

**Proposed Wood Gasification Facility  
Woodham Road, Barry**

**Human Health Risk Assessment**



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## 1 INTRODUCTION

### Background

1.1 Entran Ltd has been commissioned on behalf of Power Consulting Midlands Ltd to undertake an assessment to consider the effects on human exposure from emissions to air from a proposed wood gasification facility located adjacent to No 2 Dock, in the Port of Barry, Vale of Glamorgan, South Wales. The assessment supplements the air quality assessment provided for the proposed facility. The assessment only considers emissions to air as human exposure to any harmful pollutants discharged directly to the aquatic environment and from solid waste disposal is considered to be negligible.

1.2 The proposed facility site is located to the west and north of No 2 Dock and is located within an industrial setting. The nearest residential receptors are located along Dock View Road approximately 250 m to the northwest of the site boundary. The populated area of Barry is located to the northwest, north and northeast of the site. Therefore, the immediate surroundings is characterised by commercial/industrial with residential beyond. Rural areas where farming activities would be carried out are limited, the nearest area being around 2 km to the east. The location of the facility is presented in Figure 1.1.

1.3 The proposed plant would consist of a gas boiler utilising synthetic gas (Syngas) generated from the gasification of waste wood. The high-pressure steam generated by the boiler would be directed to a steam turbine and used to generate electricity for supply to the National Grid. The facility is designed to operate 24 hours a day, 365 days per year. Emissions to air would be via a single 43m stack and will be governed by the Industrial Emissions Directive <sup>1</sup> (IED).

1.4 The air quality assessment for the facility<sup>2</sup> provides a comparison of predicted concentrations for pollutant emissions at off-site locations with background air quality and air quality standards and guidelines for the protection of human health. The air quality assessment assumes the theoretical position that the maximum permissible emission limit values (ELVs) stipulated for compliant thermal treatment of waste plants are emitted during all times of operation. This position is considered unlikely to be a realistic operating scenario. Therefore, for this human health risk assessment (HHRA), emissions are based on typical emissions associated with this type of facility.

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<sup>1</sup> The Industrial Emissions Directive (2010/75/EU)

<sup>2</sup> Air Quality Assessment, Energy from Waste Facility, Marfleet, Hull, Entran Ltd Report (April 2016)

1.5 Given the above operating scenario, the emissions from the proposed gas boiler associated with the facility would contain a number of substances that cannot be evaluated in terms of their effects on human health simply by reference to ambient air quality standards. Health effects could occur through exposure routes other than purely inhalation. As such, an assessment needs to be made of the overall human *exposure* to the substances by the local population and then the *risk* that this exposure causes.

**Figure 1.1: Location of the Proposed Facility**



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### **Purpose of the Assessment**

This assessment has been undertaken principally to support the Planning Application and Environmental Permit application for the proposed facility and has been prepared in accordance with our understanding of the requirements of the Environment Agency for these types of development. This is a human health risk assessment of dioxin/furan emissions from the facility based on the US EPA HHRAP methodology. Human exposure to dioxins and furans has been compared against the Committee of Toxicity (COT) Tolerable Daily Intake (TDI) of 2 pg/kg per day. An assessment of exposure to dioxin-like PCBs has also been included.



1.6 It should be noted that the former HMIP method does not have the capability to consider dioxin-like PCBs and the US EPA HHRAP method is limited in this respect. The HHRAP method does not contain physical properties or exposure parameters for individual dioxin-like PCBs but does provide information for two dioxin-like PCB mixtures (Aroclor 1016 and Aroclor 1254). Therefore, for these two substances typical emissions for dioxin-like PCBs have been included in the IRAP model and these have been assumed to comprise entirely of Aroclor 1016 or Aroclor 1254 depending on which substance gives rise to the highest exposure.

### **Scope of the Assessment**

1.7 The emissions from the proposed facility during the modelled operational scenario would contain a number of substances that cannot be evaluated in terms of their effects on human health simply by reference to ambient air quality standards. Health effects could occur through exposure routes other than purely inhalation. As such, an assessment needs to be made of the overall human *exposure* to the substances by the local population and then the *risk* that this exposure causes.

1.8 The assessment presented here considers the potential impact of substances released by the facility on the health of the local population at the point of maximum exposure. These substances are those that are 'persistent' in the environment and have several pathways from the point of release to the human receptor. Essentially they can be described as dioxins/furans and dioxin-like polychlorinated biphenyls (PCBs) and are present in extremely small quantities and are typically measured in mass units of nanograms ( $\text{ng} = 10^{-9} \text{ g}$ ), picograms ( $\text{pg} = 10^{-12} \text{ g}$ ) and femtograms ( $\text{fg} = 10^{-15} \text{ g}$ ).

1.9 Unlike substances such as nitrogen dioxide, which have short term, acute effects on the respiratory system, dioxins/furans and dioxin-like PCBs have the potential to cause effects through long term, cumulative exposure. A lifetime is the conventional period over which such effects are evaluated. A lifetime is taken to be 70 years.

1.10 The exposure scenarios used here represent highly unrealistic situations in which all exposure assumptions are chosen to represent a worst case and should be treated as an extreme view of the risks to health. While individual high-end exposure estimates may represent actual exposure possibilities (albeit at very low frequency), the possibility of all high end exposure assumptions accumulating in one individual is, for practical purposes, never realised. Therefore, intakes presented here should be regarded as an extreme upper estimate of the actual exposure that would be experienced by the real population in the locality.



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## Approach to the Assessment

1.11 The risk assessment process is based on the application of the US EPA Human Health Risk Assessment Protocol (HHRAP)<sup>3</sup>. This protocol has been assembled into a commercially available model, Industrial Risk Assessment Program (IRAP, Version 4.5.6) and marketed by Lakes Environmental of Ontario.

1.12 The approach seeks to quantify the *hazard* faced by the receptor, the *exposure* of the receptor to the substances identified as being a potential hazard and then to assess the *risk* of the exposure, as follows:

1.13 *Quantification of the exposure*: an exposure evaluation determines the dose and intake of key indicator chemicals for an exposed person. The dose is defined as the amount of a substance contacting body boundaries (in the case of inhalation, the lungs) and intake is the amount of the substance absorbed into the body. The evaluation is based upon worst-case, conservative scenarios, with respect to the following:

- location of the exposed individual and duration of exposure;
- exposure rate;
- emission rate from the source.

1.14 *Risk characterisation*: following the above steps, the risk is characterised by examining the toxicity of the chemicals to which the individual has been exposed, and evaluating the significance of the calculated dose in the context of probabilistic risk. The risk is further characterised by a comparison of intakes with the tolerable daily intake (TDI) for dioxins/furans and dioxin-like PCBs.

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





## 2 METHODOLOGY FOR ESTIMATING EXPOSURE TO EMISSIONS

### Introduction

2.1 An exposure assessment for the purposes of characterising the health impact of the proposed facility emissions requires the following steps:

- (1) Measurement or estimation of emissions from the source.
- (2) Modelling the fate and transport of the emitted substances through the atmosphere and through soil, water and biota following deposition onto land. Concentrations of the emitted chemicals in the environmental media are estimated at the point of exposure, which may be through inhalation or ingestion.
- (3) *Calculation of the uptake of the emitted chemicals into humans coming into contact with the affected media and the subsequent distribution in the body.*

2.2 With regard to Step (3), the exposure assessment considers the uptake of polychlorinated dibenzo-para-dioxins and polychlorinated dibenzofurans (PCDD/Fs, often abbreviated to 'dioxins/furans') and dioxin-like PCBs by various categories of human receptors.

### Potential Exposure Pathways

2.3 There are two primary exposure 'routes' where humans may come into contact with chemicals that may be of concern:

- direct, via inhalation; or
- indirect, via ingestion of water, soil, vegetation and animals and animal products that become contaminated through the food chain.

