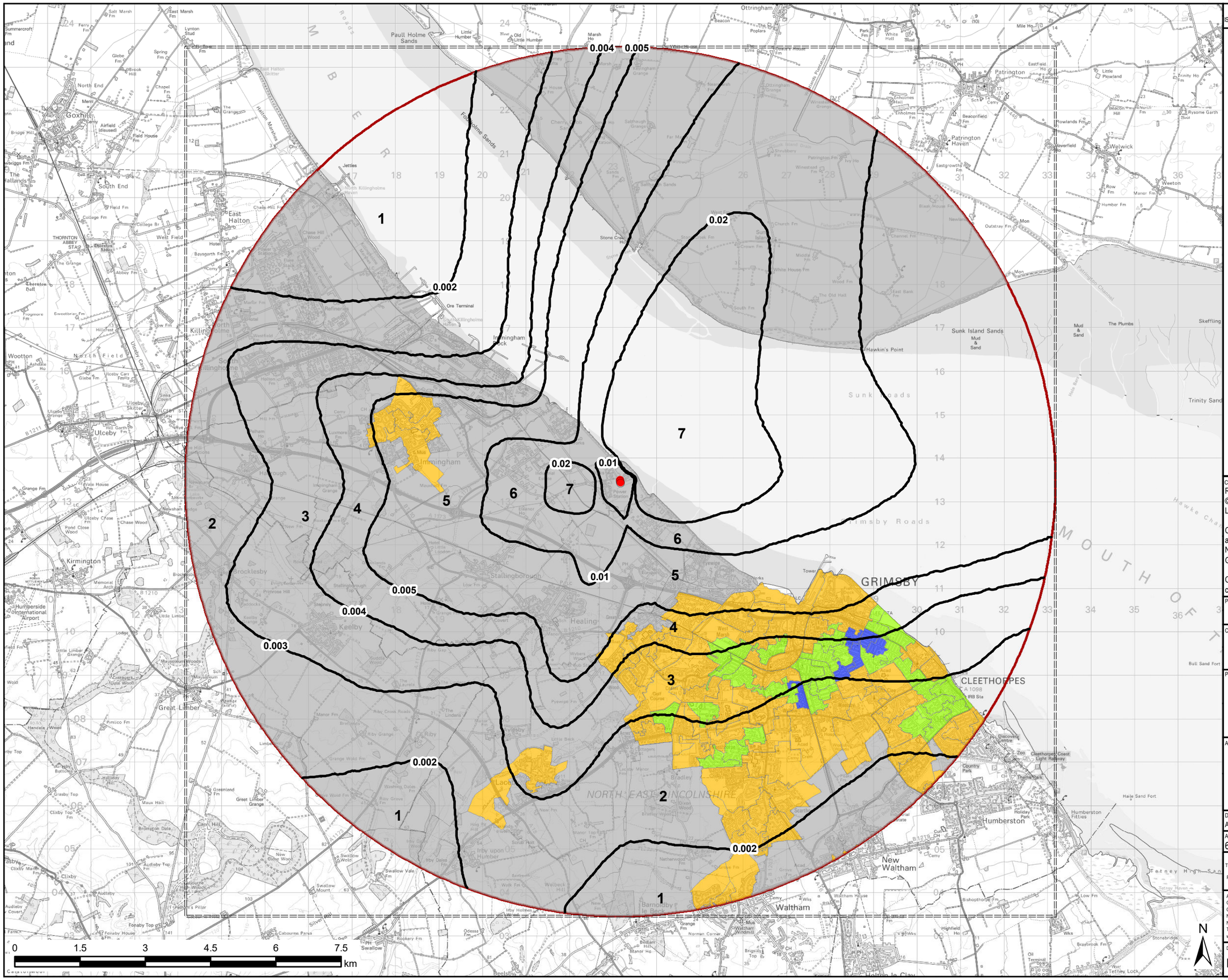
Rev

0 1.5 3 4.5 6 7.5 km

N

File Name:C:\Workspace\SHBEC\GIS\ABIFigure 7B1.1 NO2.mxd

Figure 7B.1.2 – Particulate Matter Impacts and Population Density



THIS DRAWING IS TO BE USED ONLY FOR THE PURPOSE OF ISSUE THAT IT WAS ISSUED FOR AND IS SUBJECT TO AMENDMENT

LEGEND

Population Density

(People per Hectare)

- 0.2 - 10
- 10- 50
- 50 - 100
- 100 - 150
- 150 - 200
- Emission Sources
- Modelled Domain
- Particulate Matter Process Contribution

Zone	Max Conc. µg/m³	Total Peop.
1	0.001	9,408
2	0.002	58,564
3	0.004	47,438
4	0.005	16,482
5	0.01	13,891
6	0.02	2,287
7	0.25	94

Copyright
Reproduced from Ordnance Survey digital map data © Crown copyright 2020. All rights reserved. Licence number 0100031673.

Contains National Statistics data © Crown copyright and database right 2020. Adapted from the Office for National Statistics licenced under the Open Government Licence v.1.0

Contains Ordanance Surevey data © Crown copyright and database right 2020

Purpose of Issue
ENVIRONMENTAL STATEMENT

Client
EP WASTE MANAGEMENT LTD

Project Title
SOUTH HUMBER BANK ENERGY CENTRE DCO

Application Document Ref
PARTICULATE MATTER IMPACTS AND POPULATION DENSITY

Drawn	Checked	Approved	Date
AB	MH	GG	05/03/2020

AECOM Internal Project No.	Scale @ A3
60580855	1:80,000

THIS DOCUMENT HAS BEEN PREPARED PURSUANT TO AND SUBJECT TO THE TERMS OF AECOM'S APPOINTMENT BY ITS CLIENT. AECOM ACCEPTS NO LIABILITY FOR ANY USE OF THIS DOCUMENT OTHER THAN BY ITS ORIGINAL CLIENT OR FOLLOWING AECOM'S EXPRESS AGREEMENT TO SUCH USE, AND ONLY FOR THE PURPOSES FOR WHICH IT WAS PREPARED AND PROVIDED.

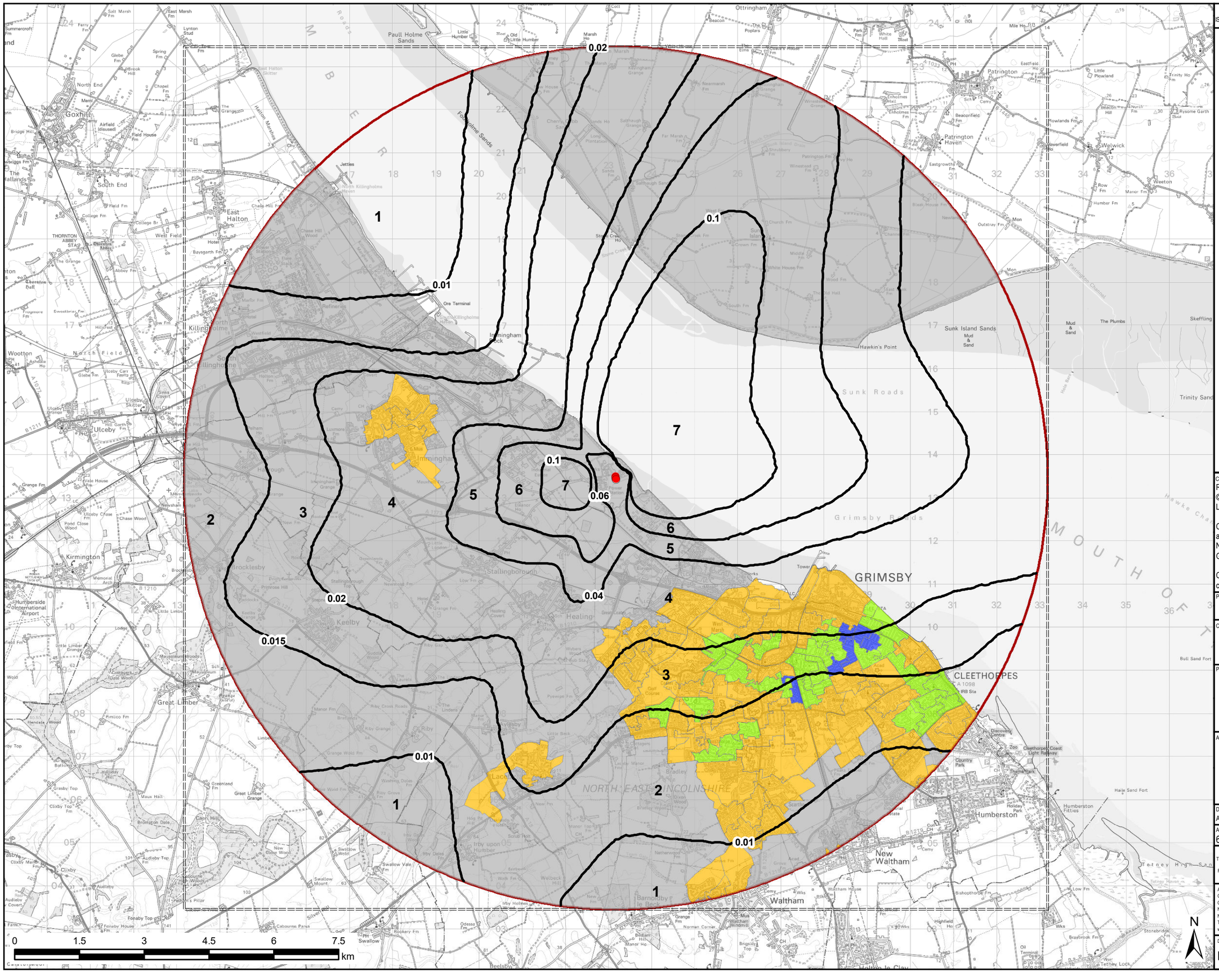
AECOM
One Trinity Gardens
Quayside
Newcastle upon Tyne
NE1 2HF
T +44 (0)191 224 6500
www.aecom.com

Drawing Ref
FIGURE 7B.1.2

Rev

File Name:W:\60580855 Project Koala AQ\2018 Application SSI\HRA\GIS\AB\Figure 7B.1.2 PM10.mxd

Figure 7B.1.3 – Sulphur Dioxide Impacts and Population Density



THIS DRAWING IS TO BE USED ONLY FOR THE PURPOSE OF ISSUE THAT IT WAS ISSUED FOR AND IS SUBJECT TO AMENDMENT

LEGEND

Population Density

(People per Hectare)

- 0.2 - 10
- 10- 50
- 50 - 100
- 100 - 150
- 150 - 200
- Emission Sources
- Modelled Domain
- SO_2 Process Contribtuion

Zone	Max Conc. µg/m³	Total Peop.
1	0.01	9,408
2	0.015	58,564
3	0.02	47,437
4	0.04	29,105
5	0.06	1,920
6	0.1	1,363
7	1.3	315

Copyright
Reproduced from Ordnance Survey digital map data © Crown copyright 2020. All rights reserved. Licence number 0100031673.

Contains National Statistics data © Crown copyright and database right 2020. Adapted from the Office for National Statistics licenced under the Open Government Licence v.1.0

Contains Ordanance Surevey data © Crown copyright and database right 2020

Purpose of Issue
ENVIRONMENTAL STATEMENT

Client
EP WASTE MANAGEMENT LTD

Project Title
SOUTH HUMBER BANK ENERGY CENTRE DCO

Application Document Ref
SULPHUR DIOXIDE IMPACTS AND POPULATION DENSITY

Drawn	Checked	Approved	Date
AB	MH	GG	05/03/2020

AECOM Internal Project No.	Scale @ A3
60580855	1:80,000

THIS DOCUMENT HAS BEEN PREPARED PURSUANT TO AND SUBJECT TO THE TERMS OF AECOM'S APPOINTMENT BY ITS CLIENT. AECOM ACCEPTS NO LIABILITY FOR ANY USE OF THIS DOCUMENT OTHER THAN BY ITS ORIGINAL CLIENT OR FOLLOWING AECOM'S EXPRESS AGREEMENT TO SUCH USE, AND ONLY FOR THE PURPOSES FOR WHICH IT WAS PREPARED AND PROVIDED.

AECOM
One Trinity Gardens
Quayside
Newcastle upon Tyne
NE1 2HF
T +44 (0)191 224 6500
www.aecom.com

Drawing Ref
FIGURE 7B.1.3

Rev

File Name:C:\Workspace\SHBEC\GIS\ABIFigure 7B1.3 SO2.mxd

ATTACHMENT B

Table 7B.1.1b: Predicted Number of Additional Cases of Selected Diseases Per Annum in the Exposed Population Based on Additional NO_2

DISEASE	EXTRA CASES PER ANNUM IN EACH EXPOSED POPULATION ZONE								TOTAL EXTRA CASES PER ANNUM
	1	2	3	4	5	6	7	8	
Cardiovascular Hospital Admissions	6.3×10^{-4}	2.9×10^{-2}	3.4×10^{-2}	1.7×10^{-2}	8.3×10^{-3}	3.1×10^{-3}	4.6×10^{-3}	4.5×10^{-3}	0.102
Respiratory hospital admissions	1.0×10^{-4}	4.8×10^{-3}	5.6×10^{-3}	2.7×10^{-3}	1.4×10^{-3}	5.0×10^{-4}	7.5×10^{-4}	7.3×10^{-4}	0.017
Deaths (non-traumatic brought forward)	9.3×10^{-5}	4.3×10^{-3}	5.1×10^{-3}	2.5×10^{-3}	1.2×10^{-3}	4.5×10^{-4}	6.8×10^{-4}	6.6×10^{-4}	0.015

Table 7B.1.2b: Predicted Number of Additional Cases of Selected Diseases Per Annum in the Exposed Population Based on Additional SO_2

DISEASE	EXTRA CASES PER ANNUM IN EACH EXPOSED POPULATION ZONE							TOTAL EXTRA CASES PER ANNUM
	1	2	3	4	5	6	7	
Respiratory hospital admissions	3.7×10^{-4}	3.4×10^{-3}	3.7×10^{-3}	4.5×10^{-3}	4.5×10^{-4}	5.3×10^{-4}	1.6×10^{-3}	0.015
Deaths (non-traumatic brought forward)	4.3×10^{-4}	4.1×10^{-3}	4.4×10^{-3}	5.4×10^{-3}	5.3×10^{-4}	6.3×10^{-4}	1.9×10^{-3}	0.017

Table 7B.1.3b: Predicted Number of Additional Cases of Selected Diseases Per Annum in the Exposed Population Based on Additional PM_{10}

DISEASE	EXTRA CASES PER ANNUM IN EACH EXPOSED POPULATION ZONE							TOTAL EXTRA CASES PER ANNUM
	1	2	3	4	5	6	7	
Chronic bronchitis (attack rates)	5.3×10^{-4}	6.6×10^{-3}	1.1×10^{-2}	4.6×10^{-3}	7.8×10^{-3}	2.6×10^{-3}	1.3×10^{-3}	0.034
Cardiovascular hospital admissions	7.9×10^{-5}	9.8×10^{-4}	1.6×10^{-3}	6.9×10^{-4}	1.2×10^{-3}	3.8×10^{-4}	2.0×10^{-4}	0.005
Respiratory hospital admissions	8.4×10^{-5}	1.0×10^{-3}	1.7×10^{-3}	7.3×10^{-4}	1.2×10^{-3}	4.1×10^{-4}	2.1×10^{-4}	0.005
Consultation with GPs (asthma, April – Sept, 15 – 64 years age)	1.9×10^{-3}	2.3×10^{-2}	3.8×10^{-2}	1.6×10^{-2}	2.8×10^{-2}	9.1×10^{-3}	4.7×10^{-3}	0.121
Lower respiratory symptoms (wheeze, shortness of breath, phlegm production) (in children)	1.2×10^{-4}	1.5×10^{-3}	2.5×10^{-3}	1.1×10^{-3}	1.8×10^{-3}	5.9×10^{-4}	3.1×10^{-4}	0.008
Lower respiratory symptoms (in adults)	3.3×10^{-5}	4.1×10^{-4}	6.6×10^{-4}	2.9×10^{-4}	4.8×10^{-4}	1.6×10^{-4}	8.2×10^{-5}	0.002

ANNEX 7B.2: ASSESSMENT OF HEALTH EFFECTS ARISING FROM EMISSIONS OF METALS AND ORGANIC SUBSTANCES

EP UK Investments

South Humber Bank Energy Centre Project

Planning Inspectorate Reference: EN010107

South Marsh Road, Stallingborough, DN41 8BZ

The South Humber Bank Energy Centre Order

Document Ref. 6.4 Appendix 7B: Human Health Risk Assessment Annex 7B.2: Assessment of Health Effects Arising from Emissions of Metals and Organic Substances

The Infrastructure Planning (Environmental Impact Assessment) Regulations 2017 (as amended)

The Infrastructure Planning (Applications: Prescribed Forms and Procedure) Regulations 2009 - Regulation 5(2)(a)

Applicant: EP Waste Management Ltd
Date: April 2020

DOCUMENT HISTORY

Document Ref	HHRA Annex 2		
Revision	1.0		
Author	M. Hill		
Signed		Date	April 2020
Approved By	G. Gray		
Signed		Date	April 2020
Document Owner	AECOM		

GLOSSARY

Term	Description
Acute effects	An effect that occurs within a short time after exposure.
Average Daily Dose	The estimated mean dose received by an individual over the course of a day.
Averaging Time	A reference time period e.g. an average daily dose is reported for an averaging time of one day.
Bioaccumulation	The process by which chemicals are taken up into an organism either directly by exposure or indirectly through consumption of contaminated material. Concentrations can accumulate higher up the food chain to levels significantly higher than the original exposure concentration.
Carcinogenic Slope Factor	An upper bound on the increased cancer risk from a lifetime of oral (ingestion) exposure to a substance based on the dose-response relationship of the substance.
Chemicals of Potential Concern	Substances identified through the risk assessment process as being of concern to human health.
Chronic effects	An effect that occurs over a long time period or following a long period of exposure.
Congeners	Substances with molecules that share slightly different chemical structures.
Dioxins/Furans	This is the abbreviated name for a family of toxic substances that share a similar chemical structure and a common mechanism of toxic action. They include the congeners polychlorinated dibenzo dioxins (PCDDs) and polychlorinated dibenzo furans (PCDFs).
Dose	The USEPA define ‘Dose’ as, the amount of a substance available for interaction with metabolic processes or biologically significant receptors after crossing the exchange boundary of an organism. An equivalent definition is, the amount of a substance taken up by an exposed individual following inhalation, ingestion or absorption across the skin.
Dose-response relationship	The relationship between the dose and the proportion of exposed individuals observed to demonstrate effects.

Term	Description
Emission	The substance or the mass of a substance emitted into the atmosphere.
Excess Lifetime Risk	The probability that an individual will develop cancer over a lifetime as a result of exposure to specific carcinogenic chemicals through multiple exposure pathways.
Exposure	The US EPA define 'exposure' as, the condition of a chemical contacting the exchange boundary of an organism. A broader definition is, the amount of a substance inhaled, ingested or present at the skin surface.
Exposure (Direct)	Inhalation of air containing substances at predicted concentrations.
Exposure (Indirect)	Results from contact of human and ecological receptors with soil, plants or water bodies on which emitted chemicals have been deposited.
Exposure Duration	The length of time that a receptor is exposed via a specific pathway.
Exposure Frequency	This is the amount of time a receptor is exposed to COPCs by all pathways. The HHRAP assumes that receptors are exposed 350 days a year, with a 2 week period away from the relevant exposure location.
Exposure Pathway	This is the route that a chemical takes from its source, through the environment to the individual being exposed.
Exposure Scenario	The combination of relevant exposure pathways to which an individual receptor may be exposed to specific substances.
Hazard	An impact to human health by chemicals of potential concern.
Hazard Index	The total chronic hazard attributable to exposure to all COPCs through a single exposure pathway.
Hazard Quotient	The comparison of oral and inhalation exposure estimates to reference dose and reference concentration values.
Human Health Risk Assessment Protocol	A structured approach to quantifying the risks to human health associated with exposure to compounds of potential concern.
Ingestion	The act of eating or drinking a substance that may then result in the substance being taken up via the digestive system.
Inhalation	The act of breathing in a substance that may then result in the substance being taken up via the respiratory system.
Industrial Risk Assessment Program	A commercially available computer programme developed to calculate excess life time risk and hazard index values following the requirements from the 2005 U.S. EPA-OSW Human Health Risk Assessment Protocol.
Industrial Emissions Directive	A directive of the European Union, the requirements of which will replace requirements of the Waste Incineration Directive (WID) by 2013.
International Toxic Equivalent	This weighs the toxicity of the less toxic compounds as a fraction of the toxicity of a reference compound. In the case of dioxins the

Term	Description
	toxicity of each individual congener is weighted to 2,3,7,8-TCDD, which is given a reference value of 1.
Lifetime	In estimating the average lifetime exposure of individual receptors or populations to substances, a lifetime is taken to be 70 years.
Lipophilic	A substance is considered lipophilic if it is readily dissolved in fat-like solvents.
Media	For the purposes of this assessment, media are parts of the wider environment that a substance could be contained within. This includes soil, water, air, biota etc.
Metals	The 12 metals, in their elemental form or contained within compounds, for which emission limit values are defined within the Waste Incineration Directive.
Nitrogen Dioxide	A molecule composed of one nitrogen atom and two oxygen atoms, present in outdoor air as a gas.
Oxides of Nitrogen	A collective term for all gases composed of nitrogen and oxygen, including nitrogen dioxide.
Particulate Matter	A solid or liquid particle (a droplet) that in the context of this report is small enough to be suspended in air.
PCB	This is the abbreviated name for a family of toxic substances that share a similar chemical structure and a common mechanism of toxic action called Polychlorinated Biphenyl.
PM10	Mass of particles per cubic metre of air passing through the inlet of a size selective sampler with a transmission efficiency of 50% at an aerodynamic diameter of 10 micrometres.
PM2.5	Mass of particles per cubic metre of air passing through the inlet of a size selective sampler with a transmission efficiency of 50% at an aerodynamic diameter of 2.5 micrometres.
Pathway	A term used to represent a series of sequential physical or chemical actions by which a substance is transported from a source, through the environment to a receptor. Typically described using a label that relates to the mechanism that receptors are exposed by, e.g. inhalation pathway.
Polycyclic aromatic hydrocarbons	A group of several hundred chemically related persistent organic compounds of various chemical structures and toxicity. Benzo[a]pyrene is used in National air quality regulations as a marker species for reporting concentrations of PAH in ambient air.
Population	All individuals living within a defined area.
Receptor	For the purposes of the human health risk assessment a receptor is, a hypothetical individual potentially exposed to chemicals of potential concern emitted to the atmosphere from the Proposed Development in question.
Reference Concentration	An estimated daily concentration of a chemical in air, the exposure to which over a specific exposure duration poses no appreciable risk of adverse health effects, even to sensitive populations.
Reference Dose	A daily oral intake rate that is estimated to pose no appreciable risk of adverse health effects, even to sensitive populations, over a 70

Term	Description
	year lifetime.
Risk	An estimation of the probability that an adverse health impact may occur as a result of exposure to chemicals in the amount and by the pathways identified.
Sulphur Dioxide	A molecule composed of one sulphur and two oxygen atoms, present in outdoor air as a gas.
Threshold	The dose or exposure level below which no appreciable effects on human health are observed.
Tolerable Daily Intake	A World Health Organisation definition of the dose of a substance that an individual could be exposed to on each day of an entire lifetime, at which appreciable health risks do not occur. See similar 'reference dose' term used by USEPA.
Unit Risk Factor	The upper bound excess lifetime cancer risk estimated to result from continuous exposure to a substance at a concentration of 1µgm-3 in air.
Waste Incineration Directive	A directive of the European Union that defines the minimum standard of environmental performance that must be achieved by installations burning waste or waste derived fuels.

