

# SKELTON GRANGE ENERGY FROM WASTE FACILITY ENVIRONMENTAL PERMIT APPLICATION

**Human Health Risk Assessment**  
Prepared for: WTI EfW Holdings Limited

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## 1.0 INTRODUCTION

WTI EfW Holdings Limited has retained SLR Consulting to prepare the Environmental Permit (EP) application as required by the Environmental Permitting Regulations 2016 (as amended) for the Skelton Grange Energy from Waste Facility (EfW) located at Skelton Grange Road, Leeds, West Yorkshire (the Site). The facility will be operated by WTI UK Ltd (WTI).

This report presents the detailed Human Health Risk Assessment (HHRA) modelling undertaken in relation to emissions to air from the thermal treatment of waste at the EfW.

The Non-Technical Summary in Section 1 of the application provides a full description of the EP application and the facility. The key details of relevance to this assessment are that the facility will comprise:

- an EfW receiving up to 410,000 tonnes per annum (tpa) as feedstock;
- Twin-line (70MW<sub>th</sub> input each) furnace/boiler units incorporating moving grate technology and steam boiler with an energy recovery system;
- Flue gas treatment (FGT) system comprising selective non catalytic reduction (SNCR – urea/ammonia based nitrogen oxides (NO<sub>x</sub>) control), acid gas scrubbing reactor (employing lime and activated carbon) and bag house filters; and
- Discharge of treated flue gases via 2 stacks at 90m above ground level.

### 1.1 Scope of Assessment

The scope of the assessment encompasses emissions to air from the thermal treatment of waste at the EfW released via the stack. Certain regulated pollutants in these emission sources may potentially affect human health via:

- the direct pathway via inhalation; and
- indirect pathways, i.e. ingestion of soil, food grown, or animals grazed on contaminated soil as a result of aerial deposition of pollutants.

Direct exposure via inhalation has been considered within the Air Emissions Risk Assessment (AERA), which compared predicted exposure to air quality limits, the purpose of which are to protect the population from the effects of direct inhalation.

The assessment presented in this document considers the impact of compounds of potential concern (COPCs) that are emitted from the facility that are 'persistent' in the environment and may lead to bioaccumulation and have several pathways from the point of release to the human receptor; specifically this addresses polychlorinated biphenyls (PCBs), polychlorinated-p-dioxins (PCDDs - dioxins) and polychlorinated dibenzofurans (PCDFs - furans).

The methodology has applied a risk screening approach, in this respect the predictions are intended to overestimate potential exposure and do not represent actual likely levels of exposure and risk. Model input values are selected based on best available data; to ensure a robust assessment input values are selected at which a reasonable level of certainty exists that actual likely values are not underestimated.

The evaluation of risk has been based on a hypothetical worst-case exposure pathway, in that it has been assumed that the most sensitive receptor is consuming vegetables grown and livestock reared at the point of maximum ground level impact.

### 1.2 Objective

The objective of the assessment is to assess the risk to human health as a result of emissions from the facility by consideration of multi-pathway uptake (i.e. inhalation and via the food chain) of dioxins, furans and dioxin-like

PCBs, with specific regard to the hypothetical worst-case scenario, i.e. a farmer family at the point of maximum ground level impact.

## 2.0 METHODOLOGY

The U.S. Environmental Protection Agency (US-EPA) Office of Solid Waste (OSW) has developed an approach for conducting multi-pathway, site-specific human health risk assessments for waste incinerators. The approach is known as the Human Health Risk Assessment Protocol (HHRAP)<sup>1</sup>.

The HHRAP methodology incorporates the following elements:

- facility characterisation;
- atmospheric dispersion modelling;
- identification of exposure scenarios;
- estimating media concentrations; and
- quantifying exposure.

Full details of the HHRAP methodology can be found in the HHRAP and updates available on the US-EPA website<sup>2</sup>. The computer model IRAP-h View (Industrial Risk Assessment Program-Health Version 4.0) used in this assessment has been designed to compute human health risk assessments following the requirements of the HHRAP (final, 2005).