2.4 There are four other potential exposure pathways of concern following the introduction of substances into the atmosphere:

- ingestion of drinking water;
- dermal (skin) contact with soil;
- incidental ingestion of soil; and
- dermal (skin) contact with water.





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## Exposure Pathways Considered in the Assessment

2.5 The possible exposure pathways included in the IRAP model are shown in Figure 2.1. Dermal contact with soil is an insignificant exposure pathway on the basis of the infrequent and sporadic nature of the events and the very low dermal absorption factors for this exposure route, coupled with the low plausible total dose that may be experienced (when considered over the lifetime of an individual). Health risk assessments of similar emissions (Pasternach (1989) *The Risk Assessment of Environmental and Human Health Hazards*, John Wiley, New York) have concluded that dermal absorption of soil is at least one order of magnitude less efficient than lung absorption.

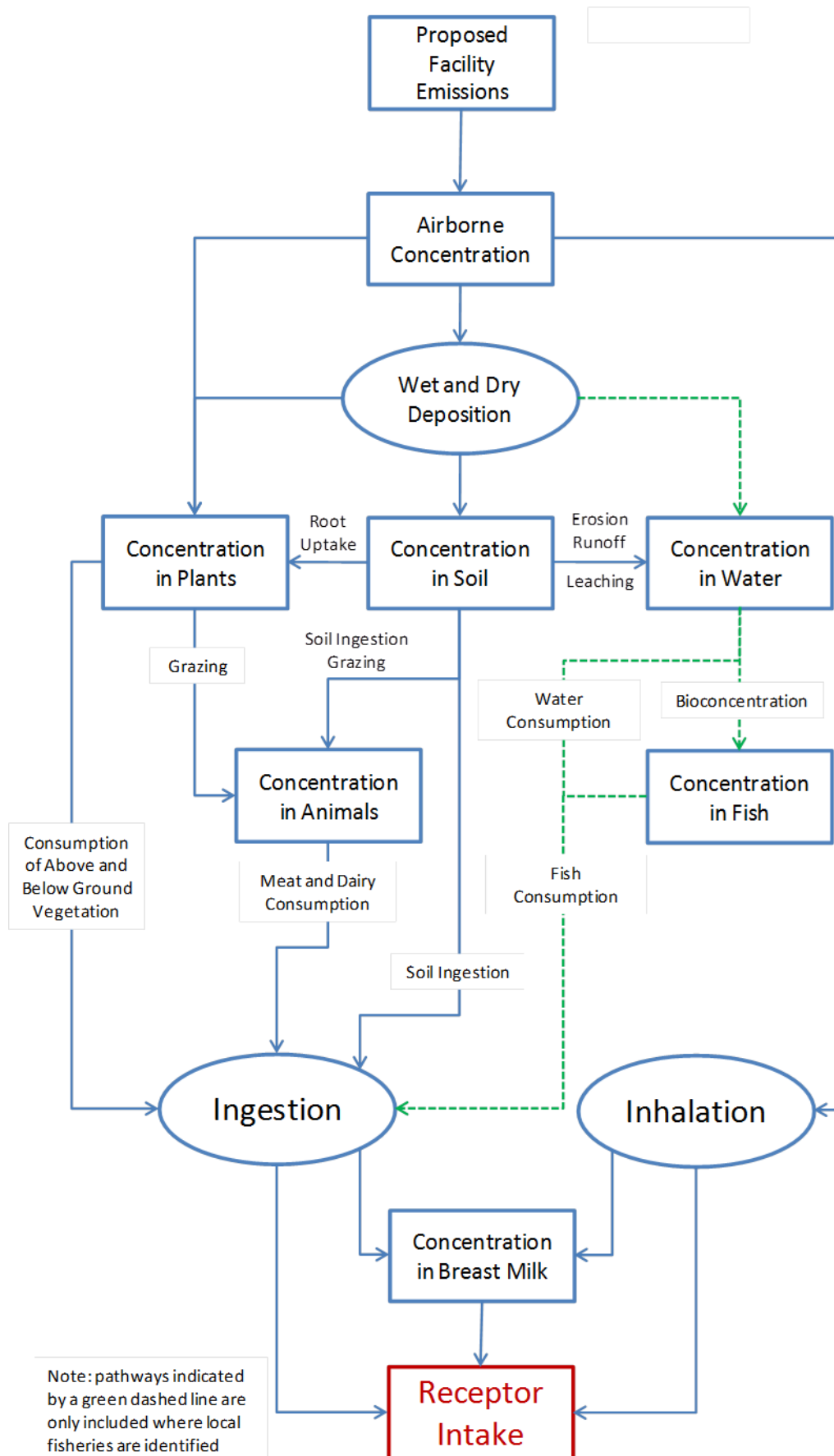
2.6 Similar arguments are relevant with respect to the elimination of aquatic pathways from consideration; swimming, fishing and other recreational activities are also sporadic and unlikely to lead to significant exposures or uptake of any contamination into the human body via dermal contact with water.

2.7 Exposure via drinking water requires contamination of surface drinking water sources local to the point of consumption. The likelihood of contamination reaching a level of concern in the local water sources and ground water supplies is extremely low, particularly where there is no large scale storage (e.g. reservoirs) or catchment areas for local water supplies. However, the US EPA's HHRAP does include the ingestion of drinking water from surface water sources as a potential exposure pathway where water bodies and water sheds have been defined within the exposure scenario. The ingestion of groundwater as a source of local drinking water is not considered by the HHRAP as it is considered to be an insignificant exposure pathway for combustion emissions.

2.8 On the basis of the assessment of the potential significance of the exposure pathways the key exposure pathways which are relevant to the assessment and, hence, subject to examination in detail are as follows:

- inhalation;
- ingestion of food; and
- ingestion of soil.

**Figure 2.1: Exposure Pathways for Receptors**





2.9 Therefore, the exposures arising from ingestion are assessed with reference to the following:

- milk from home-reared cows;
- eggs from home-reared chickens;
- home-reared beef;
- home-reared pork;
- home-reared chicken;
- home-grown vegetable and fruit produce;
- breastmilk; and
- soil (incidental).

2.10 The inclusion of all food groups in the assessment conservatively assumes that both arable and pasture land are present in the vicinity of the predicted maximum annual average ground level concentration. This is, in reality, a highly unlikely scenario, but it has been included as a means of building a high degree of conservatism into the assessment and, hence, reducing the risk of exposures being underestimated. However, it should be noted that not all exposure scenarios will result in the ingestion of home-reared meat and animal products and these food products are only considered by the HHRAP for farmers and the families of farmers. Similarly, the ingestion of fish is only considered where there is a local closed water body that is used for fishing and where the diet of the fisher (and family) may be regularly supplemented by fish caught from these local water sources. A review of local fisheries indicates that there are no fisheries within 3 km where edible fish (e.g. trout or salmon) may be taken. Therefore, the ingestion of locally caught fish has not been considered, as consumption rates are likely to be very small.

## **Emissions and Dispersion Modelling Input Data**

### Compounds of Potential Concern (COPCs)

2.11 The substances which have been considered in the assessment are referred to as the Compounds of Potential Concern (COPCs) and include the seventeen PCDD/F congeners that are known to be toxic. In addition, the IRAP model includes two dioxin-like PCBs (Aroclor 1016 and Aroclor 1254). These comprise a mixture of congeners with one to four chlorine atoms for Aroclor 1016 with a chlorine content of 41% by mass (average of three chlorine atoms). Similarly, Aroclor 1254 has between four and seven chlorine atoms and a chlorine content of 54% by mass (average of five chlorine atoms).



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## Emission Parameters

2.12 Emissions from the facility will be via a single 43 m high stack. Emission parameters assumed for the assessment are consistent with those used for the air quality assessment as follows:

- stack diameter of 1.6 m;
- emission velocity of 17.8 m/s;
- actual flow rate of 35.7 Am<sup>3</sup>/s;
- normalised flow rate of 21.6 Nm<sup>3</sup>/s; and
- emission temperature of 146 °C.