ABBREVIATIONS

Abbreviation	Description
ADD	Average Daily Dose
COPC	Compound of Potential Concern
COT	Committee on Toxicology
CSF	Cancer Slope Factor
FSA	Food Standards Agency
HHRAP	Human Health Risk Assessment Protocol
HQ	Hazard Quotient
HI	Hazard Index
IED	Industrial Emissions Directive
IRAP	Industrial Risk Assessment Program
TEF	Toxic Equivalency Factor
PAH	Polycyclic aromatic hydrocarbon
PCB	Polychlorinated Biphenyl
PCDD	Polychlorinated di benzo(p)dioxin
PCDF	Polychlorinated di benzo furans
RfD	Reference Dose
RfC	Reference Concentration
SGV	Soil Guideline Values
TDS	Total Dietary Study
TDI	Tolerable Daily Intake
URF	Unit Risk Factor
US EPA	United States Environmental Protection Agency
WHO	World Health Organisation
WID	Waste Incineration Directive

CONTENTS

TABLES

FIGURES

ANNEX 7B.2 - HEALTH EFFECTS ARISING FROM EMISSIONS OF METALS AND ORGANIC SUBSTANCES

7B.2.1 Introduction

7B.2.1.1 This Annex to Appendix 7B of the ES Volume III (Document Ref. 6.4) quantifies the human health effects associated with the exposure of the local community, within 10 km of the Proposed Development (the Study Area), to the predicted change in atmospheric concentrations of metals, PAHs and dioxins/ furans and dioxin-like PCBs. This is a much larger Study Area than is strictly necessary, but provides a consistent Study Area to that used within other elements of the air quality assessment for other air quality receptor types. The emissions have been calculated using the methodology set out in the air quality dispersion modelling report (Appendix 7A in ES Volume III, Document Ref. 6.4).

7B.2.1.2 Initially, the Waste Incineration Directive (WID) 2000/76/EC (EC, 2000) regulated the burning of waste derived fuels and waste, where waste is used as a fuel or is disposed of at a plant where energy generation or production is the main purpose. The Directive defined operating conditions for the incineration process, emission monitoring requirements and limit values for emission of substances to air and water. The WID directive was transposed into national legislation through the Environmental Permitting (England and Wales) Regulations 2010.

7B.2.1.3 The Industrial Emissions Directive (IED) 2010/75/EU (EU, 2010) incorporated a number of directives, including WID, into a single overall directive. The emission limit values and operating conditions specified within WID have been retained within the IED and have applied in respect of any installation of new facilities in England since 6 January 2013. Within the context of this assessment, the IED applies to emission limits of metals and organic substances. In addition to the IED, an updated BAT reference document (Joint Research Centre, 2017) has been produced in draft form that provides more stringent emission limits compared to the IED. Where relevant, these emission limits have been applied in this assessment.

7B.2.1.4 The methodology for assessing the effects on human health from such facilities is based on the United States Environmental Protection Agency (US EPA) Human Health Risk Assessment Protocol (HHRAP) (US EPA, 2005). This provides a systematic and transparent protocol for undertaking site-specific risk assessments of human exposure to emissions from combustion facilities. The main steps within the HHRAP are:

- characterising the source of the hazard;
- identifying the relevant pathways via which receptors could be exposed;
- calculating concentrations of COPCs in environmental media;
- calculating the magnitude of human exposure; and
- quantifying the risk of health effects.

7B.2.1.5 This report applies the HHRAP methodology published by the US EPA to quantify the risks of human health effects from exposure to metals, PAHs and dioxins/ furans and dioxin-like PCBs, associated with the operation of the Proposed Development. The HHRAP encompasses more than a decade of research into the risk assessment of combustion facilities on the subject of hazard identification and health risks. No further review of the underpinning medical literature has been undertaken in support of this document.

7B.2.1.6 The relationship between exposure to air pollutants, either singly or in combination, and the resulting effects on health remains a topic of active research. Although emissions from the Proposed Development stacks are initially airborne substances, inhalation is not the only relevant exposure pathway for some of the substances of concern. A more detailed assessment of all exposure pathways needs to be undertaken for the risks to be quantified and HHRAP adopts such a source – pathway - receptor approach.

7B.2.1.7 Taking a generic example, where a stack is the source and the substance emitted into the atmosphere is a potential hazard to human health. The people that make up the population of the land surrounding the stack are receptors that may be exposed to a dose of the substance. The substance might move through the environment via a number of available pathways before the receptors are exposed to it. One pathway might be dispersion through the atmosphere followed by inhalation into the receptor's lungs. Another pathway might be deposition from the atmosphere onto the ground, followed by uptake into plants that are then eaten by livestock, which are then in turn eaten by receptors.

7B.2.1.8 If a receptor was to live their entire life at a location where they breathed the substance at the highest airborne concentrations and they only ate locally grown food and drank local water from the location where the concentrations of the deposited substance where highest, then they would experience the maximum hypothetical level of exposure to emissions from that stack.

7B.2.1.9 Within HHRAP the health impact on the entire exposed population is characterised using six types of receptors to represent hypothetical maximum exposure scenarios:

- the resident (adult) and resident's child;
- the farmer (adult) and farmer's child; and
- the fisher (adult) and fisher's child.

7B.2.1.10 The receptor locations within the assessment have been chosen for each receptor type, based upon the predicted maximum concentrations from the air quality dispersion modelling report (Appendix 7A in ES Volume III, Document Ref. 6.4). This enables the potential health effects for the exposed population to be quantified, based on the maximum dose that a representative receptor within the study area is likely to be exposed to.

7B.2.1.11 The Compounds of Potential Concern (COPCs) considered within this report have the potential to induce long term, chronic effects on human health at

environmental concentrations. For some of these substances there is no minimum concentration below which adverse health effects will not occur and it is therefore appropriate to consider the risk of effects occurring. The receptors considered in this assessment are representative of the maximum hypothetical lifetime risk of human health effects that members of the population would be exposed to. For the purposes of this assessment, risks are presented for lifetimes of 70 years duration for an adult receptor and 6 years duration for a child receptor. The assessment quantifies the risk for carcinogenic effects and for non-carcinogenic effects and reports these risks using internationally recognised metrics.

7B.2.1.12 In this report the terminology used is necessarily technical and the meaning of the terms may differ from their use in conversational English. A glossary of the terms used is provided within this report.

7B.2.2 Methodology

Introduction

7B.2.2.1 This assessment considers the risk of effects on human health occurring within the local population when exposed to emissions to air from the Proposed Development, located near Grimsby, North East Lincolnshire. The approach to this assessment is as follows:

- characterising the source of the hazard;
- identifying the relevant pathways via which receptors could be exposed;
- calculating concentrations of COPCs in environmental media;
- calculating the magnitude of human exposure; and
- quantifying the risk of health effects.

7B.2.2.2 The hazard source consists of COPCs, which are substances emitted from waste treatment facilities at rates permitted under the Industrial Emissions Directive. The hazard source has been previously quantified through a detailed dispersion modelling exercise that has reported on substances emitted and dispersed within the atmosphere, and the amount of COPCs deposited to ground (Appendix 7A in ES Volume III (Document Ref. 6.4)).

7B.2.2.3 The relevant exposure pathways are identified as either direct (inhalation) or indirect (ingestion of water, soil, vegetation and animal products contaminated through the food chain). The receptors are chosen based on the results of the maximum predicted concentrations from the air quality dispersion modelling report and surrounding site specific conditions.

7B.2.2.4 The level of exposure and dose to COPCs via each pathway can be calculated for each receptor once the source, exposure pathways and receptors have been quantified. Ultimately a total risk for carcinogenic and non-carcinogenic effects occurring in each of the receptors from the various different exposure scenarios is calculated.

7B.2.2.5 The current and future land use, the location of water bodies and associated watersheds and any special population characteristics (e.g. infants or elderly) are considered within the assessment of exposure to COPCs.

7B.2.2.6 The risk of effects on human health arising from exposure to dioxins, furans, dioxin-like PCBs, PAHs and metals emitted from the Proposed Development are estimated for hypothetical worst case scenarios, including that of an individual exposed for a lifetime to the effects of the highest airborne concentrations and consuming mostly locally grown food.

7B.2.2.7 The methods outlined in the US EPA HHRAP have been encompassed into a commercially available risk assessment modelling tool called the Industrial Risk Assessment Program (IRAP) by Lakes Environmental Software. AECOM holds a user licence for the latest version of this software (5.0.0), which has been used to conduct the assessment of the risks to humans via the method outlined above.

7B.2.2.8 HHRAP has been specifically developed to enable the estimation of the level of exposure received by the local population via the combination of potential exposure pathways in a consistent and repeatable manner. HHRAP considers the fate and transport of substances through soil, water and biota (plant material) following deposition onto these surfaces. This is then used to calculate the potential uptake of these substances by the receptors affected by the relevant pathways.

7B.2.2.9 Within HHRAP the receptors chosen are classified as either a resident, farmer or fisher receptor types. It is also necessary to distinguish between an adult and child receptors as children are considered to be at a greater risk of experiencing health effects from a specified dose due to their lower body weights. The farmer receptor is assumed to consume proportionally more locally grown food than a resident. This means that these receptors are at a greater risk of eating food contaminated by emissions from the source. A fisher receptor type is utilised where there is the potential for the consumption of locally caught fish from water bodies affected by emissions form the source to constitute the main source of protein within the receptors diet. For resident type receptors it is assumed that they are home gardeners within an urban area and as such consume locally grown produce with some incidental ingestion of soil. All receptor types are assumed to be present at the same location all year apart from a 2 week holiday period (350 days).

7B.2.2.10 The air quality dispersion modelling report (Appendix 7A in ES Volume III, Document Ref. 6.4) generates output files that are imported into the IRAP model to calculate concentrations of COPCs within each exposure pathway that are ultimately taken up by human receptors. In order to perform this calculation IRAP requires the following input parameters:

- physical and chemical properties of COPCs;
- site specific information e.g. precipitation rate, wind speed; and
- information for each receptor type e.g. body weight, consumption rates of food, exposure rates.

7B.2.2.11 The HHRAP default values have been incorporated within IRAP and are used for the majority of input values, as discussed in the following sections.

Hazard Source

7B.2.2.12 Throughout its operational lifetime the Proposed Development `will emit a number of different substances into the atmosphere via two stacks, which are referred to in this assessment as Compounds of Potential Concern (COPCs). The IED specifies plant operating conditions (e.g. temperature and residence times) as well as emission limit values, which represent an upper limit on the permitted concentrations of COPCs that can be emitted from the Proposed Development. The emission limits specified in the IED are set out in Table 7B.2.1. The concentrations used in the assessment are presented in Table 7B2.2.

Table 7B.2.1: Daily Averaged Emissions Limit Values in the IED

POLLUTANT	**EMISSION LIMIT VALUE** (MG/M^3)	**AVERAGING PERIOD**
Total dust	10	Daily mean
Gaseous and vaporous organic substances, expressed as total organic carbon	10	Daily mean
Hydrogen Chloride (HCl)	10	Daily mean
Hydrogen Fluoride (HF)	1	Daily mean
Sulphur Dioxide (SO_2)	50	Daily mean
Nitrogen monoxide (NO) and nitrogen dioxide (NO_2), expressed as nitrogen dioxide for existing incineration plants with a nominal capacity exceeding 6 tonnes per hour or new incineration plants	200	Daily mean
Carbon Monoxide (CO)	50	Daily mean
Cadmium (Cd) and Thallium (Tl)	Total 0.05	All average values over the sampling period 30 minutes to 8 hours
Mercury (Hg)	0.05	
Antimony (Sb), Arsenic (As), Lead (Pb), Chromium (Cr), Cobalt (Co), Copper (Cu), Manganese (Mn), Nickel (Ni) and Vanadium (V)	Total 0.5	
Dioxins	0.1 ng I-TEQ / Nm^3	CEN method, sample period 6 to 8 hours

Compounds of Potential Concern

7B.2.2.13 The COPCs of relevance to this assessment are permitted emissions under IED (shown in Table 2 1). S pecific physical and chemical information on these substances is included within the US EPA HHRAP COPC companion database for the assessment of long term health effects. The particular substances considered with regards to the assessment of their effects on human health are listed below:

- polychlorinated di benzo(p)dioxins/furans (PCDD/F) as individual congeners;
- benzo(a)pyrene;
- antimony (Sb);
- arsenic (As);
- cadmium (Cd);
- chromium (Cr), trivalent and hexavalent;
- mercury (Hg);
- lead (Pb); and
- nickel (Ni).

7B.2.2.14 Benzo(a)pyrene has been included in the list of COPCs to represent polycyclic aromatic hydrocarbons (PAHs) emissions within this assessment. Although no emission limits are specified under IED, monitoring of these substances is required under the directive.

7B.2.2.15 The Committee on Carcinogenicity of Chemical in Food, Consumer Products and the Environment (CoC) reviewed the methodology for the risk assessment of polycyclic aromatic hydrocarbons (CoC, 2010) (PAHs). In the review, the Committee recommended that four PAHs should be used in order to assess the health effects of PAHs, an approach derived from the European Food Standards Agency (EFSA) (EFSA, 2008). The four PAHs to be assessed are benzo[a]pyrene, benzo[a]anthracene, benzo[b]fluoranthene, and Chrysene.

7B.2.2.16 The 2005 HHRAP excluded thallium (Tl) by virtue of there being no reference dose, reference concentration or cancer slope factors available for thallium. This is contrast to the draft 1998 HHRAP which did include compound specific parameter values for thallium in Appendix A of the draft 1998 US EPA HHRAP (US EPA, 1998). The physical and chemical properties of thallium are well known and it has been considered appropriate to include thallium in the list of COPCs for the assessment of any human health effects. Therefore, the 1998 US EPA HHRAP reference data has been used to assess the risk to human health associated with exposure of the local population to thallium.

7B.2.2.17 12 congeners of Polychlorinated Biphenyls (PCBs) have been included, in addition to PCDD and PCDF. These 12 congeners display dioxin-like properties and could contribute to possible health effects. The total additional risks to health from these congeners are much smaller than the risk from the PCDDs and PCDF, and have been included for completeness.

Emission Concentration

7B.2.2.18 The emission concentrations of the COPCs considered in this assessment have been reported in the air quality dispersion modelling report (Appendix 7A in ES Volume III, Document Ref. 6.4). The IED places limit values on the emissions of substances in the short term i.e. daily averages, which have been used as a conservative assumption within this assessment of long term health effects. In addition to the IED, alternative Best Available Technique Achievable Emission Levels (BAT-AELs) have been published in the revised draft BREF, and guidance published by the Environment Agency, which differ from the IED emission limits (EA, 2016, Joint Research Centre, 2017). These emission limits are generally lower than those included in the IED, and have been used in this assessment in place of those used in the IED.

Table 7B.2.2: Daily Averaged Emissions Limit Values used in the assessment.

METAL GROUP DEFINED IN IED	POLLUTANT	EMISSION CONCENTRATION[(A)] (mg Nm^{-3})	EMISSION RATE (gs^{-1})
Group 1	Cadmium	0.02	0.00133
	Thallium	0.02	0.00133
Group 2	Mercury	0.02	0.00133
Group 3	Antimony	0.0115	0.00077
	Arsenic	0.025	0.00166
	Total chromium	0.092	0.00612
	Chromium (vi)	0.00013	0.00001
	Lead	0.0505	0.00335
	Nickel	0.22	0.01464

a) Emission concentrations for individual metals have been based upon the Environment Agency's Interim guidance on group 3 metals for waste incineration (EA, 2016), with the exception of cadmium, mercury and Thallium, for which the 2017 Draft Waste Incineration BAT Reference document (JRC, 2017) limit values have been used.

7B.2.2.19 The individual emissions concentrations and rates for each of the inorganic COPCs are shown in

7B.2.2.20 Table 7B.2.2. Some of the metals with specified emission limits in the IED do no not pose a risk to human health in the long term and have not been included within the HHRAP e.g. cobalt, copper, manganese and vanadium. These metals have therefore been excluded from this assessment of the risks to human health.

7B.2.2.21 The concentration of mercury has been adjusted in order to take account of the loss of mercury to the global cycle. The default values within IRAP assume that 48% of total mercury is deposited as divalent mercury (mercuric chloride), 2% is deposited as elemental mercury and the rest being lost to the global cycle. IRAP assumes that the exposed population will only be exposed to elemental mercury through direct inhalation of the vapour phase whereas exposure to divalent mercury will occur via both direct and indirect inhalation of vapour and particle bound mercuric chloride. This leads to the following emission rates (in g/s) for elemental and divalent mercury:

- elemental mercury 2.66×10^{-6} ; and
- divalent mercury 6.38×10^{-4}.

7B.2.2.22 As stated above, four PAHs have been used to represent the total emission of PAHs. Monitoring of PAH releases from a municipal solid waste incinerator in the UK was undertaken (EA, 2005), and the following emission rates (in g/s) have been used:

- benzo[a]pyrene: 6.65×10^{-5};
- benzo[a]anthracene: 6.65×10^{-5};
- benzo[b]fluoranthene: 3.99×10^{-4}; and
- chrysene: 1.33×10^{-4}.

7B.2.2.23 Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are organic substances formed as a by-product of combustion processes and in the manufacture of certain chlorinated organic chemicals. PCDD/Fs have been classified as persistent organic pollutants (POPs) with a significant potential to bioaccumulate (WHO, 2010). The basic structure of the dioxin family is composed of benzene rings interconnected by two oxygen atoms. The degree and position of the chlorination to the basic structure determines the type of the individual dioxin with 75 individual compounds being possible. Furans are of a similar structure but with a carbon atom replacing one of the chlorine atoms yielding 125 individual furan compounds. Each individual compound is referred to as a congener and each has slightly different chemical and physical properties in the environment that are determined by the position and degree of chlorination within the molecule.

7B.2.2.24 The assessment of the effect of PCDD/Fs on human health takes into account the effect of the different physical and chemical properties of the individual congeners on their behaviour in the environment. Individual congeners are used to conduct the assessment of the health risk from dioxins/ furans. A standard PCDD/F emission profile for municipal waste

incinerators has previously been derived by Her Majesty's Inspectorate of Pollution (HIMP) (DOE, 1996) and will be used to represent the congener emission profile in this assessment (

7B.2.2.25 Table 7B.2.3).

7B.2.2.26 Toxic equivalency factors (TEF) are used to express the toxicities of the different PCDD/Fs in relation to the most toxic dioxin 2,3,7,8-TCDD. The TEF has been used to calculate the toxic equivalency (WHO-TEQ) for each congener, and the total TEQ for all PCDD/Fs (including dioxin-like PCBs) has been assumed to be no more than that of the maximum 2017 Draft Waste Incineration BREFBREF emission limit value of 0.06 ng WHO-TEQ Nm^{-3} for PCDD/Fs and dioxin-like PCBs. The emissions reported by the HMIP have been factored so that the total emission rate (in WHO-TEQ Nm^{-3}) is no more than the emission limit value.

Table 7B.2.3: Congener Profile for the Proposed Development for all of the PCDD/Fs

CONGENER	ANNUAL MEAN EMISSION CONCENTRATION (ng Nm^{-3})	WHO-TEF (Toxic Equivalent Factors)	ANNUAL MEAN EMISSION RATE (ng WHO-TEQ Nm^{-3})
2,3,7,8-TCDD	0.0031	1	0.00101
1,2,3,7,8-PeCDD	0.025	1	0.00812
1,2,3,4,7,8-HxCDD	0.029	0.1	0.00094
1,2,3,6,7,8-HxCDD	0.026	0.1	0.00068
1,2,3,7,8,9-HxCDD	0.021	0.1	0.00084
1,2,3,4,6,7,8-HpCDD	0.17	0.01	0.00055
OCDD	0.4	0.0003	0.00004
2,3,7,8-TCDF	0.028	0.1	0.00091
2,3,4,7,8-PeCDF	0.054	0.3	0.00526
1,2,3,7,8-PeCDF	0.028	0.03	0.00027
1,2,3,4,7,8-HxCDF	0.22	0.1	0.00715
1,2,3,6,7,8-HxCDF	0.081	0.1	0.00013
1,2,3,7,8,9-HxCDF	0.0040	0.1	0.02632
2,3,4,6,7,8-HxCDF	0.087	0.1	0.00283
1,2,3,4,6,7,8-HpCDF	0.44	0.01	0.00143
1,2,3,4,7,8,9-HpCDF	0.04	0.01	0.00013
OCDF	0.4	0.0003	0.00004
Total (ng WHO-TEQ m-3)			0.057[(a)]

a) sum of all PCDD/Fs and dioxin-like PCBs should be no more than 0.06 ng IWHO-TEQ Nm-3

7B.2.2.27 The emissions rates used in the IRAP model for each of the PCDD/Fs are shown in Table 7B.2.4. These rates have been calculated based upon the percentage contribution of each congener to the total emission rates of all dioxin/furans at the Draft emissions limits.

Table 7B.2.4: Emission Rates Used in the IRAP Model for all of the PCDD/Fs

CONGENER	EMISSION RATE PER STACK (gs^{-1})
2,3,7,8-TCDD	6.7×10^{-11}
1,2,3,7,8-PeCDD	5.4×10^{-10}
1,2,3,4,7,8-HxCDD	6.3×10^{-10}
1,2,3,6,7,8-HxCDD	4.5×10^{-10}
1,2,3,7,8,9-HxCDD	5.6×10^{-10}
1,2,3,4,6,7,8-HpCDD	3.7×10^{-9}
OCDD	8.6×10^{-9}
2,3,7,8-TCDF	6.1×10^{-10}
2,3,4,7,8-PeCDF	1.2×10^{-9}
1,2,3,7,8-PeCDF	6.1×10^{-10}
1,2,3,4,7,8-HxCDF	4.8×10^{-9}
1,2,3,6,7,8-HxCDF	8.6×10^{-11}
1,2,3,7,8,9-HxCDF	1.8×10^{-9}
2,3,4,6,7,8-HxCDF	1.9×10^{-9}
1,2,3,4,6,7,8-HpCDF	9.5×10^{-9}
1,2,3,4,7,8,9-HpCDF	8.6×10^{-10}
OCDF	8.6×10^{-9}

7B.2.2.28 Emission rates of 12 congeners of Polychlorinated Biphenyls (PCBs) were calculated using the maximum emission rates measured from 24 MWIs, provided by the Environment Agency (EA, 2015). PCBs are products of combustion, and were produced on an industrial scale for a variety of industrial and commercial uses due to their stability, low volatility, and low conductance, before being phased out in the 1970s. PCBs are therefore extremely persistent in the environment, and are likely to be detected to some degree throughout much of the UK. PCBs consist of two benzene rings connected by a single bond between a carbon atom in each ring. The position and degree of chlorination means that there are a total of 209 possible congeners of PCB. Due to the position and number of the chlorine atoms in 12 congeners, the PCB molecule is able to rotate about the bond between the two rings, and mimics PCDDs and PCDFs. These congeners are referred to as coplanar or dioxin-like, and TEFs to 2,3,7,8-TCDD have been derived for these 12 to enable an assessment of their toxicity.

