Parc Adfer Energy Recovery Facility

Environmental Permit (EP) Application

Human Health Risk Assessment

SLR Ref: 416.04097.00007/HHRA

NRW Ref: PP3733WW

August 2014

CONTENTS

TABLES

FIGURES

1.0 INTRODUCTION

WTI UK Limited (WTI) propose to develop an Energy Recovery Facility (ERF) that would treat up to 200,000 tonnes[1] of residual non-hazardous waste per annum. The ERF would be equipped with one process line.

The proposed development comprises a new road (and potentially rail) connected Combined Heat and Power (CHP) enabled energy facility. The proposed ERF would be located within the Deeside Industrial Park, Flintshire. The Deeside Industrial Park lies within an area bounded by the River Dee to the south, the A548 to the west and north and the A494 to the East.

This report presents the detailed Human Health Risk Assessment (HHRA) modelling undertaken in relation to emissions from the stack serving the ERF.

1.1 Scope

The scope of the assessment encompasses emissions to air from the ERF stack. Certain compounds released in the ERF stack emissions may potentially affect human health via:

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

Direct exposure via inhalation has been considered within the Air Quality dispersion modelling assessment (Appendix H1_1 of this EP application), which compared predicted exposure to air quality limits, the purpose of which are to protect the population from the effects of direct inhalation. The assessment undertaken in accordance with H1 guidance, incorporating screening and detailed atmospheric dispersion modelling, predicts that environmental concentrations of combustion emissions from the facility will remain below the respective environmental assessment levels for the protection of human health and the environment.

The assessment presented in this document considers the impact of compounds of potential concern (COPCs) (as detailed in Industrial Emissions Directive implemented through The Environmental Permitting (England & Wales) Regulations 2012 that are 'persistent' in the environment and may lead to bioaccumulation that have several pathways from the point of release to the human receptor. The assessment presents a site-specific HHRA that considers multiple pathways as detailed above.

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

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

[1] Although the 'design point' is for 175,000tpa this maximum tonnage allows for variability in calorific value.

1.2 Objectives

The objective of the assessment is to assess the risk to human health as a result of emissions from the ERF, by consideration of multi-pathway uptake (i.e. inhalation and via the food chain) of all compounds of potential concern, with specific regard to the hypothetical worst case scenario, i.e. a farmer family at the point of maximum ground level exposure, as well as actual receptor locations within the vicinity of the site.

2.0 METHODOLOGY

The U.S. Environmental Protection Agency (US-EPA) Office of Solid Waste (OSW) has developed an approach for conducting multi-pathway, site-specific human health risk assessments for Advanced Thermal Treatment (ATT) facilities. The approach is known as the Human Health Risk Assessment Protocol (HHRAP)[2].

The HHRA methodology incorporates the following elements:

- Facility characterisation;
- Atmospheric dispersion modelling;
- Identification of exposure scenarios;
- Estimating media concentrations;
- Quantifying exposure; and
- Characterising Risk and Hazard.

Full details of the HHRAP methodology can be found in the HHRAP and updates available on the US-EPA website[3].

The computer model IRAP-h View (Industrial Risk Assessment Program-Health Version 4.0) used in this assessment has been designed to compute human health risk assessments following the requirements of the HHRAP (final, 2005).

NRW, whilst part of the Environment Agency, considered that the use of the HHRAP approach is acceptable, as described in Environmental Permitting Decision Documents for energy from waste operations[4].

> *'Dioxin Intake Models: Two models are available to predict the dioxin intake for comparison with the Tolerable Daily Intake (TDI) recommended by the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment, known as COT. These are HHRAP and the HMIP model.*
>
> *HHRAP has been developed by the US EPA to calculate the human body uptake of a range of carcinogenic pollutants and to determine the mathematic quantitative risk in probabilistic terms. In the UK, in common with other European Countries, we consider a threshold dose below which the likelihood of an adverse effect is regarded as being very low or effectively zero. The HMIP model uses a similar approach to the HHRAP model, but does not attempt to predict probabilistic risk. Either model can however be used to make comparisons with the TDI.'*
>
> *'In addition to an assessment of risk from dioxins and furans, the HHRAP model enables a risk assessment from human intake of a range of heavy metals. The HMIP report does not consider metals. In principle, the respective EQS for these metals are protective of human health. It is not therefore necessary to model the human body uptake.*

[2] United States Environmental Protection Agency - Office of Solid Waste and Emergency Response. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Final (September 2005)

[3] http://www.epa.gov/epawaste/hazard/tsd/td/combust/risk.htm (accessed 23/06/2010)

[4] Such as the for the AmeyCespa Limited, Milton Keynes. Permit Number: EPR/UP3937ZZ Decision document recording our decision-making process. 6/11/2013 and Pye Bridge, from which this quote is taken.

Our recommended approach is therefore the use of the H1 assessment methodology comparison for most pollutants (including metals) and dioxin intake models using either the HHRA or HMIP models as described above for dioxins and furans.'

Notwithstanding this, in order to provide the most comprehensive assessment of total uptake, metals have also been considered in the risk assessment.