## Emission Concentrations for the COPCs

2.13 The general term dioxins denotes a family of compounds, with each compound composed of two benzene rings interconnected with two oxygen atoms. There are 75 individual dioxins, with each distinguished by the position of chlorine or other halogen atoms positioned on the benzene rings. Furans are similar in structure to dioxins, but have a carbon bond instead of one of the two oxygen atoms connecting the two benzene rings. There are 135 individual furan compounds. Each individual furan or dioxin compound is referred to as a congener and each has a different toxicity and physical properties with regard to its atmospheric behaviour. It is important, therefore, that the exposure methodology determines the fate and transport of PCDD/Fs on a congener specific basis. It does this by accounting for the varying volatility of the congeners and their different toxicities. Consequently, information regarding the PCDD/F annual mean ground level concentrations on a congener specific basis is required. For the purposes of the exposure assessment, the congener profile for the proposed facility is presented in Table 2.1, which is a standard profile for municipal waste incinerators derived by Her Majesty's Inspectorate of Pollution (HMIP), one of the predecessors of the Environment Agency. The international toxic equivalency factors are given and used to derive the toxic equivalent emission (I-TEQ).

2.14 It is assumed that PCDD/F emissions are at 10% of the IED emission limit (0.01 ng I-TEQ/Nm<sup>3</sup>). There is limited information on PCDD/F emissions from energy from waste facilities burning Syngas. The Defra report<sup>4</sup> indicates that gasification plant emit dioxins and furans at 2% of the IED emission limit. An uncertainty factor of five has been applied to this to derive a typical emission concentration for the facility but which may be considered to be worst-case.

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<sup>4</sup> WR 0608 Emissions from Waste Management Facilities, ERM Report on Behalf of Defra (July 2011)



**Table 2.1: PCDD/F Congener Profile for the Proposed Facility**

<b>Congener</b>	<b>Annual Mean Emission Concentration (ng/Nm<sup>3</sup>) (a)</b>	<b>I-TEF (toxic equivalent factors)</b>	<b>Annual Mean Emission Concentration (ng I-TEQ/Nm<sup>3</sup>)</b>
2,3,7,8 TCDD	0.00031	1.0	0.00031
1,2,3,7,8 PeCDD	0.0025	0.5	0.0012
1,2,3,4,7,8 HxCDD	0.0029	0.1	0.00029
1,2,3,7,8,9 HxCDD	0.0021	0.1	0.00021
1,2,3,6,7,8 HxCDD	0.0026	0.1	0.00026
1,2,3,4,6,7,8 HpCDD	0.017	0.01	0.00017
OCDD	0.040	0.001	0.000040
2,3,7,8 TCDF	0.0028	0.1	0.00028
2,3,4,7,8 PeCDF	0.0054	0.5	0.0027
1,2,3,7,8 PeCDF	0.0028	0.05	0.00014
1,2,3,4,7,8 HxCDF	0.022	0.1	0.0022
1,2,3,7,8,9 HxCDF	0.00040	0.1	0.000040
1,2,3,6,7,8 HxCDF	0.0081	0.1	0.00081
2,3,4,6,7,8 HxCDF	0.0087	0.1	0.00087
1,2,3,4,6,7,8 HpCDF	0.044	0.01	0.00044
1,2,3,4,7,8,9 HpCDF	0.0040	0.01	0.000040
OCDF	0.040	0.001	0.000040
<b>Total (ng I-TEQ/m<sup>3</sup>)</b>			<b>0.01</b>
(a) Congener profile from Table 7.2a DOE (1996) Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes Contract No. HMIP/CPR2/41/1/181			

2.15 Given the limited availability of emissions data for energy from waste facilities burning Syngas, the total emission of dioxin-like PCBs from the facility is assumed to be the same as for an Energy from Waste (EFW) plant and is considered to represent a worst case with respect to the facility emissions. Information on PCB emissions has been obtained from the Defra report WR 0608<sup>4</sup>. Based on the information provided, a maximum emission concentration of  $3.6 \times 10^{-9} \text{ mg/m}^3$  is assumed. It is not stated whether this is total PCBs or dioxin-like PCBs. Therefore, as a worst-case it is assumed to comprise entirely of dioxin-like PCBs. Furthermore, it is assumed that this is the total PCB emission and that these data are presented as the toxic equivalent concentration (i.e.  $3.6 \times 10^{-9} \text{ mg/m}^3$ ). For the dioxin-like PCBs, a toxic equivalent factor (TEF) of 0.1 has been used to provide an actual emission concentration (i.e.  $3.6 \times 10^{-8} \text{ mg/m}^3$ ). The same equivalence factor has been used to convert the total actual dose back to the total toxic equivalent dose.



2.16 The emission rates for each substance as input to the IRAP model are provided in Table 2.2.

**Table 2.2: PCDD/F Emission Rates Used in the IRAP Model**

Congener	Emission Concentration (mg/Nm <sup>3</sup> )	Emission Rate (g/s)
2,3,7,8 TCDD	$0.00031 \times 10^{-6}$	$6.7 \times 10^{-12}$
1,2,3,7,8 PeCDD	$0.0025 \times 10^{-6}$	$5.3 \times 10^{-11}$
1,2,3,4,7,8 HxCDD	$0.0029 \times 10^{-6}$	$6.3 \times 10^{-11}$
1,2,3,7,8,9 HxCDD	$0.0021 \times 10^{-6}$	$4.5 \times 10^{-11}$
1,2,3,6,7,8 HxCDD	$0.0026 \times 10^{-6}$	$5.6 \times 10^{-11}$
1,2,3,4,6,7,8 HpCDD	$0.017 \times 10^{-6}$	$3.7 \times 10^{-10}$
OCDD	$0.040 \times 10^{-6}$	$8.6 \times 10^{-10}$
2,3,7,8 TCDF	$0.0028 \times 10^{-6}$	$6.0 \times 10^{-11}$
2,3,4,7,8 PeCDF	$0.0054 \times 10^{-6}$	$1.2 \times 10^{-10}$
1,2,3,7,8 PeCDF	$0.0028 \times 10^{-6}$	$6.0 \times 10^{-11}$
1,2,3,4,7,8 HxCDF	$0.022 \times 10^{-6}$	$4.7 \times 10^{-10}$
1,2,3,7,8,9 HxCDF	$0.00040 \times 10^{-6}$	$8.6 \times 10^{-12}$
1,2,3,6,7,8 HxCDF	$0.0081 \times 10^{-6}$	$1.7 \times 10^{-10}$
2,3,4,6,7,8 HxCDF	$0.0087 \times 10^{-6}$	$1.9 \times 10^{-10}$
1,2,3,4,6,7,8 HpCDF	$0.044 \times 10^{-6}$	$9.5 \times 10^{-11}$
1,2,3,4,7,8,9 HpCDF	$0.0040 \times 10^{-6}$	$8.6 \times 10^{-10}$
OCDF	$0.040 \times 10^{-6}$	$8.6 \times 10^{-10}$
Aroclor 1016/1254	$0.036 \times 10^{-6}$	$7.8 \times 10^{-10}$

#### Dispersion Modelling Assumptions

2.17 The air quality assessment has relied upon the use of AERMOD to estimate ground level concentrations of pollutants. The HHRA model has been designed to accept output files from the US EPA ISC or AERMOD dispersion models, reflecting its North American origins and its need to follow the US EPA risk assessment protocol. The use of AERMOD is consistent with the air quality assessment undertaken for the facility and the emissions data and model set up are identical to that carried out for the air quality assessment <sup>2</sup>.