7B.2.2.29 The US HHRAP includes information relating to the assessment of PCBs and their effects on human health. While the IRAP h-View program doesn't include information of each congener of PCB, the HHRAP states that Aroclor 1254 (included as a COPC in the IRAP h-View database) can be used for their physical and chemical properties. Other factors, such as their Oral Cancer Slope Factor are given in the HHRAP, and are based on the number of chlorine atoms within each PCB molecule.

7B.2.2.30 The measured emission concentrations and associated TEFs used in the IRAP model are shown in Table 7B.2.5.

Table 7B.2.5: Congener Profile for the Proposed Development for all of the PCBs

CONGENER	ANNUAL MEAN EMISSION CONCENTRATION (ng Nm^{-3})	I-TEF(Toxic Equivalent Factors)	ANNUAL MEAN EMISSION (ng I-TEQ Nm^{-3})
PCB77	0.87	0.0001	8.71×10^{-5}
PCB81	0.05	0.0003	1.61×10^{-5}
PCB126	0.08	0.1	8.23×10^{-3}
PCB169	0.06	0.03	1.71×10^{-3}
PCB105	0.67	0.00003	2.00×10^{-5}
PCB114	0.76	0.00003	2.28×10^{-5}
PCB118	3.33	0.00003	1.00×10^{-4}
PCB123	0.33	0.00003	1.00×10^{-5}
PCB156	2.33	0.00003	7.00×10^{-5}
PCB157	0.76	0.00003	2.28×10^{-5}
PCB167	0.19	0.00003	5.70×10^{-6}
PCB189	0.15	0.00003	4.57×10^{-6}

7B.2.2.31 The emission rates of the individual PCB congeners are used in the IRAP model are shown in Table 7B.2.6. These rates have been calculated based on the concentrations reported in Table 7B.2.5 and the volumetric flow rate from the stacks.

Table 7B.2.6: Emission Rates Used in the IRAP Model for all of the PCDD/Fs

CONGENER	EMISSION RATE PER STACK (gs^{-1})
PCB77	1.9×10^{-8}
PCB81	1.2×10^{-9}
PCB126	1.8×10^{-9}
PCB169	1.2×10^{-9}
PCB105	1.4×10^{-8}
PCB114	1.6×10^{-8}
PCB118	7.2×10^{-8}
PCB123	7.2×10^{-9}
PCB156	5.0×10^{-8}
PCB157	1.6×10^{-8}
PCB167	4.1×10^{-9}
PCB189	3.3×10^{-9}

Properties of COPCs

7B.2.2.32 The HHRAP includes a database that defines the physical and chemical properties of 206 COPCs, as well as toxicity factors for each COPC. This database is the source of the default values within the IRAP model. The physical and chemical properties determine how each of the COPCs would

move within the environment and the extent to which they would bio-concentrate in different foodstuffs (e.g. meat, fish, vegetation, soil and water). An example of the range of different properties used within IRAP is presented in Table 7B.2.7. Data for lead and 2,3,7,8-TCDD are included in Table 7B.2.7 to provide an illustration of the marked differences in the properties associated with organic and inorganic substances.

7B.2.2.33 Toxicity benchmarks (e.g. reference dose/concentrations, slope factors, unit risk factors) with regards to human health effects are shown in Table 7B.2.8 for all of the COPCs considered in this assessment. These values are provided in the HHRAP and used to determine the carcinogenic and non-carcinogenic risks associated with inhalation or ingestion exposure to each of the COPCs.

7B.2.2.34 The Carcinogenic Slope Factor (CSF) and Unit Risk Factors (URF) for each COPC are used to calculate the carcinogenic risk from ingestion and inhalation respectively. The ingestion Reference Dose (RfD) and Inhalation Reference Concentration (RfC) are used to calculate the non-carcinogenic risk associated with exposure to each COPC. The detailed methodology for calculating the non-carcinogenic and carcinogenic risks to human health are provided in section 7B.2.6 and 7B.2.7 respectively.

Table 7B.2.7: Example IRAP Input Parameters for Lead and 2,3,7,8-TCDD

PARAMETER DESCRIPTION	SYMBOL	UNITS	LEAD	2,3,7,8-TCDD
Chemical abstract service number	CAS No.	-	7439-92-1	1746-01-6
Molecular weight	MW	g $mole^{-1}$	209.21	322.0
Melting point of chemical	T_m	K	603.15	578.7
Vapour pressure	V_p	atm	3.97×10^{-12}	1.97×10^{-12}
Aqueous solubility	S	mg L^{-1}	9580	1.93×10^{-5}
Henry's Law constant	H	atm-m^3 mol^{-1}	0.025	3.29×10^{-5}
Diffusivity of COPC in air	D_a	cm2 s^{-1}	0.0772	0.104
Diffusivity of COPC in water	Dw	cm2 s^{-1}	9.6×10^{-6}	5.6×10^{-6}
Octanol-water partition coefficient	K_ow	-	5.37	6,309,573
Organic carbon-water partition coefficient	K_oc	mL g^{-1}	0	3,890,451
Soil-water partition coefficient	Kd_s	mL g^{-1}	900	38,904
Suspended sediments/surface water partition	Kd_sw	L kg^{-1}	900	291,784

PARAMETER DESCRIPTION	SYMBOL	UNITS	LEAD	2,3,7,8-TCDD
coefficient				
Bed sediment/sediment pore water partition coefficient	Kd_bs	mL g^{-1}	900	155,618
COPC loss constant due to biotic and abiotic degradation	K_s_g	a^{-1}	0	0.03
Fraction of COPC air concentration	f_v		0.007	0.664
Root concentration factor	RCF	mL g^{-1}	0	39,999
Plant-soil bioconcentration factor for below ground produce	br_root_veg	-	0.009	1.03
Plant-soil bioconcentration factor for lefy-vegetables	br_leafy_veg	-	0.0136	0.00455
Plant-soil bioconcentration factor for forage	br_forage	-	0.045	0.00455
COPC air-to-plant biotransfer factor for leafy vegetables	bv_leafy_veg	-	0	65,500
COPC air-to-plant biotransfer factor for forage	bv_forage	-	0	65,500
COPC biotransfer factor for milk	ba_milk	day kg^{-1}	0.00025	0.0055
COPC biotransfer factor for beef	ba_beef	day kg^{-1}	0.0003	0.026
COPC biotransfer factor for pork	ba_pork	day kg^{-1}	0	0.032
COPC biotransfer factor for chicken	ba_chicken	day kg^{-1}	0	0.019
Plant-soil bioconcentration factor for eggs	ba_egg	-	0	0.011
Fish bioconcentration factor	BCF_fish	L kg^{-1}	0.09	34,400
Fish bioaccumulation factor	BAF_fish	L kg^{-1}	0	0
Biota-sediment	BSAF_fish	-	0	0.09

PARAMETER DESCRIPTION	SYMBOL	UNITS	LEAD	2,3,7,8-TCDD
accumulation factor				
Plant-soil bioconcentration factor for grain	br_grain	-	0.009	0.00455

Table 7B.2.8: Toxicity Factors Obtained from the HHRAP for the COPCs in this Assessment

COPC	INGESTION REFERENCE DOSE (RfD) ($mg\ kg^{-1}\ d^{-1}$)	INHALATION REFERENCE CONCENTRATION (RfC) ($mg\ m^{-3}$)	INGESTION CARCINOGENIC SLOP FACTOR (Oral CSF) (mg kg-1 d-1)$^{-1}$	INHALATION UNIT RISK FACTOR (Inhalation URF) ($\mu g\ m^{-3})^{-1}$
Metals				
Antimony	0.0004	0.0014	0	0
Arsenic	0.0003	3.0×10^{-5}	1.5	0.0043
Cadmium	0.0004	0.0002	0.38	0.0018
Chromium (iii)	1.5	5.3	0	0
Chromium (vi)	0.0030	8.0×10^{-6}	0	0.012
Lead	0.000429	0.0015	0.0085	1.2×10^{-5}
Nickel	0.02	0.0002	0	0.00024
Thallium[(a)]	0.00008	0.00028	0	0
Elemental mercury	8.57×10^{-5}	0.0003	0	0
Mercuric chloride	0.0003	0.0011	0	0
Methyl mercury	0.0001	0.00035	0	0
PAHs				
Benzo(a)anthracene	0	0	0.73	0.00011
Benzo(a)pyrene	0	0	7.3	0.0011
Benzo(b)fluoranthene	0	0	0.73	0.00011
Chrysene	0	0	0.0073	1.1×10^{-5}
PCDD/Fs				
2,3,7,8-TCDD	1×10^{-9}	0	150,000	0
1,2,3,7,8-PeCDD	0	0	0	0
1,2,3,4,7,8-HxCDD	0	0	0	0
1,2,3,6,7,8-HxCDD	0	0	6,200	1.3
1,2,3,7,8,9-HxCDD	0	0	6,200	1.3

COPC	INGESTION REFERENCE DOSE (RfD) (mg kg^{-1} d^{-1})	INHALATION REFERENCE CONCENTRATION (RfC) (mg m^{-3})	INGESTION CARCINOGENIC SLOP FACTOR (Oral CSF) (mg kg-1 d-1)$^{-1}$	INHALATION UNIT RISK FACTOR (Inhalation URF) (µg m^{-3})$^{-1}$
1,2,3,4,6,7,8-HpCDD	0	0	0	0
OCDD	0	0	0	0
2,3,7,8-TCDF	0	0	0	0
2,3,4,7,8-PeCDF	0	0	0	0
1,2,3,7,8-PeCDF	0	0	0	0
1,2,3,4,7,8-HxCDF	0	0	0	0
1,2,3,6,7,8-HxCDF	0	0	0	0
1,2,3,7,8,9-HxCDF	0	0	0	0
2,3,4,6,7,8-HxCDF	0	0	0	0
1,2,3,4,6,7,8-HpCDF	0	0	0	0
1,2,3,4,7,8,9-HpCDF	0	0	0	0
OCDF	0	0	0	0
PCBs				
PCB77	2 x10^{-5}	7 x10^{-5}	2	0
PCB81	2 x10^{-5}	7 x10^{-5}	2	0
PCB126	2 x10^{-5}	7 x10^{-5}	2	0
PCB169	2 x10^{-5}	7 x10^{-5}	2	0
PCB105	2 x10^{-5}	7 x10^{-5}	2	0
PCB114	2 x10^{-5}	7 x10^{-5}	2	0
PCB118	2 x10^{-5}	7 x10^{-5}	2	0
PCB123	2 x10^{-5}	7 x10^{-5}	2	0
PCB156	2 x10^{-5}	7 x10^{-5}	2	0
PCB157	2 x10^{-5}	7 x10^{-5}	2	0
PCB167	2 x10^{-5}	7 x10^{-5}	2	0

(a) Reference dose for Thallium is sourced from the 1998 US EPA HHRA Protocol

Dispersion Modelling

7B.2.2.35 The results of the air quality dispersion modelling report (Appendix 7A in ES Volume III, Document Ref. 6.4) have been generated through the use of the air dispersion modelling software ADMS 5.2. Ground level concentrations and deposition rates have been generated using the model parameter values e.g. emission rates, building heights, terrain data, as detailed within the air quality dispersion modelling report.

7B.2.2.36 IRAP imports the dispersion model output files generated by the US EPA ISC or ISC-AERMOD dispersion models. The output files generated by ADMS 5.2 therefore require reformatting, before the information can be imported into IRAP.

7B.2.2.37 In addition to airborne concentrations of the COPCs, the human health risk assessment requires predictions of the following properties, which have been made in the air quality dispersion modelling report:

- airborne concentrations of vapour, particle and particle bound substances emitted;
- wet deposition rates of vapour, particle and particle bound substances; and
- dry deposition rates of particle and particle bound substances

7B.2.2.38 The Proposed Development will be equipped with fabric filters, which will mean the dominant size fraction of particles will be 1-2 µm in diameter and below. For particles of this size range a dry deposition velocity of 0.01 ms^{-1} has been used in the modelling. Whereas a dry deposition velocity of 0.005 ms^{-1} has been used to calculate dry deposition rates for gaseous phase substances. Wet deposition rates have been calculated for both particulate and gaseous substances in ADMS using values for the washout coefficients A and B of 0.0001 and 0.64 respectively.

7B.2.2.39 The results from the air quality dispersion modelling report that are relevant to this assessment of the risks to human health are presented in

7B.2.2.40 Table 7B.2.9 with the parameters used for the dispersion modelling presented in the air quality dispersion modelling report (Appendix 7A in ES Volume III, Document Ref. 6.4).

7B.2.2.41 The points of maximum airborne concentration, dry deposition and wet deposition rates are represented by the relevant receptor locations as discussed in Section 7B.2.4 and shown on Figure 7B.2.1. Note that the point of maximum wet deposition is heavily influenced by the assumed washout mechanism, which is very localised, hence the location of the point of maximum wet deposition rate in close proximity to the source.

Table 7B.2.9: Maximum Annual Average Concentrations and Deposition Rates Associated with the Proposed Development

COPC	ANNUAL AVERAGE CONCENTRATIONS (a)	VAPOUR DRY DEPOSITION RATE(b)	PARTICLE DRY DEPOSITION RATE(b)	WET DEPOSITION RATE(b)
Metals	*(µg m^{-3})*	*(mg m^{-2} $year^{-1}$)*	*(mg m^{-2} $year^{-1}$)*	*(mg m^{-2} $year^{-1}$)*
Antimony	0.00029	0.678	0.045	2.112
Arsenic	$6.27x10^{-4}$	1.475	0.099	4.591
Cadmium	0.00050	1.180	0.079	3.673
Chromium (iii)	0.00226	5.313	0.356	16.539
Chromium (vi)	$3.26\ x10^{-6}$	0.008	0.001	0.024
Lead	0.00126	2.967	0.199	9.237
Nickel	0.00552	12.977	0.870	40.399
Thallium	0.00050	1.180	0.079	3.673
Elemental mercury	$1.00\ x10^{-6}$	0.002	0.000	0.007
Mercuric chloride	0.00024	0.566	0.038	1.761
PAHs				
Benzo(a)anthracene	$2.5\ x10^{-5}$	0.0590	0.0040	0.1836
Benzo(a)pyrene	$5.0\ x10^{-4}$	1.1797	0.0791	3.6726
Benzo(b)fluoranthene	$3.3\ x10^{-6}$	0.0077	0.0005	0.0239
Chrysene	$2.3\ x10^{-3}$	5.4267	0.3639	16.8941
PCDD/Fs	*(fg m^{-3})*	*(ng m^{-2} $year^{-1}$)*	*(ng m^{-2} $year^{-1}$)*	*(ng m^{-2} $year^{-1}$)*
2,3,7,8-TCDD	0.08	0.18	0.01	0.57
1,2,3,7,8-PeCDD	0.63	1.47	0.10	4.59
1,2,3,4,7,8-HxCDD	0.73	1.71	0.11	5.33
1,2,3,6,7,8-HxCDD	0.53	1.24	0.08	3.86
1,2,3,7,8,9-HxCDD	0.65	1.53	0.10	4.77
1,2,3,4,6,7,8-HpCDD	4.26	10.03	0.67	31.22
OCDD	10.03	23.59	1.58	73.45
2,3,7,8-TCDF	0.70	1.65	0.11	5.14

COPC	ANNUAL AVERAGE CONCENTRATIONS [a]	VAPOUR DRY DEPOSITION RATE[b]	PARTICLE DRY DEPOSITION RATE[b]	WET DEPOSITION RATE[b]
2,3,4,7,8-PeCDF	1.35	3.19	0.21	9.92
1,2,3,7,8-PeCDF	0.70	1.65	0.11	5.14
1,2,3,4,7,8-HxCDF	5.52	12.98	0.87	40.40
1,2,3,6,7,8-HxCDF	0.10	0.24	0.02	0.73
1,2,3,7,8,9-HxCDF	2.03	4.78	0.32	14.87
2,3,4,6,7,8-HxCDF	2.18	5.13	0.34	15.98
1,2,3,4,6,7,8-HpCDF	11.04	25.95	1.74	80.80
1,2,3,4,7,8,9-HpCDF	1.00	2.36	0.16	7.35
OCDF	10.03	23.59	1.58	73.45
PCBs	*(fg m^{-3})*	*(ng m^{-2} $year^{-1}$)*	*(ng m^{-2} $year^{-1}$)*	*(ng m^{-2} $year^{-1}$)*
PCB77	21.85	51.38	3.45	159.94
PCB81	1.35	3.17	0.21	9.85
PCB126	2.06	4.85	0.33	15.11
PCB169	1.43	3.36	0.23	10.47
PCB105	16.72	39.32	2.64	122.42
PCB114	19.06	44.83	3.01	139.56
PCB118	83.61	196.62	13.18	612.10
PCB123	8.36	19.66	1.32	61.21
PCB156	58.53	137.63	9.23	428.47
PCB157	19.06	44.83	3.01	139.56
PCB167	4.77	11.21	0.75	34.89
PCB189	3.82	8.99	0.60	27.97

Where 1 ngm^{-3} is equal to 1 x 10^{-9} and 1 fg m^{-3} is equal to 1 x 10^{-15}
Where 1 mg m^{-2} yr^{-1} is equal to 1 x 10-3 g m^{-3} yr^{-1} and 1 ng m^{-2} yr^{-1} is equal to 1 x 10^{-9} g m^{-2} yr^{-1}

7B.2.3 Exposure Pathways

7B.2.3.1 The local environment and site specific parameters within the Study Area will define the route that emissions could potentially take and lead to exposure at the relevant receptors. In order to calculate COPC specific exposure rates for each exposure pathway being considered some of the following information may be required:

- the COPC concentration in each media, as calculated in Section 7B.2.2 above;
- consumption rates of receptors in each media;
- receptor body weight; and
- the frequency and duration of exposure.

7B.2.3.2 In any given situation, regardless of site specific circumstances, two primary pathways exist where human receptors could be exposed to COPCs. These are defined as being either direct or indirect exposure pathways. The direct exposure pathway occurs via the inhalation of vapour and particulate matter emissions of COPCs from the source. Whereas, there are numerous potential indirect exposure pathways, as listed below:

- ingestion of vegetation and animal products contaminated with emissions from the Proposed Development;
- ingestion of locally grown or locally caught food (including vegetables, animals and fish);
- ingestion of drinking water from surface water sources;
- incidental ingestion of soil;
- dermal (skin) contact with contaminated soil and water;
- ingestion of breast milk.

7B.2.3.3 Exposure via the ingestion pathways can occur over a period of time and should also be expressed in terms of body weight of the receptor. The body weight of a receptor is defined by the US EPA as being 70 kg for an adult and 15 kg as a child with an exposure duration of 30 years for an adult (the expected operational lifetime of the Proposed Development) and 6 years for a child. For each exposure pathway the daily intake is defined as the dose per body weight. This highlights the importance of considering the child scenario, as for the same dose at a lower body weight the daily intake can be significantly higher.

7B.2.3.4 Plants and animals could be exposed to COPCs via deposition or direct uptake from the air. Subsequent consumption of these plants and animals via the food chain could lead to human receptors being exposed. Information on the diet of the particular receptors (type and quantity of food consumed) is used to predict the total daily intake of COPCs via the ingestion (food) pathway. Food not produced in the local vicinity will not be contaminated by COPCs and therefore only food produced and consumed at the receptor

location is considered relevant in the calculation of exposure via this pathway.

7B.2.3.5 The dermal contact exposure pathway can be disregarded from most assessments of the effects on the human health of the local population unless there are site specific requirements for its inclusion. Exposure via this pathway will occur infrequently and coupled with low dermal absorption factors will lead to a low total dose being experienced over the lifetime of an individual human receptor. Dermal contact via aquatic pathways e.g. swimming and fishing, is not a significant pathway for similar reasons.

7B.2.3.6 The HHRAP considers the ingestion of drinking water from a groundwater source as an insignificant exposure pathway from facilities similar to the Proposed Development. Surface water bodies used as a drinking water source and their associated water shed should be identified within the study area. If such water bodies exist, then the exposure via drinking water from surface water sources should be included within the assessment.

7B.2.3.7 The IRAP model requires certain site specific parameters relating to the local area with which to model the fate and transport of the COPCs via each exposure pathway. The default values within IRAP and contained within the HHRAP have been used to represent the following site specific parameters (as shown in Attachment B).

- Silage and forage grown on contaminated soils and quantity of animal feed and soil consumed by the various animal species considered.
- The interception fraction for above ground vegetation, forage and silage and length of vegetation exposure to deposition. The yield/ standing crop biomass is also required.
- Input data for assessing the risks associated with exposure to breast milk, including:
  - body weight of infant;
  - exposure duration;
  - proportion of ingested COPC stored in fat;
  - proportion of mother's weight that is fat;
  - fraction of fat in breast milk;
  - fraction of ingested contaminant that is absorbed; and
  - half-life of dioxins in adults and ingestion rate of breast milk.
- Other physical parameters (e.g. soil dry bulk density, density of air, soil mixing zone depth).
- The following site specific parameters, relating to surface conditions, are required to be defined by the user in IRAP and have been included in this assessment as follows:

- Annual average precipitation of 60.5 cma^{-1} (based on 2015 meteorological data obtained at the Humberside Airport meteorological station);
- Annual average evapotranspiration rate of 42.4 cma^{-1} (assumed to be 70% precipitation);
- Average annual irrigation of 0 cma^{-1};
- Average annual runoff of 6.05 cma^{-1} (assumed to be 10% of total precipitation);
- An average annual wind velocity of 5.3 ms^{-1} (obtained from 2015 meteorological data obtained at the Humberside Airport meteorological station); and
- The time period over which emissions would be deposited is assumed to be 30 years, expected to be the operational lifetime of the Proposed Development.

Study Specific Exposure Pathways

7B.2.3.8 Based on the local environment surrounding the Proposed Development the potential significance of all the exposure pathways, identified above, has been assessed. This has identified that the exposure pathways relevant to this assessment are as follows:

- inhalation;
- ingestion of locally grown food and locally reared animal products e.g. milk and eggs;
- incidental ingestion of soil; and
- ingestion of breast milk.