2.1 Facility Characterisation

This is the initial stage of the HHRAP and involves collecting information regarding releases from the facility. Emissions from the ERF have previously been completed for undertaking the Air Quality Assessment. Important details include:

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

2.2 Atmospheric Dispersion Modelling

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

Site-specific characteristics input for air modelling include:

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

2.3 Identification of Exposure Scenarios

Identifying exposure scenarios consists of:

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

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

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

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

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

On the basis of these standardised scenarios exposure locations have been identified within the assessment area considering the land uses and receptor types present. In relation to the Parc Adfer facility it is considered that the exposure scenario which represents the potential worst case for ingestion is ‘farmer adult’ and ‘farmer child’.

2.4 Estimating Media Concentrations

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

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

The calculation of media concentrations requires such parameters as:

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

For all of these parameters the HHRAP default values have been used.

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

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

2.5 Quantifying Exposure

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

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

The information required includes the following:

- food (meat, dairy products, fish and vegetables), water and soil consumption rates for each receptor type;
- fraction of contaminated food, water and soil which is consumed by each receptor type;
- input data for the inhalation exposure including: inhalation exposure duration, inhalation exposure frequency, inhalation exposure time; and inhalation rate; and
- input data for the ingestion exposure including: exposure duration, exposure frequency, exposure time; and body weight of receptor.

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

2.6 Characterising Risk and Hazard

Risk characterisation is undertaken by comparing the exposure quantities against the toxicity benchmarks available in the HHRAP database to calculate the excess lifetime cancer risks and non-cancer hazard for each of the pathways and receptors. Risks (and hazards) are then summed for each receptor, across all applicable exposure pathways, to obtain an estimate of total individual risk and hazard.

Cancer Risk is the probability that a human receptor will develop cancer, based on a unique set of exposure, model, and toxicity assumptions. For example, a risk of $1x10^{-5}$ is interpreted to mean that an individual has up to a one in 100,000 chance of developing cancer during their lifetime (set at 70-years) from the evaluated exposure.

Hazard is a description of the potential for developing non-cancer health effects as a result of exposure to a COPC. A hazard is not a probability but rather a comparison (calculated as a ratio) of a receptor‘s potential exposure relative to a standard exposure level. The standard exposure level is calculated over a similar exposure period and is the level at which there is no appreciable likelihood of adverse health effects to potential receptors.

2.6.1 Quantitatively Estimating Cancer Risks

Cancer risk estimates represent the incremental probability that an individual will develop cancer over a lifetime as a result of a specific exposure to a carcinogenic chemical (Lifetime Cancer Risk). The risk is calculated as follows:

- inhalation Cancer Risk = Exposure concentration x Unit Risk Factor (URF)
- ingestion Cancer Risk = Lifetime average daily dose x Cancer Slope Factor (CSF)

Receptors may be exposed to a number of carcinogenic COPCs via a single pathway. Therefore the total cancer risk for a single pathway is calculated from the sum of cancer risks for each COPC. In addition a single receptor may be exposed via multiple pathways, therefore the cumulative cancer risk is calculated from the sum of total cancer risks for each pathway. The HHRAP companion database of URFs and CSFs embedded in the IRAP model were utilised for the assessment.

The UK Government[5] considers an excess lifetime cancer risk of 1 in 100,000 as appropriate in setting Health Criteria Values for carcinogenic soil contaminants based on a “minimal risk” approach. This is the default limit value within the IRAP model and has been accepted as an acceptable threshold by the Agency in previous assessments for waste incineration facilities[6].

2.6.2 Quantitatively Estimating Non-Cancer Hazards

Standard risk assessment models assume that, for most chemicals without cancer effects, the effects exhibit a threshold response. This means, there is a level of exposure below which no adverse effects will be observed. The default approaches used by the model to assess the potential for health effects associated with a non-linear or threshold relationship involve:

- comparing an estimate of ingested exposure to a Reference Dose (RfD) for oral exposure;
- comparing an estimated chemical-specific air concentration to the Reference Concentration (RfC) for direct inhalation exposures.

An RfD is a daily oral intake rate that is estimated to pose no appreciable risk of adverse health effects, even to sensitive population over a specific exposure duration (e.g. a 70 year lifetime). Similarly an RfC is an estimated daily concentration of a chemical in air, the exposure to which over a specific exposure duration poses no appreciable risk of adverse health effects, even to sensitive populations.

The assessment has applied Agency guidance documents (as described in Section 2.7) to derive RfDs and RfCs, respectively, for use in the IRAP model to evaluate all recommended exposure pathways. The comparisons (calculated as a ratio) of oral and inhalation exposure estimates to RfD and RfC values, described above, are known as hazard quotients (HQ). In this assessment the RfD and RfC values have been adjusted to take into account

[5] DEFRA, Guidance on the Legal Definition of Contaminated Land (July 2008)
[6] Rufford Energy Recovery Facility, Decision document recording the decision making process, Permit App BP3035MG.

background exposure (i.e. Mean Daily intakes for oral exposure and background concentrations for inhalation exposure), in this respect the HQ value represents a ratio of the available 'head-room' beneath the relevant RfD or RfC (Section 2.7 provides further details).

