Human Health Risk Assessment:
Swadlincote Resource and Recovery Park (SRRP)

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Experts in air quality management & assessment

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Executive Summary

An assessment has been carried out to identify potential health risks associated with emissions of dioxins, furans and dioxin-like polychlorinated biphenyls (PCBs) the proposed Energy Recovery Facility (ERF). The assessment draws on the dispersion modelling presented in Chapter 10 of the Environmental Statement prepared to support the planning application, and additionally uses the United States Environmental Protection Agency's Human Health Risk Assessment Protocol (HHRAP). A range of worst-case assumptions has been applied and thus the assessment is precautionary.

The assessment has shown that the predicted risks to health comply with relevant benchmarks. All the impacts are assessed to be insignificant. No specific mitigation measures are required beyond those incorporated into the design of the facility.

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1 Introduction

1.1 The United States Environmental Protection Agency (USEPA) Human Health Risk Assessment Protocol (HHRAP) methodology has been used to identify potential risks to health from emissions of dioxins, furans and dioxin-like polychlorinated biphenyls (PCBs) from the Energy Recovery Facility (ERF) at the proposed Swadlincote Resource and Recovery Park at Swadlincote. The assessment has been carried out by Air Quality Consultants Ltd on behalf of R&P Clean Power Ltd Ltd.

1.2 Chapter 10 of the Environmental Statement predicts ambient pollutant concentrations and compares these against precautionary assessment levels. However, there are no suitable assessment levels for concentrations of dioxins, furans and dioxin-like PCBs in the air which adequately account for the potential for these pollutants to deposit and accumulate in the local environment, including bioaccumulation through the food chain. The HHRAP provides an approach for assessing these effects. This approach is current best-practice in the UK and is often recommended by the UK Environment Agency. This assessment has used the Industrial Risk Assessment Program-Human Health (IRAP) model, which is based on the HHRAP.

1.3 The HHRAP method was developed to assess risks in areas where much of the population derives a substantial portion of its diet from local produce. Because of the potential for accumulation in the food chain, animal husbandry (particularly the raising of pigs and cattle) forms a key part of HHRAP. In practice, relatively few people in the UK derive a substantial portion of their diet from animals raised within a small local area. In the context of the areas around the ERF, the HHRAP method will provide an extremely conservative (i.e. worst-case) assessment.

2 Scope

2.1 Following standard Environment Agency practice for studies of this nature, the assessment focuses on the uptake of polychlorinated dibenzo-para-dioxins and polychlorinated dibenzofurans, often abbreviated to 'dioxins and furans', as well as dioxin-like PCBs. While other substances such as metals, acid gases, and particulate matter would be emitted from the ERF, these can be adequately assessed by comparing the predicted ambient concentrations against relevant assessment criteria. A detailed assessment of these other pollutants is presented in Chapter 10 of the Environmental Statement.

2.2 Exposure to emissions of dioxins, furans and dioxin-like PCBs can be through a number of pathways, with inhalation and the food chain being most critical. This assessment covers exposure through the direct inhalation of dioxins, furans and dioxin-like PCBs as gases and fine airborne particles, as well as indirect exposure following the deposition of contaminants to land and subsequent transfer by biogeochemical processes through soils and vegetation into the food chain.

2.3 The assessment has not considered the potential for significant impacts associated with dermal contact with soil or water. Other studies have shown that absorption through dermal contact with soil is significantly less efficient than absorption by inhalation and can thus safely be discounted (Pasternach, 1989). Dermal contact with standing water in the area will also be sporadic and unlikely to lead to significant exposure.

3 Assessment Approach

Baseline Conditions

3.1 Background exposure to dioxins, furans and dioxin-like PCBs is dependent on a wide range of complex individual factors and will vary from one individual to another. Key issues include individual lifestyle, diet, baseline land quality, and background levels of dioxins, furans and dioxin-like PCBs. This study has thus focused on the potential impact of the ERF, rather than on the combined effect of the ERF and other potential sources of exposure. If it is possible to demonstrate that the impacts of the ERF will be insignificant, then it is not necessary to calculate the total exposure.

Emissions of Dioxins, Furans and Dioxin-like PCBs from the ERF

3.2 The general terms 'dioxins, furans and dioxin-like PCBs' describe a large number of compounds, which are individually known as 'congeners'. Each congener has a different toxicity and physical properties with regard to atmospheric behaviour.

3.3 As explained in Chapter 10 of the Environmental Statement prepared to support the planning application, the Industrial Emissions Directive (IED) (2010/75/EU, 2010) allows a maximum emission rate for total dioxins and furans of 0.1 ng/Nm^3 (averaged over a sampling period of between 6 and 8 hours). The IED also provides a range of congener-specific international toxic equivalence (I-TEQ) factors (TEFs) by which the mass concentrations of specific congeners should be multiplied before summing. Other TEF factors are provided by the WHO and are referred to as WHO-TEQs. In other words, 0.1 ng/Nm^3 refers not to the total mass emission, but to the toxic-equivalent emission. In practice, the ERF is unlikely to emit anywhere near 0.1 ng/Nm^3, but it has nevertheless been assumed that 0.1 ng I-TEQ/Nm^3 would be emitted during every hour of every year. This provides a worst-case assessment.

3.4 The emission rate for dioxin-like PCBs has been determined using the Best Available Technique Associated Emission Levels (BAT-AELs) set out in the Best Available Techniques Reference Document (BREF) for Waste Incineration (European Commission, 2019). The BREF provides a BAT-AEL for dioxins of 0.04 ng TEQ/Nm^3 and a BAT-AEL for dioxins plus dioxin-like PCBs of 0.06 ng TEQ/Nm^3. An emission concentration of 0.02 ng TEQ/Nm^3 for dioxin-like PCBs has thus been used.