The following sub-sections provide a summary of the methodology, site specific details are provided in the remainder of the report.

### 2.1 Facility Characterisation

This is the initial stage of the HHRAP and involves collecting information regarding releases from the facility. These inputs are the primarily the same as used in the AERA, and require:

- identification of stacks and buildings locations and dimensions;
- identification of COPCs for human health;
- defining stack emission parameters (velocity, temperature, volume); and
- calculating COPC release rates (g/s).

### 2.2 Atmospheric Dispersion Modelling

An atmospheric dispersion model (AERMOD) has been used to predict air concentrations and deposition rates for each COPC across the entire study area.

Site-specific characteristics input for air modelling include:

- information obtained from the 'Facility Characterisation' step;
- partitioning emissions (i.e. vapour phase / particle deposition characteristics);
- surrounding terrain topography;
- surrounding land use;
- facility building characteristics; and
- meteorological data.

### 2.3 Identification of Exposure Scenarios

Identifying exposure scenarios consists of:

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<sup>1</sup> United States Environmental Protection Agency - Office of Solid Waste and Emergency Response. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Final (September 2005)

<sup>2</sup> <http://www.epa.gov/epawaste/hazard/tsd/td/combust/risk.htm>

- characterising the exposure setting;
- identifying recommended exposure scenarios; and
- selecting exposure scenario locations.

Characterising the exposure setting includes defining the dimensions of the assessment area (or study area). It also includes identifying the current and potential human activities and land uses within those boundaries. Within the context of the exposure setting, an exposure scenario is a combination of 'exposure pathways' to which a 'receptor' may be subjected.

For the purposes of the HHRAP, receptors come into contact with COPCs via two primary exposure routes: either directly via inhalation; or indirectly via COPC deposition and subsequent ingestion of water, soil, vegetation, and animals that have been contaminated by COPCs through the food chain. The HHRAP identifies a number of generic exposure scenarios (Farmer, Farmer Child; Fisher, Fisher Child; Resident, and Resident Child), these scenarios define to what pathway human receptors would be exposed and to what degree they would be exposed to the following:

- direct inhalation of vapours and particles;
- incidental ingestion of soil;
- ingestion of drinking water from surface water sources;
- ingestion of home grown produce (i.e. fruits and vegetables);
- ingestion of home grown beef;
- ingestion of milk from home grown cows;
- ingestion of home grown chicken;
- ingestion of eggs from home grown chickens;
- ingestion of home grown pork;
- ingestion of breast milk; and
- ingestion of locally caught fish.

Parameters such as typical ingestion rates, body weights, and inhalation rates (amongst others) that affect the assessment of risk and hazard are defined in the HHRAP on the basis of national averages. Situations where the assessment uses non-default values for these parameters are stated throughout the report.

## 2.4 Estimating Media Concentrations

This step of the HHRAP estimates the concentrations of COPCs within the affected media within the identified exposure scenarios. The calculations are detailed within the HHRAP they cover:

- calculating COPC concentrations in air for direct inhalation;
- calculating COPC concentrations in soil;
- calculating COPC concentrations in produce;
- calculating COPC concentrations in beef, pork, chicken; and
- calculating COPC concentrations drinking water and fish.

The calculation of media concentrations requires parameters such as (for which default values have been applied):

- the fraction of animal feed (grain, 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
- other physical parameters (e.g. soil dry bulk density, density of air, soil mixing zone depth).

Site specific parameters are also required and are detailed in this report for the following parameters:

- annual average evapotranspiration rate;
- annual average precipitation;
- annual average irrigation;
- annual average runoff;
- an annual average wind velocity; and
- a time period over which deposition occurs.

## 2.5 Quantifying Exposure

Calculating COPC-specific exposure rates for each exposure pathway involves some or all of the following, depending upon the medium being assessed:

- the estimated COPC media concentrations;
- consumption rates of the medium;
- receptor body weight; and
- the frequency and duration of exposure.

The information required includes the following:

- food (meat, dairy products, fish and vegetables), water and soil consumption rates;
- 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.

