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NORTH BECK ENERGY CENTRE

APPENDIX 8.3 HUMAN HEALTH RISK ASSESSMENT

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1 INTRODUCTION

This Human Health Risk Assessment (HHRA) has been undertaken to support the Air Quality EIA for the proposed North Beck Energy Centre. The North Beck Energy Centre includes an energy recovery facility which will burn Refuse Derived Fuel (RDF). As explained in the ES chapter the Proposed Development will be regulated by the Environment Agency and need an Environmental Permit to operate. At the time of writing this report, the plant will be required to meet the Emission Limit Values outlined in Chapter IV and Annex VI of the Industrial Emissions Directive (IED) (2010/75/EU) for waste incineration and co-incineration plants. It is noted that the Waste Incineration BAT Reference Document (the BREF) is currently being consulting upon, once finalised any permit will need to comply with the proposed Air Emission Limits (AELs) set out in the final BREF. As currently worded the draft BREF introduces BAT AELs which are lower than those set out in the IED. However, as a worst-case for the purpose of this analysis, it has been assumed that the energy recovery facility will continually operate at the BAT AELS as currently drafted, noting actual impacts are likely to be lower.

The advice from health specialists such as the Health Protection Agency is that the damage to health from emissions from incineration and co-incineration plants is likely to be very small, and probably not detectable. Nevertheless, the specific effects on human health of the Proposed Development have been considered, and are presented in this report.

For most substances released from the energy recovery facility, the most significant effects on human health will arise by inhalation. The Air Quality Assessment Levels (AQALs) outlined in Chapter 8.0 of the ES have been set by the various authorities at a level which is considered to present minimum or zero risk to human health. It is widely accepted that, if the concentrations in the atmosphere are less than the AQALs, then the pollutant is unlikely to have an adverse effect on human health.

For some pollutants which accumulate in the environment, inhalation is only one of the potential exposure routes. Therefore, other exposure routes are considered in this HHRA. This Appendix sets out the results and approach taken.

2 ISSUE IDENTIFICATION

2.1 Issue

The key issue is the release of substances from the proposed energy recovery facility to atmosphere which have the potential to harm human health. No other sources will include emissions of either metals or dioxins. The energy recovery facility is located approximately 1.5km to the east of Immingham. The area surrounding the site is mainly industrial with the closest residential dwellings being along Queens Road approximately 560m from the main stack.

The energy recovery facility will be designed to meet the emission limits outlined in the IED (2010/75/EU). Limits have been set for pollutants known to be produced during the combustion of RDF which have the potential to impact upon the local environment either on human health or ecological receptors. These pollutants include:

- nitrogen dioxide, sulphur dioxide, particulate matter, carbon monoxide, ammonia;
- acid gases - hydrogen chloride, and hydrogen fluoride;
- total organic carbon;
- metals - mercury, cadmium, thallium, antimony, arsenic, lead, cobalt, copper, manganese, nickel and vanadium;
- dioxins and furans;
- dioxinlike PCBs; and
- polycyclic aromatic hydrocarbons (PAHs).

For most substances released from the Facility, the most significant effects on human health will arise by inhalation. The dispersion modelling assessment contained in Appendix 8.2 has been undertaken to determine the impact of atmospheric concentrations of the pollutants listed above based on the levels transposed under UK Law in the UK Air Quality Strategy and those set by the Environment Agency. These levels have been set at a level which is considered to present minimum or zero risk to human health.

Some pollutants, including dioxins, furans, dioxin-like polychlorinated biphenyls (PCBs) and heavy metals, accumulate in the environment, which means that inhalation is only one of the potential exposure routes. Therefore, impacts cannot be evaluated in terms of their effects on human health by simply reference to ambient air quality standards. An assessment needs to be made of the overall human exposure to the substances from both inhalation and ingestion by the local population and the risk that this exposure causes.

2.2 Chemicals of Potential Concern (COPC)

The substances which have been considered within this assessment are those which are authorised (as listed above). Although Emission Limit Values (ELVs) for PAHs are not currently set from installations, monitoring is required by legislation in the UK. Therefore, benzo(a)pyrene has been included in the assessment to represent PAH emissions. The following have been considered COPCs for the purpose of this assessment:

- PCDD/Fs (individual congeners) and dioxin like PCBs;
- Benzene
- Benzo(a)pyrene;
- Mercury (Hg);
- Mercuric chloride;
- Cadmium (Cd);
- Arsenic (As);
- Chromium (Cr), trivalent and hexavalent; and
- Nickel (Ni).

This risk assessment investigates the potential for long term health effect of these COPCs through other routes than just inhalation.

3 ASSESSMENT CRITERIA

The IRAP-h model calculates the total exposure through each of the different pathways so that a dose from inhalation and ingestion can be calculated for each receptor. By default, these doses are then used to calculate a cancer risk, using the United States Environmental Protection Agency (USEPA) approach. However, the results are assessed using the UK's approach, which is explained in the Environment Agency's document "Human Health Toxicological Assessment of Contaminants in Soil", ref SC050021. This approach involves two types of assessment:

- For those substances with a threshold level for toxicity, a Tolerable Daily Intake (TDI) is defined. This is "an estimate of the amount of a contaminant, expressed on a bodyweight basis, which can be ingested daily over a lifetime without appreciable health risk." A Mean Daily Intake (MDI) is also defined, which is the typical intake from background sources (including dietary intake) across the UK. In order to assess the impact of the CHP Plant, the predicted intake of a substance due to emissions from the CHP Plant is added to the MDI and compared with the TDI.
- For substances without a threshold level for toxicity, an Index Dose (ID) is defined. This is a level of exposure which is associated with a negligible risk to human health. The predicted intake of a substance due to emissions from the CHP Plant is compared directly with the ID without taking account of background levels.

Substances can reach the body either through inhalation or through ingestion (oral exposure) and the body handles chemicals differently, depending on the route of exposure. For this reason, different TDI and IDs are defined for inhalation and oral exposure.

The following table outlines the MDIs (the typical intake from existing background sources) for the pollutants released from the Facility. These figures are defined in the "Contaminants in soil: updated collation of toxicology data and intake values for humans" series of toxicological reports, available from the Environment Agency's website.

Table 3.1: Mean Daily Intake of Each Substance

Substance	Mean Daily Intake, 70 kg adult (µg/kg bw/day)		Mean Daily Intake, 20 kg child (µg/kg bw/day)	
	Intake Ingestion	Intake, Inhalation	Intake Ingestion	Intake, Inhalation
Arsenic	0.07	0.0002	0.19	0.0005
Benzene	0.04	2.9	0.11	7.4
Benzene(a)pyrene	-	-	-	-
Cadmium	0.19	0.0003	0.5	0.0007
Chromium	1.81	0.0009	4.70	0.0011
Chromium (VI)	0.18	-	0.49	-
Methyl mercury	0.007	-	0.019	-
Mercuric chloride	0.014	-	0.037	-
Nickel	1.9	0.0037	4.96	0.0096
Dioxins and dioxin like PCBs	0.7 pg WHO-TEQ/kg bw/day		1.8 pg WHO-TEQ/kg bw/day	

Table 3.2: Tolerable Daily Intake of Each Substance (µg/kg bw/day)				
Substance	**Index dose, Ingestion**	**Index dose, Inhalation**	**TDI, Ingestion**	**TDI, Inhalation**
Arsenic	0.3	0.002	-	-
Benzene	0.29	1.4	-	-
Benzene(a)pyrene	0.02	0.00007	-	-
Cadmium	-	-	0.36	0.0014
Chromium	-	-	3 *	-
Chromium (VI)	-	0.001	3 *	-
Methyl mercury	-	-	0.23	0.23
Mercuric chloride	-	-	2	0.06
Nickel	-	-	2.8	0.006
Dioxins and dioxin like PCBs	-	-	2 pg WHO-TEQ/kg bw/day	

NOTES:

* The TDI for chromium is actually based on toxicological data for chromium (VI). Therefore, an assessment has been made of the total chromium against the TDI and chromium (VI) only.

To allow comparison with the TDI for dioxins, intake values for each dioxin are multiplied by a factor known as the WHO-TEF. A full list of the WHO-TEF values for each dioxin is provided in Appendix A.

The following table presents the MDI for an adult and child as a proportion of the TDI.