2.18 For the modelling, all emission properties, building heights, and other relevant factors were retained from the air quality assessment provided for the facility. As the health risk assessment requires information on the deposition of substances to surfaces as well as airborne

concentrations of substances, the AERMOD dispersion model has also been used to predict the following:

- the airborne concentration of vapour, particle and particle bound substances emitted;
- the wet deposition rate of particle and particle bound substances; and
- the dry deposition rate of vapour, particle and particle bound substances.

2.19 For AERMOD, deposition velocities are determined from the assumed particle diameters and particle density of the emissions for three particle sizes based on information provided by the Minnesota Pollution Control Agency (MPCA)<sup>5</sup>. Details of particle sizes, density and assumed fractions are provided in Table 2.3.

**Table 2.3: Particle Sizes, Particle Density and Particle Fractions Used in the IRAP Model**

Particle Diameter	Particle Density (g/cm <sup>3</sup> )	Mass Fraction (a)	Area Fraction (b)(c)
1 µm	1	0.25	0.625
2.5 µm	1	0.25	0.25
10 µm	1	0.5	0.125
(a) Fraction assumed for the particle phase			
(b) Fraction assumed for the particle bound phase			
(c) Calculated from the mass fraction utilising the method described in the US EPA HHRAP			

## Dispersion Modelling Results

2.20 A summary of the key results from the AERMOD dispersion model is presented in Table 2.4. These have been predicted using the 2011 Rhoose (Cardiff International Airport) meteorological data set. This year was selected, as out of the five years considered, it was the year that provided highest predicted annual mean concentrations and deposition rates.

<sup>5</sup> Refined HHRAP-Based Analysis Form, AERA-26, Minnesota Pollution Control Agency (August 2011)





**Table 2.4: Maximum Annual Average Particle Phase Concentrations and Particle Phase Deposition Rates Estimated by AERMOD for the Rhoose 2011 Meteorological Data**

<b>Pollutant</b>	<b>Max Annual Average Concentration (a)</b>	<b>Max Annual Average Deposition Rate (b)</b>
<b>PCDD/Fs</b>	<b>(fg/m<sup>3</sup>)</b>	<b>(ng/m<sup>2</sup>/year)</b>
2,3,7,8 TCDD	0.0040	0.027
1,2,3,7,8 PeCDD	0.032	0.21
1,2,3,4,7,8 HxCDD	0.038	0.25
1,2,3,7,8,9 HxCDD	0.027	0.18
1,2,3,6,7,8 HxCDD	0.034	0.22
1,2,3,4,6,7,8 HpCDD	0.22	1.5
OCDD	0.52	3.5
2,3,7,8 TCDF	0.036	0.24
2,3,4,7,8 PeCDF	0.070	0.46
1,2,3,7,8 PeCDF	0.036	0.24
1,2,3,4,7,8 HxCDF	0.28	1.9
1,2,3,7,8,9 HxCDF	0.0052	0.035
1,2,3,6,7,8 HxCDF	0.11	0.70
2,3,4,6,7,8 HxCDF	0.11	0.75
1,2,3,4,6,7,8 HpCDF	0.57	3.8
1,2,3,4,7,8,9 HpCDF	0.052	0.35
OCDF	0.52	3.5
Aroclor 1016/1254	0.47	3.1
(a) Where 1 fg/m <sup>3</sup> is equal to 1 x 10 <sup>-15</sup> g/m <sup>3</sup>		
(b) Where 1 ng/m <sup>2</sup> /year is equal to 1 x 10 <sup>-9</sup> g/m <sup>2</sup> /year		

### 3 INPUT PARAMETERS FOR THE IRAP MODEL

#### Introduction

3.1 Exposure of an individual to a chemical may occur either by inhalation or ingestion (including food, water and soil). Of interest is the total dose of the chemical received by the individual through the combination of possible routes, and the IRAP model has been developed to estimate the dose received by the human body, often referred to as the external dose.

3.2 Exposure to COPCs is a function of the estimated concentration of the substance in the environmental media with which individuals may come into contact (i.e. exposure point concentrations) and the duration of contact. The concentration at the point of contact is itself a function of the transfer through air, soil, water, plants and animals that form part of the overall pathway. Exposure equations have been developed which combine exposure factors (e.g. exposure duration, frequency and medium intake rate) and exposure point concentrations. The dose equations therefore facilitate estimation of the received dose and account for the properties of the route of exposure, i.e. ingestion and inhalation.

3.3 For those substances that bio-accumulate, i.e. become more concentrated higher up the food chain, especially in body fats, the exposure to meats and milk is of particular significance.

3.4 The IRAP model user has the facility to adjust some of the key exposure factors. An example is the diet of the receptor and the proportion of which is local produce, which may be contaminated. Obviously, if a nearby resident eats no food grown locally, then that person's diet cannot be contaminated by the emissions from the source, in this case the proposed facility. It is conventional to investigate two types of receptor, a farmer and a resident. It is assumed that a farmer eats proportionately more locally grown food than a resident. Where the potential exists for the consumption of locally caught fish a fisher receptor may also be considered.

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

3.6 The IRAP model is designed to accept output files of airborne concentrations and deposition rates. From these, it proceeds to calculate the concentrations of the pollutants of concern in the environmental media, foodstuffs and the human receptor. The dose experienced by the human receptor can be compared to the tolerable daily intake (TDI) provided by the Committee on Toxicity for dioxins and dioxin like PCBs of 2 pg/kg/d.



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3.7 The model requires a wide range of input parameters to be defined, these include:

- physical and chemical properties of the COPCs;
- site information, including site specific data; and
- receptor information – for each receptor type (e.g. adult or child, resident or farmer or fisher).

3.8 The HHRAP default values, which are incorporated into the IRAP model, have been used for the majority of these input values. These data are provided in the following sections.

### **Input Parameters for the COPCs**

3.9 The IRAP model contains a database of physical and chemical parameters for each of 206 COPCs. This database is based on default values provided by the HHRAP and all default values have been used for this assessment.

3.10 These parameters are used to determine how each of the COPCs behaves in the environment and their presence and accumulation in various food products (meat, fish, animal products, vegetation, soil and water). For 2,3,7,8-TCDD (the most toxic of the PCDD/Fs), the default parameters are provided in Table 3.1.



**Table 3.1: IRAP Input Parameters for 2,3,7,8-TCDD**

Parameter Description	Symbol	Units	2,3,7,8-TCDD
Chemical abstract service number	CAS No.	-	1746-01-6
Molecular weight	MW	g/mole	322.0
Melting point of chemical	T_m	K	578.7
Vapour pressure	V_p	atm	1.97 x 10 <sup>-12</sup>
Aqueous solubility	S	mg/l	1.93 x 10 <sup>-5</sup>
Henry's Law constant	H	atm-m <sup>3</sup> /mol	3.29 x 10 <sup>-5</sup>
Diffusivity of COPC in air	D_a	cm <sup>2</sup> /s	0.104
Diffusivity of COPC in water	Dw	cm <sup>2</sup> /s	5.6 x 10 <sup>-6</sup>
Octanol-water partition coefficient	K_ow	-	6,309,573
Organic carbon-water partition coefficient	K_oc	ml/g	3,890,451
Soil-water partition coefficient	Kd_s	ml/g	38,904
Suspended sediments/surface water partition coefficient	Kd_sw	l/kg	291,784
Bed sediment/sediment pore water partition coefficient	Kd_bs	ml/g	155,618
COPC loss constant due to biotic and abiotic degradation	K_sg	/a	0.03
Fraction of COPC air concentration in vapour phase	f_v		0.664
Root concentration factor	RCF	ml/g	39,999
Plant-soil bioconcentration factor for below ground produce	br_root_veg	-	1.03
Plant-soil bioconcentration factor for leafy vegetables	br_leafy_veg	-	0.00455
Plant-soil bioconcentration factor for forage	br_forage	-	0.00455
COPC air-to-plant biotransfer factor for leafy vegetables	bv_leafy_veg	-	65,500
COPC air-to-plant biotransfer factor for forage	bv_forage	-	65,500
COPC biotransfer factor for milk	ba_milk	day/kg	0.0055
COPC biotransfer factor for beef	ba_beef	day/kg	0.026
COPC biotransfer factor for pork	ba_pork	day/kg	0.032
Bioconcentration factor for COPC in eggs	Bcf_egg	-	0.060
Bioconcentration factor for COPC in chicken	Bcf_chicken	-	3.32
Fish bioconcentration factor	BCF_fish	l/kg	34,400
Fish bioaccumulation factor	BAF_fish	l/kg	0



Biota-sediment accumulation factor	BSAF_fish	-	0.09
Plant-soil bioconcentration factor for grain	br_grain	-	0.00455
Plant-soil bioconcentration factor for eggs	br_egg	-	0.011
COPC biotransfer factor for chicken	ba_chicken	day/kg	0.019

### Site and Site Specific Parameters

3.11 The IRAP health risk assessment model requires information relating to the location and its surroundings. The parameters required include the following.