7B.2.3.9 For exposure to occur via ingestion of drinking water there must be a source of drinking water on the surface in the local area that is affected by the emissions from the Proposed Development. This exposure pathway is not considered relevant in this assessment of human health effects as the drinking water supply in the Study Area is dominated by water transferred underground into the Study Area and has therefore been excluded from any further assessment.

7B.2.3.10 The local population can be considered to fit the urban or farmer resident type for whom any fish caught would not represent the main source of protein in their diet. For these reasons it has been considered appropriate to exclude the ingestion of locally caught fish as an exposure pathway in this assessment of health effects.

7B.2.3.11 Based upon the local environment surrounding the Proposed Development the following exposure pathways have been considered within this assessment with regards to ingestion:

- soil (incidental);
- home grown produce (fruits and vegetables);

- home grown beef;
- home grown pork;
- home grown chicken;
- milk from home reared cows;
- eggs from home reared chickens; and
- breast milk.

7B.2.3.12 The inclusion of all food groups within this assessment has conservatively assumed that there is both arable and pastoral land in addition to locally grown produce and animals within the vicinity of the Proposed Development. The ingestion of home reared meat is only considered for farmers and the families of farmers.

7B.2.4 Receptors

7B.2.4.1 The HHRAP defines three generic hypothetical receptor types for use within the human health risk assessment process. The receptor types are a hypothetical adult and/ or child resident, farmer and fisher.

7B.2.4.2 The hypothetical farmer receptor is included where a member of the farming family could be exposed to COPCs. A proportion of the farmer's diet is assumed to come from home grown produce that are affected by emissions from the Proposed Development. The hypothetical resident receptor is included in the assessment where exposure could occur in an urban or non-farm rural setting. The hypothetical fisher receptor represents a receptor scenario where locally caught fresh-water fish is the main source of protein in the receptors diet in an urban or non-farm, rural setting.

7B.2.4.3 The impacts reported in the air quality dispersion modelling report (Appendix 7A in ES Volume III, Document Ref. 6.4) are used within the IRAP model to predict the location of maximum concentration and deposition rates for each particular land use type. The land use of the local area is then identified and used to define the number and location of each of the relevant hypothetical receptor types e.g. a resident receptor within a residential area.

7B.2.4.4 For each hypothetical type of receptor and within each particular land use, up to four locations are selected based on the maximum predicted airborne concentration (both long term and short term), maximum predicted dry deposition rate and maximum predicted wet deposition rate. It is not uncommon for some of these maxima points to be co-located, resulting in less than three receptor locations actually being selected.

7B.2.4.5 The calculated total exposure to each COPC via each pathway requires the use of specific information for each receptor type. The default values within the HHRAP have been used to represent the following receptor specific parameters (as shown in Attachment C):

- food (meat, dairy products, fish and vegetables), water and soil consumption rates for each receptor type - however, only fishers are

assumed to consume locally caught fish and only farmers are assumed to consume locally reared animals and animal products;

- fraction of contaminated food, water and soil which is consumed by each receptor type;
- input data for the inhalation exposure including: inhalation exposure duration, inhalation exposure frequency, inhalation exposure time; and inhalation rate; and
- input data for the ingestion exposure including: exposure duration, exposure frequency, exposure time; and body weight of receptor.

Study Specific Receptors

7B.2.4.6 The Proposed Development is situated on undeveloped land situated adjacent to South Marsh Road, between the towns of Grimsby and Immingham in North East Lincolnshire. The South Humber Bank Power Station is adjacent to the Main Development Area's western boundary, with other industrial facilities to the north and east. To the south is open agricultural land. The Humber Estuary is approximately 175 m to the east of the Site.

7B.2.4.7 There are a number of chemical and manufacturing industries throughout the area, with Immingham Docks approximately 3 km to the north-west. The towns of Immingham, Grimsby and Cleethorpes are within 10 km of the Site, as are the settlements of Healing, Laceby and Stallingborough.

7B.2.4.8 Three residential areas have been selected to represent the potential for residential exposure to emissions from the Proposed Development:

- Grimsby and Cleethorpes
- Immingham; and
- Stallingborough and Healing.

7B.2.4.9 Additional residential receptors have been placed at one additional location, R3, and is reported in Chapter 7: Air Quality in ES Volume I (Document Ref. 6.2). This is the nearest actual residential receptor.

7B.2.4.10 The land surrounding these residential areas is generally characterised by agricultural activities, with some woodland and parkland between. Hypothetical farmer type receptors have been chosen to represent the rural areas to the north east, north west, north, east, west, south, south-east and south-west of the Proposed Development based on the predicted maximum concentration locations outside of urban areas.

7B.2.4.11 The emissions from the Proposed Development have been assessed for potential effects on human health at 9 hypothetical residential receptors and 18 hypothetical famer receptors in the local vicinity. Both adult and child receptor types have been considered for each location. The selected hypothetical receptors and their locations are identified in Table 7B.2.10 and shown on Figure 7B.2.1 in Annex A.

7B.2.4.12 The hypothetical resident and farmer receptor locations shown on Figure 7B.2.1 in Annex A represent the location of maximum predicted impact of either air concentration (long term or short term), wet deposition, dry deposition, or a combination of each, in that particular land use defined area. All other locations within that particular land use defined area would be at a lower risk of experiencing human health effects than the points of maximum impact, as they would have lower levels of exposure to COPCs. The receptor locations selected for use with this assessment of human health are hypothetical scenarios and are not necessarily representative of actual receptors within the local area.

7B.2.4.13 Receptors NELN_C_2 and NELN_C_3 represent locations of potential maximum impact outside of the site boundary. In the model, the actual point of maximum impact lies within the site boundary, and so receptor locations were selected at the closest calculation grid node to the point of maximum impact. These locations have been included in order to present the full results. These receptors are located on the Site boundary, and not representative of actual receptor locations.

Table 7B.2.10: Receptor Type and Locations used for the Assessment of Human Health Effects

IDENTIFIER	HYPOTHET-ICAL RECEPTOR TYPE	LOCATION	DESCRIPTION OF MAXIMUM IMPACT	OS CO-ORDINATES
ERY_1	Farmer	Rural area of the East Riding of Yorkshire	Hourly Air Concentration	524772, 417665.69
ERY_2			Long Term Air Concentration, Dry Deposition, Wet Deposition	524972, 417465.69
G&C_1	Resident	Urban area of Grimsby and Cleethorpes	Hourly Air Concentration	524622, 411315.69
G&C_2			Long Term Air Concentration, Dry deposition	524872, 412215.69
G&C_3			Wet Deposition	524622, 411765.69
Imming_1	Resident	Urban area of Immingham	Hourly Air Concentration	518372, 413865.69
Imming_2			Long Term Air Concentration, Wet and Dry deposition	517972, 414065.69

IDENTIFIER	HYPOTHET-ICAL RECEPTOR TYPE	LOCATION	DESCRIPTION OF MAXIMUM IMPACT	OS CO-ORDINATES
LN_1	Farmer	Rural area of Lincolnshire	Hourly Air Concentration	519572, 409665.69
LN_2			Long Term Air Concentration, Wet and Dry deposition	516772, 411465.69
NELN_C_1	Farmer	Rural area of North East Lincolnshire – central section of Study Area	Long Term Air Concentration, Dry Deposition	523347, 413828.19
NELN_C_2			Hourly Air Concentration*	523097, 413228.19
NELN_C_3			Wet Deposition*	523197, 413528.69
NELN_N_1	Farmer	Rural area of North East Lincolnshire – northern section of Study Area	Hourly Air Concentration	522172, 414865.69
NELN_N_2			Long Term Air Concentration, Dry deposition	521222, 414065.69
NELN_N_3			Wet Deposition	521272, 414115.69

IDENTIFIER	HYPOTHET-ICAL RECEPTOR TYPE	LOCATION	DESCRIPTION OF MAXIMUM IMPACT	OS CO-ORDINATES
NELN_S_1	Farmer	Rural area of North East Lincolnshire – southern section of Study Area	Hourly Air Concentration	522372, 409865.69
NELN_S_2			Long Term Air Concentration, Dry deposition	522172, 409865.69
NELN_S_3			Wet Deposition	522572, 409865.69
NELN_W_1	Farmer	Rural area of North East Lincolnshire – western section of Study Area	Hourly Air Concentration	518572, 412065.69
NELN_W_2			Long Term Air Concentration, Dry Deposition	518572, 412265.69
NELN_W_3			Wet Deposition	517772, 415265.69
NLN_1	Farmer	Rural area of North Lincolnshire	Hourly Air Concentration	518772, 417265.69
NLN_2			Long Term Air Concentration, Dry Deposition, Wet Deposition	516972, 415665.69
R3	Resident	Resident located to the West-South West of the	-	521591.00, 413001.00

IDENTIFIER	HYPOTHET-ICAL RECEPTOR TYPE	LOCATION	DESCRIPTION OF MAXIMUM IMPACT	OS CO-ORDINATES
		Proposed Development		
S&H_1	Resident	Urban area of Stalling borough and Healing	Hourly Air Concentration	522322, 411665.69
S&H_2			Long Term Air Concentration, Dry deposition	520822, 412465.69
S&H_3			Wet Deposition	522772, 411415.69

* These receptor locations are the nearest point outside the site boundary for the respective type of maximum impact.

7B.2.5 Exposure Assessment for Metals, Dioxin/ Furans and Dioxin-like PCBs

7B.2.5.1 Various world government bodies have set target levels and guideline values for exposure to a variety of inorganic metals and dioxins/ furans and dioxin-like PCBs in soil and air. The Department for Environment, Food and Rural Affairs (Defra) has developed soil guideline values (SGVs) using the Contaminated Land Exposure Assessment (CLEA) model (EA, 2009). This model takes into account a number of exposure pathways including; ingestion of soil and contaminated vegetables and inhalation of dust and vapours, in order to generate limit values in soil that are set at a level for the protection for human health. The predicted soil concentrations of inorganic metals, dioxins/ furans and dioxin-like PCBs can be compared to these values to assess the effect on human health from the emissions of the Proposed Development.

7B.2.5.2 The latest UK Total Dietary Study (TDS) in 2006 (FSA, 2009) and 2001 (FSA, 2003) conducted by the Food Standards Agency (FSA) provided an estimate of the total dietary intake of metals and dioxins/furans for a range of receptors in a typical diet. The intake of metals and dioxins/ furans attributed to the Proposed Development can be compared to the intake experienced in a typical diet, as reported in the TDS, in order to assess the effect on human health.

7B.2.5.3 A separate assessment of the contribution of Dioxins and Furans from the Proposed Development to various food products has been made by comparison with the maximum levels specified by the European Commission (EC, 2006). The assessment within this report specifically reports results on dioxin and furan concentrations in milk and eggs, whereas food products are defined within the regulation as meat and meat products, fish, milk, eggs, oils and fats.

7B.2.5.4 The World Health Organisation (WHO) and UK Committee on Toxicity (COT) have defined Tolerable Daily Intakes (TDI) for dioxins/ furans of 1 to 4 pg I-TEQ kg-$BW^{-1}d^{-1}$ and 2 pg I-TEQ kg-$BW^{-1}d^{-1}$ respectively (WHO, 1998)(COT, 2001). The units of the TDI are defined as picogrammes of the International Toxic Equivalent per kilogram bodyweight per day. The predicted lifetime daily intake of dioxins/ furans and dioxin-like PCBs at each receptor associated with the Proposed Development has been compared to the above TDIs in order to assess the health risks over the lifetime of a single receptor.

7B.2.5.5 An additional exposure pathway considered in this assessment is the infant exposure to dioxins/ furans and dioxin-like PCBs via the ingestion of their mother's breast milk. This pathway is of particular importance as dioxin like compounds are extremely lipophilic (fat soluble) and could bioaccumulate in breast milk. In addition, the lower infant body weight means they will experience a disproportionately higher impact than in an adult from the same initial exposure. The HHRAP reports a national (US) average background exposure level of 60 pg TEQ $kg^{-1}d^{-1}$ for all dioxins and furans in nursing infants. Predicted Average Daily Dose (ADD) associated with the Proposed Development for each of the infant receptors is compared to this background exposure level in order to assess the impact on breast-fed infants from

exposure to the sum of all dioxin/ furans and dioxin-like PCBs via ingestion of their mother's breast milk.

7B.2.6 Method of Assessment for Non-Carcinogenic Effects

7B.2.6.1 It is assumed that for most COPCs there is a threshold dose, below which no adverse effects will be observed. A reference dose is used to assess any potential health effects against exposure to COPCs exhibiting a threshold relationship. The reference dose (RfD) and reference concentration (RfC) represent a daily ingestion intake rate and a daily concentration in air respectively, at which there is no appreciable risk of adverse health effects. These reference values only identify the level below which effects are unlikely and they do not state anything about the risk for higher exposures. The reference dose and reference concentration for each COPC is provided in Table 7B.2.8.

7B.2.6.2 A Hazard Quotient (HQ) is used to assess the non-carcinogenic effects of emissions from the Proposed Development on human health. This represents the potential to develop non-cancer health effects as a result of exposure to concentrations of COPCs. When assessing the level of exposure via the ingestion pathway the HQ is calculated as the Average Daily Dose (ADD) divided by the reference dose (RfD), as shown in equations (1) and (2) below.

$$HQ_{Ing,} = \frac{ADD_{Ing,}}{RfD_{Ing,}} \quad (1)$$

Where:

$$ADD_{Ing,} = \frac{I_{Ing,} \times ED \times EF}{AT \times 365} \quad (2)$$

7B.2.6.3 Where: ADD_{Ing} = ingestion dose for COPC; ED is the exposure duration (dependent on the receptor type); EF is the exposure frequency (350 days per year); and AT is the averaging time (equal to ED for non-carcinogenic effects and 70 years for carcinogenic risks).

7B.2.6.4 The HQ for the assessment of exposure via the inhalation pathway is calculated by dividing the exposure concentration by a reference concentration (RfC), as shown in equations (3) and (4) below.

$$HQ_{Inh} = \frac{EC}{RfC_{Inh}} \quad (3)$$

Where:

$$EC = \frac{C_a \times ED \times EF}{AT \times 365} \quad (4)$$

7B.2.6.5 Where: EC is the exposure concentration of a COPC (μgm^{-3}), RfC_{Inh} is the reference concentration for a COPC (mgm^{-3}) and C_a is the concentration of the COPC in air.

7B.2.6.6 If the daily intake is less than or equal to the reference dose, the hazard quotient would be less than or equal to 1 and this is considered to be a level that is protective of human health. A hazard quotient of greater than 1 would indicate the potential for non-carcinogenic human health effects.

7B.2.6.7 A particular receptor has the potential to be exposed to multiple COPCs with non-carcinogenic effects. The total hazard quotient for all the COPCs exposed to a single receptor via one exposure pathway is defined by a Hazard Index (HI). The HI sums up all the individual hazard quotients from each COPC for a single pathway and assumes that the health effects from the emissions of the Proposed Development are additive.

7B.2.6.8 In addition, a receptor could be exposed to the health effects of COPCs via numerous exposure pathways. The total hazard index is the sum of the individual hazard indices for each exposure pathway relevant to that receptor. This generates a total non-carcinogenic life-time risk for each individual receptor encompassing the exposure experienced via all COPCs and all relevant pathways.

7B.2.7 Method of Assessment for Carcinogenic Effects

7B.2.7.1 Carcinogenic risks associated with exposure to the emissions from the Proposed Development are calculated in terms of the excess lifetime risk of developing cancer. For each of the individual COPCs, the US EPA has calculated a Carcinogenic Slope Factor (CSF) for the ingestion exposure pathway and a Unit Risk Factor (URF) for the inhalation exposure pathway. The CSF represents an upper bound estimate of the carcinogenic risk for ingestion exposure to an individual COPC based on the does-response relationship. The URF represents a similar linear dose-response relationship albeit for concentrations in the air.

7B.2.7.2 The probability that an individual will develop cancer over a lifetime (excess life-time risk) as a result of a specific exposure to a certain carcinogenic COPC is calculated for the ingestion pathway using equation (5).

$$Risk_{Ing} = ADD_{Ing} \times CSF_{Ing} \quad (5)$$

7B.2.7.3 Where ADD_{Ing} is the sum of the average daily dose from all ingestion exposure routes (mg/kg-day) and CSF is the cancer slope factor associated with ingestion exposure to a specific COPC $(mg/kg\text{-}day)^{-1}$.

7B.2.7.4 The excess life-time risk of developing cancer associated with the inhalation of a specific COPC is calculated using equation (6).

$$Risk_{Inh} = EC \times URF_{Inh} \quad (6)$$

7B.2.7.5 Where EC is the exposure concentration of a COPC (μgm^{-3}) and URF is the unit risk factor for inhalation exposure to a COPC (μgm^{-3}).

7B.2.7.6 It is possible for a single receptor to be exposed to multiple COPCs within an individual pathway. Therefore the excess lifetime cancer risk for an exposure pathway is calculated as the sum of the cancer risks for individual COPCs for that pathway. Similarly a single receptor is at risk of being exposed to COPCs via multiple pathways. Therefore the total excess life time cancer risk for a single receptor is the sum of the total risk for all the individual exposure pathways relevant to that receptor.

7B.2.8 Summary of Information

Inputs

7B.2.8.1 The Chemicals of Potential Concern considered relevant to this assessment of human health effects on the local population exposed to emissions from the Proposed Development, fall into the following three main classes: dioxins/ furans and dioxin-like PCBs; PAHs; and trace metals (including antimony, arsenic, cadmium, chromium (III) & (VI), mercury, lead and nickel). Table 7B.2.11 shows the exposure scenarios for the each of the generic receptor types recommended by the HHRAP. An exposure scenario is defined as the relevant exposure pathways for each receptor at a specific location.

7B.2.8.2 The study specific pathways and receptors discussed in Sections 7B.2.1 and 7B.2.4 have been selected and considered relevant based upon Table 7B.2.11 below.

Table 7B.2.11: Exposure Scenarios Recommended by the US EPA HHRAP for each Receptor Type (US EPA, 2005)

EXPOSURE PATHWAY	FARMER[a]	FARMER CHILD[a]	RESIDENT[a]	RESIDENT CHILD[a]
Inhalation of vapour and particulates	✓	✓	✓	✓
Incidental ingestion of soil	✓	✓	✓	✓
Ingestion of home grown produce	✓	✓	✓	✓
Ingestion of home grown beef	✓	✓	×	×

EXPOSURE PATHWAY	FARMER[a]	FARMER CHILD[a]	RESIDENT[a]	RESIDENT CHILD[a]
Ingestion of milk from home grown cows	✓	✓	×	×
Ingestion of home grown chicken	✓	✓	b)	b)
Ingestion of eggs from home grown chickens	✓	✓	b)	b)
Ingestion of home grown pork	✓	✓	×	×
Ingestion of breast milk	c)	×	c)	×

a) acute receptor scenario evaluates short-term 1 hour maximum COPC air concentrations at any land use area that would support the other recommended exposure scenarios
b) Site specific exposure setting characteristics (e.g. ponds on farm or presence of small livestock within residential areas) may warrant the consideration of this scenario
c) Infant exposure to dioxins/furans via the ingestion of their mothers breast milk is evaluated as a separate exposure pathway

Outputs

7B.2.8.3 This assessment considers the effects on the human health of the local population within the Study Area when exposed to emissions from the Proposed Development by using a number of different methods. The IRAP model calculates exposure concentrations and average daily doses experienced at each individual hypothetical receptor.

7B.2.8.4 The exposure of receptors to metals and dioxin/ furans from the Proposed Development via concentrations in soil and in the diet of the local population is considered in this assessment by comparison to relevant standards and typical dietary values. The human health effects of the additional dioxin/furan concentrations associated with the emissions from the Proposed Development are assessed by comparison with the TDI derived by the WHO and the UK COT. A separate exposure pathway is used to assess the infant exposure to dioxin/ furans via the mother's breast milk by comparison to the US EPA background values.

7B.2.8.5 In the assessment of the non-carcinogenic effects on human health a hazard quotient is calculated for each COPC for the ingestion and inhalation pathway by comparing the average dose received by a receptor to a reference dose, below which there is no appreciable risk of adverse human effects. A hazard index sums up the risk to human health experienced by a

receptor to all the relevant COPCs via a single pathway and a total hazard index is calculated by combining the risks to all COPCs via all pathways.

7B.2.8.6 Carcinogenic risk associated with exposure to the emissions from the Proposed Development is calculated in terms of the excess lifetime risk of developing cancer at a single receptor for each COPC via the inhalation or ingestion pathway. This is done by multiplying the exposure concentration by a particular factor that takes into account the risk of developing cancer based on the dose response relationship for that COPC. The excess lifetime cancer risk for an exposure pathway at a single receptor sums up the risk associated with the exposure to all the relevant COPCs. The total excess lifetime risk of developing cancer at a single receptor takes into account the risks associated with all the relevant COPCs via all the relevant pathways.

7B.2.9 Results

Exposure Assessment

Metals

7B.2.9.1 The maximum additional contribution to soil concentrations associated with the emissions of arsenic, cadmium, mercury, nickel and lead from the Proposed Development, predicted at the resident and farmer receptors at the point of maximum impact in the Study Area are presented in Table 7B.2.12 below (resident receptors G&C_2, Imm_2 and S&H_2 and farmer receptors NELN_C_1, NELN_C_3 and NELN_N_2). The three reported farmer receptors represent hypothetical exposure locations at the predicted points of maximum impact in their respective areas. These areas are unrealistic, as NELN_C_1 lies on the banks of the Humber Estuary, NELN_C_3 lies nearest the point of maximum impact outside the site boundary, and NELN_N_2 lies within an industrial estate to the west of the Proposed Development, areas where there are not expected to be any receptors. These receptors are located at similar distance from the Proposed Development to other potential receptor locations, and therefore represent points of maximum potential exposure. One other receptor has been assessed, in order to demonstrate realistic exposure scenarios: resident receptor R3 is the closest receptor to the Proposed Development.