3.5 Her Majesty's Inspectorate of Pollution (HMIP) previously published a dioxin and furan congener profile based on measurements at municipal waste incinerators (HMIP, 1996). This provides the most robust dataset to describe the ERF at this time and has thus been used to define the relative quantities of each congener that would be emitted.

3.6 The congener profile from HMIP has been combined with the IED toxic equivalence factor and the volumetric emission rate from the ERF to calculate g/s mass emissions of the individual congeners. These calculations are shown in Table 1.

Table 1: Assumed Dioxin and Furan Emissions from the ERF

Congener	Sum of Emissions (ng-TEQ/Nm^3)	% of Total Toxic-Equivalent Conc from Each Congener	Toxic Eq Factor (ng TEQ / ng)	Mass Emission Conc (ng/Nm^3) [a]	Mass Emission Rate (g/s) [b]
2,3,7,8-TCDD	-	3.1%	1	0.0031	1.49×10^{-10}
1,2,3,7,8-PeCDD	-	12.4%	0.5	0.025	1.20×10^{-9}
1,2,3,4,7,8-HxCDD	-	2.9%	0.1	0.029	1.39×10^{-9}
1,2,3,7,8,9-HxCDD	-	2.1%	0.1	0.021	1.01×10^{-9}
1,2,3,6,7,8-HxCDD	-	2.6%	0.1	0.026	1.25×10^{-9}
1,2,3,4,6,7,8-HpCDD	-	1.7%	0.01	0.17	8.17×10^{-9}
OCDD	-	0.4%	0.001	0.4	1.92×10^{-8}
2,3,7,8-TCDF	-	2.7%	0.1	0.027	1.30×10^{-9}
2,3,4,7,8-PeCDF	-	26.8%	0.5	0.054	2.60×10^{-9}
1,2,3,7,8-PeCDF	-	1.4%	0.05	0.028	1.35×10^{-9}
1,2,3,4,7,8-HxCDF	-	21.8%	0.1	0.22	1.06×10^{-8}
1,2,3,7,8,9-HxCDF	-	0.4%	0.1	0.0042	2.02×10^{-10}
1,2,3,6,7,8-HxCDF	-	8.0%	0.1	0.081	3.89×10^{-9}
2,3,4,6,7,8-HxCDF	-	8.6%	0.1	0.087	4.18×10^{-9}
1,2,3,4,6,7,8-HpCDF	-	4.4%	0.01	0.44	2.11×10^{-8}
1,2,3,4,7,8,9-HpCDF	-	0.4%	0.01	0.043	2.07×10^{-9}
OCDF	-	0.4%	0.001	0.36	1.73×10^{-8}
Sum	**0.1**	**100%**	**-**	**2.02**	**9.70×10^{-8}**

[a] i.e. 0.1 ng TEQ/Nm^3 divided by the toxic equivalence factor and multiplied by the percentage of total toxic-equivalent concentration from each congener. Calculated from unrounded numbers.

[b] i.e. mass emission concentration (ng/Nm^3) divided by 1 billion, multiplied by the volume flow rate (173,000 Nm^3/hr – see Chapter 10 of the Environmental Statement) divided by 3,600.

3.7 As the fate, transport and bioaccumulation properties are not known for all PCB congeners, the USEPA approach (also commonly applied in the UK) is to use a surrogate substance to represent a combination of PCBs. Aroclor 1254 is representative of higher chlorinated (i.e. greater toxicity) PCBs and has therefore been used to provide a robust assessment. There is no TEQ factor available for

aroclor 1254, therefore a TEQ factor of 0.1 ng TEQ/ng has been used as a worst-case, as this is the highest TEQ factor defined for any PCB. This TEQ factor has been combined with the volumetric emission rate from the ERF to calculate a mass emissions rate for aroclor 1254 of 9.61 x 10^{-9} g/s.

Dispersion Modelling

3.8 The transport of emissions though air has been modelled using the ADMS-5 dispersion model (v5.2). The model, and the input parameters used for this assessment, are described in Chapter 10 of the Environmental Statement prepared to support the planning application.

3.9 Dry and wet deposition of particle-bound and vapour-phase congeners has been simulated within the ADMS-5 model. In terms of the dry deposition of particles, because large particles will be filtered out from the stack emissions, particulate emissions are expected to be predominantly in the 1-2 μm diameter range. For particles of this size, deposition velocities are likely to be in the range of 0.001 m/s to 0.01 m/s. For dry deposition of particle-bound congeners, a fixed worst-case deposition velocity of 0.01 m/s has thus been assumed. The dry deposition of vapour-phase congeners has been modelled using the ADMS-5 routine for non-reactive gases.

3.10 The ADMS model has been run using five separate yearly meteorological datasets (2015 to 2019 inclusive) from the Sutton Bonnington meteorological monitoring site. These are the data used in Chapter 10 of the Environmental Statement and are described therein. For each receptor, the maximum predicted concentration, dry deposition flux, and wet deposition flux during any year was taken and used for the assessment.

Receptors for Dispersion Modelling

3.11 Modelling has been carried out across a 10 km x 10 km nested Cartesian grid of receptors, centred on the proposed ERF. The gridded receptors have been modelled at a height of 1.5 m, to represent ground-level human exposure.

Human Health Risk Assessment

3.12 The assessment has used the IRAP model, which is based on HHRAP.

Receptors for IRAP Model

3.13 Within the IRAP model, there are six receptor types of relevance to this assessment:

- resident adult;
- resident child;
- farmer adult;
- farmer child;

- fisher adult; and
- fisher child.