For the purposes of this assessment the default HHRAP parameters have been used to define the characteristics of the receptors, with exception in that longer exposure durations have been considered. The exposures calculated using the HHRAP are intended to represent reasonable maximum exposure (RME) conditions.

## 2.6 Health Criteria Values for Risk Assessment

### 2.6.1 Toxic Equivalence Factors

Dioxins comprise a number of congeners. The relative contribution of individual congeners to the overall toxicity of a mixture of dioxins is calculated by the use of toxicity equivalence factors (TEFs). It is generally acknowledged that the toxicity of individual dioxins is mediated by the same mechanism of action with the dioxin 2,3,7,8-TCDD being the most potent and best studied congener so TEFs define potency in relation to 2,3,7,8-TCDD.

The WHO European Centre for Environment and Health and the International Program on Chemical Safety have developed a set of criteria for TEF calculations for the relevant dioxin and furan congeners (Van den Berg et al., 2006)<sup>3</sup>. These TEFs have since been endorsed by the Committee on Toxicity (COT) for use in UK assessments of

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<sup>3</sup> Van den Berg M et al (2006) The 2005 World Health Organization Re-evaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-like Compounds. Toxicological Sciences Advance Access, July 2006.

dioxin exposure<sup>4</sup>. The potential dioxin-like activity contributed by each congener is determined by multiplying the concentration of the congener by its WHO-designated TEF to yield the dioxin toxic equivalent (TEQ) for that congener. The net TEQ is the sum of the individual TEQs for each dioxin or dioxin-like compound.

## 2.6.2 EA Tolerable Daily Intake Value

The EA recommend<sup>5</sup> a Tolerable Daily Intake (TDI) of 2pg WHO-TEQ kg<sup>-1</sup> bodyweight (bw) to protect against the induction of developmental effects in humans. As these are the most sensitive effects, the TDI is also expected to protect against all other toxic and carcinogenic effects. The TDI is the limit against which combined exposure from all pathways (inhalation and oral) is compared.

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<sup>4</sup> COT (2006). 2005 WHO toxic equivalency factors for dioxins and dioxin-like compounds. Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment. COT statements 2006, viewed on-line at <http://cot.food.gov.uk/pdfs/cotstatementdioxintef200613.pdf> November 2008.

<sup>5</sup> Environment Agency, Contaminants in soil: updated collation of toxicological data and intake values for humans: Dioxins, furans and dioxin-like PCBs in soil (September 2009)

## 3.0 FACILITY AND EMISSION CHARACTERISATION

The sections below describe the release parameters, emission concentrations and the resultant predicted emission rates.

### 3.1 Emission Source Process Conditions

The emission characteristics applied in the dispersion model and process conditions used to determine the pollutant emission rates used in the dispersion modelling process are presented in Table 3-1. These are consistent with Air Emissions Risk Assessment (AERA) report.

**Table 3-1**  
**Stack Emission Characteristics**

Parameter	Stack 1	Stack 2
Stack Location (NGR x/y)	433470/431232	433472/431235
Stack Internal Diameter (m)	1.9	1.9
Stack Exhaust Height (m AGL)	90m	90m
Volume Flow (Nm <sup>3</sup> /s) (273K, 11% O <sub>2</sub> , dry)	37.92	37.92
Emission Temperature (°C)	140	140
Oxygen Content (% O <sub>2</sub> dry gas)	7.64	7.64
Moisture content (% H <sub>2</sub> O)	19.07	19.07
Actual Flow Rate (Am <sup>3</sup> /s) (wet, at stack conditions)	52.88	52.88
Emission velocity (m/s)	18.8	18.8

### 3.2 Emissions of COPCs

#### 3.2.1 Dioxins

The emission rate of dioxins from the EfW has been determined on the basis of continuous operation at the IED emission limit for dioxins of 0.1 TEQng/Nm<sup>3</sup>.<sup>6</sup>