As with carcinogenic chemicals, a receptor might be exposed to multiple chemicals associated with non-cancer health effects through a single pathway. Therefore the total chronic hazard from each exposure pathway has been calculated by summing the HQ's, this is known as a hazard index (HI).

Each receptor's cumulative hazard has then been calculated as the sum of hazards from each individual COPC (i.e. the sum total of HI's). Whilst this is a simplification, it can be considered a screening approach i.e., if the total HI is not above the target hazard level, then no further investigation, i.e. segregation of the HI for each COPC, is necessary. The HHRAP states '*this is because the toxicological effects associated with exposure to multiple chemicals, often through different exposure pathways, may not be additive. The total HI might therefore overestimate the potential for non-cancer health effects'*.

2.7 Toxicity Factors for COPCs considered in the Assessment

The toxicity factors considered in the assessment are presented in Table 2-1 to Table 2-3. These are taken from Agency TOX reports and H1 guidance[7] where available (as indicated) or from the Hazardous Waste Companion Database[8] that is referenced in the HHRAP. To account for background levels, the RfD values and RfC values have been adjusted down on the basis of oral mean daily intakes (MDI) and background air concentrations respectively. Following Environment Agency guidance[9], in situations where the MDI exceeds the TDI, '*a minimum of 50% of the TDI is reserved for exposure from land'*.

Table 2-1
Toxicity Factors for Metals – RfD and RfC

COPC	Ingestion Reference Dose	Adjusted Ingestion Reference Dose - Adult	Adjusted Ingestion Reference Dose - Child	Inhalation Reference Conc.	Adjusted Inhalation Reference Conc.
Symbol	RfD	RfD	RfD	RfC	RfC
Units	mg/kg/d	mg/kg/d	mg/kg/d	mg/m^3	mg/m^3
Antimony	0.00040	US-EPA[f]	US-EPA[f]	0.0014	0.0014
Arsenic	0.00030[a]	0.00023	0.00015	0.0000030[e]	0.0000025
Cadmium	0.00036[b]	0.00018	0.00018	0.0000049[b]	0.0000048
Chromium (III)	1.5	1.4998	1.4996	5.3	5.3
Chromium (VI)	0.0030	0.0028	0.0026	2.0E-07[e]	1.6E-07
Lead	0.00043	US-EPA[f]	US-EPA[f]	0.0015	0.0015
Mercury (elemental)	8.57E-05	8.57E-05	8.57E-05	0.00030	0.0003
Mercury (inorganic)	0.002[c]	0.00199	0.00196	0.00021[c]	0.00021
Mercuric Chloride	0.0003	US-EPA[f]	US-EPA[f]	0.0011	0.0011
Methyl Mercury	0.00023[c]	0.00022	0.00021	0.00081[c]	0.00081

[7] Environment Agency, How to comply with your environmental permit Additional guidance for: Horizontal Guidance Note H1 - Annex (f) (April 2010)

[8] http://www.epa.gov/epawaste/hazard/tsd/td/combust/risk.htm

[9] Environment Agency, Human Health Toxicological Assessment of Contaminants in Soil (January 2009)

COPC	Ingestion Reference Dose	Adjusted Ingestion Reference Dose - Adult	Adjusted Ingestion Reference Dose - Child	Inhalation Reference Conc.	Adjusted Inhalation Reference Conc.
Nickel	0.012 (d)	0.0101	0.0072	0.000020 (e)	0.000019
Thallium (I)	0.00008	US-EPA (f)	US-EPA (f)	0.00028	0.00028

Table Notes:
a) Environment Agency, Contaminants in soil: updated collation of toxicological data and intake values for humans: Arsenic (May 2009)
b) Environment Agency, Contaminants in soil: updated collation of toxicological data and intake values for humans: Cadmium (July 2009)
c) Environment Agency, Contaminants in soil: updated collation of toxicological data and intake values for humans: Mercury (March 2009)
d) Environment Agency, Contaminants in soil: updated collation of toxicological data and intake values for humans: Nickel (May 2009)
e) Environment Agency, How to comply with your environmental permit Additional guidance for: Horizontal Guidance Note H1 - Annex (f) (April 2010)
f) In the absence of MDI values or guidance from UK Environment Agency the US-EPA approach and benchmarks have been used.

Table 2-2
Toxicity Factors for Metals – Carcinogenic Risk Factors

COPC	Ingestion Carcinogenic Slope Factor	Inhalation Unit Risk Factor
Symbol	Ing_CSF	Inh_URF
Units	$(mg/kg/d)^{-1}$	$(mg/m^3)^{-1}$
Antimony	No data	No data
Arsenic	1.5	0.0043
Cadmium	0.38	0.0018
Chromium III	No data	No data
Chromium VI	No data	0.012
Lead	0.0085	1.2E-05
Mercury (elemental)	No data	No data
Mercury (inorganic)	No data	No data
Mercuric Chloride	No data	No data
Methyl Mercury	No data	No data
Nickel	No data	2.40E-04
Thallium (I)	No data	No data