Table 3.3: Mean Daily Intake of Each Substance as a % of the TDI				
Substance	**Mean Daily Intake, 70 kg adult (µg/kg bw/day)**		**Mean Daily Intake, 20 kg child (µg/kg bw/day)**	
	Intake Ingestion	**Intake, Inhalation**	**Intake Ingestion**	**Intake, Inhalation**
Cadmium	53.2%	20.4%	137.7%	52.9%
Chromium	60.5%	-	156.6%	-
Chromium (VI)	6.0%	-	15.7%	-
Methyl mercury	3.1%	-	8.0%	-
Mercuric chloride	0.7%	-	1.9%	-
Nickel (screening)	68.4%	61.7%	177.1%	159.7%
Nickel (based on monitoring data)	-	23.8%	-	61.7%
Dioxins and dioxin like PCBs	35.0%		90.7%	

As shown, the MDI of cadmium, chromium and nickel from existing sources exceeds the TDI for children.

The MDI for **chromium** is set for chromium III and taken from the DEFRA report "Contaminants in Soil: Collation of Toxicological Data and Intake Values for Humans. Chromium". This states that there are no published reports on the adverse effects in humans resulting from ingested chromium III. Almost all toxicological opinion is that chromium III compounds are of low oral toxicity, and indeed the UK Committee on Medial Aspects of Food Policy recommends chromium III in the diet. The World Health Organisation (WHO) have reviewed the daily intake of chromium from foods and found that existing levels do not represent a toxicity problem. The WHO conclude that "in the form of trivalent compounds, chromium is an essential nutrient and is relatively non-toxic for man and other mammalian species".

The DEFRA report explains that the TDI has been derived from the USEPA's Reference Dose of 3µg/kg bw/day for chromium VI. This is the only explicitly derived safety limit for oral exposures of chromium. DEFRA recommends that the USEPA Reference Dose is applied to all the chromium content as a starting point. Therefore, the TDI presented in Table 3.2 is actually the TDI for chromium VI not chromium. Assessing the total dietary intake of chromium against this TDI is highly conservative. When the MDI chromium VI is compared against the TDI the MDI is well below the TDI. When considering chromium impacts, the total chromium and chromium (VI) have been assessed separately against the TDI.

The key determinant of **cadmium**'s toxicity potential is its chronic accumulation in the kidney. The Environment Agency in their toxicology report "SC050021/TOx 3) explain that chronic exposure to levels in excess of the TDI might be associated with an increase in kidney disease in a proportion of those exposed, but (small) exceedances lasting for shorter periods are of less consequence. Therefore, assessing a lifetime exposure is appropriate. If we assess the exposure of a receptor over a lifetime (i.e. a period as a child and adult) the lifetime MDI is below the TDI.

The MDI and TDI (oral) for **nickel** has been revised following the publication by the European Food Safety Authority of new expert opinion relating to the reproductive and developmental effects in experimental animals. The MDI exceeds the TDI for children for both inhalation and ingestion. The MDI for inhalation is 0.26µg/day for an adult which, assuming an inhalation rate of 20m^3/day, equates to an atmospheric concentration of 13.0ng/m^3. A review of the monitoring data of nickel across the UK between 2011 and 2015 has shown that concentrations at urban background locations (excluding the sites at Sheffield Tinsley and Swansea Coedgwilym which are close to significant sources of nickel) are 1.38ng/m^3 on average, with a maximum of 3.04ng/m^3. At urban industrial sites the concentration is slightly higher with average concentrations (again excluding those located close to significant sources of nickel) is 1.81ng/m^3 on average, with a maximum of 5.00ng/m^3 which is significantly below the MDI

Therefore, the recommended MDI for inhalation is conservative for the proposed Facility, as the proposed location is in an industrial areas away from significant sources of nickel. Applying this maximum background concentration, the MDI would be 0.10µg/day or 23.8% of the inhalation TDI for an adult and 61.7% of the TDI for a child. This has been used as the value of the MDI for the remainder of this analysis.

4 CONCEPTUAL SITE MODEL

4.1 Conceptual site model

A detailed Human Health Risk Assessment has been carried out using the Industrial Risk Assessment Program-Human Health (IRAP-h View – Version 4.0). The programme, created by Lakes Environmental, is based on the United States Environment Protection Agency (USEPA) Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities[1]. This Protocol is a development of the approach defined by Her Majesties Inspectorate on Pollution (HMIP) in the UK in 1996[2], taking account of further research since that date. The exposure pathways included in the IRAP model are shown in Figure 1.

Exposure to gaseous contaminants has the potential to occur by direct inhalation or vapour phase transfer to plants. In addition, exposure to particulate phase contaminants may occur via indirect pathways following the deposition of particles to soil. These pathways include:

- Ingestion of soil and dust;
- Uptake of contaminants from soil into the food-chain (through home-grown produce and crops); and
- Direct deposition of particles onto above-ground crops.

The pathways through which inhalation and ingestion occur and the receptors that have been considered to be impacted via each pathway are:

• Direct inhalation	All receptors
• Ingestion of soil	All receptors
• Ingestion of home-grown produce	All receptors
• Ingestion of drinking water	All receptors
• Ingestion of eggs from home-grown chickens	Agricultural receptors
• Ingestion of home-grown chickens	Agricultural receptors
• Ingestion of home-grown beef	Agricultural receptors
• Ingestion of home-grown pork	Agricultural receptors
• Ingestion of home-grown milk	Agricultural receptors
• Ingestion of breast milk	Infants only

It is noted that some households may keep chickens and consume eggs and potentially the birds. The impact on these households is considered to be between the impact at an agricultural receptor and a standard resident receptor. The approach used considers an agricultural receptor at the point of maximum impact as a complete worst case.

As shown in Figure 1, the pathway from the ingestion of mother's milk in infants is considered within the assessment. This considers all dioxins and dioxin-like PCBs. The IRAP model calculates the amount of these COPCs entering the mother's milk and being passed on to the infants. The impacts are then compared against the TDI.

[1] USEPA (2005) Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities.

[2] HMIP (1996) Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes.