As each specific congener has different physico-chemical properties, congener-specific emission data are required. The congener profile will be dependent on various factors including the type of waste being burnt, the temperature of combustion, the type of combustion chamber being operated and the air pollution control devices (APCDs) installed. The most complete dataset for EfW emissions available is from measurements undertaken on behalf of the USEPA in 2000, which included congener profiles for over 150 large facilities (defined as over 250 tonnes per day treating over 28 million tonnes of municipal solid waste per annum). The USEPA

<sup>6</sup> The IED requires dioxins to be reported using the I-TEQ reporting convention to assess compliance against an emission limit of 0.1ng I-TEQ / Nm<sup>3</sup>. The UK's independent health advisory committee, Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT), has adopted the World Health Authority (WHO) toxicity equivalence factors (TEF) for both dioxins and dioxin-like PCBs in their recent review of Tolerable Daily Intake (TDI) criteria.

derived congener split<sup>7</sup> has been applied in this assessment given the number of datasets which support this data.

The mass emission of each congener has therefore been calculated on the basis of the USEPA congener profile, factored on the basis of WHO Toxic Equivalency Factors (TEF) at the IED emission concentration. The congener specific emission rates applied are detailed in the following table.

**Table 3-2**  
**Applied Dioxin Congener Emission Rates**

Compound	WHO TEFs for Dioxins	Emission Concentration (ng/Nm <sup>3</sup> )			Emission Rate (TEQ ng/s) (Total from 2-flues)
		US-EPA Measured	Factored to WHO-TEF <sup>(a)</sup>	Factored to ELV <sup>(b)</sup>	
2,3,7,8-TCCD	1	0.005	0.0050	0.0059	0.4460
1,2,3,7,8-	1	0.016	0.0160	0.0188	1.4272
1,2,3,4,7,8-Hx	0.1	0.016	0.0016	0.0019	0.1427
1,2,3,6,7,8-	0.1	0.037	0.0037	0.0044	0.3300
1,2,3,7,8,9-	0.1	0.032	0.0032	0.0038	0.2854
1,2,3,4,6,7,8-	0.01	0.219	0.0022	0.0026	0.1954
OCCD	0.0003	0.345	0.0001	0.0001	0.0092
2,3,7,8-TCDF	0.1	0.072	0.0072	0.0085	0.6423
1,2,3,7,8-	0.03	0.05	0.0015	0.0018	0.1338
2,3,4,7,8-	0.3	0.069	0.0207	0.0243	1.8465
1,2,3,4,7,8-	0.1	0.082	0.0082	0.0096	0.7315
1,2,3,6,7,8-	0.1	0.059	0.0059	0.0069	0.5263
1,2,3,7,8,9-	0.1	0.013	0.0013	0.0015	0.1160
2,3,4,6,7,8-	0.1	0.066	0.0066	0.0078	0.5887
1,2,3,4,6,7,8-	0.01	0.156	0.0016	0.0018	0.1392
1,2,3,4,7,8,9-	0.01	0.024	0.0002	0.0003	0.0214
OCDF	0.0003	0.09	0.00003	0.00003	0.0024
TOTAL		1.351	0.0850	0.1000	7.584

Table Note:

- a) US-EPA measured concentrations factored to WHO Toxic Equivalency Factors.
- b) US-EPA TEF Factored emission factored to meet emission limit of 0.1ng(TEQ)/m<sup>3</sup>

### 3.2.2 PCBs

The latest draft Waste Incineration BREF Note (December 2018), with respect to PCB's, states that '*emission levels are mostly at levels that are low and well below the levels of PCCD/F*'. The emission data used to inform the BREF (presented in Figure 3.50 of the BREF) has been reviewed and the highest concentration is 0.04ng WHO-TEQ/Nm<sup>3</sup> (measured at an old installation without a bag filter before publication of 2006 WI BREF).