3.14 Resident receptors are assumed to have the potential to intake pollutants via inhalation, by eating above-ground home-grown vegetables and by eating soil[1].

3.15 Farmer receptors are assumed to intake pollutants by these same pathways, but also from eating home-reared beef, chicken and pork, drinking milk from cows kept at home, and eating home-produced eggs. It is important to recognise that, when a receptor is included as a farmer, the assumption is made that the location is an active farm at which only beef, pork, poultry, eggs, milk and vegetables produced at that farm are consumed. It is unlikely that there are any such locations in the vicinity of the ERF.

3.16 Fisher receptors are assumed to intake pollutants by the same pathways as resident receptors, but also from eating locally caught fish. When a receptor is included as a fisher, the assumption is made that fish is the main component of the receptor diet. It is unlikely that there are any such locations within the study area.

3.17 Occasionally, residents keep animals in domestic gardens. Where this occurs, it is highly unlikely that local bioaccumulation would affect the dominant source of consumed food. While it is possible that, for example, chickens kept at home could provide the only source of eggs consumed, it remains unlikely that a dominant proportion of the chickens' food would be grown within the immediate area.

3.18 The results from the ADMS-5 dispersion model runs for the Cartesian grid of receptors have been plotted on a map and used to select the worst-case receptors for the human health risk assessment (i.e. the human health risk assessment receptors were chosen to represent locations where the gridded concentrations and deposition fluxes were highest). The receptors used for the human health risk assessment are described in Table 2 and shown in Figure 1.

[1] This is usually accidental and associated with home-grown vegetables.

Table 2: Receptors Used in this Assessment

Receptor	Description	Type
A	Farmland south west of the development	Farmer
B	Farmland north of the development	Farmer
C	Residential property south east of the development	Residential
D	Residential property north of the development.	Residential
E	Residential property north of the development	Residential
F	Residential property north east of the development	Residential
G	Residential property south west of the development	Residential
H	Foremark Reservoir / Fishery north east of the development	Fisher
I	Cadwell Pool Fish Pond south west of the development	Fisher

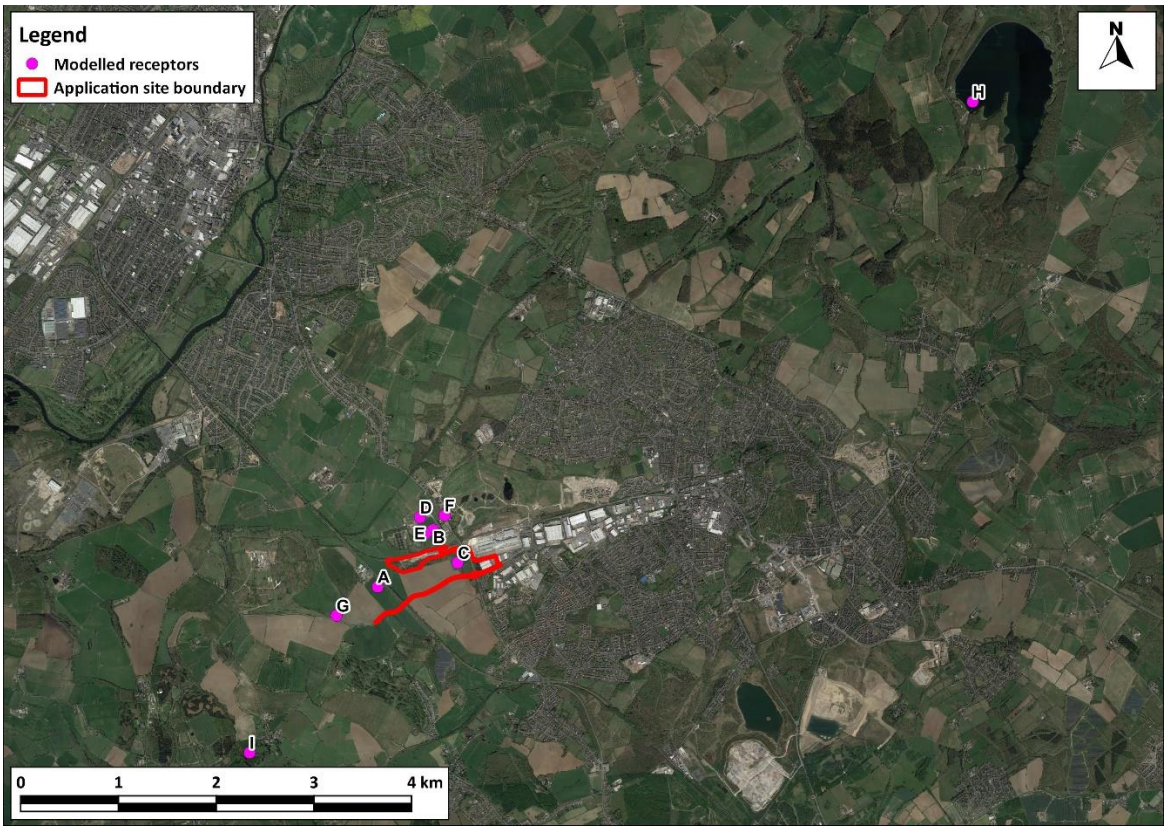

Figure 1: Receptors Used in this Assessment

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IRAP Model Inputs

3.19 The outputs from the ADMS-5 dispersion model have been used as inputs to the IRAP model. From these, IRAP calculated the concentrations of the various congeners in the environmental media, foodstuffs, and the human population.

3.20 The IRAP model requires a wide range of input data to be defined as set out below. In many cases the IRAP model default parameters have been used, as is common practice for such assessments. These parameters are extensive and detailed and set out in the documentation provided by the model developers[2].