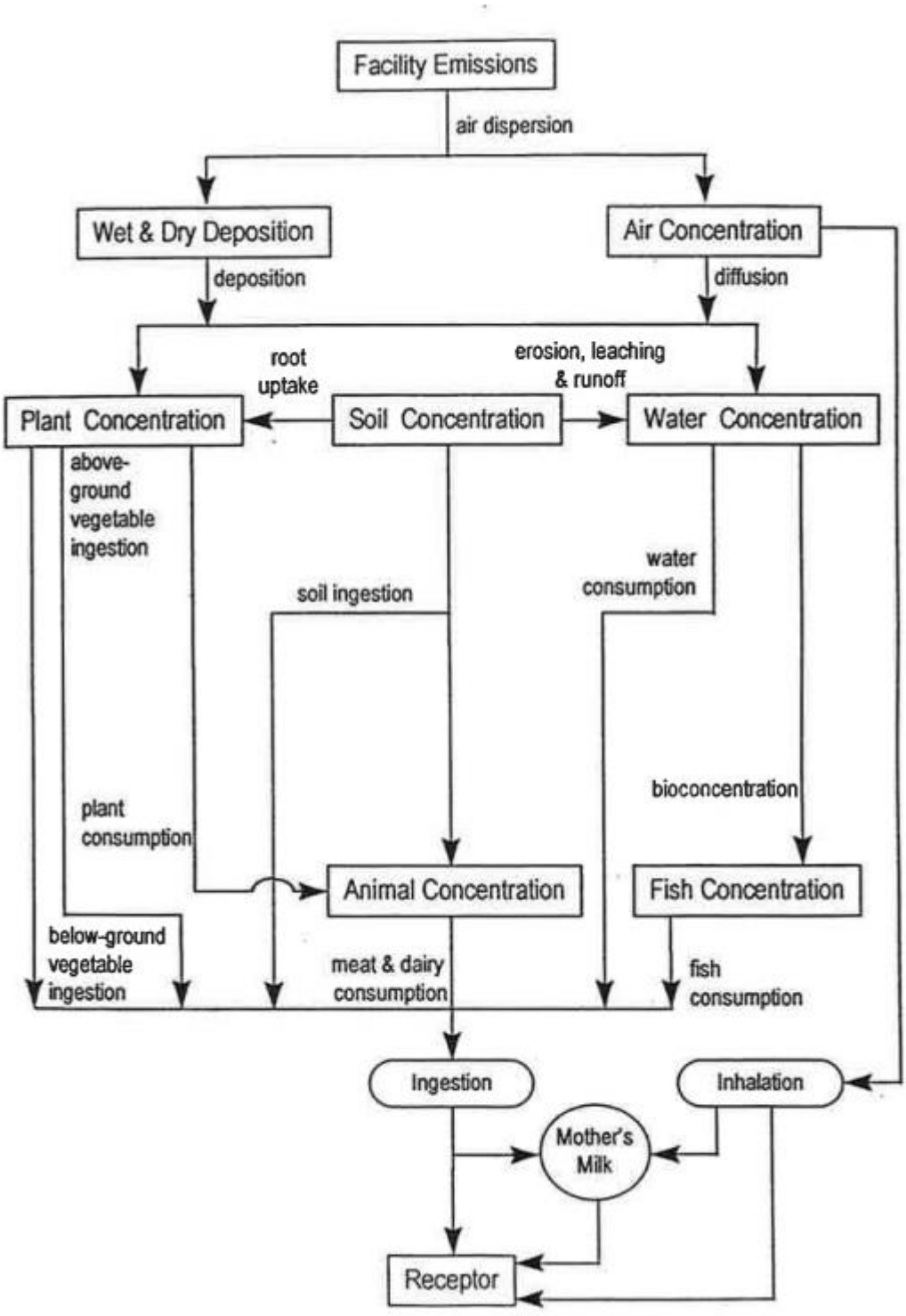

Figure 1: Conceptual Site Model – Exposure Pathways

4.2 Pathways excluded from assessment

The intake of dioxins via dermal absorption, groundwater, surface water exposure and consumption of fish pathways is very limited and as such these pathways are excluded from the HHRA. The justification for excluding these pathways is highlighted in the following sections.

4.2.1 Dermal absorption

Both the HMIP and the USEPA note that the contribution from dermal exposure to soils impacted from thermal treatment facilities is typically a very minor pathway and is very small relative to contributions resulting from exposures via the food chain.

The USEPA[3] provide an example from the risk assessment conducted for the Waste Technologies Industries, Inc. hazardous thermal treatment plant in East Liverpool, Ohio. This indicated that for an adult subsidence farmer in a subarea with high exposures, the risk resulting from soil ingestion and dermal contact was 50-fold less than the risk from any other pathway and 300-fold less than the total estimated risk.

The HMIP document[4] provides a screening calculation using conservative assumptions, which states that for a 1pg I-TEQ/m^3 value the intake via dermal absorption is 30 times lower than the intake via inhalation, which is itself a minor contributor to the total risk.

As such, the pathway from dermal absorption is deemed to be an insignificant risk and has been excluded from this assessment.

4.2.2 Groundwater

Exposure via groundwater can only occur if it is contaminated and consumed untreated by an individual.

The USEPA[5] have concluded that the build up of dioxins in the aquifer over realistic travel times relevant to human exposure was predicted to be so small as to be essentially zero.

As such, the pathway from groundwater is deemed to be an insignificant risk and has been excluded from this assessment.

4.2.3 Surface water

It is noted that a possible pathway is via deposition of emissions directly onto surface water – i.e. local drinking water supplies or rainwater storage tanks.

Surface water generally goes through several treatment steps and so any contaminants would be removed from the water before consumption. It is noted that run off to rainwater tanks may not go through the same treatment. However, rain water tanks have a very small surface area and as such the potential for deposition and build-up of COPCs is limited. As such, the pathway from contaminated surface water is deemed to be an insignificant risk and has been excluded from this assessment.

4.2.4 Consumption of fish

The consumption of locally caught fish has been excluded from the assessment. Whilst it is noted that fish make up a proportion of the UK diet, it is not likely that this would be sourced in significant amounts from inland fisheries in close proximity to the Facility. Therefore this pathway has been excluded from this assessment.

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[3] USEPA (2005) Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities.

[4] HMIP (1996) Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes.

[5] USEPA (2005) Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities.

5 SENSITIVE RECEPTORS

This assessment considers the possible effects on human health at key receptors, where humans are likely to be exposed to the greatest impact from the Facility, and at the point of maximum impact of annual mean emissions.

For the purposes of this assessment, 'Residential' and 'Agricultural' receptors have been identified and can be defined as follows:

- Residential: A known place of residence that is occupied within the study area;
- Agricultural: A farm holding or area of land of horticultural interest.

The emissions from the Facility are expected to be significant only in the locality of the plant. The specific receptors identified in Chapter 8 of the ES have been considered in this assessment. In addition, a 'Point of maximum impact' receptor has been selected at the point of maximum impact, although it should be noted that this point is actually within the industrial area adjacent to the Humber Estuary and uninhabited.

These sensitive receptors are listed in Table 5.1. These receptors are the same as those used in the dispersion modelling assessment. Reference should be made to Figure 8.33 of the ES which shows the location of these receptors with respect to the Facility. An impact for an agricultural receptor is typically predicted to be greater than a residential receptor, due to the additional pathways considered.

Table 5.1: Sensitive Receptors

ID	Receptor Name	Location		Type of Receptor
		X	Y	
MAX	Point of maximum impact	521060	415410	Agricultural / Residential
R1	Queens Road, Immingham	520077	414761	Residential
R2	Chestnut Avenue, Immingham	519237	415004	Residential
R3	North Moss Lane, Stallingborough	521283	413113	Residential
R4	Brickpit Farm, South Marsh Road, Stallingborough	521167	412586	Residential
R5	South Marsh Road, Stallingborough	520844	412118	Residential
R6	Church Lane, Stallingborough	519605	411701	Residential
R7	Keelby Road, Stallingborough	518675	412019	Residential
R8	Mauxhall Farm, Immingham Road, Stallingborough	519194	413216	Agricultural
R9	Havenmere Residential Care Home	518767	414853	Residential
R10	Stark Lincolnshire and Goole Hospitals	518797	414983	Residential
R11	Canon Peter Hall Church of England Primary School	518654	414749	Agricultural
R12	Eastfield Primary School	518341	414021	Agricultural
R13	Oasis Academy Immingham	518428	414585	Agricultural

6 IRAP Model Assumptions and Inputs

The following section details the user defined assumptions used within the IRAP model and provides justifications where appropriate.

6.1 Concentration in soil

The concentration of each chemical in the soil is calculated from the deposition results of the air quality modelling for vapour phase and particle phase deposition. The critical variables in calculating the accumulation of pollutants in the soil are as follows:

- The lifetime of the Facility is taken as 30 years.
- The soil mixing depth is taken as 2 cm in general and 15 cm for produce.

The split between the solid and vapour phase for the substance considered depends on the specific physical properties of each chemical.

In order to assess the amount of substance which is lost from the soil each year through volatilisation, leaching and surface run-off, a soil loss constant is calculated. The rates for leaching and surface runoff are taken as constant, while the rate for volatilisation is calculated from the physical properties of each substance.

6.2 Concentration in plants

The concentrations in plants are determined by considering direct deposition and air-to-plant transfer for above ground produce, and root uptake for above-ground and below-ground produce. The calculation takes account of the different types of plant; for example, uptake of substances through the roots will differ for below-ground and above-ground vegetables, and deposition onto plants will be more significant for above-ground vegetables.

6.3 Concentration in animals

The concentrations in animals, based on consumption of plants, are calculated from the concentrations in plants, assumed consumption rates and bio-concentration factors. These vary for different animals and different substances, since the transfer of chemicals between the plants consumed and animal tissue varies.

It is also assumed that 100% of the plant materials eaten by animals is grown on soil contaminated by emission sources. This is likely to be a highly pessimistic assumption for UK farming practice.

6.4 Concentration in humans

6.4.1 Intake via inhalation

This is calculated from inhalation rates of typical adults and children and atmospheric concentrations. The inhalation rates used for adults and children are:

- Adults - 20m^3/day; and
- Children – 7.2m^3/day.

These are as specified within the Environment Agency series of reports: "Contaminants in soil: updated collation of toxicology data and intake values for humans". The calculation also takes account of time spent outside, since most people spend most of their time indoors.

6.4.2 Intake via soil ingestion

This calculation allows for the ingestion of soil and takes account of different exposure frequencies. It allows for ingestion of soil attached to unwashed vegetables, unintended ingestion when farming or gardening and, for children, ingestion of soil when playing.