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

3.12 For all of these parameters the IRAP/EPA HHRAP default values have been used and these are presented in **Appendix A**. Other site specific parameters are also required which are not provided by the IRAP model. These parameters were specified for the proposed facility as follows:

- Annual average evapotranspiration rate of 70.0 cm/a (assumed to be 70% of total precipitation);
- Annual average precipitation of 100.0 cm/a (based on the average for the five year data set for 2009 to 2013 meteorological data);



- 
- Annual average irrigation of 0 cm/a;
  - Annual average runoff of 10.0 cm/a (assumed to be 10% of total precipitation);
  - An annual average wind velocity of 4.6 m/s (average for the five years); and
  - A time period over which deposition occurs of 30 years.

### **Receptor Information**

3.13 Within the IRAP model there are three receptor types; Resident, Farmer and Fisher. Information relating to each receptor type (adult and/or child) is required by the model where these receptor types are used. The information required includes the following:

- Food (meat, dairy products, fish and vegetables), water and soil consumption rates for each receptor type. However, only Fishers are assumed to consume fish and only Farmers are assumed to consume locally reared animals and animal products.
- Fraction of contaminated food, water and soil which is consumed by each receptor type.
- Input data for the inhalation exposure including: inhalation exposure duration, inhalation exposure frequency, inhalation exposure time; and inhalation rate.
- Input data for the ingestion exposure including: exposure duration, exposure frequency, exposure time; and body weight of receptor.

3.14 For the purposes of this assessment the default IRAP/HHRAP parameters have been used to define the characteristics of the receptors. The input data used are presented in **Appendix B**.



## 4 EXPOSURE ASSESSMENT

### Selection of Receptors

4.1 In addition to defining specific locations for assessment, IRAP can be used to determine the location of maximum impact over an area based on the results of the dispersion model. For each defined land-use area, IRAP selects the locations which represent the maximum predicted concentrations or deposition rates for the area selected. The locations of these various maxima are often co-located resulting in the selection of one to nine receptor locations per defined area. This approach is adopted by IRAP since the maximum receptor impact may occur at any one of the maximum concentration or deposition locations identified.

4.2 Residential exposure within the immediate vicinity of the facility is limited. The immediate locality is industrial and dominated by the docks to the east and southwest of the site. The nearest residential areas are to the north along Dock View Road and the extensive areas of Barry beyond to the north, northeast and northwest. Therefore, seven areas where residential exposure may occur have been defined based on residential areas around the proposed facility. These are the nearest residential settlements.

4.3 There are limited rural areas within the immediate vicinity of the facility. The nearest extensive rural area where farming activities might be carried out lie approximately 2 km to the north and east of the site on the eastern boundary of Barry and 2.5 km to the north and west on the western boundary. There is also a small area to the 1.2 km to the east of the site above Sully Bay which has been used for growing crops. Therefore, two areas where the potential for farming exists have been defined with the area above Sully Bay included within the Farmer East area.

4.4 For each type of receptor up to nine locations are selected based on the maximum predicted airborne concentration, maximum predicted wet deposition rate and maximum dry deposition rate. However, often these maxima are co-located and, therefore, each receptor type will have between one and nine identified receptors per defined area. For the assessment, 22 Residential receptors and 6 Farmer receptors have been assessed. It is considered that the likelihood of locally caught fish being consumed is low and fisher receptors have not been included in the assessment. For all of the receptor types, adult and child receptors have been considered. The locations of the Resident and Farmer receptors are presented in Table 4.1 and Figure 4.1. At other locations not specifically considered in the assessment, the predicted hazards and risks will be lower than predicted for the discrete receptors considered.