The fate, transport and bioaccumulation properties for all PCB congeners are not available. Therefore the US-EPA approach (that is also commonly applied in the UK) of adopting a surrogate substance to represent a

<sup>7</sup> U.S. EPA. An Inventory of Sources and Environmental Releases of Dioxin-Like Compounds in the U.S. for the Years 1987, 1995, and 2000 (EPA/600/P-03/002f, Final Report, November 2006). U.S. Environmental Protection Agency, Washington, DC, EPA/600/P-03/002F.

combination of PCB's has been applied. Aroclor 1254 has been selected as this is representative of higher chlorinated PCB's (i.e. greater toxicity) and is therefore considered to represent a precautionary approach.

The model has assumed an emission of 3.0 TEQ ng/s (total from 2 flues).

### 3.3 Abnormal Emissions

Abnormal emissions concentrations for dioxins, furans and PCB emissions have been sourced from a study commissioned by the EA<sup>8</sup>. The study found a maximum concentration of 0.58ng(TEQ)/Nm<sup>3</sup> at any point during the start-up and shut-down process. As such this concentration has been applied to dioxins and the same factor (x 5.8) increase for the PCB's for the allowable 60 hours per year and is considered a precautionary approach.

### 3.4 Deposition Modelling

The air dispersion model has been used to provide deposition rates for dioxins as input to the HHRAP model. In the absence of specific UK guidance, this has been primarily undertaken based on guidance<sup>9</sup> issued by the United States Environmental Protection Agency (USEPA) as described in the following sections.

#### 3.4.1 Assignment of Phase

The emissions to atmosphere from the stacks occur as either vapour or particulate matter and the modelling methodology depends on the phase in which the pollutant is emitted from the facility.

Guidance indicates that in general it can be assumed that:

- most organic pollutants with very low volatility (i.e. fraction of the pollutant in the vapour phase is less than 0.05) occur only in the particle phase;
- highly volatile organic pollutants occur only in the vapour phase (i.e. the fraction of the pollutant in the vapour phase is 1.0);and
- the remaining pollutants are condensed onto the surface of particulate matter (particle-bound).

The fraction of the identified pollutants in the vapour phase, and the assigned phase for the EfW stack dispersion modelling, are presented in Table 3-3.

**Table 3-3**  
**Assigned Phases Dioxins and PCBs**

	Pollutant	Fraction in Vapour Phase (Fv) <sup>(a)</sup>	Assigned phase
Dioxins and Furans	2,3,7,8–TCCD	0.664	Vapour
	1,2,3,7,8–PeCDD	0.117	Particle-bound
	1,2,3,4,7,8–HxCDD	0.024	Particle
	1,2,3,6,7,8–HxCDD	0.029	Particle
	1,2,3,7,8,9–HxCDD	0.016	Particle
	1,2,3,4,6,7,8–HpCDD	0.003	Particle
	OCCD	0.002	Particle
	2,3,7,8–TCDF	0.77	Vapour
	1,2,3,7,8–PeCDF	0.268	Particle-bound
	2,3,4,7,8–PeCDF	0.221	Particle-bound

<sup>8</sup> AEA, Investigation of Waste Incinerator Dioxins During start-up and shutdown operating phases (2008)

<sup>9</sup> USEPA, Office of Solid Waste and Emergency Response, Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, FINAL September 2005. Chapter 3: Air Dispersion and Deposition Modelling.

	Pollutant	Fraction in Vapour Phase (Fv) <sup>(a)</sup>	Assigned phase
	1,2,3,4,7,8-HxCDF	0.049	Particle
	1,2,3,6,7,8-HxCDF	0.052	Particle
	1,2,3,7,8,9-HxCDF	0.09	Particle
	2,3,4,6,7,8-HxCDF	0.058	Particle
	1,2,3,4,6,7,8-HpCDF	0.01	Particle
	1,2,3,4,7,8,9-HpCDF	0.057	Particle
	OCDF	0.002	Particle
PCB's	Aroclor 1254	0.992	Vapour

Table Notes:

a) Data from the HHRAP companion database as detailed in Appendix A of the USEPA, OSW, Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, FINAL September 2005.