Physical and Chemical Properties of the Pollutants

3.21 These parameters determine how the pollutants behave in the environment and their presence and accumulation in various food sources. The HHRAP and IRAP default values have been used in this assessment.

Site-specific Information

3.22 The IRAP model requires information about the location and its surroundings including:

- the fraction of animal feed grown on local soils and the amount of animal feed and soil consumed by various species;
- the interception fraction for above ground vegetation, forage and silage and lengths of vegetation exposure to deposition;
- data for assessing the risks with exposure to breast milk, such as infant bodyweight, fraction of mother's body weight that is fat etc.; and
- other physical parameters such as soil and air density etc.

The HHRAP and IRAP default parameters have been used for all of this information.

3.23 Other parameters need to be taken from site-specific data. These are set out in Table 3.

[2] https://www.weblakes.com/software/risk-assessment/irap-h-view/protocol/

Table 3: Site-specific Parameters Used

Parameter	Value
Annual Mean Precipitation	25.7 cm/yr [a]
Annual Mean Wind Velocity	1.30 m/s [b]
Annual Mean Evapotranspiration	18.0 cm/yr [c]
Annual Mean Irrigation	2.64 cm/yr [d]
Annual Mean Runoff	2.57 cm/yr [e]
Time period over which deposition occurs	30 yrs

[a] The average of five year's meteorological data.

[b] The average of five year's meteorological data.

[c] Assumed to be 70% of precipitation.

[d] Estimated from previous projects.

[e] Assumed to be 10% of precipitation.

Receptor Information

3.24 For each receptor type, IRAP requires information on receptor behaviour including absolute and relative food consumption rates, body weight, inhalation rates, and exposure frequency etc. The HHRAP and IRAP default values have been used in this assessment.

IRAP Model Outputs

Cancer Risk

3.25 The HHRAP approach is to calculate the probability of affected receptors developing cancer over their lifetime as a result of emissions from the ERF. This risk is presented as an absolute number which represents a probability. For example, a value of 1×10^{-6} (or 0.000001) represents a one in one million chance of an effect over a lifetime's exposure.

Hazard Risk

3.26 IRAP has also been used to calculate the 'Hazard Quotient' for each receptor. The 'Hazard Quotient' is a way of expressing the ratio of the predicted exposure level and a 'reference dose' which represents the level at which no adverse effects are expected. Hazard Quotients are pollutant and pathway specific and can then be summed to calculate a 'Hazard Index'. Of the congeners considered in this assessment, a reference dose has only been set for the most toxic congener (2,3,7,8-TCDD) via the ingestion pathway. Thus, for this assessment, the Hazard Quotient and the Hazard Index are the same. The Hazard Quotient is simply the exposure level divided by the reference dose and is thus unitless.

Oral Intake of All Congeners

3.27 While the Hazard Quotient only takes account of intake of the most toxic congener (2,3,7,8-TCDD), the total oral intake of all modelled congeners has been derived from the IRAP model and compared against defined 'Tolerable Daily Intake Levels' (TDIs). The units for this assessment are toxic-equivalent-picogrammes[3] per kg of bodyweight per day (pg-TEQ/kg/d).

Infant Exposure through Breast Milk

3.28 Dioxins, furans and dioxin-like PCBs are extremely fat soluble and hence prone to accumulation in breast milk. The accumulated compounds can then be passed to the infant. Exposure through breast milk is measured by the Average Daily Dose (ADD) on the basis of an averaging time of one year. The units for this assessment are also pg-TEQ/kg/d.

Concentrations in Soils

3.29 The final set of outputs derived from the IRAP model is the concentrations of dioxins, furans and dioxin-like PCBs in soils. These are expressed as the sum of all congeners in μg per kg (μg/kg) of soil (dry weight).

[3] One picogramme is equal to 0.000000000001 grammes.

4 Assessment Criteria

Cancer Risk

4.1 One definition of 'acceptable risk' that has been widely used in the UK, is if exposure to a substance increases a person's chance of dying in any one year by one chance in a million (1:1,000,000 or 1 x 10^{-6}) or less[4] (Hunter and Fewtrell, 2010). The one in one million figure used in the UK is an annual figure. Since risk is cumulative, assuming a 70 year lifetime, the equivalent lifetime risk is one in fourteen thousand (1:14,300 or 7 x 10^{-5}).

4.2 HHRAP uses a value of one in one hundred thousand (1:100,000 or 1 x 10^{-5}) for lifetime cancer risk. This is effectively more stringent than the 1 in 1 million annual risk figure and has thus been used for this study. The assessment criterion for cancer risk is thus 1 x 10^{-5}.

Hazard Risk

4.3 As explained in Paragraph 3.26, the Hazard Quotient is the predicted exposure level divided by the 'reference dose'. The HHRAP reference dose relevant to this study is given in Table 4. If the Hazard Quotient is less than 1 (i.e. the predicted exposure is less than the reference dose), then no adverse health effects are expected. If the Hazard Quotient is greater than 1, then adverse health effects are possible. The Hazard Quotient is different from the cancer risk estimates in that it cannot be translated to a probability that adverse health effects will occur and it is unlikely to be proportional to risk. It is important to note that a Hazard Quotient exceeding 1 does not necessarily mean that adverse effects will occur; it simply indicates the potential for an effect.

Table 4: Reference Dose and Hazard Quotient

Description	Value
Reference Dose (of 2,3,7,8-TCDD per kg body weight) (mg/kg/d)	1 x 10^{-9}
Hazard Quotient	1

Oral Intake of All Congeners

4.4 The World Health Organisation (WHO) has recommended a range of TDIs (Table 5). These represent a lifetime's exposure; short-term exceedances of the TDI are not of concern if the long-term average is not exceeded (WHO, 1998).