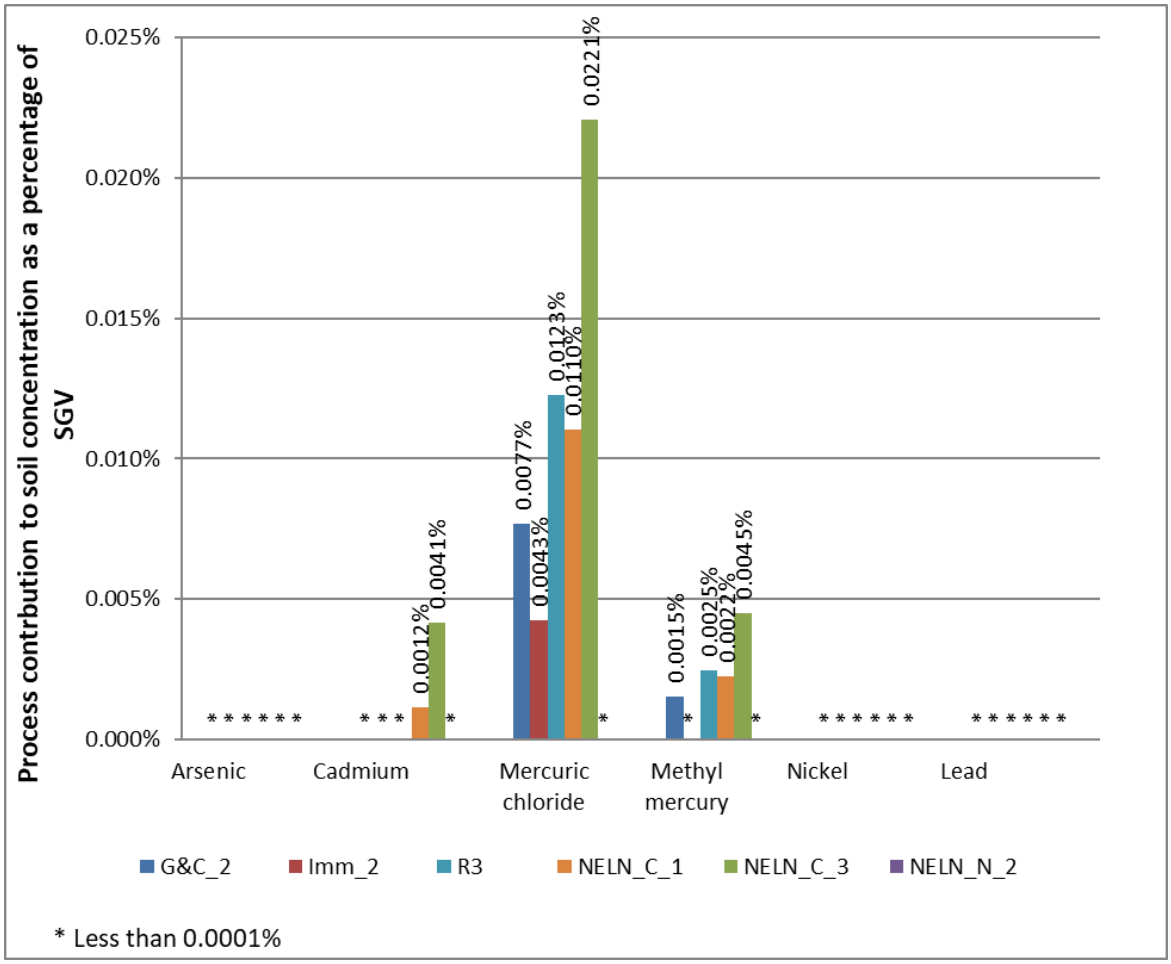
Table 7B.2.12: Maximum Contribution to Trace Metal Concentrations in Soil Associated With The Proposed Development for the Resident and Farmer Receptor Located at the Point of Maximum Impact in the Study Area

METAL	SOIL CONCENTRATION (mg/Kg soil)						SGV (mg/Kg soil)
	G&C_2	Imm_2	NELN_C_1	NELN_C_3	NELN_N_2	R3	
Arsenic	2.4×10^{-9}	1.4×10^{-9}	4.0×10^{-7}	1.5×10^{-6}	2.9×10^{-8}	3.8×10^{-9}	32
Cadmium	1.2×10^{-7}	7.2×10^{-8}	2.1×10^{-5}	7.5×10^{-5}	1.5×10^{-6}	1.9×10^{-7}	1.8
Inorganic Mercury	6.1×10^{-3}	3.4×10^{-3}	8.8×10^{-3}	1.8×10^{-2}	6.8×10^{-4}	9.8×10^{-3}	80
Methyl Mercury	1.2×10^{-4}	6.8×10^{-5}	1.8×10^{-4}	3.6×10^{-4}	1.4×10^{-5}	2.0×10^{-4}	8
Nickel	1.5×10^{-6}	8.5×10^{-7}	2.5×10^{-4}	8.8×10^{-4}	1.7×10^{-5}	2.3×10^{-6}	130
Lead	4.7×10^{-6}	2.7×10^{-6}	7.8×10^{-4}	2.8×10^{-3}	5.5×10^{-5}	7.2×10^{-6}	450

7B.2.9.2 A comparison of the predicted contribution to the soil concentrations associated with the Proposed Development for each metal as a percentage of the most stringent SGV is presented in Figure 7B.2.2.

7B.2.9.3 The highest contribution to soil concentrations are predicted for inorganic mercury at the resident R3 and farmer NELN_C_3 locations, as they are near the points of maximum impact. The contributions to the concentrations of inorganic mercury are predicted to be less than 0.025% of the SGV at these two receptors. At the hypothetical farmer receptor NELN_N_2, the contribution to the concentrations of inorganic mercury are predicted to be a factor of 100 less than for the highest predicted contribution at NELN_C_3. At other resident receptor locations, both hypothetical and real world, contributions of inorganic mercury are all less than at R3. All other predicted contributions to soil concentrations for arsenic, cadmium, methyl mercury, nickel and lead are less than 0.005% of the relevant SGV.

Figure 7B.2.2: Predicted Maximum Contribution to Metal Concentrations in Soil as a Percentage of the Most Stringent SGV for Receptors Located at the Point of Maximum Impact



7B.2.9.4 The predicted additional dietary intake of metals associated with the emissions from the Proposed Development for the resident and farmer receptors types located at the point of maximum impact in the study area are

shown in Table 7B.2.13 below. The typical dietary intake of these substances obtained from the UK TDS in 2006 (FSA, 2009) has been provided in Table 7B.2.13 for comparison purposes.

Table 7B.2.13: Dietary Intake of Metals Associated with the Proposed Development for the Resident and Farmer Receptors Located at the Points of Maximum Impact (µg Kg-BW^{-1} d^{-1})

METAL	TOTAL DIETARY INTAKE (µg Kg-BW-1 d-1)						TDS INTAKE (a)
	G&C_2	Imm_2	NELN_C_1	NELN_C_3	NELN_N_2	R3	
Arsenic	3.5 x10^{-5}	1.9 x10^{-5}	1.0 x10^{-3}	1.2 x10^{-3}	1.0 x10^{-4}	5.3 x10^{-5}	1.65 - 1.68
Cadmium	2.8 x10^{-5}	1.5 x10^{-5}	4.8 x10^{-4}	5.6 x10^{-4}	4.8 x10^{-5}	4.3 x10^{-5}	0.14 - 0.17
Chromium (b)	1.4 x10^{-4}	7.6 x10^{-5}	2.0 x10^{-2}	2.4 x10^{-2}	2.0 x10^{-3}	2.1 x10^{-4}	0.28 - 0.37
Lead	7.2 x10^{-5}	3.9 x10^{-5}	2.6 x10^{-3}	3.0 x10^{-3}	2.6 x10^{-4}	1.1 x10^{-4}	0.09 - 0.10
Mercury (c)	1.8 x10^{-5}	1.0 x10^{-5}	8.7 x10^{-4}	1.7 x10^{-3}	7.8 x10^{-5}	2.9 x10^{-5}	0.02 - 0.05
Nickel	3.1 x10^{-4}	1.7 x10^{-4}	3.6 x10^{-2}	4.2 x10^{-2}	3.7 x10^{-3}	4.7 x10^{-4}	1.49 - 1.63
Thallium	3.1 x10^{-5}	1.7 x10^{-5}	9.9 x10^{-3}	1.2 x10^{-2}	9.9 x10^{-4}	4.6 x10^{-5}	0.11 - 0.012

(a) -Mean exposure for an adult
(b) - Total chromium (trivalent and hexavalent)
(c) - Total mercury (organic and inorganic)

7B.2.9.5 The hypothetical farmer receptor locations (NELN_C_1, NELN_C_3 and NELN_N_2) would experience a greater impact on dietary intake of the metals (chromium, lead, mercury, nickel and thallium) emitted from the Proposed Development, than would be experienced at any other location within the Study Area. The greatest impact on the dietary intake of all metals for hypothetical resident receptors would be experienced at R3 and G&C_2. The impact on dietary intake varies in magnitude for each metal. The largest absolute change in dietary intake is predicted for the metal nickel. The locations R3 and G&C_2 are representative of a hypothetical resident receptor whose main exposure pathway is via the ingestion of above ground vegetables and some incidental ingestion of soil. The dietary intake obtained from the TDS in 2006 is typical of intake rates of metals for adults in the UK population that obtain the majority of their food from retail stores. The maximum predicted intake at this location within the study area can be considered conservative as it ignores the fact that most consumed food stuffs will be sourced from retail operations in the vicinity and as such represents a robust assessment of the impact of emissions from the Proposed Development on daily intake rates.

7B.2.9.6 The predicted maximum additional dietary intake for the hypothetical receptor scenarios can be compared to the typical dietary intake rates for each of the metals obtained from the UK TDS in 2006 listed in Table 7B.2.13. For example the predicted additional dietary intake of lead in the maximum exposed resident type receptor in the study area (R3) of 1.1 x 10^{-4} µg kg-$BW^{-1}d^{-1}$ is markedly less than the equivalent typical dietary intake value of 9.0x10^{-2} – 1.0x10^{-1}µg kg-$BW^{-1}d^{-1}$. For mercury (both organic and inorganic) an additional dietary intake of 2.9 x10^{-5}µg kg-$BW^{-1}d^{-1}$ was predicted at the maximally impacted hypothetical resident type receptor in the study area (R3), while a typical dietary intake value of 2.0x10^{-2} - 5.0x10^{-2}µg kg-$BW^{-1}d^{-1}$ was obtained from the UK TDS in 2006.

7B.2.9.7 The maximum exposed hypothetical farmer type receptor (NELN_C_3) would experience a greater impact on the dietary intake rate of each metal emitted from the Proposed Development, than would be experienced at any other rural location within the study area. This receptor is a hypothetical receptor location and conservatively assumes that a significant proportion of the farmer's diet comes from home grown/ reared food and animal produce. At this location the predicted maximum additional dietary intake of chromium for the hypothetical farmer NELN_C_2 receptor scenario of 2.4 x10^{-2}µg kg-$BW^{-1}d^{-1}$ is less than the typical dietary value of 2.8x10^{-1} – 3.7x10^{-1}µg kg-$BW^{-1}d^{-1}$ obtained from the UK TDS. The predicted additional dietary intake of mercury (both organic and inorganic) of 1.7 x10^{-3}µg kg-$BW^{-1}d^{-1}$ can be compared to the typical dietary values of 2.0x10^{-2} - 5.0x10^{-2}µg kg-$BW^{-1}d^{-1}$ obtained from the UK TDS in 2006. However, the predicted maximum additional dietary intake of thallium is 1.2 x 10^{-2} µg kg-$BW^{-1}d^{-1}$, equal to the upper typical dietary intake of thallium. By comparison, the predicted maximum additional dietary intake of chromium and mercury at the hypothetical farmer receptor NELN_N_2 is 2.0 x10^{-3} µg kg-$BW^{-1}d^{-1}$ and 7.8

x10^{-5} µg kg-$BW^{-1}d^{-1}$ respectively, a factor of 10-20 less and well below their respective TDS values.

7B.2.9.8 In practice the maximum impact on dietary intake of all metals at farmer type receptors would fall between the hypothetical scenario represented by NELN_C_3 and the nearby hypothetical urban resident scenario for Grimsby and Cleethorpes (G&C_2), Immingham (Imm_2) and R3. The greater the proportion of shop bought food in the household diet of these receptors the closer the dietary intake values for these metals would be to the typical values presented in the UK TDS.

Dioxins/ Furans and Dioxin-like PCBs

7B.2.9.9 The maximum additional contribution to soil concentrations associated with the emissions of dioxins/ furans form the Proposed Development, predicted at the resident and farmer receptors located at the point of maximum impact in the Study Area, are presented in Table 7B.2.14 below.

Table 7B.2.14: Maximum Contributions to Soil Concentrations of Dioxins/ Furans and Dioxin-Like PCBs Associated with the Proposed Development for the Resident and Farmer Receptors Located at the Point of Maximum Impact in the Study Area

COPC	SOIL CONCENTRATION (µg/Kg soil)						SGV (µg/Kg soil)
	G&C_2	Imm_2	NELN_C_1	NELN_C_3	NELN_N_2	R3	
Total PCDD/ PCDF and dioxin-like PCBs	0.00059	0.00035	0.0011	0.0048	0.000073	0.00089	7B.2.9.10

7B.2.9.11 The largest additional contribution of dioxins, furans and dioxin-like PCBs to soil concentrations associated with the Proposed Development is predicted to occur at the hypothetical farmer NELN_C_3 scenario. This additional contribution to soil concentrations represents 0.06% of the Soil Guideline Value for total dioxins and furans, with the resident R3 predicted to experience a lower contribution (0.011%). All other additional contributions of dioxins and furans to the soil concentration at the other hypothetical farmer and resident receptor locations are predicted to be below 0.015% of the Soil Guideline Value.

7B.2.9.12 The additional contribution of the Proposed Development to the concentrations of dioxins and furans in milk and eggs at the top 5 maximally impacted farmer receptors in each of the rural areas considered in this assessment are shown in Table 7B.2.15.

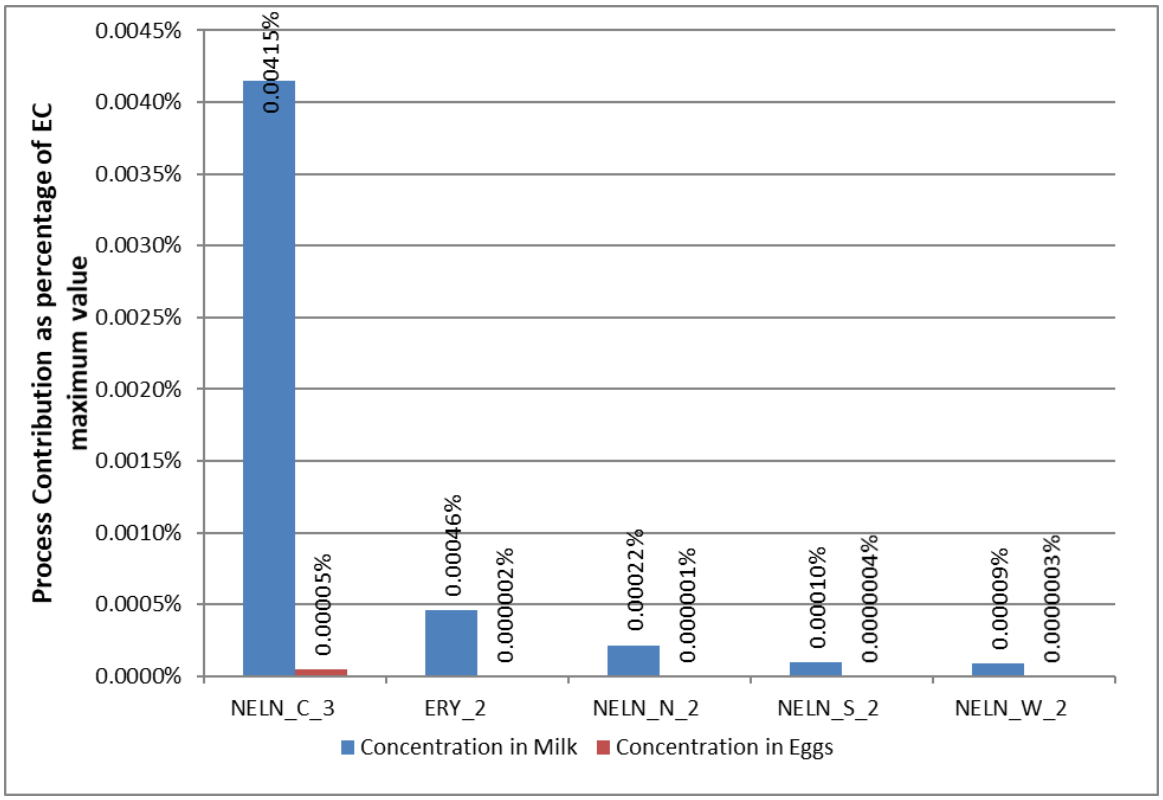
Table 7B.2.15: Predicted Contributions to Dioxin/Furan and Dioxin-Like PCBs Concentrations in Milk and Eggs Associated with the Proposed Development for the Maximally Predicted Farmer Receptors in each of the Rural Areas Considered in this Assessment

FARMER RECEPTOR	CONCENTRATION IN MILK(a) (pg WHO-TEQ g^{-1} fat)	CONCENTRATION IN EGGS(b) (pg WHO-TEQ g^{-1} fat)
NELN_C_3	0.000124	0.00000136
ERY_2	0.0000139	0.00000005
NELN_N_2	0.00000650	0.00000003
NELN_S_2	0.00000294	0.00000001
NELN_W_2	0.00000274	0.00000001

(a) - Assuming a fat content of milk of 3%
(b) - Assuming a fat content of eggs of 12%

7B.2.9.13 A comparison of the predicted additional dioxin/furan and dioxin-like PCBs concentrations in milk and eggs associated with the Proposed Development, as a percentage of the maximum European levels (EC, 2006) is presented in Figure 7B.2.3.

Figure 7B.2.3: Predicted Additional Dioxin/Furan and Dioxin-like PCBs Concentrations in Milk And Eggs as a Percentage of the Maximum European Permitted Levels at the Maximally Impacted Farmer Receptors



7B.2.9.14 The largest additional contribution to the concentration of dioxins, furans and dioxin-like PCBs in milk associated with the Proposed Development occurs in

the hypothetical farmer NELN_C_3 scenario. This largest additional concentration represents less than 0.00415% of the maximum European level (EC, 2006). The largest additional contribution to the concentration of dioxin, furans and dioxin-like PCBs in eggs is predicted to occur in the hypothetical farmer NELN_C_3 scenario, which represents less than 0.00005% of the maximum permitted European level. By comparison, the next highest concentrations in milk and eggs (beyond the point of maximum impact), are found at ERY_2, with concentrations which represent 0.00046% and 0.000002% of the respective maximum European level.

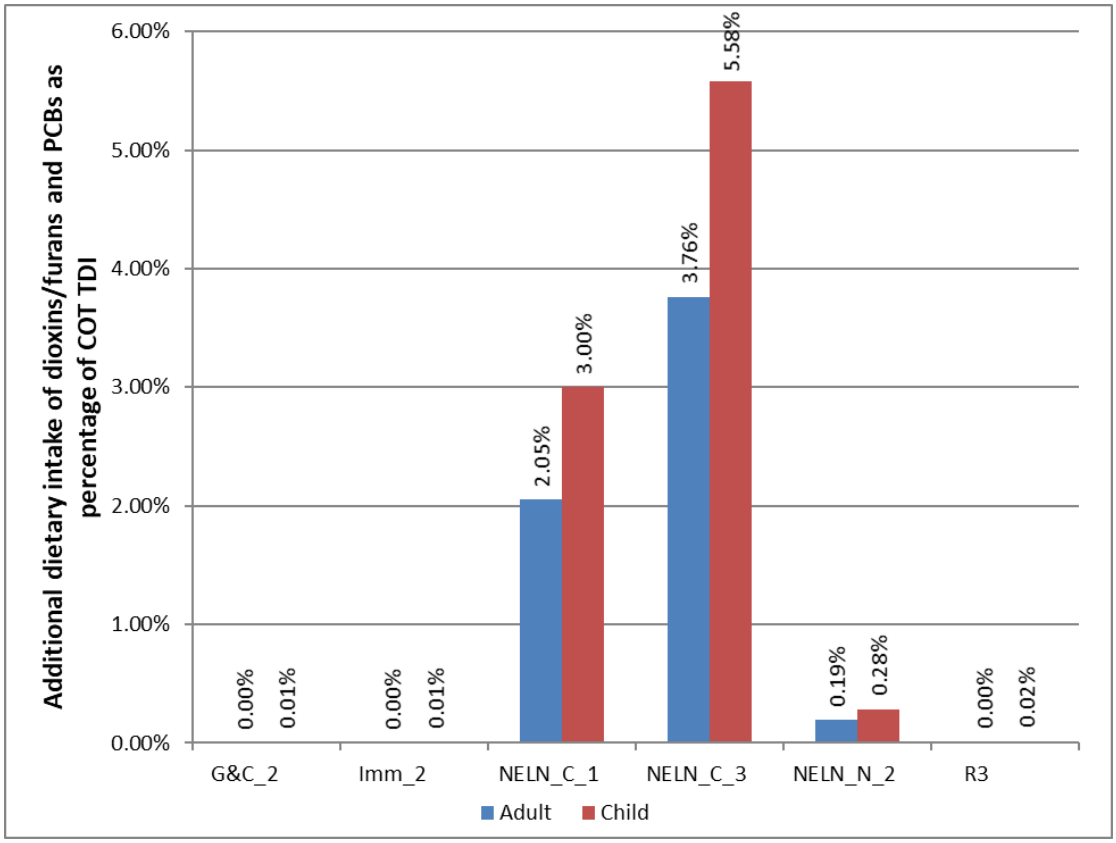
7B.2.9.15 The additional average daily intake of dioxins, furans and dioxin-like PCBs associated with the Proposed Development over the lifetime of the resident and farmer receptors, located at the point of maximum impact in the Study Area, is shown in Table 7B.2.16. These values are presented along with the WHO and COT tolerable daily intake values for comparison purposes.

Table 7B.2.16: Average Daily Intake of Dioxins/Furans and Dioxin-Like PCBs Associated with the Proposed Development for the Adult and Child of each Resident and Farmer Receptor, Located at the Point of Maximum Impact in the Study Area

RECEPTOR	ADULT (pg WHO-TEQ kg-BW^{-1}d^{-1})	CHILD (pg WHO-TEQ kg-BW^{-1}d^{-1})
G&C_2	0.00006	0.00023
Imm_2	0.00003	0.00013
NELN_C_1	0.041	0.06
NELN_C_3	0.075	0.11
NELN_N_2	0.0039	0.0057
R3	0.0001	0.0003
COT TDI (COT, 2001)	2 pg I-TEQ kg-BW^{-1} d^{-1}	
WHO TDI (WHO, 1998)	1 to 4 pg I-TEQ kg-BW^{-1} d^{-1}	

7B.2.9.16 The predicted additional average daily intake of dioxins, furans and dioxin-like PCBs have been directly compared as a percentage of the COT TDI value, as shown in Figure 7B.2.4.