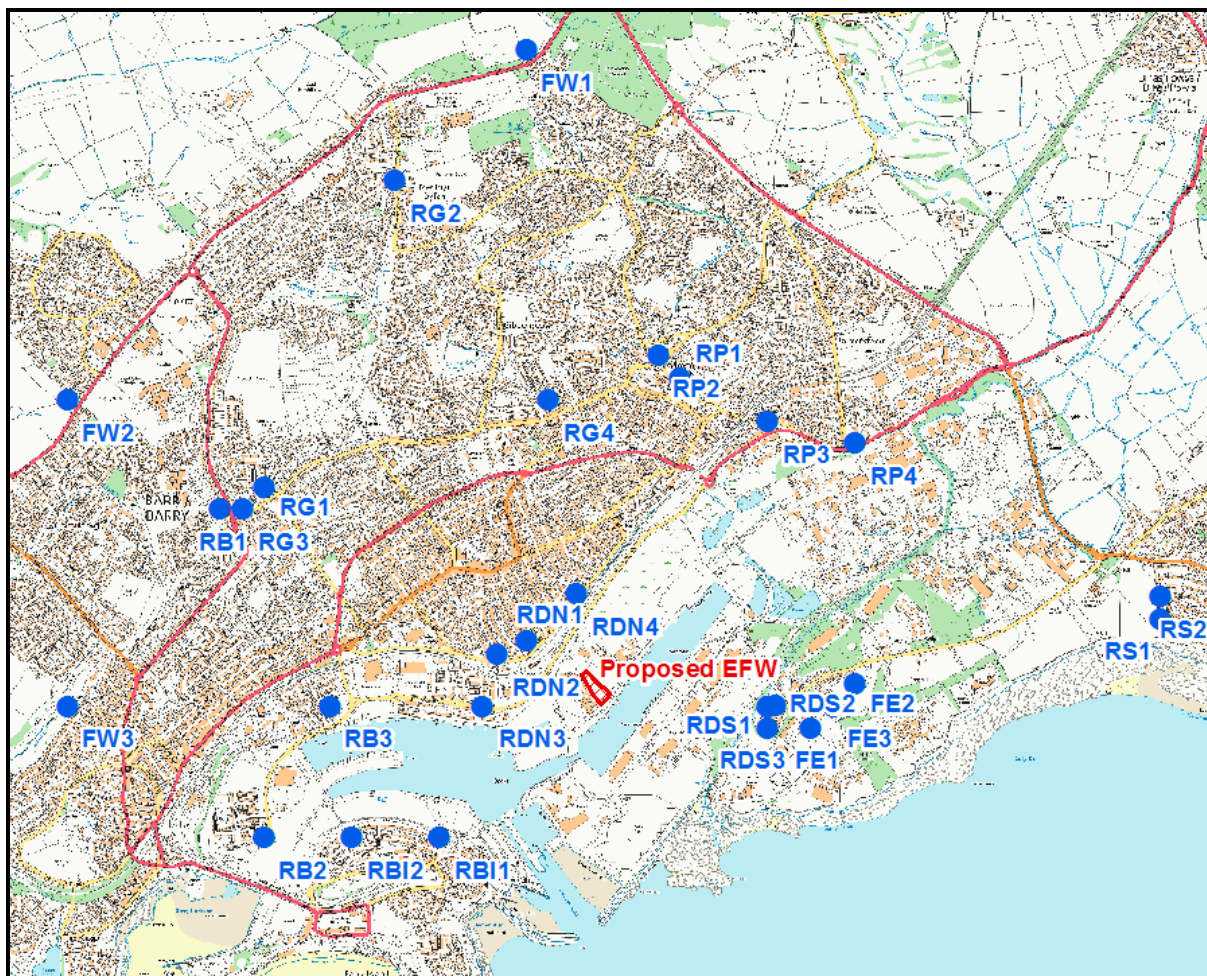




**Table 4.1: Description of Resident and Farmer Receptors**

Reference	Name	Type	Easting	Northing
FE1	Farmer East 1	Farmer	313600	167500
FE2	Farmer East 2	Farmer	313800	167700
FE3	Farmer East 3	Farmer	313700	167600
FW1	Farmer West 1	Farmer	312300	170600
FW2	Farmer West 2	Farmer	310200	169000
FW3	Farmer West 3	Farmer	310200	167600
RB1	Residential Barry 1	Residential	310900	168500
RB2	Residential Barry 2	Residential	311100	167000
RB3	Residential Barry 3	Residential	311400	167600
RBI1	Residential Barry Island 1	Residential	311900	167000
RBI2	Residential Barry Island 2	Residential	311500	167000
RDN1	Residential Docks North 1	Residential	312300	167900
RDN2	Residential Docks North 2	Residential	312162	167836
RDN3	Residential Docks North 3	Residential	312100	167600
RDN4	Residential Docks North 4	Residential	312528	168111
RDS1	Residential Docks South 1	Residential	313400	167600
RDS2	Residential Docks South 2	Residential	313437	167606
RDS3	Residential Docks South 3	Residential	313400	167500
RG1	Residential Gibbonsdown 1	Residential	311100	168600
RG2	Residential Gibbonsdown 2	Residential	311700	170000
RG3	Residential Gibbonsdown 3	Residential	311000	168500
RG4	Residential Gibbonsdown 4	Residential	312400	169000
RP1	Residential Palmerstown 1	Residential	313000	169100
RP2	Residential Palmerstown 2	Residential	312900	169200
RP3	Residential Palmerstown 3	Residential	313400	168900
RP4	Residential Palmerstown 4	Residential	313800	168800
RS1	Residential Sully 1	Residential	315200	168000
RS2	Residential Sully 2	Residential	315200	168100

**Figure 4.1: Location of the Resident and Farmer Receptors**



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## Assessment of Intake

### Ingestion Dose

4.5 The ingestion intake is calculated as the Average Daily Dose (ADD) from all ingestion exposure routes (e.g. soil, above ground vegetables, meat and dairy products) where for example:

$$ADD_{Ing, TCDD} = \frac{I_{Ing, TCDD} \bullet ED \bullet EF}{AT \bullet 365}$$

4.6 Where:  $ADD_{Ing, TCDD}$  = total ingestion dose for TCDD; ED is the exposure duration (dependent on the receptor type); EF is the exposure frequency (350 days per year); and AT is the averaging time, and for determining the TDI, is assumed to be equal to the ED. The total dose is the sum of the dose for each of the individual congeners.

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## Inhalation Dose

4.7 For inhalation, the ADD from inhalation exposure is calculated as follows:

$$ADD_{Inh, TCDD} = \frac{C_a \bullet IR \bullet ED \bullet EF}{AT \bullet 365}$$

4.8 Where:  $ADD_{Inh, TCDD}$  is the total inhalation dose for TCDD,  $C_a$  is the concentration of TCDD in air and IR is the daily inhalation rate. The total dose is the sum of the dose for each of the individual congeners.

## **Exposure to Dioxins and Furans**

### Comparison of Dioxin/Furan Exposure with WHO and UK COT Guidance

#### *Facility Contribution to Intake*

4.9 The World Health Organization (WHO) recommends a tolerable daily intake for dioxins/furans of 1 to 4 pg I-TEQ/kg-BW/d (picogrammes as the International Toxic Equivalent per kilogram bodyweight per day)<sup>6</sup>. The TDI represents the tolerable daily intake for lifetime exposure and short-term excursions above the TDI would have no consequence provided that the average intake over long periods is not exceeded. The average (lifetime) daily intake of dioxins/furans for the receptors considered is presented in Table 4.2. These are also compared to the Committee on Toxicity (COT) TDI for dioxins and dioxin-like PCBs of 2 pg I-TEQ/kg-BW/d.

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<sup>6</sup> Assessment of the Health Risk of Dioxins: Re-evaluation of the Tolerable Daily Intake (TDI), WHO Consultation, May 25-29 1998, Geneva, Switzerland



**Table 4.2: Comparison of Average Daily Intakes with the UK COT and WHO's TDI for Dioxins/ Furans (pg I-TEQ/kg-BW/d)**

Receptor Name	Adult	Child
Farmer East 1	0.013	0.018
Farmer East 2	0.0095	0.014
Farmer East 3	0.012	0.017
Farmer West 1	0.00036	0.00052
Farmer West 2	0.00048	0.00070
Farmer West 3	0.00066	0.00098
Residential Barry 1	0.000039	0.00012
Residential Barry 2	0.000086	0.00027
Residential Barry 3	0.00012	0.00036
Residential Barry Island 1	0.00018	0.00057
Residential Barry Island 2	0.00013	0.00042
Residential Docks North 1	0.00029	0.00092
Residential Docks North 2	0.00033	0.0010
Residential Docks North 3	0.00038	0.0012
Residential Docks North 4	0.00020	0.00062
Residential Docks South 1	0.00085	0.0027
Residential Docks South 2	0.00074	0.0023
Residential Docks South 3	0.00076	0.0024
Residential Gibbonsdown 1	0.000042	0.00013
Residential Gibbonsdown 2	0.000019	0.000058
Residential Gibbonsdown 3	0.000041	0.00013
Residential Gibbonsdown 4	0.000053	0.00016
Residential Palmerstown 1	0.000045	0.00014
Residential Palmerstown 2	0.000043	0.00013
Residential Palmerstown 3	0.000074	0.00023
Residential Palmerstown 4	0.000081	0.00025
Residential Sully 1	0.000086	0.00027
Residential Sully 2	0.000083	0.00026
<i>WHO TDI</i>	<i>1 to 4 pg I-TEQ/kg-BW/d</i>	
<i>Committee on Toxicity (COT) TDI</i>	<i>2 pg I-TEQ/kg-BW/d</i>	

4.10 The maximum contribution of the facility to the COT TDI is 0.9% for the Farmer East 1 child receptor and 0.1% for the Resident Docks South 1 child receptor. However, this assumes as a worst-case that these receptors produce their own home reared and grown food at the



location of maximum impact for the area. Therefore, the contribution of the facility to the intake of dioxins/furans and dioxin-like PCBs is negligible for all receptors.

#### *Total Intake*

4.11 The contribution of the facility to total intake is provided as follows:

- predicted incremental intake due to emissions from the facility;
- average daily background intake (i.e. that arising from other sources), referred to as the mean daily intake (MDI);
- the total intake (i.e. the sum of the predicted incremental intake and the MDI);
- a comparison of the total intake with the TDI for dioxin/furans.

4.12 For the key receptors (i.e. those which represent the predicted highest exposure for the receptor types considered) the results are presented in Table 4.3. Results are presented for both adult and child receptors.

4.13 The MDI is derived from data provided by the Environment Agency <sup>7</sup> and a value of 49 pg WHO-TEQ/d. The MDI for an adult receptor and child receptor is calculated as follows:

- for an adult receptor a MDI of 0.7 pg I-TEQ/kg/d <sup>8</sup> is derived by dividing the Environment Agency MDI by a bodyweight of 70 kg;
- for a child receptor a MDI of 2.1 pg I-TEQ/kg/d is derived by dividing the Environment Agency MDI by a bodyweight of 15 kg and applying an adult to child correction factor of 0.65.