4.5 In the UK, the Health Protection Agency and the Department of Health are advised by the Committee on the Toxicity of Chemicals in Food, Consumer Products and the Environment (COT). The COT has also provided recommendations on TDIs, which are based on adverse effects on a developing

[4] By way of comparison, the likelihood of dying in a road traffic accident is approximately 1 in 17,500 per year http://www.medicine.ox.ac.uk/bandolier/booth/Risk/trasnsportpop.html

foetus (since this represents the most sensitive exposure pathway) (COT, 2001). The TDIs used in this study are set out in Table 5.

Table 5: Tolerable Daily Intake (TDI) Values

Reference Source	TDI (pg-TEQ/kg/d)
(WHO, 1998)	1 – 4
(COT, 2001)	2

Infant Exposure through Breast Milk

4.6 There is no official UK or USEPA assessment criterion for acceptable infant exposure. One approach that is often taken is to compare the ADD against average background exposure levels, while other studies have cited a threshold value of 50 pg-TEQ/kg/d of the congener 2,3,7,8-TCDD as being potentially harmful (Gair Consulting Ltd, 2020).

4.7 Two separate approaches have been taken here. The first is to compare the ADD for all congeners against an assumed nominal baseline dose of 100 pg-TEQ/kg/day [5]. The second is to compare the ADD for the congener 2,3,7,8-TCDD against the threshold value of 50 pg-TEQ/kg/day.

Concentrations in Soils

4.8 The Environment Agency has developed Soil Guideline Values (SGVs) for dioxins, furans, and dioxin-like PCBs (Environment Agency, 2009). These are assessment criteria that can be used to evaluate the risk to human health from long-term exposure to chemicals in soil.

4.9 For residential and allotment land uses, the SGVs are based on estimates representative of exposure of young children because they are generally more likely to have higher exposures to soil contaminants. The toxic equivalence of the various congeners has been taking into account in the derivation of the SGVs. Thus, the mass sum of all congeners in the soil may be compared directly with the SGVs.

4.10 The SGVs used in this assessment are set out in Table 6. These are based on a generic profile of congeners which is representative of general diffuse pollution, but within the context of this assessment are considered to be sufficiently accurate.

[5] A compilation of European dioxin exposure data (DETR, 1999) found that, in 1993, the dioxin intake of firstborn infants, up to 2 months of age, might be around 106 pg-TEQ/kg/day in rural areas of the EU and 144 pg TEQ/kg/day in industrial areas. These intake levels fall rapidly after the first few months. These are well in excess of the TDIs, but, as explained in Paragraph 3.5, the TDIs refer to an average lifetime's exposure. Exposure is likely to have fallen since 1993 but these values nevertheless provide a basis for assessment.

Table 6: Soil Guideline Values (SGVs) for Dioxins, Furans, and Dioxin-like PCBs [a]

Land use	Sum of Congeners (µg/kg DW)
Residential	8
Allotment	8
Commercial	240

[a] Based on an assumed soil profile for urban soils and on a sandy loam soil.

Generic Environment Agency Screening Criteria

4.11 The Environment Agency has considered potential impacts from industrial emissions in its guidance (Environment Agency, 2016). This explains that regardless of what the baseline environmental conditions are, a process can be considered as insignificant if the long-term (annual mean) process contribution is <1% of the long-term environmental standard.

4.12 This criterion is typically applied to process contributions to airborne concentrations, but it provides helpful context to the assessment in this report. It should be recognised that this criterion determines when an impact can be screened out as insignificant. It does not imply that impacts will necessarily be significant above this level merely that above this level there is a potential for significant impacts.

Judgement on Significance

4.13 To aid interpretation and decision-making, the impacts assessed in this report have been classified as either significant or insignificant. This judgement takes account of all factors described above, as well as the degree of precaution built into the dispersion modelling. It ultimately relies on the professional experience of the consultants involved in the assessment, which has been gained over several decades and through carrying out several thousand studies in the UK and internationally which have compared changes in air quality and deposition against health-based assessment criteria. The professional experience of the consultants involved in the project is summarised in Appendix A1.

5 Results

Cancer Risk

5.1 The total lifetime cancer risks associated with emissions from the facility for each of the receptors are presented in Table 7.

5.2 The data in Table 7 show that the highest residential risk is predicted at receptor C, where the predicted lifetime risk of developing cancer as a result of emissions from the ERF is 1.4 x 10^{-8} (1 in 71 million). The highest risk at any of the receptors that have been treated as farms is at receptor A, where the risk is 6.5 x 10^{-6} (1 in 154 thousand), while for fishers it is receptor I, where the risk is 2.4 x 10^{-8} (1 in 41 million). All of the results are below the assessment criterion of 1 x 10^{-5} (1 in one hundred thousand). Such risks are conventionally considered to be acceptable in the UK.