6.4.3 Ingestion of food

The calculation of exposure due to ingestion of food draws on the calculations of concentrations in animals and plants and takes account of different ingestion rates for the various food groups by different age groups.

For most people, locally-produced food is only a fraction of their diet and so exposure factors are applied to allow for this.

6.4.4 Breast milk ingestion

For infants, the primary route of exposure is through breast milk. The calculation draws on the exposure calculation for adults and then allows for the transfer of chemicals in breast milk to an infant who is exclusively breast-fed.

The only pathway considered for dioxins for a breast feeding infant is through breast milk. The modelled scenario consists of the accumulation of pollutants in the food chain up to an adult receptor, the accumulation of pollutants in breast milk and finally the consumption of breast milk by an infant.

The assumptions used were:

- Exposure duration of infant to breast milk 1 year
- Proportion of ingested dioxin that is stored in fat 0.9%
- Proportion of mother's weight that is stored in fat 0.3%
- Fraction of fat in breast milk 0.04%
- Fraction of ingested contaminant that is absorbed 0.9%
- Half life of dioxins in adults 2,555 days
- Ingestion rate of breast milk 0.688kg/day

6.5 Estimation of COPC concentration in media

The IRAP-h model uses a database of physical and chemical parameters to calculate the COPC concentrations through each of the different pathways identified. The base physical and chemical parameters have been used in this assessment.

In order to calculate the COPC concentrations, a number of site specific pieces of information are required.

- Weather data was obtained from the Monks Wood meteorological recording station for the years 2012 – 2016, as used within the Air Quality Assessment. This provides the annual average precipitation which can be used to calculate the general IRAP-h input parameters:

Table 6.1: Ground Type Dependent Properties		
Input Variable	**Assumption**	**Value (cm/year)**
Annual average evapo-transpiration	70% of annual average precipitation	43.9
Annual average irrigation	0% of annual average precipitation	0.00
Annual average precipitation	100% of annual average precipitation	62.7
Annual average runoff	10% of annual average precipitation	6.3

- The average wind speed was taken as 5.1 m/s, calculated from the average of the 5 years of weather data for the period 2012-2016 from the Humberside Airport weather station.

A number of assumptions have been made with regard to the deposition of the different phases. These are summarised in the following table.

Table 6.2: Deposition Assumptions			
Deposition Phase	**Dry Deposition Velocities (m/s)**	**Ratio Dry deposition to Wet deposition**	
		Dry Deposition	**Wet Deposition**
Vapour	0.005	1.0	2.0
Particle	0.010	1.0	2.0
Bound particle	0.010	1.0	2.0
Mercury vapour	0.029	1.0	0
The above deposition velocities have been agreed with the UK Environment Agency for all IRAP based assessments where modelling of specific deposition of pollutants is not undertaken. These are considered to be conservative.			

These deposition assumptions have been applied to the annual mean concentrations predicted using the dispersion modelling, to generate the inputs needed for the IRAP modelling. For details of the dispersion modelling methodology please refer to Appendix 8.2 of the ES.

6.6 Modelled emissions

For the purpose of this assessment it is assumed that the Facility operates at the IED ELVs for its entire operational life. In reality, the Facility will be shut down for periods of maintenance and monitoring of similar facilities in the UK shows that they operate below the ELVs. In addition, it is likely that the limits set out in the permit will be lower than these if the WID BREF is finalised before the granting of an Environmental Permit to operate.

The following tables give the emissions rates of each COPC modelled and the associated IED ELVs which have been used to derive the emission rate.

Table 6.3: COPC Emissions Modelled			
COPC	**Emission Limit Value (mg/Nm^3)**	**Emission rate**	**Units**
Benzene	10	1249.1	µg/s
PAHs (Benzo(a)pyrene)	0.0003	37.473	ng/s
Elemental mercury	0.0001	12.491	ng/s
Mercuric chloride	0.024	2.998	µg/s
Cadmium	0.025	3.123	µg/s
Arsenic	0.025	3.123	µg/s
Chromium	0.092	11.492	µg/s
Chromium, hexavalent	0.00013	16.238	ng/s
Nickel	0.220	27.48	µg/s

Table 6.4: COPC Emissions Modelled		
COPC	**Emission Limit Value (ng I-TEQ/Nm^3)**	**Emission rate (ng/s)**
TetraCDD,2,3,7,8	0.1	0.387
HexaCDD,1,2,3,7,8,9		2.561
OctaCDD,1,2,3,4,6,7,8,9		50.489
HeptaCDD,1,2,3,4,6,7,8		21.285
OctaCDF,1,2,3,4,6,7,8,9		44.543
HexaCDD,1,2,3,4,7,8		3.585
PentaCDD,1,2,3,7,8		3.060
TetraCDF,2,3,7,8		3.460
HeptaCDF,1,2,3,4,7,8,9		5.359
PentaCDF,2,3,4,7,8		6.683
PentaCDF,1,2,3,7,8		3.460
HexaCDF,1,2,3,6,7,8		10.080
HexaCDD,1,2,3,6,7,8		3.223
HexaCDF,2,3,4,6,7,8		10.880
HeptaCDF,1,2,3,4,6,7,8		54.898
HexaCDF,1,2,3,4,7,8		27.218
HexaCDF,1,2,3,7,8,9		0.525
Dioxin like PCBs	0.0092	1.149

A number of points should be noted for each group of COPCs:

(1) Benzene (Table 6.3).

a) It has been assumed that the entire TOC emissions consist of only benzene.

b) It has been assumed that TOC emissions are emitted at the daily ELV.

(2) PAHs (Table 6.3).

a) It has been assumed that the entire PAH emissions consist of only benzo(a)pyrene.

b) Benzo(a)pyrene is not a regulated pollutant within the IED. The highest recorded emission concentration of Benzo(a)pyrene from the UK Environment Agency's public register was 0.105ug/m³, or 0.000105mg/m³ (dry, 11% oxygen, 273K). As this is not a regulated pollutant and only monitored periodically we have applied a safety factor of 2.

(3) Group 1 metals - mercury and compounds (Table 6.3).

a) It has been assumed that the ELV of total mercury is 0.05mg/Nm^3

b) The concentration of elemental mercury has been taken as 0.2% of the total mercury and compounds ELV

c) The concentration of mercury chloride has been taken as 48% of the total mercury and compounds ELV.

d) The losses to the global cycle have been taken as 51.8% of the total mercury and compounds ELV.

(4) Group 2 metals - cadmium compounds (Table 6.3).

a) The assessment is based on the IED ELV of 0.05mg/Nm³ for cadmium, thallium and compounds.

b) It is assumed that the emissions of cadmium are half of the combined ELV for cadmium and thallium, whereas monitoring of existing EfW Facilities has shown that these are typically 14% of the limit as set out in Appendix 8.2 of the ES

(5) Group 3 metals – antimony, arsenic, chromium, lead and nickel (Table 6.3).

a) The assessment is based on the IED ELV of 0.5mg/Nm³ for "other metals".

b) The emissions of each of the nine "other metals" in the third group have been taken as no worse than a currently operating facility as detailed in Table A1 of the Environment Agency "Guidance on assessing group 3 metals stack emissions from incinerators – v4", which is reproduced in Table 6.5. This data is based on monitoring at 18 MWI and Waste Wood Co-Incinerators between 2007 and 2015 operating under the IED in the UK.

(6) Dioxins and furans (Table 6.4).

These are a group of similar halogenated organic compounds, which are generally found as a complex mixture. The toxicity of each compound is different and is generally expressed as a Toxic Equivalent Factor (TEF), which relates the toxicity of each individual compound to the toxicity of 2,3,7,8-TCDD, the most toxic dioxin. A full list of the TEF values for each dioxin is provided in Appendix A. The total concentration is then expressed as a Toxic Equivalent (TEQ).

The split of the different dioxins and furans is based on split of congeners for a release of 0.1ng I-TEQ/Nm³ as presented in Table A.7.

To determine the Emission Rate, the concentration of each compound has been derived from the split of the different dioxins for a total emission concentration of 0.1ng I-TEQ/Nm³. This concentration has then been multiplied by the normalised flow rate as shown in Table 6.6 and IRAP has been used to model the distribution of each compound, taking account of the physical and chemical properties of the compound. The output of IRAP, which is the expected dose for each compound, has then been multiplied by the TEF value set out in Table A.7 at the end of this report to determine the total dioxin dose in ITEQ.

(7) Dioxin like PCBs (Table 6.4).