Figure 7B.2.4: Predicted Daily Intake of Dioxin/ Furan and Dioxin-like PCBs for Receptors Located at the Point of Maximum Impact as a Percentage of the COT Tolerable Daily Intake



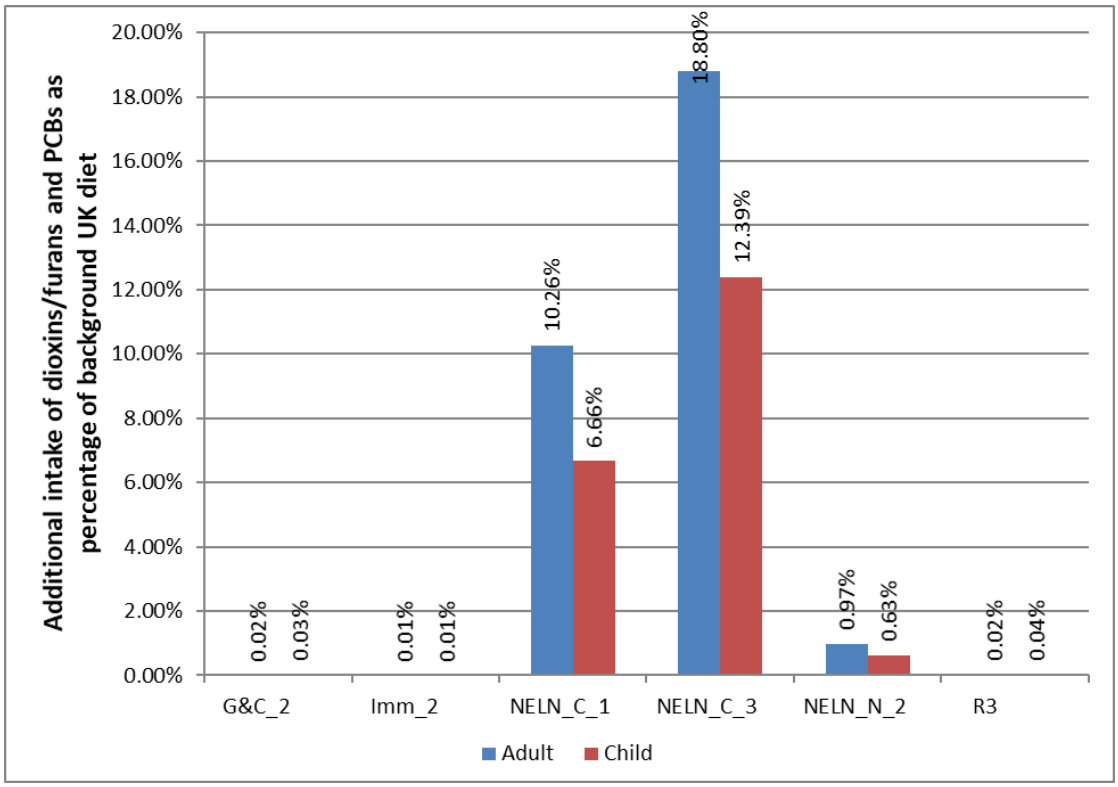
7B.2.9.17 The total dioxins and furans associated with the Proposed Development across all hypothetical receptor scenarios are predicted to contribute less than 6% of the COT TDI value. The hypothetical farmer's child receptor type (NELN_C_3) is predicted to experience an impact that equates to 5.6% of the COT TDI value and the impact at all other child or adult receptors within the Study Area would be smaller in magnitude. The additional daily intake predicted at R3 for the Resident's child receptor type is approximately a factor of 1000 below the COT TDI value and the lower range value of the WHO TDI.

7B.2.9.18 The additional daily intake of dioxins, furans and dioxin-like PCBs in the hypothetical resident G&C_2 and Imm_2 scenarios for the child receptor type is predicted to contribute a maximum of 0.01% of the COT TDI. The other hypothetical farmer NELN_N_2 scenario is predicted to contribute less than 0.3% to the COT TDI for both the adult and child receptor types. Hypothetical farmer receptors beyond the point of maximum impact experience a slightly higher contribution level than the resident receptor types.

7B.2.9.19 The predicted additional average daily intake of dioxins, furans and dioxin-like PCBs associated with the Proposed Development over the lifetime of the same receptors identified above can also be compared to the typical dietary intake of these substances, as obtained from the UK TDS undertaken in

2001 (FSA, 2003). The predicted additional intake of dioxins, furans and dioxin-like PCBs as a percentage of the typical UK dietary intake is presented in Figure 7B.2.5.

Figure 7B.2.5: Predicted Daily Intake of Dioxin/Furan and Dioxin-like PCBs for Receptors Located at the Point of Maximum Impact as a Percentage of UK Background Dietary Values



7B.2.9.20 The most recently available data from the FSA have shown that dioxin and furan levels in the UK diet are declining. The analysis of the 2001 TDS samples for dioxin and furan concentrations have reported average daily intakes for adults and children (aged 4 – 6 years) of 0.4 and 0.9 pg kg-BW^{-1}day^{-1} respectively. This is a decrease from the 1997 values of 0.9 and 2.1 pg kg-BW^{-1}day^{-1} for an adult and child respectively.

7B.2.9.21 The predicted additional dietary intake of dioxins, furans and dioxin-like PCBs associated with the Proposed Development represents less than the background contribution of the 2001 typical UK dietary values for all hypothetical receptor scenarios. The largest contributions to the typical dietary values are predicted to occur in the hypothetical farmer NELN_C_3 scenario for the adult receptor type with a contribution of 18.8%. The largest contribution to the typical dietary values for resident receptor types of 0.02% and 0.04% are predicted to occur at R3 for the adult and child receptor types respectively. The receptors R3 and NELN_C_3 are located closer to the Proposed Development than the other receptors and demonstrate that there is a significant reduction in values for a similar geographical location and reflect the conservative nature of the assessment for impacts on the rural community.

7B.2.9.22 The predicted additional average daily dose of dioxins/ furans and dioxin like PCBs associated with the Proposed Development experienced by infants via their mother's breast milk for the resident and farmer receptor types, located at the point of maximum impact in the Study Area, is shown in Table 7B.2.17.

Table 7B.2.17: Additional Average Daily Dose of Dioxins/Furans Associated with the Proposed Development for Infants via Exposure from their Mother's Breast Milk at the Resident and Farmer Receptor Types Located at the Point of Maximum Impact in the Study Area

RECEPTOR	AVERAGE DAILY DOSE FROM BREAST FEEDING (pg I-TEQ $kg^{-1}d^{-1}$)
G&C_2	0.0008
Imm_2	0.0004
NELN_C_1	0.5601
NELN_C_3	0.9455
NELN_N_2	0.054
R3	0.0012
US EPA Criteria	60 pg I-TEQ kg-BW^{-1} d^{-1}
COT TDI	2 pg I-TEQ kg-BW^{-1} d^{-1}
WHO TDI	1 to 4 pg I-TEQ kg-BW^{-1} d^{-1}

7B.2.9.23 The largest additional average daily dose (ADD) in an infant from breast feeding is predicted to occur in the hypothetical farmer NELN_C_3 scenario, which represents less than 1.6% of the US EPA criteria value, and 47.3% of the UK COT value. Concentrations at NELN_N_2 are a factor of 30 less than at NELN_C_3. The corresponding additional ADD predicted in the resident R3, and hypothetical resident G&C_2 scenarios are approximately a factor of 100-1000 less than the ADD predicted in the farmer NELN_C_3 scenario. The farmer receptor scenarios are assumed to consume locally grown and reared animal products, which are the most significant exposure route for dioxins and furans, whereas the resident scenario assumes a more varied and predominantly non local food source for its diet. The predicted additional ADDs for farmer receptor scenarios are therefore larger than those for resident scenarios at similar distances from the Proposed Development, as exposure to dioxins/ furans mainly occurs through the food chain, while exposure to dioxin-like PCBs occurs through inhalation.

7B.2.9.24 The predicted additional ADD for all the hypothetical receptor scenarios are below both the COT TDI value and the lower range of the WHO TDI value. The duration of exposure via the breast fed infant pathway to these additional ADD values is short, with the ADD over the lifetime of an individual significantly lower and similar to the values presented in Table 7B.2.16.

Assessment of Non-Carcinogenic Effects

Non-Carcinogenic Effects by Receptor Type

7B.2.9.25 The exposure concentrations experienced at the most sensitive receptors from emissions of each COPC associated with the Proposed Development via inhalation and ingestion, represented by exposure concentrations and average daily doses respectively, are presented in Table 7B.2.18 to Table

7B.2.23. The individual HQs, calculated for each COPC for each receptor using the method in Section 7B.2.6 by dividing the predicted exposure concentrations by reference concentrations, are also presented in the same tables below. In addition, the HI for each exposure pathway for all the COPCs along with the total HI for that receptor has been calculated.

Table 7B.2.18: Summary of the Exposure Experienced by the Resident G&C_2 Child Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION (µg m^{-3}) INHALATION	AVERAGE DAILY DOSE (mg kg^{-1} day^{-1}) INGESTION	HAZARD QUOTIENT	
			INHALATION	INGESTION
Antimony	1.6×10^{-5}	2.9×10^{-11}	1.1×10^{-5}	6.9×10^{-8}
Arsenic	3.5×10^{-5}	8.5×10^{-8}	1.1×10^{-3}	2.7×10^{-4}
Cadmium	2.8×10^{-5}	6.9×10^{-8}	1.4×10^{-4}	1.6×10^{-4}
Chromium (III)	1.3×10^{-4}	3.8×10^{-7}	2.4×10^{-8}	2.4×10^{-7}
Chromium (VI)	1.8×10^{-7}	5.4×10^{-10}	2.2×10^{-5}	1.7×10^{-7}
Lead	7.1×10^{-5}	1.7×10^{-7}	4.6×10^{-5}	3.9×10^{-4}
Mercuric Chloride	1.4×10^{-5}	1.2×10^{-7}	1.2×10^{-5}	3.9×10^{-4}
Methyl mercury	5.7×10^{-8}	7.0×10^{-9}	1.8×10^{-7}	6.7×10^{-5}
Nickel	3.1×10^{-4}	7.5×10^{-7}	1.5×10^{-3}	3.6×10^{-5}
Thallium	2.8×10^{-5}	1.0×10^{-8}	1.0×10^{-4}	1.2×10^{-3}
Total Dioxin-like PCBs (I-TEQ)	4.7×10^{-12}	7.1×10^{-15}	6.1×10^{-8}	3.2×10^{-7}
2,3,7,8-TCDD	-	4.0×10^{-15}	-	3.9×10^{-6}
HI for Exposure Pathway			0.003	0.0026
Total Hazard Index			0.0055	

Table 7B.2.19: Summary of the Exposure Experienced by the Resident Imm_2 Child Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION (µg m^3) INHALATION	AVERAGE DAILY DOSE (mg kg^{-1} day^{-1}) INGESTION	HAZARD QUOTIENT	
			INHALATION	INGESTION
Antimony	8.6×10^{-6}	1.7×10^{-11}	5.9×10^{-6}	4.2×10^{-8}
Arsenic	1.9×10^{-5}	4.6×10^{-8}	6.0×10^{-4}	1.5×10^{-4}
Cadmium	1.5×10^{-5}	3.7×10^{-8}	7.2×10^{-5}	8.9×10^{-5}
Chromium (III)	6.9×10^{-5}	2.1×10^{-7}	1.2×10^{-8}	1.3×10^{-7}
Chromium (VI)	9.7×10^{-8}	2.9×10^{-10}	1.2×10^{-5}	9.4×10^{-8}
Lead	3.8×10^{-5}	9.4×10^{-8}	2.4×10^{-5}	2.1×10^{-4}
Mercuric Chloride	7.2×10^{-6}	6.7×10^{-8}	6.3×10^{-6}	2.2×10^{-4}
Methyl mercury	3.0×10^{-8}	3.8×10^{-9}	9.6×10^{-8}	3.6×10^{-5}
Nickel	1.6×10^{-4}	4.0×10^{-7}	7.9×10^{-4}	1.9×10^{-5}
Thallium	1.5×10^{-5}	5.5×10^{-8}	5.3×10^{-5}	6.9×10^{-4}
Total Dioxin-like PCBs (I-TEQ)	2.5×10^{-12}	4.3×10^{-15}	3.2×10^{-8}	1.9×10^{-7}
2,3,7,8-TCDD	-	2.3×10^{-15}	-	2.2×10^{-6}
HI for Exposure Pathway			0.0016	0.0014
Total Hazard Index			0.003	

Table 7B.2.20: Summary of the Exposure Experienced by the Resident R3 Child Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION (µg m^{-3}) INHALATION	AVERAGE DAILY DOSE (mg kg^{-1} day^{-1}) INGESTION	HAZARD QUOTIENT	
			INHALATION	INGESTION
Antimony	2.5×10^{-5}	4.3×10^{-11}	1.7×10^{-5}	1.0×10^{-7}
Arsenic	5.3×10^{-5}	1.3×10^{-7}	1.7×10^{-3}	4.1×10^{-4}
Cadmium	4.3×10^{-5}	1.0×10^{-7}	2.1×10^{-4}	2.5×10^{-4}
Chromium (III)	2.0×10^{-4}	5.7×10^{-7}	3.6×10^{-8}	3.6×10^{-7}
Chromium (VI)	2.8×10^{-7}	8.1×10^{-10}	3.3×10^{-5}	2.6×10^{-7}
Lead	1.1×10^{-4}	2.6×10^{-7}	6.9×10^{-5}	5.8×10^{-4}
Mercuric Chloride	2.1×10^{-5}	1.9×10^{-7}	1.8×10^{-5}	6.2×10^{-4}
Methyl mercury	8.6×10^{-8}	1.1×10^{-8}	2.7×10^{-7}	1.0×10^{-4}
Nickel	4.7×10^{-4}	1.1×10^{-6}	2.3×10^{-3}	5.4×10^{-5}
Thallium	4.3×10^{-5}	1.5×10^{-7}	1.5×10^{-4}	1.9×10^{-3}
Total Dioxin-like PCBs (I-TEQ)	7.2×10^{-12}	1.1×10^{-14}	9.1×10^{-8}	4.8×10^{-7}
2,3,7,8-TCDD	-	6.0×10^{-15}	-	5.8×10^{-6}
HI for Exposure Pathway			0.0045	0.0039
Total Hazard Index			0.0084	

Table 7B.2.21: Summary of the Exposure Experienced by the Farmer NELN_C_1 Child Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION (µg m^{-3}) INHALATION	AVERAGE DAILY DOSE (mg kg^{-1} day^{-1}) INGESTION	HAZARD QUOTIENT	
			INHALATION	INGESTION
Antimony	1.5×10^{-4}	8.0×10^{-10}	1.1×10^{-4}	1.9×10^{-6}
Arsenic	3.3×10^{-4}	1.8×10^{-6}	1.1×10^{-2}	5.7×10^{-3}
Cadmium	2.7×10^{-4}	1.1×10^{-6}	1.3×10^{-3}	2.7×10^{-3}
Chromium (III)	1.2×10^{-3}	3.2×10^{-5}	2.2×10^{-7}	2.1×10^{-5}
Chromium (VI)	1.7×10^{-6}	4.6×10^{-8}	2.1×10^{-4}	1.5×10^{-5}
Lead	6.7×10^{-4}	5.0×10^{-6}	4.3×10^{-4}	1.1×10^{-2}
Mercuric Chloride	1.3×10^{-4}	1.8×10^{-6}	1.1×10^{-4}	5.6×10^{-3}
Methyl mercury	5.3×10^{-7}	1.2×10^{-7}	1.7×10^{-6}	1.1×10^{-3}
Nickel	2.9×10^{-3}	5.5×10^{-5}	1.4×10^{-2}	2.6×10^{-3}
Thallium	2.7×10^{-4}	1.2×10^{-5}	9.5×10^{-4}	1.4×10^{-1}
Total Dioxin-like PCBs (I-TEQ)	4.5×10^{-11}	3.2×10^{-13}	5.7×10^{-7}	1.4×10^{-5}
2,3,7,8-TCDD	-	1.6×10^{-12}	-	1.5×10^{-3}
HI for Exposure Pathway			0.0278	0.1755
Total Hazard Index			0.2033	

Table 7B.2.22: Summary of the Exposure Experienced by the Farmer NELN_C_3 Child Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION (µg m^{-3}) INHALATION	AVERAGE DAILY DOSE (mg kg^{-1} day^{-1}) INGESTION	HAZARD QUOTIENT	
			INHALATION	INGESTION
Antimony	1.5×10^{-8}	3.8×10^{-9}	1.0×10^{-8}	9.1×10^{-6}
Arsenic	3.3×10^{-8}	2.1×10^{-6}	1.1×10^{-6}	6.6×10^{-3}
Cadmium	2.7×10^{-8}	1.3×10^{-6}	1.3×10^{-7}	3.1×10^{-3}
Chromium	1.2×10^{-7}	4.1×10^{-5}	2.2×10^{-11}	2.6×10^{-5}
Chromium (VI)	1.7×10^{-10}	5.8×10^{-8}	2.1×10^{-8}	1.9×10^{-5}
Lead	6.7×10^{-8}	5.9×10^{-6}	4.3×10^{-8}	1.3×10^{-2}
Mercuric Chloride	1.3×10^{-8}	3.4×10^{-6}	1.1×10^{-8}	1.1×10^{-2}
Methyl mercury	5.3×10^{-11}	1.7×10^{-7}	1.7×10^{-10}	1.6×10^{-3}
Nickel	2.9×10^{-7}	6.3×10^{-5}	1.4×10^{-6}	3.0×10^{-3}
Thallium	2.7×10^{-8}	1.5×10^{-5}	9.5×10^{-8}	1.8×10^{-1}
Total Dioxin-like PCBs (I-TEQ)	4.5×10^{-15}	6.8×10^{-13}	5.7×10^{-11}	3.0×10^{-5}
2,3,7,8-TCDD	-	1.4×10^{-12}	-	1.4×10^{-3}
HI for Exposure Pathway			2.8×10^{-6}	0.2213
Total Hazard Index			0.2213	

Table 7B.2.23: Summary of the Exposure Experienced by the Farmer NELN_N_2 Child Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION (µg m^{-3}) INHALATION	AVERAGE DAILY DOSE (mg kg^{-1} day^{-1}) INGESTION	HAZARD QUOTIENT	
			INHALATION	INGESTION
Antimony	1.7×10^{-5}	5.3×10^{-11}	1.2×10^{-5}	1.3×10^{-7}
Arsenic	3.7×10^{-5}	1.8×10^{-7}	1.2×10^{-3}	5.8×10^{-4}
Cadmium	3.0×10^{-5}	1.1×10^{-7}	1.4×10^{-4}	2.7×10^{-4}
Chromium (III)	1.4×10^{-4}	3.2×10^{-6}	2.5×10^{-8}	2.1×10^{-6}
Chromium (VI)	2.0×10^{-7}	4.6×10^{-9}	2.3×10^{-5}	1.5×10^{-6}
Lead	7.6×10^{-5}	5.1×10^{-7}	4.8×10^{-5}	1.1×10^{-3}
Mercuric Chloride	1.4×10^{-5}	1.5×10^{-7}	1.3×10^{-5}	4.9×10^{-4}
Methyl mercury	6.0×10^{-8}	1.1×10^{-8}	1.9×10^{-7}	1.1×10^{-4}
Nickel	3.3×10^{-4}	5.6×10^{-6}	1.6×10^{-3}	2.7×10^{-4}
Thallium	3.0×10^{-5}	1.2×10^{-6}	1.1×10^{-4}	1.5×10^{-2}
Total Dioxin-like PCBs (I-TEQ)	5.0×10^{-12}	2.9×10^{-14}	6.4×10^{-8}	1.3×10^{-6}
2,3,7,8-TCDD	-	1.7×10^{-13}	-	1.6×10^{-4}
HI for Exposure Pathway			0.0031	0.0175
Total Hazard Index			0.0207	

7B.2.9.26 The HQ is a comparison of the predicted oral and inhalation exposure estimates to the reference dose and concentration values. A total Hazard Index value of 1 or less represents a level of exposure below which no appreciable risk of adverse health effects, even to sensitive populations, over a 70 year time period would occur.

7B.2.9.27 The largest HQs for the inhalation pathway are predicted for nickel in the resident and farmer hypothetical receptor scenarios, which represent approximately 50% of the total HI for that pathway. Larger HQs are predicted for the child type of receptor for both the farmer and resident receptor scenarios. The ingestion HQ for thallium is predicted to be the largest for the child farmer NELN_C_1 and NELN_C_3 receptor scenarios out of all the hypothetical receptor scenarios and represents approximately 80% of the total HI for that exposure pathway. In the other resident scenarios, both actual and hypothetical, (R3 and G&C_2) for the child receptor type, the largest HQ is predicted for inorganic mercury, which represents approximately 15% of the total HI for the ingestion exposure pathway at both locations.

7B.2.9.28 Contributions to the hazard index for the ingestion exposure pathway are also predicted for lead and organic mercury, antimony, arsenic, chromium VI, and lead are predicted to provide a contribution to the HI for the inhalation exposure pathway for each hypothetical receptor scenario.

Non-Carcinogenic Effects by Pathway

7B.2.9.29 The HIs calculated for each exposure pathway, which takes into account the HQs for exposure to all COPCs via this pathway, for the most sensitive receptors are shown in Table 7B.2.24.

Table 7B.2.24: Summary of the Hazard Indices for each Exposure Pathway for the Most Sensitive Receptors

CHILD RECEPTOR	G&C_2	Imm_2	R3	NELN_C_1	NELN_C_3	NELN_N_2
Inhalation	0.0029	0.0015	0.0043	0.027	0.000003	0.0030
Ingestion of above ground vegetables	0.0010	0.0006	0.0016	0.017	0.0203	0.0016
Ingestion of beef	-	-	-	0.001	0.0017	0.00013
Ingestion of chicken	-	-	-	0.000007	0.00002	0.000001
Ingestion of drinking water	-	-	-	-	-	-

CHILD RECEPTOR	G&C_2	Imm_2	R3	NELN_C_1	NELN_C_3	NELN_N_2
Ingestion of eggs	-	-	-	0.000009	0.00002	0.000001
Ingestion of fish	-	-	-	-	-	-
Ingestion of milk	-	-	-	0.012	0.0168	0.0012
Ingestion of pork	-	-	-	0.000008	0.00001	0.000001
Ingestion of soil	0.0003	0.0002	0.0004	0.00043	0.0009	0.000033
Hazard Index (HI)	0.0042	0.0022	0.0063	0.0575	0.0398	0.0060

7B.2.9.30 The total HIs for the resident R3 and hypothetical resident G&C_2 child receptor scenarios are approximately a factor of 10 to 15 smaller than that of the farmer NELN_C_1 child receptor scenario. For receptors located beyond the point of maximum impact, the hazard indices are broadly similar, regardless of receptor type. For all the hypothetical child resident receptor scenarios the largest non-carcinogenic risk occurs via the inhalation pathways, which represents approximately 60-70% of the total HI. The inhalation pathway is predicted to be the largest non-carcinogenic pathway risk for the hypothetical child farmer receptor scenario, which represents approximately 50% of the total HI.