Table 7: Total Lifetime Cancer Risk for all Receptors [a]

R	Description	Type [b]	Child	Adult
A	Farmland south west of the development	Fa	**1.4 x 10^{-6}**	**6.5 x 10^{-6}**
B	Farmland north of the development	Fa	1.1 x 10^{-6}	5.2 x 10^{-6}
C	Residential property south east of the development	R	**7.5 x 10^{-9}**	**1.4 x 10^{-8}**
D	Residential property north of the development.	R	5.5 x 10^{-9}	1.0 x 10^{-8}
E	Residential property north of the development	R	7.5 x 10^{-9}	1.4 x 10^{-8}
F	Residential property north east of the development	R	6.8 x 10^{-9}	1.3 x 10^{-8}
G	Residential property south west of the development	R	4.3 x 10^{-9}	8.1 x 10^{-9}
H	Foremark Reservoir / Fishery north east of the development	Fi	8.3 x 10^{-10}	3.8 x 10^{-9}
I	Cadwell Pool Fish Pond south west of the development	Fi	**4.1 x 10^{-9}**	**2.4 x 10^{-8}**
Assessment Criterion			**1 x 10^{-5}**	

1 x 10^{-5} = 1 in one hundred thousand chance of an effect over a lifetime's exposure

1 x 10^{-6} = 1 in one million chance of an effect over a lifetime's exposure

1 x 10^{-8} = 1 in one hundred million chance of an effect over a lifetime's exposure

1 x 10^{-10} = 1 in one billion chance of an effect over a lifetime's exposure

[a] The highest relevant values are shown in bold.

[b] R = residential, Fa = farmer, Fi = fisher.

5.3 Table 8 shows the pathways of exposure for receptors A, C and I. The most significant pathway for the residential receptors is eating home-grown vegetables, the most significant pathway for farm receptors is drinking home-produced milk, while the most significant pathway for fisher receptors is eating locally-caught fish. As explained in Paragraph 3.15, by including receptors as farms, it is

assumed that the location is an active farm at which only beef, pork, poultry, eggs, milk and vegetables produced at that farm are consumed. As there are unlikely to be any such locations in the vicinity of the proposed ERF (i.e. all local residents who consume these foodstuffs are likely to purchase at least some of their food), the assessment is worst-case. It is also assumed that, for fisher receptors, fish is the main component of the receptor diet. It is unlikely that there are any such locations in the vicinity of the proposed ERF, thus the assessment is worst-case.

Table 8: Total Lifetime Carcinogenic Risk by Pathway at Worst-case Receptors [a]

Pathway	Receptor C (Resident)		Receptor A (Farmer)		Receptor I (Fisher)	
	Child	Adult	Child	Adult	Child	Adult
Drinking home-produced milk	0	0	**1.2×10^{-6}**	**5.0×10^{-6}**	0	0
Eating home-reared beef	0	0	1.3×10^{-7}	1.4×10^{-6}	0	0
Eating above ground vegetables	**6.1×10^{-9}**	**1.3×10^{-8}**	1.2×10^{-8}	3.3×10^{-8}	7.6×10^{-10}	1.6×10^{-9}
Eating home-reared pork	0	0	6.6×10^{-9}	5.9×10^{-8}	0	0
Eating soil	1.3×10^{-9}	7.1×10^{-10}	1.8×10^{-9}	1.5×10^{-9}	1.9×10^{-10}	1.0×10^{-10}
Eating home-reared chicken	0	0	2.7×10^{-11}	3.0×10^{-10}	0	0
Eating home-produced eggs	0	0	1.9×10^{-11}	1.9×10^{-10}	0	0
Inhalation	1.1×10^{-10}	5.4×10^{-10}	1.4×10^{-10}	9.5×10^{-10}	1.2×10^{-11}	6.2×10^{-11}
Eating locally-caught fish	0	0	0	0	**3.1×10^{-9}**	**2.2×10^{-8}**
Drinking water	1.5×10^{-12}	3.3×10^{-12}	1.5×10^{-12}	4.4×10^{-12}	1.1×10^{-11}	2.4×10^{-11}
Total Lifetime Risk	7.5×10^{-9}	1.4×10^{-8}	1.4×10^{-6}	6.5×10^{-6}	4.1×10^{-9}	2.4×10^{-8}

1×10^{-5} = 1 in one hundred thousand chance of an effect over a lifetime's exposure

1×10^{-6} = 1 in one million chance of an effect over a lifetime's exposure

1×10^{-8} = 1 in one hundred million chance of an effect over a lifetime's exposure

1×10^{-10} = 1 in one billion chance of an effect over a lifetime's exposure

[a] Highest relevant values in each column are shown in bold.

Hazard Risk

5.4 The Hazard Quotient for each of the receptors is set out in Table 9. All of the values are well below 1 and the risk of significant health effects is thus discounted. The Hazard Risk pathway breakdown for receptors A, C and I is given in Table 10. Again, this shows that eating home-grown vegetables is the dominant risk pathway for residents, while for farmers it is drinking home-produced milk and for fishers it is eating locally-caught fish.

Table 9: Hazard Quotient for All Receptors [a]

R	Description	Type [b]	Child	Adult
A	Farmland south west of the development	Fa	**6.6×10^{-3}**	**4.6×10^{-3}**
B	Farmland north of the development	Fa	5.3×10^{-3}	3.7×10^{-3}
C	Residential property south east of the development	R	**2.9×10^{-5}**	**1.1×10^{-5}**
D	Residential property north of the development.	R	2.1×10^{-5}	7.6×10^{-6}
E	Residential property north of the development	R	2.8×10^{-5}	1.0×10^{-5}
F	Residential property north east of the development	R	2.6×10^{-5}	9.7×10^{-6}
G	Residential property south west of the development	R	1.6×10^{-5}	6.1×10^{-6}
H	Foremark Reservoir / Fishery north east of the development	Fi	3.6×10^{-6}	3.6×10^{-6}
I	Cadwell Pool Fish Pond south west of the development	Fi	**1.8×10^{-5}**	**2.2×10^{-5}**
Assessment Criterion			**1**	

1×10^{-3} = 0.001

1×10^{-6} = 0.000001

[a] Highest relevant values are shown in bold.

[b] R = residential, F = farm, Fi = fisher.