Table 2-3
Toxicity Factors for Dioxins and Furans

COPC	Ingestion Reference Dose (a)	Inhalation Carcinogenic Slope Factor	Ingestion Carcinogenic Slope Factor	Inhalation Unit Risk Factor
Symbol	RfD	Inh_CSF	Ing_CSF	Inh_URF
Units	mg/kg/d	$(mg/kg/d)^{-1}$	$(mg/kg/d)^{-1}$	$(\mu g/m^3)^{-1}$
TetraCDD, 2,3,7,8-	1.3E-09 (b)	150000	150000	No data
PentaCDD, 1,2,3,7,8-	No data	75000	150000	No data
HexaCDD, 1,2,3,4,7,8-	No data	15000	15000	No data
HexaCDD, 1,2,3,6,7,8-	No data	15000	6200	1.3

COPC	Ingestion Reference Dose [(a)]	Inhalation Carcinogenic Slope Factor	Ingestion Carcinogenic Slope Factor	Inhalation Unit Risk Factor
HexaCDD, 1,2,3,7,8,9-	No data	15000	6200	1.3
HeptaCDD, 1,2,3,4,6,7,8-	No data	1500	1500	No data
OctaCDF, 1,2,3,4,6,7,8,9-	No data	150	15	No data
TetraCDF, 2,3,7,8-	No data	150	15	No data
TetraCDF, 2,3,7,8-	No data	15000	15000	No data
PentaCDF, 2,3,7,8-	No data	7500	7500	No data
PentaCDF, 2,3,4,7,8-	No data	75000	75000	No data
HexaCDF, 1,2,3,4,7,8-	No data	15000	15000	No data
HexaCDF, 1,2,3,6,7,8-	No data	15000	15000	No data

Table Note:
a) The majority of Dioxins and Furans do not have specific toxicity factors, therefore at each receptor location all Dioxins and Furans are summed and assessed using the criteria for 2,3,7,8- TetraCDD.
b) Environment Agency, Contaminants in soil: updated collation of toxicological data and intake values for humans: Dioxins, furans and dioxin-like PCBs in soil Science report: SC050021/TOX 12 (September 2009). On this basis 1.3E-09 has been applied as the RfD for adults and 1.0E-09 for children to account for the mean daily intake.

2.8 Evaluation of infant exposure via breast milk to Dioxins and Furans

The ingestion of mother's breast milk by infants is identified as an exposure pathway to PCDDs and PCDFs warranting separate evaluation in the HHRAP. The potential for contamination of breast milk is high for dioxin-like compounds such as these, as they are highly lipophilic (fat soluble) and hence likely to accumulate in breast milk. This exposure is measured by the Average Daily Dose (ADD) on the basis of an averaging time of 1 year. In the UK the Committee on Toxicity have recommended[10] a Tolerable Daily Intake level of 2 pg I-TEQ/kg BW per day, this has been carried forward into the Agency CLEA framework[11].

2.9 PCBs and PAH

The view of the national regulators in relation to HHARP assessment of PCB (polychlorinated biphenyl) and Polycyclic aromatic hydrocarbons (PAHs) is as follows:

The release of dioxins and furans to air is required by the IED to be assessed against the I-TEQ (International Toxic Equivalence) limit of 0.1 ng/m^3. Further development of the understanding of the harm caused by dioxins has resulted in the World Health Organisation (WHO) producing updated factors to calculate the WHO-TEQ value. Certain PCBs have structures which make them behave like dioxins (dioxin-like PCBs), and these also have toxic equivalence factors defined by WHO to make them capable of being considered together with dioxins. The UK's independent health advisory committee, the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) has adopted WHO-TEQ values for both dioxins and dioxin-like PCBs in their review of Tolerable Daily Intake (TDI) criteria. EPR requires that, in addition to the requirements of the IED, the WHO-TEQ values for both dioxins and dioxin-like PCBs should be specified for monitoring and reporting purposes, to enable evaluation of exposure to dioxins and dioxin-like PCBs to be made using the revised TDI recommended by COT. The release of dioxin-like PCBs and PAHs is expected to be low where measures have been taken to control dioxin releases. We require monitoring of a range of PAHs and dioxin-like PCBs in waste incineration Permits at the same frequency as dioxins

[10] Committee On Toxicity Of Chemicals In Food, Consumer Products and The Environment, 'COT statement on 2005 WHO toxic equivalency factors for dioxins and dioxin-like compounds' (2005)
[11] Environment Agency, Contaminants in soil: updated collation of toxicological data and intake values for humans: Dioxins, furans and dioxin-like PCBs in soil (September 2009)

are monitored. We have included a requirement to monitor and report against these WHO-TEQ values for dioxins and dioxin-like PCBs and the range of PAHs identified by Defra in the Environmental Permitting Guidance on the IED. We are confident that the measures taken to control the release of dioxins will also control the releases of dioxin-like PCBs and PAHs.

3.0 FACILITY AND EMISSION CHARACTERISATION

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

3.1 Abatement of POPs

The 1998 Protocol to the Stockholm Convention recommended that unintentionally produced Persistent Organic Pollutants (POPs) should be controlled by imposing emission limits (e.g. 0.1 ng/m^3 for facilities such as the Parc Adfer ERF) and using BAT for incineration. UN Economic Commission for Europe (Executive Body for the Convention) (ECE-EB) produced BAT guidance for the parties to the Convention in 2009.