There are a total of 209 PCBs, which act in a similar manner to dioxins, are generally found in complex mixtures and also have TEFs.

The UK Environment Agency has advised that 44 measurements of dioxin-like PCBs have been taken at 24 MWIs between 2008 and 2010. The following data summarises the measurements, all at 11% reference oxygen content:

- Maximum = 9.2 x 10^{-3}ng[TEQ]/m^3
- Mean = 2.6 x 10^{-3}ng[TEQ]/m^3
- Minimum = 5.6 x 10^{-5}ng[TEQ]/m^3

For the purpose of this assessment, as a conservative assumption, the maximum monitored PCB concentration has been used which has been converted to an emission rate using the volumetric flow rate at reference conditions.

The IRAP software, and the HHRAP database which underpins it, does not include any data on individual PCBs, but it does include data for take-up and accumulation rates within the food chain for two groups of PCBs, known as Aroclor 1254 and Aroclor 1016. Each Arocolor is based on a fixed composition of PCBs. Since we are not aware of any data on the specification of PCBs within incinerator emissions, as a worst case assumption we have assumed that the PCBs are released in each of the two Aroclor compositions.

Table 6.5: Monitoring Data from Municipal Waste Incinerators

Pollutant	Measured Concentration as % of IED Group 3 Limit		
	Mean	Max	Min
Antimony	0.28%	2.30%	0.02%
Arsenic	0.20%	5.00%	0.04%
Chromium	1.68%	18.40%	0.04%
Chromium (VI)	0.007%	0.026%	0.00046%
Cobalt	2.20%	1.12%	0.04%
Copper	1.50%	5.80%	0.38%
Lead	2.18%	10.06%	0.06%
Manganese	3.36%	12.00%	0.30%
Nickel	3.00%	44.00%	0.50%
Tin	-	-	-
Vanadium	0.08%	1.20%	0.02%
Total (calculated)	**14.5%**	**99.9%**	**1.4%**

NOTES:

The two highest nickel concentrations are outliers being 44%, as above, and 27% of the ELV. The third highest concentration is 0.53 mg/Nm³ or 11% of the ELV.

Table 6.6: Basis for the Emission Rate of Dioxins and Furans		
Dioxin / furan	**Concentration of Congeners (ng/Nm^3) for a total release of 0.1 ng I-TEQ/Nm^3**	**Emission rate (ng/s)**
2,3,7,8-TCDD	0.0031	0.387
1,2,3,7,8-PeCDD	0.0245	3.060
1,2,3,4,7,8-HxCDD	0.0287	3.585
1,2,3,6,7,8-HxCDD	0.0258	3.223
1,2,3,7,8,9-HxCDD	0.0205	2.561
1,2,3,4,6,7,8-HpCDD	0.1704	21.285
1,2,3,4,6,7,8,9-OctaCDD	0.4042	50.489
2,3,7,8-TCDF	0.0277	3.460
1,2,3,7,8-PCDF	0.0277	3.460
2,3,4,7,8-PCDF	0.0535	6.683
1,2,3,4,7,8-HxCDD	0.2179	27.218
1,2,3,6,7,8-HxCDF	0.0807	10.080
1,2,3,7,8,9-HxCDF	0.0042	0.525
2,3,4,6,7,8-HxCDF	0.0871	10.880
1,2,3,4,6,7,8-HpCDF	0.4395	54.898
1,2,3,4,7,8,9-HpCDF	0.0429	5.359
1,2,3,4,6,7,8,9-OctaCDF	0.3566	44.543
Total	2.0150	-

7 RESULTS

7.1 At point of maximum impact

The following tables outline the impact of emissions from the Facility at the point of maximum impact for an 'Agricultural' receptor. As explained in section 4, this receptor type includes the direct inhalation pathway, and ingestion from soil, drinking water, and home-grown eggs and chicken, beef, pork, and milk. This specific receptor assumes that the person lives at the point of maximum impact and consumes home-grown produce, etc. This is considered an unlikely worst-case scenario, especially given that this point is located within the non-residential industrial area adjacent to the Humber Estuary. Reference should be made to Figure 8.33 of the ES for the location of the point in relation to the Facility. Where appropriate a comparison has been made to the TDI or ID.

The TDI is an estimate of the amount of a contaminant, expressed on a bodyweight basis, which can be ingested daily over a lifetime without appreciable health risk. As shown in Table 7.1 for the worst-case receptor the overall impact (including the contribution from existing dietary intakes) is less than the TDI for methyl mercury, mercuric chloride and dioxins. Therefore, there would not be an appreciable health risk based on the emission of these pollutants.

For a child receptor, the cadmium, chromium and nickel MDI (that sourced from existing dietary intake) exceeds the TDI. However, the process contribution is exceptionally small and the exceedance is a reflection of the fact the MDI is over 100% of the TDI. On this basis, it is not considered that the Facility would increase the health risks from cadmium, chromium or nickel for children significantly.

As noted in Section 3, the key determinant of cadmium's toxicity potential is its chronic accumulation in the kidney. The Environment Agency explains that chronic exposure to levels in excess of either the TDI might be associated with an increase in kidney disease in a proportion of those exposed, but (small) exceedances lasting for shorter periods are of less consequence. If we assess the lifetime exposure (i.e. a period being a child and an adult) the overall impact is well below the TDI. Therefore, there would not be an appreciable health risk based on the emission of cadmium over a lifetime of an individual.

As explained in Section 3, almost all toxicological opinion is that chromium III compounds are of low oral toxicity and the WHO states that "in the form of trivalent compounds, chromium is an essential nutrient and is relatively non-toxic for man and other mammalian species". Although the TDI for chromium is predicted to be exceeded, this is due to existing dietary intake. The WHO has reviewed the daily intake of chromium from foods and found that existing levels do not represent a toxicity problem, and state that "in the form of trivalent compounds, chromium is an essential nutrient and is relatively non-toxic for man and other mammalian species". The TDI is based on the USEPA's Reference Dose for chromium VI. Assessing the total dietary intake of chromium against this TDI is highly conservative. As the process contribution is small, the existing levels of chromium do not represent a toxicity problem, and as the TDI is highly conservative there would not be an appreciable health risk based on the emission of chromium over a lifetime of an individual. If the intake of chromium VI is assessed against the TDI for chromium the total intake for a child is only 15.67%, of which the Facility contributes 0.00203% at the point of maximum impact which is within the Humber Estuary.

For nickel, the MDI for ingestion again exceeds the TDI for the child receptor. However, the maximum process contribution is only 2.44% for an agricultural child receptor, and 0.36% for a residential child receptor. This is based on the conservative assumption that the process contribution is based on emissions of nickel at 44% of the group ELV. As outlined in Table 6.5, this is the maximum of the monitoring data and is an outlier. The third highest concentration was 11%, and the mean 3%, of the group ELV. If we assume the plant operates as per the 3rd highest – i.e. 11% of the group ELV – then the process contribution would be 1.12% of the ingestion TDI at the point of maximum impact for the agricultural child receptor. On this basis, it is not considered that the Facility would increase the health risks from nickel for children significantly.

Table 7.1: Impact Analysis – TDI – Point of Maximum Impact						
Substance	**MDI (% of TDI)**		**Process Contribution (% of TDI)**		**Overall (% of TDI)**	
	Inhalation	**Ingestion**	**Inhalation**	**Ingestion**	**Inhalation**	**Ingestion**
Adult – Agricultural						
Cadmium	20.41%	53.17%	7.51%	0.21%	27.92%	53.38%
Chromium	-	60.48%	-	0.87%	-	61.35%
Chromium VI	-	6.05%	-	0.00125%	-	6.05%
Methyl mercury	-	3.11%	-	0.08%	-	3.19%
Mercuric chloride	-	0.71%	-	0.30%	-	1.02%
Nickel(1)	23.81%	68.37%	15.42%	1.60%	39.22%	69.97%
Dioxins(2)	35.00%		9.13%		44.13%	
Child – Agricultural						
Cadmium	52.86%	137.72%	9.46%	0.48%	62.32%	138.20%
Chromium	-	156.63%	-	1.43%	-	158.06%
Chromium VI	-	15.66%	-	0.00203%	-	15.67%
Methyl mercury	-	8.04%	-	0.18%	-	8.22%
Mercuric chloride	-	1.85%	-	0.51%	-	2.36%
Nickel(1)	61.67%	177.07%	19.42%	2.44%	81.09%	179.51%
Dioxins(2)	90.65%		12.98%		103.63%	
Adult – Residential						
Cadmium	20.41%	53.17%	7.51%	0.13%	27.92%	53.31%
Chromium	-	60.48%	-	0.07%	-	60.55%
Chromium VI		6.05%				6.05%
Methyl mercury	-	3.11%	-	0.03%	-	3.14%
Mercuric chloride	-	0.71%	-	0.04%	-	0.76%
Nickel(1)	23.81%	68.37%	15.42%	0.15%	39.22%	68.52%
Dioxins(2)	35.00%		0.21%		35.21%	
Child – Residential						
Cadmium	52.86%	137.72%	9.46%	0.32%	62.32%	138.04%
Chromium	-	156.63%	-	0.21%	-	156.85%
Chromium VI		15.66%		0.00030%		15.66%
Methyl mercury	-	8.04%	-	0.09%	-	8.14%
Mercuric chloride	-	1.85%	-	0.17%	-	2.02%
Nickel(1)	61.67%	177.07%	19.42%	0.36%	81.09%	177.43%
Dioxins(2)	90.65%		0.72%		91.37%	