### 3.4.2 Particulate Deposition

Particle deposition is determined mainly by the particle size (aerodynamic) and density, with the terminal velocity of a particle determining how far and soon it will deposit. AERMOD incorporates 2 methods for modelling deposition of particles:

- Method 1 is used when a significant fraction (> 10%) of the total particulate mass has a diameter greater than 10 microns and the particle size distribution is reasonably well known.
- Method 2 is used when the particle size distribution is not well known and when a small fraction (less than 10% of the mass) consists of particles with a diameter of 10 microns or larger.

For this assessment, as data relating to particle size and density is limited, the Method 2 approach has been applied using published data<sup>10</sup>. As shown in Table 3-3, for the purposes of this assessment all dioxins (and furans) have been assumed to be particle (or particle-bound) with the exception of 2,3,7,8-TCDD and 2,3,7,8-TCDF which have been assumed to be vapour phase (gaseous).

**Table 3-4**  
**Assigned Deposition Parameters for Particulates**

Compound	Fine Mass Fraction	Mean Particle Diameter (µm)
Dioxins (particulate)	0.9	0.1

### 3.4.3 Vapour Deposition

Vapour phase compounds are deposited via both wet and dry processes, dependent on factors relating to their solubility etc. and not by particle size, mass or surface area. Published data<sup>11</sup> has been applied to individual pollutants as shown in the Table 3-5.

<sup>10</sup> Deposition Parameterizations for the Industrial Source Complex (ISC3) Model. Environmental Research Division, Argonne National Laboratory on behalf of US Department of Energy, June 2002.

<sup>11</sup> Deposition Parameterizations for the Industrial Source Complex (ISC3) Model. Environmental Research Division, Argonne National Laboratory on behalf of US Department of Energy, June 2002.

**Table 3-5**  
**Assigned Deposition Parameters for Vapours**

Compound	Diffusivity in air (cm <sup>2</sup> /s)	Diffusivity in water (cm <sup>2</sup> /s)	Cuticular resistance (s/cm)	Henry's constant (Pa m <sup>3</sup> /mol)
2,3,7,8-TCCD	0.05196	0.000004392	7.84	3.34
2,3,7,8-TCDF	0.05269	0.000004544	7.84	1.46
Aroclor 1254	0.04929	0.4000	3.3E-2	24

## 4.0 EXPOSURE SCENARIOS

### 4.1 Site and Surroundings

The Site is located in Skelton at NGR SE 334 312. The site is approximately 4.5km south east of Leeds city centre in an industrial area. The closest residential areas in the surrounding environment are Hunslet (approximately 1.3km northwest), Belle Isle (approximately 2.0km west), Rothwell (approximately 2.0km south), and Halton (approximately 1.6km north east).

### 4.2 Assessment Exposure Pathways

On the basis of all the potential exposure scenarios as defined in Section 2.3 the following specific exposure scenarios have been identified as of relevance to this HHRA (as illustrated in Figure 1):

- inhalation;
- ingestion of food - beef, pork, chicken and home-grown produce (including breast milk); and
- ingestion of soil.

Exposure pathways are determined by the diet of the receptor and the proportion of which is local produce. For example a residential receptor is unlikely to habitually ingest home-reared pork. However a residential receptor is reasonably likely to ingest home-grown vegetables. As a worst case the farmer exposure route has been assessed (summarised in Table 4-1).

The consumption of fish from local water bodies, as a result of recreational fishing is likely to be very small (i.e. does not form a regular supplement in the diet). Therefore no 'Fisher' scenario has been investigated.