Successful control techniques for waste incinerators listed in the ECE-EB BAT are:

- maintaining furnace temperature of 850^{o}C and a combustion gas residence time of at least 2 seconds
- rapid cooling of flue gases to avoid the de novo reformation temperature range of 250-450^{o}C
- use of bag filters and the injection of activated carbon or coke to adsorb residual POPs components.

Using the methods listed above, the UN-ECE BAT document concludes that incinerators can achieve an emission concentration of 0.1 ng TEQ/m^3.

3.2 Emission Source Process Conditions

The emission characteristics applied in the dispersion model and process conditions used to determine the pollutant emission rates used in the dispersion modelling process are presented in Table 3-1.

Table 3-1
Stack Emission Characteristics

Parameter	Value
Stack Diameter (m)	2.3
Stack Location (X,Y)	331093.4, 371418.0
Stack Height (m AoD)	85.0
Volume Flow (a) (Nm3/s) (273K, 11%, dry)	37.6
Emission Temperature (a) (ºC)	140.0
Oxygen Content (a) (% O2 in dry gas)	10.1
Moisture content (a) (% H2O)	18.14
Actual Flow Rate (Am3/s)	63.8
Emission velocity (m/s)	15.35

A typical ERF line requires a minimum of 2-weeks shutdown for maintenance per year (over 2 periods), therefore operational hours would never be in excess of 8256 hours and ERF's in the UK typically achieve between 7800 and 8200 operational hours per annum.

The ERF has been modelled as emitting continuously throughout the year. Therefore on this basis, it can be concluded that actual long-term (annual) emissions, (and resultant impacts) are likely to between 6.4% and 11.0% lower than modelled for this assessment.

3.3 Emissions of COPCs

For substances that are of sufficient concern with regard to human health and potentially persistent in the environment the IED sets out limit values for emissions to air as detailed in the Table 3-2. Therefore the substances that have been included in this assessment are those for which emission limits are defined (and which are included in the EPA HHRAP COPC database for the assessment of long term health effects). These emission limits would typically be set as Environmental Permit conditions by NRW as part of the permitting process.

Table 3-2
Emission Limit Values

Pollutant	Emission Limits (mg/Nm^3) [(a)]
Group 1 metals cadmium (Cd) and thallium (Tl)	0.05
Group 2 metals - mercury (Hg)	0.05
Group 3 metals - antimony (Sb), arsenic (As), lead (Pb), chromium (Cr), cobalt (Co), copper (Cu), manganese (Mn), nickel (Ni), and vanadium (V).	0.5
Dioxins and furans [(b)]	0.0000001

Notes:
(a) Concentrations referenced to temperature 273 K, pressure 101.3 kPa, 11% oxygen, dry gas.
(b) The emission limit value refers to the total concentration of dioxins and furans calculated using the concept of toxic equivalence (TEQ).

Some of the metals for which emission limits are defined in the IED are excluded from this assessment on the grounds that they pose little or no hazard in the context of long term health impacts, and as such are not included in the EPA HHRAP COPC database or Environment Agency / NRW risk framework[12]; these are cobalt, copper, manganese and vanadium. Therefore, the following substances have been considered as COPCs for the proposed ERF:

- Dioxins and furans (PCDD/Fs individual congeners);
- Antimony (Sb);
- Arsenic (As);
- Cadmium (Cd);
- Chromium (Cr), trivalent and hexavalent;
- Mercury (Hg);
- Thallium (Tl);
- Lead (Pb); and
- Nickel (Ni).

[12] These were excluded from the list of potentially significant pollutants in CLR 8: Potential Contaminants for the Assessment of Land Contamination (Environment Agency 2002 now withdrawn) and follows through in the current CLEA framework.

3.3.1 COPC Specific Emission Details

Dioxins

The emission rate of dioxins from the Installation has been determined on the basis of the ERF process lines operating continuously (typical actual operating hours are approximately 8000 per year) at the IED and EP emission limit for dioxins of 0.1 WHO-TEF ng/m[3, 13].

As each specific congener has different physico-chemical properties, congener-specific emission data are required. The congener profile will be dependent on various factors including the type of waste being burnt, the temperature of combustion, the type of combustion chamber being operated and the air pollution control devices (APCDs) installed. A summary of the relative contribution of each of the 17 congeners from site-specific and published data is presented in Appendix A.

From this it is evident that whilst there is considerable variation in the congener profile between both published data, general trends are apparent across datasets. The most complete dataset available is from measurements undertaken on behalf of the USEPA in 2000, which included congener profiles for over 150 large facilities (defined as over 250 tonnes per day treating over 28 million tonnes of MSW per annum). The USEPA derived congener split[14] has been applied in this assessment given the number of datasets which support this data.