NOTES:
(1) Based on Nickel released at 44% of the IED ELV
(2) total dioxins and dioxin like PCBs

The ID is the level of exposure which is associated with a negligible risk to human health. As shown, for this worst-case receptor the process contribution is well below the ID. Therefore, emissions from the Facility are considered to have a negligible impact on human health.

Table 7.2: Impact Analysis – ID – Point of Maximum Impact

Substance	Inhalation (% of ID)	Ingestion (% of ID)
Adult – Agricultural		
Arsenic	5.26%	0.42%
Benzene	3.00%	0.35%
Benzo[a]pyrene	1.80%	3.78%
Chromium (VI)	0.05%	-
Child – Agricultural		
Arsenic	6.62%	0.74%
Benzene	3.78%	0.82%
Benzo[a]pyrene	2.27%	5.47%
Chromium (VI)	0.07%	-
Adult – Residential		
Arsenic	5.26%	0.16%
Benzene	3.00%	0.37%
Benzo[a]pyrene	1.80%	0.04%
Chromium (VI)	0.05%	-
Adult – Residential		
Arsenic	6.62%	0.38%
Benzene	3.78%	0.66%
Benzo[a]pyrene	2.27%	0.10%
Chromium (VI)	0.07%	-

The total accumulation of dioxins in an infant, considering the breast milk pathway and based on the agricultural adult receptor at the point of maximum impact feeding an infant, is 1.096 pg WHO-TEQ / kg-bw / day which is 54.8% of the TDI. Again this is highly unlikely as this point is located within the Humber Estuary. The following section details the impact at the most impacted receptor which is a more appropriate measure of the likely impact (albeit still conservative) of the Facility on the local environment.

7.2 Maximum impact at a receptor

The following tables outline the impact of emissions from the Facility at the most affected receptor (i.e the receptor with the greatest impact from ingestion and inhalation of emissions) (R8 – Mauxhall Farm). Where appropriate a comparison has been made to the TDI or ID.

Table 7.3: Impact Analysis – TDI –Maximum Impacted Receptor						
Substance	**MDI (% of TDI)**		**Process Contribution (% of TDI)**		**Overall (% of TDI)**	
	Inhalation	**Ingestion**	**Inhalation**	**Ingestion**	**Inhalation**	**Ingestion**
Adult						
Cadmium	20.41%	53.17%	1.04%	0.03%	21.45%	53.20%
Chromium	-	60.48%	-	0.12%	-	60.60%
Chromium VI		6.05%	-	0.00017%	-	6.05%
Methyl mercury	-	3.11%	-	0.012%	-	3.12%
Mercuric chloride	-	0.71%	-	0.042%	-	0.76%
Nickel(1)	23.81%	68.37%	2.13%	0.221%	25.94%	68.59%
Dioxins(2)	35.00%		1.261%		36.26%	
Child						
Cadmium	52.86%	137.72%	1.31%	0.07%	54.16%	137.79%
Chromium	-	156.63%	-	0.20%	-	156.83%
Chromium VI	-	15.66%	-	0.00028%	-	15.66%
Methyl mercury	-	8.04%	-	0.02%	-	8.07%
Mercuric chloride	-	1.85%	-	0.07%	-	1.92%
Nickel(1)	61.67%	177.07%	2.68%	0.356%	64.35%	177.43%
Dioxins(2)	90.65%		1.793%		92.44%	

NOTES:
(1) Based on Nickel released at 44% of the IED ELV
(2) total dioxins and dioxin like PCBs

As shown, for the most impacted receptor the overall impact (including the contribution from existing dietary intakes) is less than the TDI for methyl mercury, mercuric chloride and dioxins. Therefore, there would not be an appreciable health risk based on the emission of these pollutants.

For a child receptor, the cadmium, chromium and nickel MDI (that sourced from existing dietary intake) exceeds the TDI for ingestion. However, the process contribution is exceptionally small and the exceedance is a reflection of the fact that the MDI is over 100% of the TDI. On this basis, it is not considered that the Facility would increase the health risks from cadmium, chromium or nickel for children significantly.

The total accumulation of dioxins in an infant, considering the breast milk pathway and based on the most impacted residential receptor, R8 feeding an infant, is 0.151 pg WHO-TEQ / kg bw / day which is 7.57% of the TDI.

Table 7.4: Impact Analysis – ID – Maximum Impacted Receptor		
Substance	**Inhalation (% of ID)**	**Ingestion (% of ID)**
Adult		
Arsenic	0.73%	0.06%
Benzene	0.42%	0.048%
Benzo[a]pyrene	0.25%	0.523%
Chromium (VI)	0.01%	-
Child		
Arsenic	0.92%	0.10%
Benzene	0.52%	0.114%
Benzo[a]pyrene	0.31%	0.755%
Chromium (VI)	0.01%	-

As shown, for this worst-case receptor, the process contribution is well below the ID. Therefore, emissions from the Facility are considered to have a negligible impact on human health.

7.3 Uncertainty and sensitivity analysis

To account for uncertainty in the modelling, the impact on human health was assessed for a receptor at the point of maximum impact.

To account for uncertainty in the dietary intake of a person, both residential and agricultural receptors have been assessed. The agricultural receptor is assumed to consume a greater proportion of home grown produce, which has the potential to be contaminated by the COPCs released, than for a residential receptor. In addition, the agricultural receptor includes the pathway from consuming animals grazed on land contaminated by the emission source. This assumes that 100% of the plant materials eaten by the animals is grown on soil contaminated by emission sources.

The agricultural receptor, being at the point of maximum impact, is considered the upper maximum of the impact of the Facility.

7.4 Upset process conditions

Article 46(6) of the IED (Directive 2010/75/EU) states that:

> *"… the waste incineration plant … shall under no circumstances continue to incinerate waste for a period of more than 4 hours uninterrupted where emission limit values are exceeded.*
>
> *The cumulative duration or operation in such conditions over 1 year shall not exceed 60 hours."*

Article 47 continues with:

> *"In the case of a breakdown, the operator shall reduce or close down operations as soon as practicable until normal operations can be restored."*

In addition Annex VI, Part 3, 2 of the IED states the emission limit values applicable in the circumstances described in Article 46(6) and Article 47:

> *"The total dust concentration in the emissions into the air of a waste incineration plant shall under no circumstances exceed 150 mg/Nm^3 expressed as a half-hourly average. The air emission limit values for TOC and CO set out in points 1.2 and 1.5(b) shall not be exceeded."*

The conditions detailed in Article 46(6) are considered to be "Upset Operating Conditions". As identified, these periods are short term events which can only occur for a maximum of 60 hours per year.

Start-up of the Facility from cold will be conducted with clean support fuel (low sulphur light fuel oil). During start-up, RDF will not be introduced onto the grate unless the temperature within the oxidation zone is above the 850ºC as required by Article 50, paragraph 4(a) of the IED. During start-up, the flue gas treatment plant will be operational as will the combustion control systems and emissions monitoring equipment.

The same is true during plant shutdown, where RDF will cease to be introduced to the grate. The RDF remaining on the grate will be combusted, the temperature not being permitted to drop below 850ºC through the combustion of clean support auxiliary fuel. During this period the flue gas treatment equipment will be fully operational, as will be the control systems and monitoring equipment. After complete combustion of the RDF, the auxiliary burners will be turned off and the plant will be allowed to cool.