7B.2.9.31 The ingestion of thallium is not included with the IRAP program (see Section 7B.2.2.16) and is therefore not considered in the assessment of hazard indices through different exposure pathways or in the summary of non-carcinogenic effects. The additional risk posed by exposure to thallium has only been considered at the maximum affected receptors, and the results presented in Table 7B.2.18 to Table 7B.2.23 represent the maximum hazard indices predicted for any receptor within the study area.

7B.2.9.32 The total HIs for the farmer NELN_C_1 is approximately a factor of 10 larger than the nearest receptors (R3) indicating that the extra risk for the farmer type of receptor occurs via the ingestion of locally grown vegetable products. The resident child receptors are approximately equal to the farmer child NELN_N_2, representative of farmer receptors in the wider study area. The relative contributions of each pathway to the total hazard index value are consistent with experience in most studies. None of the total hazard index values determined in this study represents a significant effect.

Summary of Non-Carcinogenic Effects

7B.2.9.33 The total Hazard Index for each receptor, which takes into account the cumulative risk for each COPC via each pathway, calculated by IRAP is presented in Table 7B.2.25.

Table 7B.2.25: Summary of the Total Hazard Index for each Receptor

RECEPTOR NAME	RECEPTOR TYPE	TOTAL HAZARD INDEX (HI)	RECEPTOR NAME	RECEPTOR TYPE	TOTAL HAZARD INDEX (HI)
ERY_1	Adult	0.0126	NELN_N_2	Child	0.0101
ERY_1	Child	0.0204	NELN_N_3	Adult	0.0060
ERY_2	Adult	0.0137	NELN_N_3	Child	0.0098
ERY_2	Child	0.0222	NELN_S_1	Adult	0.0027
G&C_1	Adult	0.0016	NELN_S_1	Child	0.0044
G&C_1	Child	0.0026	NELN_S_2	Adult	0.0027
G&C_2	Adult	0.0031	NELN_S_2	Child	0.0045
G&C_2	Child	0.0049	NELN_S_3	Adult	0.0026
G&C_3	Adult	0.0020	NELN_S_3	Child	0.0042
G&C_3	Child	0.0032	NELN_W_1	Adult	0.0027
Imm_1	Adult	0.0013	NELN_W_1	Child	0.0044
Imm_1	Child	0.0020	NELN_W_2	Adult	0.0027
Imm_2	Adult	0.0016	NELN_W_2	Child	0.0044
Imm_2	Child	0.0026	NELN_W_3	Adult	0.0021
LN_1	Adult	0.0016	NELN_W_3	Child	0.0035
LN_1	Child	0.0025	NLN_1	Adult	0.0009
LN_2	Adult	0.0018	NLN_1	Child	0.0015
LN_2	Child	0.0030	NLN_2	Adult	0.0018
NELN_C_1	Adult	0.0594	NLN_2	Child	0.0029
NELN_C_1	Child	0.0982	R3	Adult	0.0046
NELN_C_2	Adult	0.0044	R3	Child	0.0074
NELN_C_2	Child	0.0084	S&H_1	Adult	0.0025
NELN_C_3	Adult	0.0509	S&H_1	Child	0.0040
NELN_C_3	Child	0.0984	S&H_2	Adult	0.0027
NELN_N_1	Adult	0.0039	S&H_2	Child	0.0044
NELN_N_1	Child	0.0063	S&H_3	Adult	0.0024

RECEPTOR NAME	RECEPTOR TYPE	TOTAL HAZARD INDEX (HI)	RECEPTOR NAME	RECEPTOR TYPE	TOTAL HAZARD INDEX (HI)
NELN_N_2	Adult	0.0062	S&H_3	Child	0.0039

7B.2.9.34 All of the Total Hazard Indices presented in Table 7B.2.25 for each of the individual hypothetical receptor scenarios represent values that are approximately one order of magnitude lower than the reference dose at which there is an appreciable risk of non-carcinogenic health effects occurring over a 70 year lifetime.

7B.2.9.35 The maximum predicted non-carcinogenic impact within an urban area would occur at the hypothetical receptor called G&C_2 and the maximum predicted impact in a rural area would occur at the hypothetical receptor called NELN_C_3. The hypothetical child resident type receptor (G&C_2) and hypothetical child farmer type receptor (NELN_C_2), which are located in the Grimsby and Cleethorpes urban area and the rural area in the vicinity of the Site at the point of maximum deposition, have a total Hazard Index of 0.0049 and 0.0984 respectively. These are over an order of magnitude lower than the reference dose (HI value of 1.0), at which there is an appreciable risk of non-carcinogenic health effects occurring over the lifetime of an individual. The risk of the operation of the Proposed Development resulting in non-carcinogenic health effects at locations within the Study Area is low near the point of maximum impact and decreases to very low with increasing distance from the Proposed Development.

Assessment of Carcinogenic Effects

Carcinogenic Effects for each COPC

7B.2.9.36 The exposure concentrations experienced at the most sensitive receptors from the emissions of each COPC associated with the Proposed Development via inhalation and ingestion, represented by exposure concentrations and average daily doses respectively, are presented in Table 7B.2.26 to

7B.2.9.38 Table 7B.2.31. The individual lifetime risk of developing cancer are also presented in the same tables below and are calculated for each COPC at each receptor using the method in Section 7B.2.7 by multiplying the predicted exposure concentrations by the relevant carcinogenic risk factor for inhalation and ingestion. In addition, the excess lifetime cancer risk for each exposure pathway encompassing all the COPCs and the total excess lifetime cancer risk for that receptor has been calculated.

Table 7B.2.26: Summary of the Exposure Experienced by the Resident G&C_2 Adult Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION ($\mu g\ m^{-3}$) INHALATION	AVERAGE DAILY DOSE ($mg\ kg^{-1}\ d^{-1}$) INGESTION	LIFETIME CANCER RISK	
			INHALATION	INGESTION
Arsenic	3.5×10^{-5}	3.5×10^{-8}	6.3×10^{-8}	2.2×10^{-8}
Benzo(a)anthracene	1.4×10^{-6}	9.7×10^{-10}	6.4×10^{-11}	2.9×10^{-10}
Benzo(a)pyrene	1.4×10^{-6}	1.4×10^{-9}	6.4×10^{-10}	4.1×10^{-9}
Benzo(b)fluoranthene	8.5×10^{-6}	6.3×10^{-10}	3.8×10^{-10}	1.9×10^{-10}
Cadmium	2.8×10^{-5}	2.8×10^{-8}	2.1×10^{-8}	4.4×10^{-9}
Chromium (VI)	1.8×10^{-7}	2.0×10^{-10}	9.1×10^{-10}	-
Chrysene	2.8×10^{-6}	1.0×10^{-9}	1.3×10^{-11}	3.1×10^{-12}
Lead	7.1×10^{-5}	7.2×10^{-8}	3.5×10^{-10}	2.5×10^{-10}
Nickel	3.1×10^{-4}	3.1×10^{-7}	3.1×10^{-8}	-
Total Dioxins, Furans and PCBs	5.3×10^{-11}	6.1×10^{-14}	1.2×10^{-11}	3.8×10^{-9}
Total Lifetime Risk for Exposure Pathway			1.2×10^{-7}	3.5×10^{-8}
Total Lifetime Risk for Receptor			1.5×10^{-7}	

Table 7B.2.27: Summary of the Exposure Experienced by the Resident Imm_2 Adult Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION ($\mu g\ m^{-3}$) INHALATION	AVERAGE DAILY DOSE ($mg\ kg^{-1}\ d^{-1}$) INGESTION	LIFETIME CANCER RISK	
			INHALATION	INGESTION
Arsenic	1.9×10^{-5}	1.9×10^{-8}	3.3×10^{-8}	1.2×10^{-8}
Benzo(a)anthracene	7.5×10^{-7}	5.4×10^{-10}	3.4×10^{-11}	1.6×10^{-10}
Benzo(a)pyrene	7.5×10^{-7}	7.6×10^{-10}	3.4×10^{-10}	2.3×10^{-9}
Benzo(b)fluoranthene	4.5×10^{-6}	3.6×10^{-10}	2.0×10^{-10}	1.1×10^{-10}
Cadmium	1.5×10^{-5}	1.5×10^{-8}	1.1×10^{-8}	2.4×10^{-9}
Chromium (VI)	9.7×10^{-8}	1.1×10^{-10}	4.8×10^{-10}	-
Chrysene	1.5×10^{-6}	5.9×10^{-10}	6.8×10^{-12}	1.8×10^{-12}
Lead	3.8×10^{-5}	3.9×10^{-8}	1.9×10^{-10}	1.4×10^{-10}
Nickel	1.6×10^{-4}	1.7×10^{-7}	1.6×10^{-8}	-
Total Dioxins, Furans and PCBs	2.8×10^{-11}	3.4×10^{-14}	6.1×10^{-12}	2.1×10^{-9}
Total Lifetime Risk for Exposure Pathway			6.2×10^{-8}	1.9×10^{-8}
Total Lifetime Risk for Receptor			8.1×10^{-8}	

Table 7B.2.28: Summary of the Exposure Experienced by the Resident R3 Adult Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION ($\mu g\ m^{-3}$) INHALATION	AVERAGE DAILY DOSE ($mg\ kg^{-1}\ d^{-1}$) INGESTION	LIFETIME CANCER RISK	
			INHALATION	INGESTION
Arsenic	5.3×10^{-5}	5.3×10^{-8}	9.4×10^{-8}	3.3×10^{-8}
Benzo(a)anthracene	2.1×10^{-6}	1.4×10^{-9}	9.7×10^{-11}	4.3×10^{-10}
Benzo(a)pyrene	2.1×10^{-6}	2.0×10^{-9}	9.7×10^{-10}	6.1×10^{-9}
Benzo(b)fluoranthene	1.3×10^{-5}	9.4×10^{-10}	5.8×10^{-10}	2.8×10^{-10}
Cadmium	4.3×10^{-5}	4.3×10^{-8}	3.2×10^{-8}	6.7×10^{-9}
Chromium (VI)	2.8×10^{-7}	3.0×10^{-10}	1.4×10^{-9}	-
Chrysene	4.3×10^{-6}	1.5×10^{-9}	1.9×10^{-11}	4.6×10^{-12}
Lead	1.1×10^{-4}	1.1×10^{-7}	5.3×10^{-10}	3.8×10^{-10}
Nickel	4.7×10^{-4}	4.7×10^{-7}	4.6×10^{-8}	-
Total Dioxins, Furans and PCBs	8.0×10^{-11}	9.0×10^{-14}	1.7×10^{-11}	5.6×10^{-9}
Total Lifetime Risk for Exposure Pathway			1.8×10^{-7}	5.2×10^{-8}
Total Lifetime Risk for Receptor			2.3×10^{-7}	

Table 7B.2.29: Summary of the Exposure Experienced by the Farmer NELN_C_1 Adult Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION (µg m^{-3}) INHALATION	AVERAGE DAILY DOSE (mg kg^{-1} d^{-1}) INGESTION	LIFETIME CANCER RISK	
			INHALATION	INGESTION
Arsenic	3.3×10^{-4}	1.0×10^{-6}	7.9×10^{-7}	8.4×10^{-7}
Benzo(a)anthracene	1.3×10^{-5}	1.1×10^{-6}	8.0×10^{-10}	4.3×10^{-7}
Benzo(a)pyrene	1.3×10^{-5}	2.0×10^{-6}	8.0×10^{-9}	7.8×10^{-6}
Benzo(b)fluoranthene	8.0×10^{-5}	5.4×10^{-7}	4.8×10^{-9}	2.2×10^{-7}
Cadmium	2.7×10^{-4}	4.8×10^{-7}	2.6×10^{-7}	1.0×10^{-8}
Chromium (VI)	1.7×10^{-6}	2.9×10^{-8}	1.1×10^{-8}	-
Chrysene	2.7×10^{-5}	8.8×10^{-7}	1.6×10^{-10}	3.5×10^{-9}
Lead	6.7×10^{-4}	2.6×10^{-6}	4.4×10^{-9}	1.2×10^{-8}
Nickel	2.9×10^{-3}	3.6×10^{-5}	3.9×10^{-7}	-
Total Dioxins, Furans and PCBs	5.0×10^{-10}	4.1×10^{-11}	1.5×10^{-10}	3.6×10^{-6}
Total Lifetime Risk for Exposure Pathway			1.5×10^{-6}	1.3×10^{-5}
Total Lifetime Risk for Receptor			1.4×10^{-5}	

Table 7B.2.30: Summary of the Exposure Experienced by the Resident NELN_C_3 Adult Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION (µg m^{-3}) INHALATION	AVERAGE DAILY DOSE (mg kg^{-1} d^{-1}) INGESTION	LIFETIME CANCER RISK	
			INHALATION	INGESTION
Arsenic	3.3×10^{-8}	1.2×10^{-6}	7.8×10^{-11}	9.7×10^{-7}
Benzo(a)anthracene	1.3×10^{-9}	2.1×10^{-6}	8.0×10^{-14}	8.4×10^{-7}
Benzo(a)pyrene	1.3×10^{-9}	2.7×10^{-6}	8.0×10^{-13}	1.1×10^{-5}
Benzo(b)fluoranthene	8.0×10^{-9}	8.3×10^{-7}	4.8×10^{-13}	3.3×10^{-7}
Cadmium	2.7×10^{-8}	5.6×10^{-7}	2.6×10^{-11}	1.2×10^{-7}
Chromium (VI)	1.7×10^{-10}	3.5×10^{-8}	1.1×10^{-12}	-
Chrysene	2.7×10^{-9}	2.1×10^{-6}	1.6×10^{-14}	8.5×10^{-9}
Lead	6.7×10^{-8}	3.0×10^{-6}	4.4×10^{-13}	1.4×10^{-8}
Nickel	2.9×10^{-7}	4.2×10^{-5}	3.8×10^{-11}	-
Total Dioxins, Furans and PCBs	5.0×10^{-14}	7.5×10^{-11}	1.4×10^{-14}	6.4×10^{-6}
Total Lifetime Risk for Exposure Pathway			1.5×10^{-10}	1.9×10^{-5}
Total Lifetime Risk for Receptor			1.9×10^{-5}	

Table 7B.2.31: Summary of the Exposure Experienced by the Resident NELN_N_2 Adult Receptor for Each COPC via Inhalation and Ingestion

COPC	EXPOSURE CONCENTRATION ($\mu g\ m^{-3}$) INHALATION	AVERAGE DAILY DOSE ($mg\ kg^{-1}\ d^{-1}$) INGESTION	LIFETIME CANCER RISK	
			INHALATION	INGESTION
Arsenic	3.7×10^{-5}	1.0×10^{-7}	8.8×10^{-8}	8.6×10^{-8}
Benzo(a)anthracene	1.5×10^{-6}	1.0×10^{-7}	9.0×10^{-11}	4.1×10^{-8}
Benzo(a)pyrene	1.5×10^{-6}	1.9×10^{-7}	9.0×10^{-10}	7.8×10^{-7}
Benzo(b)fluoranthene	9.0×10^{-6}	5.3×10^{-8}	5.4×10^{-10}	2.1×10^{-8}
Cadmium	3.0×10^{-5}	4.8×10^{-8}	3.0×10^{-8}	1.0×10^{-8}
Chromium (VI)	2.0×10^{-7}	2.9×10^{-9}	1.3×10^{-9}	-
Chrysene	3.0×10^{-6}	7.8×10^{-8}	1.8×10^{-11}	3.1×10^{-10}
Lead	7.6×10^{-5}	2.6×10^{-7}	5.0×10^{-10}	1.2×10^{-9}
Nickel	3.3×10^{-4}	3.7×10^{-6}	4.3×10^{-8}	-
Total Dioxins, Furans and PCBs	5.6×10^{-11}	3.9×10^{-12}	1.6×10^{-11}	3.4×10^{-7}
Total Lifetime Risk for Exposure Pathway			1.6×10^{-7}	1.3×10^{-6}
Total Lifetime Risk for Receptor			1.4×10^{-6}	

7B.2.9.39 The largest predicted lifetime cancer risk via the inhalation exposure pathway is for arsenic for the hypothetical resident and farmer receptor scenarios. Exposure via inhalation of cadmium represents approximately 55% of the total lifetime cancer risk for exposure to all COPCs via the inhalation pathway for each hypothetical receptor.

7B.2.9.40 For the hypothetical resident receptor scenario the largest contribution to the lifetime cancer risk via the ingestion exposure pathway is predicted to occur for arsenic, while for the hypothetical farmer receptor scenario, the largest risk is from benzo[a]pyrene. The ingestion of arsenic and benzo[a]pyrene both represent approximately 60-65% of the total lifetime cancer risk via the ingestion pathway for the hypothetical resident and farmer receptor types.

Carcinogenic Effects for each Pathway

7B.2.9.41 The total lifetime cancer risks calculated for each exposure pathway, which takes into account the risk for exposure to all COPCs via this pathway, for the most sensitive receptors are shown in Table 7B.2.32.

Table 7B.2.32: Summary of the Total Lifetime Cancer Risk for each Exposure Pathway for the Most Sensitive Receptors

CHILD RECEPTOR	G&C_2	Imm_2	R3	NELN_C_1	NELN_C_3	NELN_N_2
Inhalation	1.2×10^{-7}	6.2×10^{-8}	1.8×10^{-7}	1.5×10^{-6}	1.5×10^{-10}	1.6×10^{-7}
Ingestion of above ground vegetables	3.4×10^{-8}	1.9×10^{-8}	5.1×10^{-8}	7.5×10^{-7}	1.1×10^{-6}	7.4×10^{-8}
Ingestion of beef	-	-	-	2.8×10^{-6}	4.2×10^{-6}	2.8×10^{-7}
Ingestion of chicken	-	-	-	2.6×10^{-10}	1.0×10^{-9}	1.8×10^{-11}
Ingestion of drinking water	-	-	-	-	-	-
Ingestion of eggs	-	-	-	1.7×10^{-10}	6.4×10^{-10}	1.2×10^{-11}
Ingestion of fish	-	-	-	-	-	-
Ingestion of milk	-	-	-	9.3×10^{-6}	1.4×10^{-5}	9.2×10^{-7}
Ingestion of pork	-	-	-	8.1×10^{-8}	1.1×10^{-7}	8.0×10^{-9}
Ingestion of soil	4.8×10^{-10}	2.8×10^{-10}	7.4×10^{-10}	1.2×10^{-9}	4.4×10^{-9}	8.1×10^{-11}
Total Lifetime Risk	1.5×10^{-7}	8.1×10^{-8}	2.3×10^{-7}	1.4×10^{-5}	1.9×10^{-5}	1.4×10^{-6}

7B.2.9.42 The total lifetime cancer risk for the hypothetical farmer NELN_C_3 receptor scenario is approximately 100 times larger than that of the resident R3 and G&C_2 receptors, and approximately 10 times larger than farmer NELN_N_2 receptor scenario. For the hypothetical resident receptors, the largest risk to carcinogenic health effects occurs via the inhalation exposure pathway. The inhalation exposure pathway represents approximately 75-80% of the total carcinogenic risk via all pathways for these receptors.

7B.2.9.43 The ingestion of food products and in particular the ingestion of milk is predicted to be the exposure pathway with the largest risk of carcinogenic effects for the hypothetical farmer receptor scenario. This exposure pathway represents approximately 65-70% of the total overall carcinogenic risk via all ingestion exposure pathways for the farmer receptor scenarios.

Summary of Carcinogenic Effects

7B.2.9.44 The total lifetime cancer risk for each receptor, which takes into account the cumulative risk for each COPC via each pathway, calculated by IRAP is presented in Table 7B.2.33.

Table 7B.2.33: Summary of the Total Hazard Index for each Receptor

RECEPTOR NAME	RECEPTOR TYPE	TOTAL LIFETIME CANCER RISK	RECEPT NAME	RECEPTOR TYPE	TOTAL LIFETIME CANCER RISK
ERY_1	Adult	2.9×10^{-6}	NELN_N_2	Child	3.1×10^{-7}
ERY_1	Child	6.1×10^{-7}	NELN_N_3	Adult	1.4×10^{-6}
ERY_2	Adult	3.1×10^{-6}	NELN_N_3	Child	3.0×10^{-7}
ERY_2	Child	6.7×10^{-7}	NELN_S_1	Adult	6.5×10^{-7}
G&C_1	Adult	7.7×10^{-8}	NELN_S_1	Child	1.4×10^{-7}
G&C_1	Child	2.1×10^{-8}	NELN_S_2	Adult	6.5×10^{-7}
G&C_2	Adult	1.5×10^{-7}	NELN_S_2	Child	1.4×10^{-7}
G&C_2	Child	4.1×10^{-8}	NELN_S_3	Adult	6.2×10^{-7}
G&C_3	Adult	9.9×10^{-8}	NELN_S_3	Child	1.3×10^{-7}
G&C_3	Child	2.7×10^{-8}	NELN_W_1	Adult	6.2×10^{-7}
Imm_1	Adult	6.3×10^{-8}	NELN_W_1	Child	1.3×10^{-7}
Imm_1	Child	1.7×10^{-8}	NELN_W_2	Adult	6.2×10^{-7}
Imm_2	Adult	8.1×10^{-8}	NELN_W_2	Child	1.3×10^{-7}
Imm_2	Child	2.2×10^{-8}	NELN_W_3	Adult	5.0×10^{-7}
LN_1	Adult	3.6×10^{-7}	NELN_W_3	Child	1.1×10^{-7}
LN_1	Child	7.6×10^{-8}	NLN_1	Adult	2.1×10^{-7}
LN_2	Adult	4.2×10^{-7}	NLN_1	Child	4.5×10^{-8}
LN_2	Child	8.9×10^{-8}	NLN_2	Adult	4.2×10^{-7}
NELN_C_1	Adult	1.4×10^{-5}	NLN_2	Child	8.9×10^{-8}
NELN_C_1	Child	3.1×10^{-6}	R3	Adult	2.3×10^{-7}
NELN_C_2	Adult	1.6×10^{-6}	R3	Child	6.1×10^{-8}
NELN_C_2	Child	3.6×10^{-7}	S&H_1	Adult	1.2×10^{-7}
NELN_C_3	Adult	1.9×10^{-5}	S&H_1	Child	3.3×10^{-8}
NELN_C_3	Child	4.3×10^{-6}	S&H_2	Adult	1.3×10^{-7}

RECEPTOR NAME	RECEPTOR TYPE	TOTAL LIFETIME CANCER RISK	RECEPT NAME	RECEPTOR TYPE	TOTAL LIFETIME CANCER RISK
NELN_N_1	Adult	9.0×10^{-7}	S&H_2	Child	3.6×10^{-8}
NELN_N_1	Child	1.9×10^{-7}	S&H_3	Adult	1.2×10^{-7}
NELN_N_2	Adult	1.4×10^{-6}	S&H_3	Child	3.2×10^{-8}

7B.2.9.45 The largest carcinogenic risk within an urban area is predicted to occur at the hypothetical receptor called G&C_2 adult and the maximum predicted impact in a rural area would occur at the farmer NELN_C_3 adult receptor scenario. The additional total lifetime (70 year period) carcinogenic risks to health at these hypothetical receptors associated with the Proposed Development are 1.5 $x10^{-7}$ for the resident G&C_2 and 1.9 $x10^{-5}$ for the farmer NELN_C_3. The additional total lifetime carcinogenic risk at the resident receptor R3 is 2.3 $x10^{-7}$. The additional total lifetime carcinogenic risk for the farmer and resident receptor scenarios NELN_N_2 and Imm_2 are 1.4 $x10^{-6}$ and 8.1 $x10^{-8}$. Expressing these values in terms of a probabilistic risk estimate of developing cancer over the lifetime of an individual, results in a 1 in 6,599,379 for G&C_2, a 1 in 51,287 for NELN_C_3, a 1 in 4,382,214 for R3, a 1 in 692,674 for NELN_N_2, and a 1 in 12,414,833 for Imm_2 probability of developing cancer. The risks of developing cancer over the lifetime of an individual are significantly smaller than the $1x10^{-5}$ (1 in 100,000) lifetime risk of developing cancer considered acceptable by the US EPA, with the exception of the hypothetical farmer receptors NELN_C_1 and NELN_C_3, where the predicted risk of developing cancer over the lifetime of an individual are greater than the 1 in 100,000 criteria. These receptors represent the potential risk at the point of maximum impact and are not representative of real world receptors. Beyond the point of maximum impact, the predicted risk drops to a 1 in 607,940 at NELN_C_2. This degree of risk is considered to be more representative of farmer receptors in the vicinity of the Proposed Development.