Toxic Equivalence Factors

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

The WHO European Centre for Environment and Health and the International Program on Chemical Safety have developed a set of criteria for TEF calculations for the relevant dioxin and furan congeners (Van den Berg et al., 2006)[15]. These TEFs have since been endorsed by the COT for use in UK assessments of dioxin exposure[16]. The potential dioxin-like activity contributed by each congener is determined by multiplying the concentration of the congener by its WHO-designated TEF to yield the dioxin toxic equivalent (TEQ) for that congener. The

[13] The IED requires dioxins to be reported using the I-TEQ reporting convention to assess compliance against an emission limit of 0.1ng I-TEQ / Nm3. The UK's independent health advisory committee, Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT), has adopted the World Health Authority (WHO) toxicity equivalence factors (TEF) for both dioxins and dioxin-like PCBs in their recent review of Tolerable Daily Intake (TDI) criteria.

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

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

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

net TEQ is the sum of the individual TEQs for each dioxin or dioxin-like compound. WHO TEFs for dioxins and furans are detailed in Table 3-3 below.

Table 3-3
WHO TEFs for Dioxins (Van den Berg et al, 2006)

	Congener	WHO TEF
Dioxins (PCDDs)	2,3,7,8-TCDD	1
	1,2,3,7,8-PeCDD	1
	1,2,3,4,7,8-HxCDD	0.1
	1,2,3,7,8,9-HxCDD	0.1
	1,2,3,6,7,8-HxCDD	0.1
	1,2,3,4,6,7,8-HpCDD	0.01
	OCDD	0.0003
Furans (PCDFs)	2,3,7,8-TCDF	0.1
	1,2,3,7,8-PeCDF	0.03
	2,3,4,7,8-PeCDF	0.3
	1,2,3,4,7,8-HxCDF	0.1
	1,2,3,7,8,9-HxCDF	0.1
	1,2,3,6,7,8-HxCDF	0.1
	2,3,4,6,7,8-HxCDF	0.1
	1,2,3,4,6,7,8-HpCDF	0.01
	1,2,3,4,7,8,9-HpCDF	0.01
	OCDF	0.0003

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

Table 3-4
Applied Congener Emission Rates

Compound	Emission Concentration (ng/Nm^3)		Emission Rate (ng/s)
	Measured (factored)[(a)]	WHO-TEF[(b)]	
2,3,7,8 –TCCD	0.00588	0.0059	0.2
1,2,3,7,8–PeCDD	0.01882	0.0188	0.7
1,2,3,4,7,8–Hx CDD	0.01882	0.0019	0.7
1,2,3,6,7,8–HxCDD	0.04352	0.0044	1.6
1,2,3,7,8,9–HxCCD	0.03764	0.0038	1.4
1,2,3,4,6,7,8–HpCCD	0.25758	0.0026	9.7
OCCD	0.40578	0.0001	15.3
2,3,7,8-TCDF	0.08469	0.0085	3.2
1,2,3,7,8-PeCDF	0.05881	0.0018	2.2
2,3,4,7,8-PeCDF	0.08116	0.0243	3.1
1,2,3,4,7,8-HxCDF	0.09645	0.0096	3.6
1,2,3,6,7,8-HxCDF	0.06940	0.0069	2.6
1,2,3,7,8,9-HxCDF	0.01529	0.0015	0.6
2,3,4,6,7,8-HxCDF	0.07763	0.0078	2.9
1,2,3,4,6,7,8-HpCDF	0.18349	0.0018	6.9

1,2,3,4,7,8,9-HpCDF	0.02823	0.0003	1.1
OCDF	0.10586	<0.0001	4.0
TOTAL	**1.58903**	**0.1000**	**59.8**

Table Note:
a) US-EPA measured concentrations factored to WHO Toxic Equivalency Factors.
b) US-EPA TEF Factored emission factored to meet emission limit of 0.1ng(TEF)/m^3

Metals

As shown in Table 3-2, the IED emission limits for metals are based on total emission rates for 3 different groups of compounds. A review of public records data from operational EfW facilities in the UK indicates that the typical emission rate of each metal within Group 1 is less than 10% of the IED emission limit, therefore the emission rate for Group 1 Metals has been divided by 2 (i.e. each metal at 50% of the IED emission limit for the group). Similarly, the typical emission rate of each metal within Group 3 is less than 30% of the IED emission limit, therefore the emission rate for Group 3 Metals has been divided by 9 (i.e. each metal at 11.1% of the IED emission limit for the group).

Hexavalent Chromium

In relation to chromium, it is important to note that different EALs apply depending on the oxidation state of chromium. The EPAQS recommended annual mean limit of 0.2ηg/m^3 relates specifically to chromium (VI) (i.e. hexavalent chromium), with the long-term EAL of 5µg/m^3 applying to all other oxidation states of chromium.

Annex B of the EA (2012) *Releases from municipal waste incinerators: Guidance to applicants on impact assessment for group 3 metals stack*. [sic] September 2012, version 3 provides chromium VI analysis data from 10 EfWs in the UK. The data shows a maximum emission concentration of $1.3*10^{-4}$ mg/m^3 although an extremely precautionary $3*10^{-4}$ mg/m^3 has been used in this assessment

3.4 Deposition Modelling – Metals and Dioxins

In order to inform the assessment of potential impacts on human health, the air dispersion model has been used to provide deposition rates of metals and dioxins. In the absence of suitable UK guidance, this has been primarily undertaken based on guidance[17] issued by the United States Environmental Protection Agency (USEPA) as described in the following sections.