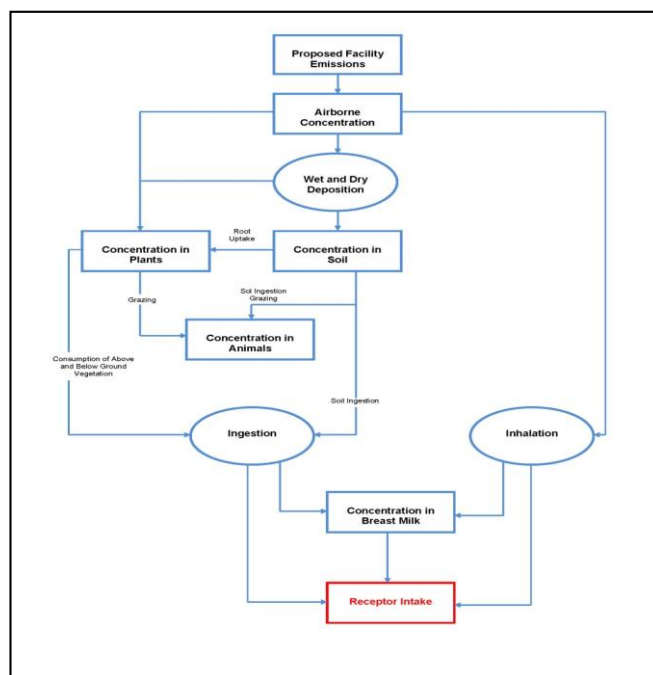
The local population does not obtain its drinking water from local surface water sources and has treated drinking water provided by Yorkshire Water Treatment Plants; therefore ingestion of water as a pathway has been excluded.

The receptor types are divided into adults and children. Children are sensitive receptors because they are more likely to ingest soil and dusts directly and have lower body weights, so that the effect of the same dose is likely to be greater in the child than in the adult.

**Table 4-1**  
**Ingestion Exposure by Receptor Type**

Ingestion of:	Farmer Adult	Farmer Child
Milk From Home-Reared Cows	✓	✓
Eggs From Home-Reared Chickens	✓	✓
Home-Reared Beef	✓	✓
Home-Reared Pork	✓	✓
Home-Reared Chicken	✓	✓
Home-Grown Vegetable/Fruit	✓	✓
Local Fish	X	X
Breastmilk	X	✓
Soil (Incidental)	✓	✓





**Figure 1**  
**Exposure Scenario**

#### 4.2.1 Evaluation of infant exposure via breast milk to Dioxins and Furans

The ingestion of mother’s breast milk by infants is identified as an exposure pathway to PCDDs and PCDFs warranting separate evaluation in the HHRAP. The potential for contamination of breast milk is high for dioxin-like compounds such as these, as they are highly lipophilic (fat soluble) and hence likely to accumulate in breast milk. This exposure is measured by the Average Daily Dose (ADD) on the basis of an averaging time of 1 year as has been compared to the EA TDI of 2pg I-TEQ/kg BW per day.

#### 4.3 Identification of Sensitive Receptor Locations

The dispersion modelling domain was an area of 5km radius from the Site (the point of maximum ground level impact occurs within this limit) with a nested grid.

The HHRA has considered a hypothetical worst-case scenario i.e., a Farmer receptor (child and adult) present at the point of maximum (long term average) impact of the emissions from the facility consuming predominantly home-grown plants and home-reared animals which occurs approximately 1km east on land allocated for logistics and light manufacturing use (i.e. this is not an actual ‘farmer’ exposure location).

#### 4.4 Site Parameters for Estimating Media Concentrations

Site parameters for estimating media concentrations were specified for the study area as follows:

- annual average precipitation of 603.2 mm/annum<sup>12</sup>;
- annual average evapo-transpiration rate of 487.3 mm/annum (CEH<sup>13</sup>);
- annual average irrigation of 0 mm/annum (assumed not to be significant);
- annual average runoff of 115.9 mm/annum;
- an annual average wind velocity of 4.65m/s (from Leeds-Bradford Meteorological Station);

<sup>12</sup> Church Fenton Met Office Station 1981 -2010 Average

<sup>13</sup> <https://eip.ceh.ac.uk/apps/chess/>

- a time period over which deposition occurs of 50 years (highly precautionary assumption considering likely life of installation); and
- exposure period for adult of 90 years.

## 5.0 ASSESSMENT OF IMPACT

### 5.1 Summary of Exposure to Dioxins and Furans

The total exposure (i.e. including both inhalation and indirect pathways) for all dioxins, furans and dioxin like PCB's as a percentage of the TDI is presented in Table 5-1 below. The intake as a result of the process contribution is small at less than 1% of the TDI and can therefore be considered insignificant.