Table 10: Hazard Quotient by Pathway at Worst-case Receptors [a]

Pathway	Receptor C (Resident)		Receptor A (Farmer)		Receptor I (Fisher)	
	Child	Adult	Child	Adult	Child	Adult
Drinking home-produced milk	0	0	**5.9×10^{-7}**	**3.6×10^{-3}**	0	0
Eating home-reared beef	0	0	6.1×10^{-4}	1.0×10^{-3}	0	0
Eating home-reared pork	0	0	3.1×10^{-5}	4.1×10^{-5}	0	0
Eating above ground vegetables	**2.4×10^{-5}**	**1.0×10^{-5}**	4.5×10^{-5}	1.9×10^{-5}	2.9×10^{-6}	1.2×10^{-6}
Eating soil	4.8×10^{-6}	5.1×10^{-7}	6.6×10^{-6}	7.1×10^{-7}	6.8×10^{-7}	7.3×10^{-8}
Eating home-reared chicken	0	0	9.9×10^{-8}	1.5×10^{-7}	0	0
Eating home-produced eggs	0	0	6.8×10^{-8}	9.4×10^{-8}	0	0
Inhalation	5.9×10^{-8}	5.9×10^{-8}	7.8×10^{-8}	7.8×10^{-8}	6.8×10^{-9}	6.8×10^{-9}
Eating locally-caught fish	0	0	0	0	**1.4×10^{-5}**	**2.1×10^{-5}**
Drinking water	4.3×10^{-9}	1.9×10^{-9}	4.3×10^{-9}	1.9×10^{-9}	2.9×10^{-8}	1.3×10^{-8}

1×10^{-3} = 0.001

1×10^{-6} = 0.000001

1×10^{-9} = 0.000000001

[a] Highest relevant values in each column shown in bold.

Oral Intake of all Congeners

5.5 Table 11 sets out the predicted oral intake of all congeners for receptors A, C and I. The results are separately compared with the TDIs from both WHO and COT. The predicted intakes at receptors C and I are well below 1% of the TDIs. The predicted intakes at receptor A exceed 1% of both TDIs, predominantly as a result of drinking home-produced milk. It should be recognised that exceedance of the assessment criterion does not necessarily mean that impacts will be significant, merely that there is a potential for significant impacts and that further consideration is required, as set out below.

5.6 The impacts at receptor A, as shown in Table 11, are up to 9.4% of the lower-bound WHO assessment criterion and up to 4.7% of the COT assessment criterion. While it is not possible to immediately discount these changes as insignificant, they remain small. It must also be recognised that this assessment is very much worst-case. The figures in Table 11 are based on the assumption that the proposed ERF will emit continuously at the maximum emission rates allowed under the IED. Furthermore, when a receptor is included as a farmer, the assumption is made that the location is an active farm at which only beef, pork, poultry, eggs, milk and vegetables produced at that farm are consumed. It is highly unlikely that receptor A meets these conditions. Taking these factors into

account, it is considered that the predicted oral intake presented in Table 11 represents an extreme worst case. The actual impacts are expected to be much lower and are therefore judged to be insignificant.

Table 11: Predicted Oral Intake of Dioxins, Furans and Dioxin-like PCBs at Worst-case Receptors (pg-TEQ/kg/d)

Pathway	Receptor C (Resident)		Receptor A (Farmer)		Receptor I (Fisher)	
	Child	Adult	Child	Adult	Child	Adult
Drinking home-produced milk	0	0	8.4×10^{-2}	5.1×10^{-2}	0	0
Eating home-reared beef	0	0	8.8×10^{-3}	1.4×-10^{-2}	0	0
Eating home-reared pork	0	0	4.9×10^{-4}	6.4×10^{-4}	0	0
Eating above ground vegetables	5.0×10^{-4}	2.1×10^{-4}	9.3×10^{-4}	3.9×10^{-4}	6.4×10^{-5}	2.7×10^{-5}
Eating soil	1.7×10^{-4}	1.8×10^{-5}	2.4×10^{-4}	2.5×10^{-5}	2.4×10^{-5}	2.6×10^{-6}
Eating home-reared chicken	0	0	3.4×10^{-6}	5.0×10^{-6}	0	0
Eating home-produced eggs	0	0	2.3×10^{-6}	3.2×10^{-6}	0	0
Eating locally-caught fish	0	0	0	0	2.2×10^{-4}	3.1×10^{-4}
Drinking water	1.0×10^{-7}	4.6×10^{-8}	1.0×10^{-7}	4.6×10^{-8}	7.6×10^{-7}	3.4×10^{-7}
Total Intake	0.0007	0.0002	0.094	0.066	0.0003	0.0003
WHO Assessment Criteria (TDI)	**1-4**					
COT Assessment Criteria (TDI)	**2**					
Intake as % of 1 pg-TEQ/kg/d	0.07	0.02	9.4	6.6	0.03	0.03
Intake as % of 2 pg-TEQ/kg/d	0.03	0.01	4.7	3.3	0.02	0.02

1×10^{-3} = 0.001

1×10^{-6} = 0.000001

1 x 10-9 = 0.000000001

Infant Exposure through Breast Milk

5.7 Table 12 sets out the estimated ADDs for infant exposure through breast milk for receptors A, C and I. The predicted ADDs have been compared with the assessment criteria described in Paragraph 4.7. All of the ADDs are less than 1% of the respective criteria and the impacts can thus be discounted as insignificant.