Start-up and shut-down are infrequent events. The Facility is designed to operate continuously, and ideally only shut down for its annual maintenance programme.

In relation to the magnitude of dioxin emissions during plant start-up and shut-down, research has been undertaken by AEA Technology on behalf of the Environment Agency[6]. Whilst elevated emissions of dioxins (within one order of magnitude) were found during shutdown and start-up phases where the fuel was not fully established in the combustion chamber, the report concluded that:

> *"The mass of dioxin emitted during start-up and shut-down for a 4-5 day planned outage was similar to the emission which would have occurred during normal operation in the same period. The emission during the shutdown and restart is equivalent to less than 1 % of the estimated annual emission (if operating normally all year)."*

There is therefore no reason why such start-up and shut-down operations or upset operating conditions will affect the long term impact of the Facility.

[6] AEA Technology (2012) Review of research into health effects of Energy from Waste facilities.

8 CONCLUSIONS

Of all the pollutants considered with a Tolerable Daily Intake (TDI), nickel is the pollutant that results in the highest level of existing exposure (MDI). The combined impact of nickel from existing background sources and contributions from the proposed Facility at the point of maximum impact is 179.51% of the ingestion TDI for an agricultural child type receptor. However, the process contribution from the Facility for nickel is small, being only 2.44% of the TDI at the point of maximum impact, and 0.36% or less at receptors. This is based on the worst case assumption that emissions of nickel are 44% of the group ELV. Analysis by the Environment Agency states that this is an outlier[7]. If it is assumed that emissions of nickel are 11% of the group ELV then the impact is less than 1% of the TDI for ingestion at the point of maximum impact for an agricultural child type receptor. On this basis it is not considered that the Facility would increase the health risks from nickel for children significantly.

Similarly, the ingestion of cadmium and chromium from existing background sources and contributions from the proposed Facility also exceeds the ingestion TDI for children. However, the process contribution from the proposed Facility for cadmium is again small, being only 0.48% of the TDI at the point of maximum impact for an agricultural receptor, and 0.07% or less at actual receptors. The process contribution for chromium is also small, being only 1.43% of the TDI at the point of maximum impact, and 0.20% or less at receptors.

The TDI is set at a level "that can be ingested daily over a lifetime without appreciable health risk" (WHO). The ingestion of cadmium and chromium by children as a result of background sources, based on UK-wide data, is already above the TDI. On the basis that the process contribution of these substances is exceptionally small it is not considered that the Facility would increase the health risks from this pollutant significantly.

For all other pollutants, the combined impact from the Facility plus the existing MDI is below the TDI, so there would not be an appreciable health risk based on the emission of these pollutants.

Although the MDI exceeds the cadmium TDI for children, the Environment Agency explains that chronic exposure to levels in excess of either the TDI might be associated with an increase in kidney disease in a proportion of those exposed, but (small) exceedances lasting for shorter periods are of less consequence. Therefore, assessing a lifetime exposure is appropriate. If we assess the exposure over the lifetime (i.e. a period as a child and adult) the overall impact is well below the TDI, so there would not be an appreciable health risk based on the emission of cadmium.

Again, the TDI for chromium for children is predicted to be exceeded due to existing dietary intake. Toxicological opinion is that chromium III is of low oral toxicity and is needed as part of a healthy diet. The UK Committee on Medial Aspects of Food Policy recommend a minimum safe and adequate intake, but do not restrict an upper limit. The WHO have analysed human intake for chromium through food and conclude that existing levels do not represent a toxicity problem. The TDI is based on the USEPA's Reference Dose for chromium VI. Assessing the total dietary intake of chromium against this TDI is highly conservative. The total intake of chromium VI is significantly below the TDI and the process contribution of chromium VI is extremely small. Therefore, as the process contribution is so small and the TDI is set at a highly conservative level, it is concluded that there would not be an appreciable health risk based on the emission of chromium.

[7] An **outlier** is an observation point that is distant from other observations and may be due to variability in the measurement or it may indicate experimental error; the latter are sometimes excluded from the data set.

For pollutants which do not have a TDI, a comparison has been made against an Index Dose (ID). The ID is a threshold below which there are considered to be negligible risks to human health. The greatest contribution from the Facility is from arsenic, which is only 6.6% of the Index Dose for children at the point of maximum impact. Therefore, emissions from the Facility of arsenic and all other pollutants are considered to have a negligible impact on human health.

In conclusion, the Facility will not result in appreciable health risks from its operation.

Appendix A - Detailed Results Tables

Table A.1: Comparison with ID Limits for Adult Receptors							
Receptor	Ingestion (% of ID)			Inhalation (% of ID)			
	Arsenic	Benzene	Benzo(a)pyrene	Arsenic	Benzene	Benzo(a)pyrene	Chromium (VI)
Point of maximum impact - agricultural	0.424%	0.350%	3.783%	5.255%	3.003%	1.802%	0.055%
Point of maximum impact - agricultural	0.157%	0.371%	0.037%	5.255%	3.003%	1.802%	0.055%
R1	0.010%	0.024%	0.002%	0.339%	0.194%	0.116%	0.004%
R2	0.015%	0.036%	0.004%	0.513%	0.293%	0.176%	0.005%
R3	0.011%	0.026%	0.003%	0.367%	0.210%	0.126%	0.004%
R4	0.011%	0.026%	0.003%	0.369%	0.211%	0.126%	0.004%
R5	0.010%	0.024%	0.002%	0.347%	0.198%	0.119%	0.004%
R6	0.011%	0.027%	0.003%	0.379%	0.216%	0.130%	0.004%
R7	0.012%	0.029%	0.003%	0.410%	0.234%	0.141%	0.004%
R8	0.059%	0.048%	0.523%	0.726%	0.415%	0.249%	0.008%
R9	0.015%	0.035%	0.004%	0.498%	0.284%	0.171%	0.005%
R10	0.014%	0.033%	0.003%	0.464%	0.265%	0.159%	0.005%
R11	0.039%	0.032%	0.350%	0.486%	0.278%	0.167%	0.005%
R12	0.055%	0.045%	0.487%	0.676%	0.386%	0.232%	0.007%
R13	0.041%	0.034%	0.362%	0.503%	0.288%	0.173%	0.005%
Max R1 to R13	0.059%	0.048%	0.523%	0.726%	0.415%	0.249%	0.008%

Table A.2: Comparison with ID Limits for Child Receptors							
Receptor	Ingestion (% of ID)			Inhalation (% of ID)			
	Arsenic	Benzene	Benzo(a)pyrene	Arsenic	Benzene	Benzo(a)pyrene	Chromium (VI)
Point of maximum impact - agricultural	0.742%	0.822%	5.469%	6.621%	3.784%	2.270%	0.069%
Point of maximum impact - agricultural	0.379%	0.659%	0.101%	6.621%	3.784%	2.270%	0.069%
R1	0.024%	0.043%	0.007%	0.427%	0.244%	0.146%	0.004%
R2	0.037%	0.064%	0.010%	0.647%	0.370%	0.222%	0.007%
R3	0.026%	0.046%	0.007%	0.463%	0.264%	0.159%	0.005%
R4	0.027%	0.046%	0.007%	0.465%	0.266%	0.159%	0.005%
R5	0.025%	0.043%	0.007%	0.437%	0.250%	0.150%	0.005%
R6	0.027%	0.048%	0.007%	0.477%	0.273%	0.164%	0.005%
R7	0.030%	0.051%	0.008%	0.517%	0.295%	0.177%	0.005%
R8	0.102%	0.114%	0.755%	0.915%	0.523%	0.314%	0.010%
R9	0.036%	0.062%	0.010%	0.627%	0.358%	0.215%	0.007%
R10	0.033%	0.058%	0.009%	0.585%	0.334%	0.201%	0.006%
R11	0.069%	0.076%	0.506%	0.613%	0.350%	0.210%	0.006%
R12	0.095%	0.106%	0.703%	0.852%	0.487%	0.292%	0.009%
R13	0.071%	0.079%	0.524%	0.634%	0.362%	0.217%	0.007%
Max R1 to R13	0.102%	0.114%	0.755%	0.915%	0.523%	0.314%	0.010%