4.14 For the child receptor, the MDI for dioxin/furans exceeds the TDI without the contribution from the facility. However, in deriving the MDI for the child receptors, a bodyweight of 15 kg has been used in order to be consistent with the IRAP predictions which assume a bodyweight of 15 kg. As a consequence the MDI is higher than it would be for a 20 kg child (typically assumed in the UK) and also represents a worst case. For a 20 kg child (adult to child correction factor of 0.74) the MDI for a child would be 1.8 pg I-TEQ/kg/d and below the COT TDI.

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<sup>7</sup> Soil Guideline Values for dioxins, furans and dioxin-like PCBs in soil, Environment Agency, Science Report SC050021/Dioxins SGV, September 2009

<sup>8</sup> No correction is provided between the WHO-TEF and the I-TEF but a sensitivity analysis indicates that correcting between the two systems would have negligible impact on the results



4.15 A comparison of predicted intakes with the MDI and TDI is presented in Table 4.3. Results are presented for the Farmer East 1 and Resident Docks South 1 receptors where highest farmer and resident exposures are predicted, respectively.

**Table 4.3: Comparison of Total Intake with the COT TDI**

Receptor	Total Intake from the Facility (pg I-TEQ/kg/d)	Total Intake + MDI (pg I-TEQ/kg/d)	Facility as %age of TDI	Total Intake as %age of TDI
Farmer East 1 Adult	0.013	0.71	0.6%	35.6%
Farmer East 1 Child	0.018	2.12	0.9%	105.9%
Resident Docks South 1 Adult	0.00085	0.70	0.04%	35.0%
Resident Docks South 1 Child	0.0027	2.10	0.13%	105.1%
<i>COT TDI</i>	2	2	-	-

4.16 For inhalation and oral intake of PCDD/Fs for adults, total intake is well below the TDI. Background exposure represents approximately 35% of total exposure. At worst, the facility contributes 0.6% to the TDI for adults.

4.17 For inhalation and oral intake of PCDD/Fs for children, the background intake is in excess of the TDI. At worst the additional contribution from the facility for a child is 0.018 pg I-TEQ/kg/d (0.9% of the COT TDI). Combined with the background exposure for a 20 kg child (1.8 pg I-TEQ/kg/d) the total intake would be below the TDI (90.9%). Furthermore, it should be noted that the TDI for PCDD/Fs is set for the purposes of assessing lifetime exposure and these elevated background exposures for children are therefore not representative of long term exposure.

#### **Infant Breast Milk Exposure to Dioxins and Furans**

4.18 Another exposure pathway of interest is infant exposure to dioxins and furans via the ingestion of their mother's breast milk. This is because the potential for contamination of breast milk is particularly high for dioxin-like compounds such as these, as they are extremely lipophilic (fat soluble) and hence likely to accumulate in breast milk. Further, the infant body weight is smaller and it could be argued that the effect is therefore proportionately greater than in an adult.

4.19 This exposure is measured by the Average Daily Dose (ADD) on the basis of an averaging time of 1 year. In the US, a threshold value of 50 pg/kg/d of 2,3,7,8-TCDD TEQ is





cited as being potentially harmful. The IRAP model calculates the ADD that would result from an adult receptor breast feeding an infant. It should be noted that the ADD calculated by IRAP does not consider dioxin-like PCBs. A summary of the ADD for each of the infants of adult receptors considered for the assessment is presented in Table 4.4.

4.20 The highest ADDs are calculated for the infants of farmer receptors and represent at worst less than 0.3% of the US EPA criterion of 50 pg/kg/d of 2,3,7,8-TCDD. The calculated ADDs for residential receptors are lower compared to the farmer since the most significant exposure to dioxins/furans is via the food chain, particularly animals and animal products. The farmer receptors are assumed to consume contaminated meat and dairy products. However, residential receptors are only assumed to consume vegetable products which are less significant with regard to exposure to dioxins/furans.

4.21 As a worst case, the ADD for the highest exposure for the infants of farmers is 7% of the COT TDI. However, for these receptors it is assumed, as a worst-case, that all of their food produce is reared and grown locally at the location of maximum impact in their area. Furthermore, the duration of exposure is short and the average daily intake over the lifetime of the individual would be substantially less. For residential receptors, the maximum exposure via breast milk is 0.4% of the COT TDI.

4.22 The WHO recognises that breast-fed infants will be exposed to higher intakes for a short duration, but also that breast feeding itself provides associated benefits.





**Table 4.4: Assessment of the Average Daily Dose for a Breast-fed Infant of an Adult Receptor**

<b>Receptor Name</b>	<b>Average Daily Dose from Breast Feeding (pg/kg/d of 2,3,7,8-TCDD)</b>
Farmer East 1	0.14
Farmer East 2	0.10
Farmer East 3	0.13
Farmer West 1	0.0040
Farmer West 2	0.0052
Farmer West 3	0.0073
Residential Barry 1	0.00033
Residential Barry 2	0.00075
Residential Barry 3	0.0010
Residential Barry Island 1	0.0016
Residential Barry Island 2	0.0012
Residential Docks North 1	0.0026
Residential Docks North 2	0.0030
Residential Docks North 3	0.0034
Residential Docks North 4	0.0018
Residential Docks South 1	0.0078
Residential Docks South 2	0.0068
Residential Docks South 3	0.0070
Residential Gibbonsdown 1	0.00036
Residential Gibbonsdown 2	0.00015
Residential Gibbonsdown 3	0.00035
Residential Gibbonsdown 4	0.00044
Residential Palmerstown 1	0.00037
Residential Palmerstown 2	0.00036
Residential Palmerstown 3	0.00063
Residential Palmerstown 4	0.00070
Residential Sully 1	0.00075
Residential Sully 2	0.00073
<i>US EPA Criterion</i>	<i>50</i>
<i>WHO Criterion</i>	<i>1 to 4</i>
<i>UK Criterion (COT)</i>	<i>2</i>

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## 5 SUMMARY AND CONCLUSIONS

### Summary

5.1 The possible impacts on human health arising from dioxins and furans (PCDD/F) and dioxin-like PCBs emitted from the proposed energy from waste facility to the south of Barry, Vale of Glamorgan have been assessed under the very worst case scenario, namely that of an individual exposed for a lifetime to the effects of the highest airborne concentrations and consuming mostly locally grown food. This equates to a hypothetical farmer consuming food grown on the farm, situated at the closest proximity to the proposed facility. Where there are no active farming areas in close proximity, a residential receptor is considered where it is assumed that the resident consumes locally grown vegetables.

5.2 The assessment has identified and considered the most plausible pathways of exposure for the individuals considered (farmer and resident). Deposition and subsequent uptake of the compounds of potential concern (COPCs) into the food chain is likely to be the more numerically significant pathway over direct inhalation.

The maximum contribution of the facility to the COT TDI is 0.9% for the farmer receptors and 0.1% for the residential receptors. However, for the farmer this assumes as a worst-case that these receptors are located at the closest farming area to the facility and all of their food is reared and grown at this location. Therefore, the impact of emissions on local sensitive receptors is considered to be not significant.

### Conclusions

5.3 The risk assessment methodology used in this assessment has been structured so as to create worst case estimates of risk. A number of features in the methodology give rise to this degree of conservatism. It has been demonstrated that for the maximally exposed individual, exposure to dioxins, furans and dioxin-like PCBs is not significant.