3.4.1 Assignment of Phase

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

Guidance indicates that in general it can be assumed that:

- Most metals and organic pollutants with very low volatility (i.e. fraction of the pollutant in the vapour phase is less than 0.05) occur only in the particle phase;

[17] USEPA, Office of Solid Waste and Emergency Response, Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, FINAL September 2005. Chapter 3: Air Dispersion and Deposition Modelling.

- Highly volatile organic pollutants occur only in the vapour phase (i.e. the fraction of the pollutant in the vapour phase is 1.0);and
- The remaining pollutants are condensed onto the surface of particulate matter (particle-bound).

The fraction of the identified pollutants in the vapour phase, and the assigned phase for the ERF stack dispersion modelling, are presented in Table 3-5.

Table 3-5
Assigned Phases for Metals and Dioxins

	Pollutant	Fraction in Vapour Phase (Fv) [(a)]	Assigned phase
Group 1 Metal	Cadmium	0.009	Particle
	Thallium	0.009	Particle
Group 2	Mercury	1	Particle-bound & vapour[(b)]
Group 3 Metals	Antimony	1[(c)]	Particle-bound
	Arsenic	0.006	Particle
	Lead	0.007	Particle
	Chromium	0.009	Particle
	Nickel	0.008	Particle
	Vanadium	No data available	Particle
Dioxins and Furans	2,3,7,8–TCCD	0.664	Vapour
	1,2,3,7,8–PeCDD	0.117	Particle-bound
	1,2,3,4,7,8-HxCDD	0.024	Particle
	1,2,3,6,7,8–HxCDD	0.029	Particle
	1,2,3,7,8,9–HxCCD	0.016	Particle
	1,2,3,4,6,7,8–HpCCD	0.003	Particle
	OCCD	0.002	Particle
	2,3,7,8-TCDF	0.77	Vapour
	1,2,3,7,8-PeCDF	0.268	Particle-bound
	2,3,4,7,8-PeCDF	0.221	Particle-bound
	1,2,3,4,7,8-HxCDF	0.049	Particle
	1,2,3,6,7,8-HxCDF	0.052	Particle
	1,2,3,7,8,9-HxCDF	0.09	Particle
	2,3,4,6,7,8-HxCDF	0.058	Particle
	1,2,3,4,6,7,8-HpCDF	0.01	Particle
	1,2,3,4,7,8,9-HpCDF	0.057	Particle
	OCDF	0.002	Particle

Table Notes:

a) Data from the HHRAP companion database as detailed in Appendix A of the USEPA, OSW, Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, FINAL September 2005.
b) The classification of emission of Mercury is discussed further in the following section on metal deposition.
c) Despite a published vapour fraction of 1, Antimony has a melting point of over 600ºC and therefore has been assigned to the particle-bound category.

3.4.2 Particulate Deposition

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

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

For this assessment, as data relating to particle size and density is limited, the Method 2 approach has been applied using published data[18] relating to particle size distribution for individual pollutants as shown in Table 3-6.

Dioxin Deposition Modelling from ERF

As shown in Table 3-5, for the purposes of this assessment all dioxins (and furans) have been assumed to be particle (or particle-bound) with the exception of 2,3,7,8-TCCD and 2,3,7,8-TCDF which have been assumed to be vapour phase (gaseous). The applied congener split is as detailed previously in Table 3-4.

Deposition of Metals

As shown in Table 3-5, for the purposes of this assessment all metals have been assumed to be particle (or particle-bound) with the exception of mercury.

In relation to stack emissions of mercury, these will comprise both vapour and particle-bound forms of mercury, speciated as both elemental and divalent mercury. USEPA guidance[19] indicates that for the purposes of assessments such as these it is to be assumed that 80% of emission of mercury will be present as vapour, and 20% as particle-bound. In terms of speciation, the vapour is considered to be three-quarters divalent and one-quarter elemental mercury and the particle-bound mercury to be entirely divalent.

In accordance with the HHRA Protocol mercury loss to the global cycle has been incorporated (using HHRAP default values).

Table 3-6
Assigned Deposition Parameters for Particulates

Compound	Fine Mass Fraction	Mean Particle Diameter
Cadmium	0.7	0.6
Thallium	0.7	0.6
Antimony	0.6	1
Arsenic	0.75	0.5

[18] Deposition Parameterizations for the Industrial Source Complex (ISC3) Model. Environmental Research Division, Argonne National Laboratory on behalf of US Department of Energy, June 2002.
[19] USEPA, Office of Solid Waste and Emergency Response, Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, FINAL September 2005. Chapter 2: Facility Characterization.

Chromium	0.55	1.2
Lead	0.75	0.5
Nickel	0.6	1
Vanadium	0.6	1
Hg(0) particulate	0.8	0.4
Dioxins (particulate)	0.9	0.1

3.4.3 Vapour Deposition

Vapour phase compounds are deposited via both wet and dry processes, dependent on factors relating to their solubility etc. and not by particle size, mass or surface area. Published data[20] relating to solubility etc has been applied to individual pollutants as shown in the following Table.