Table A.3: Comparison with TDI Limits for Adult Receptors							
Receptor	Ingestion (% of ID)					Inhalation (% of ID)	
	Cadmium	Chromium	Methyl Mercury	Mercuric Chloride	Nickel	Cadmium	Nickel
MDI of TDI (%)	**53.17%**	**60.48%**	**3.11%**	**0.71%**	**68.37%**	**20.41%**	**23.81%**
Point of maximum impact - agricultural	53.381%	61.350%	3.190%	1.019%	69.967%	27.916%	39.225%
Point of maximum impact - agricultural	53.307%	60.549%	3.139%	0.755%	68.515%	27.916%	39.225%
R1	53.183%	60.481%	3.108%	0.717%	68.377%	20.893%	24.804%
R2	53.188%	60.483%	3.109%	0.718%	68.382%	21.142%	25.316%
R3	53.184%	60.481%	3.108%	0.717%	68.378%	20.933%	24.886%
R4	53.184%	60.481%	3.108%	0.717%	68.378%	20.935%	24.892%
R5	53.183%	60.481%	3.108%	0.717%	68.377%	20.903%	24.826%
R6	53.184%	60.481%	3.108%	0.717%	68.378%	20.949%	24.921%
R7	53.185%	60.482%	3.108%	0.717%	68.379%	20.994%	25.012%
R8	53.203%	60.597%	3.117%	0.756%	68.588%	21.446%	25.940%
R9	53.187%	60.483%	3.109%	0.718%	68.381%	21.119%	25.270%
R10	53.186%	60.483%	3.109%	0.718%	68.380%	21.072%	25.172%
R11	53.194%	60.557%	3.113%	0.742%	68.515%	21.103%	25.236%
R12	53.201%	60.589%	3.116%	0.754%	68.573%	21.374%	25.793%
R13	53.194%	60.560%	3.114%	0.743%	68.521%	21.127%	25.286%
Max R1 to R13	53.203%	60.597%	3.117%	0.756%	68.588%	21.446%	25.940%

Table A.4: Comparison with TDI Limits for Child Receptors							
Receptor	Ingestion (% of ID)					Inhalation (% of ID)	
	Cadmium	Chromium	Methyl Mercury	Mercuric Chloride	Nickel	Cadmium	Nickel
MDI of TDI (%)	**137.72%**	**156.63%**	**8.04%**	**1.85%**	**177.07%**	**52.86%**	**24.67%**
Point of maximum impact - agricultural	138.203%	158.058%	8.219%	2.356%	179.510%	62.316%	81.090%
Point of maximum impact - agricultural	138.041%	156.847%	8.137%	2.023%	177.428%	62.316%	81.090%
R1	137.743%	156.647%	8.049%	1.861%	177.094%	53.467%	62.920%
R2	137.753%	156.654%	8.053%	1.867%	177.106%	53.781%	63.564%
R3	137.745%	156.648%	8.050%	1.862%	177.096%	53.518%	63.024%
R4	137.745%	156.648%	8.050%	1.862%	177.096%	53.521%	63.030%
R5	137.743%	156.647%	8.050%	1.861%	177.095%	53.481%	62.948%
R6	137.745%	156.649%	8.050%	1.862%	177.097%	53.539%	63.067%
R7	137.747%	156.650%	8.051%	1.864%	177.099%	53.595%	63.182%
R8	137.789%	156.830%	8.068%	1.920%	177.408%	54.164%	64.351%
R9	137.752%	156.654%	8.052%	1.866%	177.105%	53.753%	63.507%
R10	137.750%	156.652%	8.052%	1.865%	177.103%	53.693%	63.383%
R11	137.767%	156.765%	8.060%	1.897%	177.297%	53.732%	63.464%
R12	137.784%	156.817%	8.066%	1.915%	177.385%	54.074%	64.166%
R13	137.768%	156.770%	8.060%	1.898%	177.305%	53.763%	63.527%
Max R1 to R13	137.789%	156.830%	8.068%	1.920%	177.408%	54.164%	64.351%

Table A.5: Comparison with Total Dioxin TDI Limits for Adult Receptors				
Receptor	**Total Inhalation, (pg WHO-TEQ kg^{-1} bw day^{-1})**	**Total Ingestion, (pg WHO-TEQ kg^{-1} bw day^{-1})**	**Total uptake, (pg WHO-TEQ kg^{-1} bw day^{-1})**	**Comparison (% of limit)**
MDI (% of TDI)				**35.00%**
Point of maximum impact - agricultural	4.74E-04	1.82E-01	1.83E-01	44.13%
Point of maximum impact - agricultural	4.74E-04	3.80E-03	4.28E-03	35.21%
R1	3.06E-05	2.45E-04	2.76E-04	35.01%
R2	4.63E-05	3.71E-04	4.18E-04	35.02%
R3	3.31E-05	2.66E-04	2.99E-04	35.01%
R4	3.33E-05	2.67E-04	3.00E-04	35.01%
R5	3.13E-05	2.51E-04	2.82E-04	35.01%
R6	3.42E-05	2.74E-04	3.08E-04	35.02%
R7	3.70E-05	2.96E-04	3.33E-04	35.02%
R8	6.55E-05	2.52E-02	2.52E-02	36.26%
R9	4.49E-05	3.60E-04	4.05E-04	35.02%
R10	4.19E-05	3.36E-04	3.77E-04	35.02%
R11	4.39E-05	1.68E-02	1.69E-02	35.84%
R12	6.10E-05	2.34E-02	2.35E-02	36.17%
R13	4.54E-05	1.74E-02	1.75E-02	35.87%
Max R1 to R13	6.55E-05	2.52E-02	2.52E-02	36.26%

Table A.6: Comparison with Total Dioxin TDI Limits for Adult Receptors				
Receptor	**Total Inhalation, (pg WHO-TEQ kg^{-1} bw day^{-1})**	**Total Ingestion, (pg WHO-TEQ kg^{-1} bw day^{-1})**	**Total uptake, (pg WHO-TEQ kg^{-1} bw day^{-1})**	**Comparison (% of limit)**
MDI (% of TDI)				**90.65%**
Point of maximum impact - agricultural	5.98E-04	2.59E-01	2.60E-01	103.63%
Point of maximum impact - agricultural	5.98E-04	1.39E-02	1.45E-02	91.37%
R1	3.85E-05	8.95E-04	9.33E-04	90.70%
R2	5.84E-05	1.36E-03	1.41E-03	90.72%
R3	4.17E-05	9.69E-04	1.01E-03	90.70%
R4	4.19E-05	9.73E-04	1.02E-03	90.70%
R5	3.94E-05	9.15E-04	9.54E-04	90.70%
R6	4.31E-05	9.99E-04	1.04E-03	90.70%
R7	4.66E-05	1.08E-03	1.13E-03	90.71%
R8	8.26E-05	3.58E-02	3.59E-02	92.44%
R9	5.66E-05	1.31E-03	1.37E-03	90.72%
R10	5.28E-05	1.22E-03	1.28E-03	90.71%
R11	5.53E-05	2.40E-02	2.40E-02	91.85%
R12	7.69E-05	3.33E-02	3.34E-02	92.32%
R13	5.72E-05	2.48E-02	2.49E-02	91.89%
Max R1 to R13	8.26E-05	3.58E-02	3.59E-02	92.44%

Table A.7: TEF Factors	
Compound	**WHO-TEF Multiplier**
HeptaCDD, 1,2,3,4,6,7,8-	0.0031
HeptaCDF, 1,2,3,4,6,7,8-	0.0245
HeptaCDF, 1,2,3,4,7,8,9-	0.0287
HexaCDD, 1,2,3,4,7,8-	0.0258
HexaCDD, 1,2,3,6,7,8-	0.0205
HexaCDD, 1,2,3,7,8,9-	0.1704
HexaCDF, 1,2,3,4,7,8-	0.4042
HexaCDF, 1,2,3,6,7,8-	0.0277
HexaCDF, 1,2,3,7,8,9-	0.0277
HexaCDF, 2,3,4,6,7,8-	0.0535
OctaCDD, 1,2,3,4,6,7,8,9-	0.2179
PentaCDD, 1,2,3,7,8-	0.0807
PentaCDF, 1,2,3,7,8-	0.0042
PentaCDF, 2,3,4,7,8-	0.0871
TetraCDD, 2,3,7,8-	0.4395
TetraCDF, 2,3,7,8-	0.0429
Source: Van den Berg et al, 2006	

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