Table 12: Estimated Infant Average Daily Dose (ADD) of Dioxins, furans and Dioxin-like PCBs (pg-TEQ'kg/d)

Pathway	Receptor C (Resident)	Receptor A (Farmer)	Receptor I (Fisher)
All Congeners			
ADD (pg TEQ/kg/day)	0.005	0.99	0.005
Assessment Criterion (pg TEQ/kg/day)	**100**		
ADD as percentage of Criterion	0.005	0.99	0.005
2,3,7,8-TCDD			
ADD (pg TEQ/kg/day)	0.0005	0.14	0.0007
Assessment Criterion (pg TEQ/kg/day)	**50**		
ADD as percentage of Criterion	0.001	0.28	0.001

Concentrations in Soils

5.8 Table 13 sets out the maximum process contributions to dioxin and furan concentrations in soils at receptors A, C and I. The predicted concentration has been compared with the residential SGV defined in Table 6. The predicted concentrations are well below 1% of the SGV. The impacts can thus be discounted as insignificant.

Table 13: Maximum Process Contribution to Dioxin and Furan Concentrations in Soils

Parameter	Receptor C (Resident)	Receptor A (Farmer)	Receptor I (Fisher)
Process Contribution to Maximum Soil Concentration (µg/kg)	0.0003	0.0004	0.00004
Soil Guideline Value (SGV) (µg/kg)	**8**		
Process Contribution as % of Guideline Value	0.004	0.005	0.0005

6 Conclusions

6.1 This assessment has shown that:

- **cancer risk** - all of the predicted process contributions are less than the assessment criterion. They are at levels which are conventionally considered to be acceptable and are thus discounted as **insignificant**;
- **hazard risk -** all of the predicted process contributions are less than 1 and the risk of effects can be discounted as **insignificant**;
- **oral intake of all congeners -** all of the predicted process contributions at relevant locations are extremely small and are judged to be **insignificant**;
- **infant exposure through breast milk** -all of the predicted process contributions are less than 1% of the relevant assessment criteria and are judged to be **insignificant**; and
- **concentrations in soils** - all of the predicted process contributions are less than 1% of the relevant assessment criterion and are judged to be **insignificant**.

6.2 These conclusions are on the basis that the ERF will emit dioxins, furans and dioxin-like PCBs constantly at the maximum level permitted by the European Directive (2010/75/EU, 2010). In practice, emissions are expected to be a small fraction of those assessed; meaning that the impacts will have been grossly over-stated.

6.3 This assessment has found no requirement for specific mitigation measures beyond those incorporated into the design of the facility.

7 References

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8 Glossary

2,3,7,8-TCDD	A highly toxic congener
ADMS	Atmospheric Dispersion Modelling System
Congener	An individual dioxin or furan compound
COT	Committee on the Toxicity of Chemicals in Food, Consumer Products and the Environment
Dioxins	polychlorinated dibenzo-para-dioxins
ES	Environmental Statement
Furans	polychlorinated dibenzofurans
Hazard Quotient	The ratio of the predicted exposure level and a 'reference dose' which represents the level at which no adverse effects are expected
HHRAP	Human Health Risk Assessment Protocol
HMIP	Her Majesty's Inspectorate of Pollution
IED	Industrial Emissions Directive
IRAP	Industrial Risk Assessment Program-Human Health
PCB	Polychlorinated Biphenyl
SGV	Soil Guideline Value
TDI	Tolerable Daily Intake
TEQ	International Toxic Equivalence units
UK	United Kingdom
USEPA	United States Environmental Protection Agency
WHO	World Health Organisation

Units of Mass

kg	Kilogramme = 1,000 grammes
g	Gramme
µg	Microgramme = 0.000001 grammes
ng	Nanogramme = 0.000000001 grammes
pg	Picogramme = 0.000000000001 grammes

Other units

m^3	Cubic metre
Nm^3	Normalised cubic metre
s	Second
µm	Micrometer= 0.000001 metres

9 Appendices

A1 Professional Experience

Adam Clegg, BSc (Hons) MIEnvSc MIAQM

Mr Clegg is a Technical Director with AQC, with over 17 years' experience, specialising in industrial emissions. He is a member of the Institute of Air Quality Management, has previously contributed his time to, and authored publications on behalf of, the Energy Institute's Emissions Working Group, and has acted as peer reviewer for the Journal of Air & Waste Management. His expertise includes ambient and stack emissions monitoring, emission inventory development and reporting, atmospheric dispersion modelling, abatement of air emissions, environmental permitting, Best Available Technique (BAT) assessments, cost-benefit analysis and compliance assessment. He has extensive experience in the quantification and assessment of emissions from a variety of releases, covering point source emissions, flare emissions, fugitive emissions and emissions from mobile transport sources, including marine vessels, on-road and off-road vehicles and rail locomotives. He has detailed knowledge of the technologies and techniques to reduce concentrations of combustion and non-combustion related pollutants, including oxides of nitrogen, acid gases (e.g., SO_2, HF, HCl), volatile organic compounds (VOCs), particulates, heavy metals and odour.

Dr Imogen Heard, BSc (Hons) MSc PhD MInstPhys

Dr Heard is an Associate of AQC with over ten years' experience in the field of air quality. She has been involved in numerous development projects including road schemes, energy from waste facilities, urban extensions and energy centres. These have included the use of ADMS-5 and ADMS-Roads dispersion models to study the impacts of a variety of pollutants, including nitrogen dioxide, PM_{10} and $PM_{2.5}$, and the preparation of air quality assessment reports and air quality chapters for Environmental Statements. Dr Heard has prepared HHRAs in support of planning permissions and permit applications for a large number of facilities across the UK. By way of example, she led the HHRA assessments submitted as part of the planning applications for the Kingmoor Energy Facility in Carlisle and the Avondale Energy from Waste facility in Falkirk, both of which have since been granted planning permission. Prior to joining AQC she worked as a scientist in the Atmospheric Dispersion and Air Quality area at the UK Met Office for four years, modelling the dispersion of a range of pollutants over varying spatial and temporal scales.