## APPENDIX A – SITE PARAMETERS

**Table A1: Site Parameters Defined for the Health Risk Assessment**

Parameter	Parameter Value	IRAP Symbol	Units
Soil dry bulk density	1.5	bd	g/cm <sup>3</sup>
Forage fraction grown on contam. soil eaten by CATTLE	1.0	beef_fi_forage	-
Grain fraction grown on contam. soil eaten by CATTLE	1.0	beef_fi_grain	-
Silage fraction grown on contam. eaten by CATTLE	1.0	beef_fi_silage	-
Qty of forage eaten by CATTLE each day	8.8	beef_qp_forage	kg DW/d
Qty of grain eaten by CATTLE each day	0.47	beef_qp_grain	kg DW/d
Qty of silage eaten by CATTLE each day	2.5	beef_qp_silage	kg DW/d
Grain fraction grown on contam. soil eaten by CHICKEN	1.0	chick_fi_grain	-
Qty of grain eaten by CHICKEN each day	0.2	chick_qp_grain	kg DW/d
Fish lipid content	0.07	f_lipid	-
Fraction of CHICKEN's diet that is soil	0.1	fd_chicken	-
Universal gas constant	$8.2 \times 10^{-5}$	gas_r	atm-m <sup>3</sup> /mol/K
Plant surface loss coefficient	18	kp	/a
Fraction of mercury emissions NOT lost to the global cycle	0.48	merc_q_corr	-
Fraction of mercury speciated into methyl mercury in produce	0.22	mercmethyl_ag	-
Fraction of mercury speciated into methyl mercury in soil	0.02	mercmethyl_sc	-
Forage fraction grown contam. soil, eaten by MILK CATTLE	1.0	milk_fi_forage	-
Grain fraction grown contam. soil, eaten by MILK CATTLE	1.0	milk_fi_grain	-
Silage fraction grown contam. soil, eaten by MILK CATTLE	1.0	milk_fi_silage	-



Qty of forage eaten by MILK CATTLE each day	13.2	milk_qp_forage	kg DW/d
Qty of grain eaten by MILK CATTLE each day	3.0	milk_qp_grain	kg DW/d
Qty of silage eaten by MILK CATTLE each day	4.1	milk_qp_silage	kg DW/d
Averaging time	1	milkfat_at	a
Body weight of infant	9.4	milkfat_bw_infant	kg
Exposure duration of infant to breast milk	1	milkfat_ed	a
Proportion of ingested dioxin that is stored in fat	0.9	milkfat_f1	-
Proportion of mothers weight that is fat	0.3	milkfat_f2	-
Fraction of fat in breast milk	0.04	milkfat_f3	-
Fraction of ingested contaminant that is absorbed	0.9	milkfat_f4	-
Half-life of dioxin in adults	2555	milkfat_h	d
Ingestion rate of breast milk	0.688	milkfat_ir_milk	kg/d
Viscosity of air corresponding to air temp.	$1.81 \times 10^{-4}$	mu_a	g/cm/s
Fraction of grain grown on contam. soil eaten by PIGS	1.0	pork_fi_grain	-
Fraction of silage grown on contam. soil and eaten by PIGS	1.0	pork_fi_silage	-
Qty of grain eaten by PIGS each day	3.3	pork_qp_grain	kg DW/d
Qty of silage eaten by PIGS each day	1.4	pork_qp_silage	kg DW/d
Qty of soil eaten by CATTLE	0.5	qs_beef	kg/d
Qty of soil eaten by CHICKEN	0.022	qs_chick	kg/d
Qty of soil eaten by DAIRY CATTLE	0.4	qs_milk	kg/d
Qty of soil eaten by PIGS	0.37	qs_pork	kg/d
Density of air	$1.2 \times 10^{-3}$	rho_a	g/cm <sup>3</sup>
Solids particle density	2.7	rho_s	g/cm <sup>3</sup>



Interception fraction - edible portion ABOVEGROUND	0.39	rp	-
Interception fraction - edible portion FORAGE	0.5	rp_forage	-
Interception fraction - edible portion SILAGE	0.46	rp_silage	-
Ambient air temperature	298	t	K
Temperature correction factor	1.026	theta	-
Soil volumetric water content	0.2	theta_s	ml/cm <sup>3</sup>
Length of plant expos. to depos. - ABOVEGROUND	0.16	tp	a
Length of plant expos. to depos. - FORAGE	0.12	tp_forage	a
Length of plant expos. to depos. – SILAGE	0.16	tp_silage	a
Average annual wind speed	3.9	u	m/s
Dry deposition velocity	0.5	vdv	cm/s
Dry deposition velocity for mercury	2.9	vdv_hg	cm/s
Wind velocity	3.9	w	m/s
Yield/standing crop biomass - edible portion ABOVEGROUND	2.24	yp	kg DW/m <sup>2</sup>
Yield/standing crop biomass - edible portion FORAGE	0.24	yp_forage	kg DW/m <sup>2</sup>
Yield/standing crop biomass - edible portion SILAGE	0.8	yp_silage	kg DW/m <sup>2</sup>
Soil mixing zone depth	2.0	z	cm



## APPENDIX B – SCENARIO PARAMETERS

**Table B1: Exposure Scenario Parameters**

Parameter Description	Adult Resident	Child Resident	Adult Farmer	Child Farmer	Adult Fisher	Child Fisher	Units
Averaging time for carcinogens	70	70	70	70	70	70	a
Averaging time for noncarcinogens	30	6	40	6	30	6	a
Consumption rate of BEEF	0.0	0.0	0.00122	0.00075	0.0	0.0	kg/kg FW/d
Body weight	70	15	70	15	70	15	kg
Consumption rate of POULTRY	0.0	0.0	0.00066	0.00045	0.0	0.0	kg/kg FW/d
Consumption rate of ABOVEGROUND PRODUCE	0.00032	0.00077	0.00047	0.00113	0.00032	0.00077	kg/kg DW/d
Consumption rate of BELOWGROUND PRODUCE	0.00014	0.00023	0.00017	0.00028	0.00014	0.00023	kg/kg DW/d
Consumption rate of DRINKING WATER	1.4	0.67	1.4	0.67	1.4	0.67	l/d
Consumption rate of PROTECTED ABOVEGROUND PRODUCE	0.00061	0.0015	0.00064	0.00157	0.00061	0.0015	kg/kg DW/d
Consumption rate of SOIL	0.0001	0.0002	0.0001	0.0002	0.0001	0.0002	kg/d
Exposure duration	30	6	40	6	30	6	yr
Exposure frequency	350	350	350	350	350	350	d/a
Consumption rate of EGGS	0.0	0.0	0.00075	0.00054	0.0	0.0	kg/kg FW/d
Fraction of contaminated ABOVEGROUND PRODUCE	1.0	1.0	1.0	1.0	1.0	1.0	-
Fraction of contaminated DRINKING WATER	1.0	1.0	1.0	1.0	1.0	1.0	-
Fraction contaminated SOIL	1.0	1.0	1.0	1.0	1.0	1.0	-



Consumption rate of FISH	0.0	0.0	0.0	0.0	0.0	0.0	kg/kg FW/d
Fraction of contaminated FISH	1.0	1.0	1.0	1.0	1.0	1.0	-
Inhalation exposure duration	30	6	40	6	30	6	a
Inhalation exposure frequency	350	350	350	350	350	350	d/a
Inhalation exposure time	24	24	24	24	24	24	h/d
Fraction of contaminated BEEF	1	1	1	1	1	1	-
Fraction of contaminated POULTRY	1	1	1	1	1	1	-
Fraction of contaminated EGGS	1	1	1	1	1	1	-
Fraction of contaminated MILK	1	1	1	1	1	1	-
Fraction of contaminated PORK	1	1	1	1	1	1	-
Inhalation rate	0.83	0.30	0.83	0.30	0.83	0.30	m <sup>3</sup> /h
Consumption rate of MILK	0.0	0.0	0.01367	0.02268	0.0	0.0	kg/kg FW/d
Consumption rate of PORK	0.0	0.0	0.00055	0.00042	0.0	0.0	kg/kg FW/d
Time period at the beginning of combustion	0	0	0	0	0	0	a
Length of exposure duration	30	6	40	6	30	6	a