**Table 5-1**  
**Dioxin, Furan and PCBs Daily Intake**

Location	Type	Dioxins PC (pg TEQ/kg(BW)/day)	PCBs PC (pg TEQ/kg(BW)/day)	Dioxins + PCBs PC (pg TEQ/kg(BW)/day)	As % of TDI
Maximum Ground Level Impact (hypothetical exposure)	Farmer Adult	2.9E-03	4.6E-03	7.5E-03	0.37%
	Farmer Child	4.2E-03	6.9E-03	1.1E-02	0.55%

The IRAP model calculates the Average Daily Dose (ADD) that would result from an adult receptor breast feeding an infant; a summary of the ADD (total sum of all PCDDs and PCDFs) for the infants of a Farmer adult receptor considered for the assessment is presented in Table 5-2. The ADD is below the TDI.

**Table 5-2**  
**Assessment of Infant ADD to Dioxins and Furans via Breast Milk**

Location	Infant ADD [pg TEQ/kg BW/day]
Maximum Ground Level Impact (hypothetical exposure)	0.40

### 5.2 Consideration of worst-case 'Abnormal emissions'

The dioxin and PCB intake has been pro-rated on the basis of the abnormal emissions as described in Section 3.3 and then combined to present the cumulative potential impact (of dioxins and dioxin-like PCB's). The intake at all receptors remains well below the TDI.

**Table 5-3**  
**Abnormal Emissions Dioxin Daily Intake**

Location	Type	Dioxins + PCBs PC (pg TEQ/kg(BW)/day)	As % of TDI
Maximum Ground Level Impact (hypothetical exposure)	Farmer Adult	7.7E-03	0.39%
	Farmer Child	1.1E-02	0.57%

## 6.0 CONCLUSIONS

The findings of the assessment are that the predicted impacts as a consequence of emissions from the EfW are all within limits for the protection of human health as defined by the Environment Agency and intake of dioxins and PCBs at all receptors are well below the EA's adopted Tolerable Daily Intake value of 2pg I-TEQ/kg BW/day.

This conclusion is considered robust on the basis of the worst-case approach adopted in the characterisation of emissions, the safety factors incorporated into the US-EPA HHRA Protocol, and the hypothetical worst case exposure scenario considered in the assessment.

## APPENDIX A

Model Files (electronic only)

**Model files provided separately**

## EUROPEAN OFFICES

### United Kingdom

#### AYLESBURY

T: +44 (0)1844 337380

#### BELFAST

T: +44 (0)28 9073 2493

#### BRADFORD-ON-AVON

T: +44 (0)1225 309400

#### BRISTOL

T: +44 (0)117 906 4280

#### CAMBRIDGE

T: +44 (0)1223 813805

#### CARDIFF

T: +44 (0)29 2049 1010

#### CHELMSFORD

T: +44 (0)1245 392170

#### EDINBURGH

T: +44 (0)131 335 6830

#### EXETER

T: +44 (0)1392 490152

#### GLASGOW

T: +44 (0)141 353 5037

#### GUILDFORD

T: +44 (0)1483 889800

#### LEEDS

T: +44 (0)113 258 0650

#### LONDON

T: +44 (0)203 691 5810

#### MAIDSTONE

T: +44 (0)1622 609242

#### MANCHESTER

T: +44 (0)161 872 7564

#### NEWCASTLE UPON TYNE

T: +44 (0)191 261 1966

#### NOTTINGHAM

T: +44 (0)115 964 7280

#### SHEFFIELD

T: +44 (0)114 245 5153

#### SHREWSBURY

T: +44 (0)1743 23 9250

#### STAFFORD

T: +44 (0)1785 241755

#### STIRLING

T: +44 (0)1786 239900

#### WORCESTER

T: +44 (0)1905 751310

### Ireland

#### DUBLIN

T: +353 (0)1 296 4667

### France

#### GRENOBLE

T: +33 (0)4 76 70 93 41