7B.2.9.46 If these lifetime risks over a 70 year period are converted into annual risks of carcinogenic effects then the risk of developing cancer over a year becomes 1 in 461,956,558 for G&C_2, a 1 in 3,590,088 for NELN_C_3, a 1 in 306,754,972 for R3, a 1 in 48,487,172 for NELN_N_2, and a 1 in 869,038,289 for Imm_2. These probabilistic estimates of risk are significantly smaller than the annual risk of $1x10^{-6}$ (1 in 1,000,000), considered acceptable for industry within the UK (CIWEM, 2001).

7B.2.10 Summary of Results

7B.2.10.1 The assessment of health effects from exposure to metals and organic substances associated with the operation of the Proposed Development reported the following:

7B.2.10.2 The contribution of emissions from the Proposed Development to soil concentrations of each metal and the total dioxins/ furans and dioxin-like PCBs are low. The impacts represent an additional contribution of less than 0.025% of the respective soil guideline concentration values for metals and less than 0.06% of the soil guideline concentration values for total dioxins/ furans and dioxin-like PCBs.

7B.2.10.3 A relatively low additional dietary intake of metals and dioxins/ furans and dioxin-like PCBs, when compared to the typical dietary intake values, is predicted to be associated with the operation of the Proposed Development. The predicted additional dietary intake of total mercury in the hypothetical resident G&C_2 and resident R3 receptor scenarios of less than 1.8 $x10^{-5}$ µg

kg-BW^{-1}d^{-1} is markedly less than the equivalent typical UK dietary value of 9.0x10^{-2} – 1.0x10^{-1} µg kg-BW^{-1}d^{-1}. The additional dietary intake of total dioxins/furans and dioxin-like PCBs at resident receptors is predicted to be approximately 0.03% of typical background UK dietary values, with the daily intake predicted to be approximately 5.6% of the COT TDI value at the farmer receptor location with the highest predicted impact, NELN_C_3;

7B.2.10.4 A low additional exposure to total dioxins/ furans and dioxin-like PCBs of infants via their mother's breast milk is predicted. Additional daily intake values at resident receptors are predicted to be 0.001% of the US EPA criteria and approximately 0.04% of the UK COT TDI value. At farmer receptors, the highest concentrations represent approximately 1.6% of the US EPA criteria, and 47.28% of the UK COT TDI at NELN_C_3;

7B.2.10.5 The maximum predicted non-carcinogenic impact within an urban area would occur at the hypothetical receptor called G&C_2 and the maximum predicted impact in a rural area would occur at the hypothetical receptor called NELN_C_1. The maximum predicted non-carcinogenic impact at any resident receptor would occur at receptor R3. The location of these three receptors and other receptors predicted to experience smaller impacts are illustrated on Figure 7B.2.1 within Attachment A. These receptors represent locations with larger risks of non-carcinogenic health effects predicted to be associated with the operation of the Proposed Development than at any of the other resident and farmer receptor scenarios. A range of chemicals of potential concern have been assessed and of these arsenic, nickel, inorganic mercury and thallium are predicted as having the largest contribution to non-carcinogenic health effects via the inhalation and ingestion pathway. The exposure pathways predicted to contain the largest risk to non-carcinogenic health effects is by inhalation for the hypothetical resident receptor and the hypothetical farmer receptor. The total hazard indices for these hypothetical receptors locations are predicted to be approximately a factor of 15 - 100 below the reference dose at which there is an appreciable risk of health effects occurring over a 70 year lifetime.

7B.2.10.6 The maximum predicted carcinogenic impact within an urban area would occur at the hypothetical receptor called G&C_2 and the maximum predicted impact in a rural area would occur at the hypothetical receptor called NELN_C_3. The maximum predicted carcinogenic impact at any resident receptor would occur at receptor R3. These receptors represent locations with larger risks to carcinogenic health effects predicted to be associated with the Proposed Development than at any other of the other resident and farmer receptor scenarios. A range of chemicals of potential concern have been assessed and of these arsenic and cadmium are predicted as having the largest contribution to carcinogenic health effects via the ingestion pathway for resident type receptors, while benzo[a]pyrene and total dioxins/ furans and dioxin-like PCBs are predicted as having the largest contribution to carcinogenic health effects via the ingestion pathway for farmer type receptors. The largest risk of carcinogenic health effects is predicted to occur for arsenic via the inhalation exposure pathway in the hypothetical resident and farmer receptor scenarios. The ingestion of milk and inhalation

are predicted to be the exposure pathways with the largest risk of carcinogenic health effects in the hypothetical farmer and resident receptor scenarios respectively. The total lifetime risk at these locations is a 1 in 6,599,379 for receptor G&C_2, 1 in 4,382,214 for R3, 1 in 51,287 for NELN_C_3 and 1 in 607,940 for NELN_C_2 risk of developing cancer over the entire lifetime of an individual receptor, which translates into an annual risk of 1 in 461,956,558, 1 in 306,754,972, 1 in 3,590,088 and 1 in 42,555,778 respectively. This is well within the acceptable annual risk of 1 in 1,000,000 for UK industrial operations (CIWEM, 2001).

7B.2.11 Conclusion

7B.2.11.1 This assessment has quantified the risks to human health in the local population within the Study Area from exposure to various different chemicals of potential concern associated with the emissions of the Proposed Development. The methodology used is consistent with the US EPA Human Health Risk Assessment Protocol (US EPA, 2005). The assessment has encompassed conservative assumptions regarding the exposure of a hypothetical individual receptor to the maximum concentrations of compounds of potential concern (COPCs) over the lifetime of an individual receptor and that a larger than average proportion of locally grown food is consumed. The COPCs emitted from the Proposed Development have been identified, along with the exposure pathways of greatest concern and the potentially most sensitive hypothetical receptors within the vicinity. The commercially available human health risk assessment modelling tool IRAP and the results from the air dispersion modelling exercise (Appendix 7A in ES Volume III, Document Ref. 6.4) have been used to calculate exposure concentrations and the risk of health effects at the most sensitive hypothetical receptors via the inhalation and ingestion pathways.

7B.2.11.2 This report has assessed the health effects from metals and organic substances, namely dioxins/ furans and dioxin-like PCBs. Despite dioxin-like PCBs comprising a significant proportion of the total organic substance concentrations at receptors close to the Proposed Development, the toxic equivalency factors of each congener of dioxin-like PCB is several orders of magnitude lower than for PCDDs and PCDFs, with the exception of PCB126 which is comparable. Consequently, this assessment of the health effects from metals and organic substances has shown that there is not a significant risk to human health associated with emissions from the Proposed Development via the inhalation and ingestion exposure pathway. The annual carcinogenic risks at the most sensitive receptor locations are predicted to achieve the UK industry acceptable annual risk of 1 in 1,000,000. The total non-carcinogenic risks for all COPCs via all exposure pathways predicted concentrations significantly below the reference dose and reference concentrations, at which there is an appreciable risk of health effects occurring. A relatively low dietary intake of metals and dioxins/ furans and dioxin-like PCBs is predicted to be associated with the Proposed Development, when compared to the typical UK dietary intake values.

7B.2.11.3 The assessment methodology has deliberately used assumptions to generate scenarios that will lead to overestimations of the risk to human health. Such conservative assumptions include the duration and frequency of exposure to an individual i.e. they are assumed to live their entire lives in the area of maximum impact and that a significant portion of their diet is obtained from animal and vegetable products grown/ reared in the local area where deposition occurs, whereas in reality it will originate from further afield. Taking into account the conservative nature of this assessment, it can be concluded with confidence that actual receptors within Immingham, Grimsby and Cleethorpes, or other communities, would not be subject to a significant risk of carcinogenic and non-carcinogenic health effects from exposure to COPCs via the inhalation and ingestion pathways as a consequence of the proposed operation of the Proposed Development.

7B.2.12 References

Committee on Carcinogenicity of Chemical in Food, Consumer Products and the Environment (2010) *Statement on the Risk Assessment of the Effects of Combined Exposures to Chemical Carcinogens, July 2010*

COT (2001) *Statement on the Tolerable Daily Intake for Dioxins and Dioxin like Polychlorinated Biphenyls, Committee on Toxicity, October 2001*

CIWEM (2001) *Risk Assessment for Environmental Professional, CIWEM Publication, December 2001*

DOE (1996) *Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes Contract No. HMIP/CPR2/41/1/181*

EFSA (2008) *Polycyclic Aromatic Hydrocarbons in Food: Scientific Opinion of the Panel on Contaminants in the Food Chain, (Question No. EFSA-Q-2007-136), July 2008*

Environment Agency (2005) *PCB and PAH Releases from Incineration and Power Generation Processes, R&D Technical Report P4-052, June 2005*

Environment Agency (2009) *Soil Guideline Values for dioxins, furans and dioxin-like PCBs in soil, Science Report SC050021 / Dioxins SGV*

Environment Agency (2015) *Email from Dean Sullivan, Environment Agency, to James Sturman, Fichtner GB, Assessment of dioxin-like PCBs*, Reference: S1515-0510-0007, date: 05/03/2015

Environment Agency (2016) *Releases from waste incinerators, Guidance on assessing group 3 metal stack emissions from incinerators, July 2016*

European Commission (2000) *Directive 2000/76/EC on the Incineration of Waste*

European Commission (2006) *Regulation 1881/2006, Setting of Maximum Levels for Certain Contaminants in Foodstuffs (19th December 2006)*

European Union (2010) *Directive 2010/75/EU on Industrial Emissions (integrated pollution prevention and control) (recast)*

FSA (2003) *Dioxins and Dioxin-like PCBs in the UK Diet: 2001 Total Diet Study Samples, Food Standards Agency July 2003*

FSA (2009) *Measurement of the Concentrations of Metals and Other Elements from the 2006 UK Total Diet Study, Food Standards Agency January 2009*

H.M. Government (2010) *Environmental Permitting Regulations (England and Wales). SI 675, the Stationary Office*

Joint Research Centre (2017), *Best Available Techniques (BAT) Reference Document on Waste Incineration, Draft 1, May 2017*

US EPA (1998) *Human Health Risk Assessment for Hazardous Waste Combustion Facilities, U.S. EPA Office of Solid Waste, Peer Review Draft, July 1998*

US EPA Office of Solid Waste (September 2005) *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities*

WHO (1998) *Assessment of the Health Risk of Dioxins: Re-evaluation of the Tolerable Daily Intake (TDI), WHO Consultation, May 25-29 1998, Geneva, Switzerland*

WHO (2010) *Dioxins and their effects on human health, Factsheet No. 225, May 2010*

ATTACHMENT A

Figure 7B.2.1 – Human Health Risk Assessment Receptor Locations

THIS DRAWING IS TO BE USED ONLY FOR THE PURPOSE OF ISSUE THAT IT WAS ISSUED FOR AND IS SUBJECT TO AMENDMENT

LEGEND

- IRAP Human Health Receptors
- Emission Source
- Order Limits
- IRAP Modelled Domain - 10km Buffer

Copyright
Reproduced from Ordnance Survey digital map data © Crown copyright 2018. All rights reserved. Licence number 0100031673.

Purpose of Issue
ENVIRONMENTAL STATEMENT

Client
EP WASTE MANAGEMENT LTD

Project Title
SOUTH HUMBER BANK ENERGY CENTRE DCO

Application Document Ref
HUMAN HEALTH RISK ASSESSMENT RECEPTOR LOCATIONS

Drawn	Checked	Approved	Date
MH	GG	GG	30/03/2020

AECOM Internal Project No.	Scale @ A3
60580855	1:75,000

THIS DOCUMENT HAS BEEN PREPARED PURSUANT TO AND SUBJECT TO THE TERMS OF AECOM'S APPOINTMENT BY ITS CLIENT. AECOM ACCEPTS NO LIABILITY FOR ANY USE OF THIS DOCUMENT OTHER THAN BY ITS ORIGINAL CLIENT OR FOLLOWING AECOM'S EXPRESS AGREEMENT TO SUCH USE, AND ONLY FOR THE PURPOSES FOR WHICH IT WAS PREPARED AND PROVIDED.

AECOM
One Trinity Gardens
Quayside
Newcastle upon Tyne
NE1 2HF
T +44 (0)191 224 6500
www.aecom.com

Drawing Ref
FIGURE 7B.2.1

Rev

File Name C:\Workspace\SHBEC\GIS\Humber South Bank HHRA.mxd

0 1 2 3 4 5 km

N

ATTACHMENT B

Default values used within IRAP for selected site specific parameters

PARAMETER	PARAMETER VALUE	IRAP SYMBOL	UNITS
Soil dry bulk density	1.5	Bd	g cm^{-3}
Forage fraction grown on contam. soil eaten by CATTLE	1.0	beef_fi_forage	-
Grain fraction grown on contam. soil eaten by CATTLE	1.0	beef_fi_grain	-
Silage fraction grown on contam. eaten by CATTLE	1.0	beef_fi_silage	-
Qty of forage eaten by CATTLE each day	8.8	beef_qp_forage	kg DW day^{-1}
Qty of grain eaten by CATTLE each day	0.47	beef_qp_grain	kg DW day^{-1}
Qty of silage eaten by CATTLE each day	2.5	beef_qp_silage	kg DW day^{-1}
Grain fraction grown on contam. soil eaten by CHICKEN	1.0	chicken_fi_grain	-
Qty of grain eaten by CHICKEN each day	0.2	chick_qp_grain	kg DW day^{-1}
Average annual evapotranspiration	42.4	e_v	cm yr^{-1}
Fish lipid content	0.07	f_lipid	-
Fraction of CHICKEN's diet that is soil	0.1	fd_chicken	-
Universal gas constant	8.205×10^{-5}	gas_r	atm-m^3 mol^{-1} K^{-1}
Average annual irrigation	0	i	cm yr^{-1}
Plant surface loss coefficient	18	kp	yr^{-1}
Fraction of mercury emissions NOT lost to the global cycle	0.48	merc_q_corr	-
Fraction of mercury speciated into methyl mercury in produce	0.22	mercmethyl_ag	-
Fraction of mercury speciated into methyl mercury in soil	0.02	mercmethyl_sc	-
Forage fraction grown contam. soil, eaten by MILK CATTLE	1.0	milk_fi_forage	-
Grain fraction grown contam. soil, eaten by MILK CATTLE	1.0	milk_fi_grain	-
Silage fraction grown contam. soil, eaten by MILK CATTLE	1.0	milk_fi_silage	-
Qty of forage eaten by MILK CATTLE each day	13.2	milk_qp_forage	kg DW d^{-1}
Qty of grain eaten by MILK CATTLE each day	3.0	milk_qp_grain	kg DW d^{-1}
Qty of silage eaten by MILK CATTLE each day	4.1	milk_qp_silage	kg DW d^{-1}

PARAMETER	PARAMETER VALUE	IRAP SYMBOL	UNITS
Averaging time	1	milkfat_at	yr
Body weight of infant	9.4	milfat_bw_infant	kg
Exposure duration of infant to breast milk	1	milkfat_ed	a
Proportion of ingested dioxin that is stored in fat	0.9	milkfat_f1	-
Proportion of mothers weight that is fat	0.3	milkfat_f2	-
Fraction of fat in breast milk	0.04	milkfat_f3	-
Fraction of ingested contaminant that is absorbed	0.9	milkfat_f4	-
Half-life of dioxin in adults	2555	milkfat_h	day
Ingestion rate of breast milk	0.688	milkfat_ir_milk	kg day^{-1}
Viscosity of air corresponding to air temp.	1.81×10^{-04}	mu_a	g cm^{-1} s^{-1}
Average annual precipitation	60.5	p	cm yr^{-1}
Fraction of grain grown on contam. soil eaten by PIGS	1.0	pork_fi_grain	-
Fraction of silage grown on contam. soil and eaten by PIGS	1.0	pork_fi_silage	-
Qty of grain eaten by PIGS each day	3.3	pork_qp_grain	kg DW day^{-1}
Qty of silage eaten by PIGS each day	1.4	pork_qp_silage	kg DW day^{-1}
Qty of soil eaten by CATTLE	0.5	qs_beef	kg day^{-1}
Qty of soil eaten by CHICKEN	0.022	qs_chick	kg day^{-1}
Qty of soil eaten by DAIRY CATTLE	0.4	qs_milk	kg day^{-1}
Qty of soil eaten by PIGS	0.37	qs_pork	kg day^{-1}
Average annual runoff	6.05	r	cm yr^{-1}
Density of air	1.2×10^{-3}	rho_a	g cm^{-3}
Solids particle density	2.7	rho_s	g cm^{-3}
Interception fraction - edible portion ABOVEGROUND	0.39	rp	-
Interception fraction - edible portion FORAGE	0.5	rp_forage	-
Interception fraction - edible portion SILAGE	0.46	rp_silage	-
Ambient air temperature	298	t	K
Temperature correction factor	1.026	theta	-

PARAMETER	PARAMETER VALUE	IRAP SYMBOL	UNITS
Soil volumetric water content	0.2	theta_s	mL cm^{-3}
Length of plant expos. to depos. - ABOVEGROUND	0.16	tp	year
Length of plant expos. to depos. - FORAGE	0.12	tp_forage	year
Length of plant expos. to depos. - SILAGE	0.16	tp_silage	year
Dry deposition velocity	0.5	vdv	cm s^{-1}
Dry deposition velocity for mercury	2.9	vdv_hg	cm s^{-1}
Wind velocity	5.3	w	m s^{-1}
Yield/standing crop biomass - edible portion ABOVEGROUND	2.24	yp	kg DW m^{-2}
Yield/standing crop biomass - edible portion FORAGE	0.24	yp_forage	kg DW m^{-2}
Yield/standing crop biomass - edible portion SILAGE	0.8	yp_silage	kg DW m^{-2}
Soil mixing zone depth	2.0	z	cm
Soil mixing depth for produce	2.0	z_p	cm

ATTACHMENT C

Default values used within IRAP for Receptor Specific Parameters

PARAMETER DESCRIPTION	ADULT RESIDENT	CHILD RESIDENT	ADULT FARMER	CHILD FARMER	ADULT FISHER	CHILD FISHER	UNITS
Averaging time for carcinogens	70	70	70	70	70	70	year
Averaging time for noncarcinogens	30	6	3030	6	30	6	year
Consumption rate of BEEF	0.0	0.0	0.00122	0.00075	0.0	0.0	Kg/kg-day FW
Body weight	70	15	70	15	70	15	kg
Consumption rate of POULTRY	0.0	0.0	0.00066	0.00045	0.0	0.0	Kg/kg-day FW
Consumption rate of ABOVEGROUND PRODUCE	0.00032	0.00077	0.00047	0.00113	0.00032	0.00077	Kg/kg-day FW
Consumption rate of BELOWGROUND PRODUCE	0.00014	0.00023	0.00017	0.00028	0.00014	0.00023	Kg/kg-day FW
Consumption rate of DRINKING WATER	1.4	0.67	1.4	0.67	1.4	0.67	$L\ day^{-1}$
Consumption rate of	0.00061	0.0015	0.00064	0.00157	0.00061	0.0015	Kg/kg-day FW

PARAMETER DESCRIPTION	ADULT RESIDENT	CHILD RESIDENT	ADULT FARMER	CHILD FARMER	ADULT FISHER	CHILD FISHER	UNITS
PROTECTED ABOVEGROUND PRODUCE							
Consumption rate of SOIL	0.0001	0.0002	0.0001	0.0002	0.0001	0.0002	kg day^{-1}
Exposure duration	30	6	3030	6	30	6	year
Exposure frequency	350	350	350	350	350	350	day/year
Consumption rate of EGGS	0.0	0.0	0.00075	0.00054	0.0	0.0	Kg/kg-day FW
Fraction of contaminated ABOVEGROUND PRODUCE	1.0	1.0	1.0	1.0	1.0	1.0	-
Fraction of contaminated DRINKING WATER	1.0	1.0	1.0	1.0	1.0	1.0	-
Fraction contaminated SOIL	1.0	1.0	1.0	1.0	1.0	1.0	-
Consumption rate of FISH	0.0	0.0	0.0	0.0	0.00125	0.00088	Kg/kg-day FW
Fraction of contaminated FISH	1.0	1.0	1.0	1.0	1.0	1.0	-
Inhalation	30	6	3030	6	30	6	year

PARAMETER DESCRIPTION	ADULT RESIDENT	CHILD RESIDENT	ADULT FARMER	CHILD FARMER	ADULT FISHER	CHILD FISHER	UNITS
exposure duration							
Inhalation exposure frequency	350	350	350	350	350	350	day/year
Inhalation exposure time	24	24	24	24	24	24	hrs/day
Fraction of contaminated BEEF	1	1	1	1	1	1	-
Fraction of contaminated POULTRY	1	1	1	1	1	1	-
Fraction of contaminated EGGS	1	1	1	1	1	1	-
Fraction of contaminated MILK	1	1	1	1	1	1	-
Fraction of contaminated PORK	1	1	1	1	1	1	-
Inhalation rate	0.83	0.30	0.83	0.30	0.83	0.30	$m^3\ hr^{-1}$
Consumption rate of MILK	0.0	0.0	0.01367	0.02268	0.0	0.0	Kg/kg-day FW
Consumption rate of PORK	0.0	0.0	0.00055	0.00042	0.0	0.0	Kg/kg-day FW
Time period at	0	0	0	0	0	0	year

PARAMETER DESCRIPTION	ADULT RESIDENT	CHILD RESIDENT	ADULT FARMER	CHILD FARMER	ADULT FISHER	CHILD FISHER	UNITS
the beginning of combustion							
Length of exposure duration	30	6	3030	6	30	6	year