Table 3-7
Assigned Deposition Parameters for Vapours

Compound	Diffusivity in air (cm^2/s)	Diffusivity in water (cm^2/s)	Cuticular resistance (s/cm)	Henry's constant (Pa m^3/mol)
Hg(2+)	0.06	0.00032	10000000	0.000006
Hg(0)	0.07	0.0000063	10000000	150
2,3,7,8 -TCCD	0.05196	0.000004392	7.84	3.34
2,3,7,8-TCDF	0.05269	0.000004544	7.84	1.46

[20] Deposition Parameterizations for the Industrial Source Complex (ISC3) Model. Environmental Research Division, Argonne National Laboratory on behalf of US Department of Energy, June 2002.

4.0 ATMOSPHERIC DISPERSION MODELLING

The atmospheric dispersion modelling for the HHRA has been completed using the AERMOD model[21]. The AERMOD dispersion modelling program is widely used and accepted by NRW in the UK for undertaking such assessments and its predictions have been validated against real-time monitoring data by the USEPA[22]. It is therefore considered a suitable model for this assessment. All details relevant to the use of the dispersion model, such as meteorology and topography are described in the detailed dispersion modelling report for the Parc Adfer facility (Appendix H1_1).

4.1 Modelling Scenarios

For the purposes of the dispersion modelling of emissions (i.e. process contribution) from the ERF one scenario has been defined.

In order to predict a realistic 'worst case' scenario, the proposed ERF has been assumed to be in operation continuously throughout the year, and to have pollutant emission rates at the daily average emission limits permitted by the IED. This ensures that the maximum possible impacts on air quality are predicted with the facility in operation within its authorisation limits. In reality operational hours will typically be between 7800 and 8200 per year and emissions will be below IED limits.

4.2 Assessment Area

The detailed dispersion modelling report describes that the potential air quality impact of the proposed plant was assessed over an area of 10km radius from the Application site. The receptor grid spacing used was: 100m across a 5km radius circular grid and 200m across the 5km – 10km radius (13696 data points).

From the dispersion modelling results, 5 receptor locations were chosen for this HHRA assessment as described in section 5.3.

4.3 Model Output

The AERMOD dispersion model has been used to produce the following outputs (as an annual average based on 5 years of meteorological data):

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

[21] Software used: BREEZE AERMOD GIS Pro, v7.
22 AERMOD: Latest Features and Evaluation Results. USEPA Report: EPA-454/R-03-003 June 2003, (http://www.epa.gov/scram001/dispersion_prefrec.htm#aermod)

5.0 EXPOSURE SCENARIOS

5.1 Site and Surroundings

The proposed application site is remote from any large areas of residential development or other sensitive uses, such as schools, hospitals and care homes; it is also similarly remote from any individual dwellings.

The nearest residential areas are located in Connah's Quay more than 2km away to the south (in the vicinity of the B5129); Garden City (off Sealand Avenue) to the southeast. The villages of Puddington and Burton, which are within England, are also more than 2km away to the northeast and north respectively. Examination of aerial photography and OS mapping show individual properties lying to the south of Burton, in the vicinity of Burton Mere Fisheries, which are around 1.7km from the northern boundary of the proposed application site.

5.2 Assessment Exposure Pathways

On the basis of all the potential exposure scenarios as defined in Section 2.3 the following specific exposure scenarios have been identified as of relevance to this HHRA, and hence, subject to examination in detail are as follows (and presented in Figure 1):

- inhalation;
- ingestion of food (including breast milk); and
- ingestion of soil.

Exposure pathways are determined by the diet of the receptor and the proportion of which is local produce. For example a residential receptor is unlikely to habitually ingest home-reared pork compared to a farmer. However a residential receptor is reasonably likely to ingest home-grown vegetables. The exposures scenarios assessed arising from ingestion are summarised in Table 5-1.

The consumption of fish from local water bodies, as a result of recreational fishing, is likely to be very small (i.e. does not form a regular supplement in the diet). The waters of the Dees Estuary are tidal and bioaccumulation as a result of emissions from the ERF will not be a significant issue due to the regular refreshing of the aquatic environment.

The local population does not obtain its drinking water from local surface water sources. The local population has treated drinking water provided by water companies. Therefore ingestion of water as a pathway has been excluded.

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

Table 5-1
Ingestion Exposure by Receptor Type

Ingestion of:	Resident Adult	Resident Child	Farmer Adult	Farmer Child	Fisher Adult	Fisher Child
Milk From Home-Reared Cows	X	X	✓	✓	X	X
Eggs From Home-Reared Chickens	X	X	✓	✓	X	X
Home-Reared Beef	X	X	✓	✓	X	X

Ingestion of:	Resident Adult	Resident Child	Farmer Adult	Farmer Child	Fisher Adult	Fisher Child
Home-Reared Pork	X	X	✓	✓	X	X
Home-Reared Chicken	X	X	✓	✓	X	X
Home-Grown Vegetable/Fruit	✓	✓	✓	✓	✓	✓
Local Fish	X	X	X	X	✓	✓
Breastmilk	X	✓	X	✓	X	✓
Soil (Incidental)	✓	✓	✓	✓	✓	✓

Only the results for farmer adult and farmer child have been presented here as these are the pathways with the highest exposure potential.

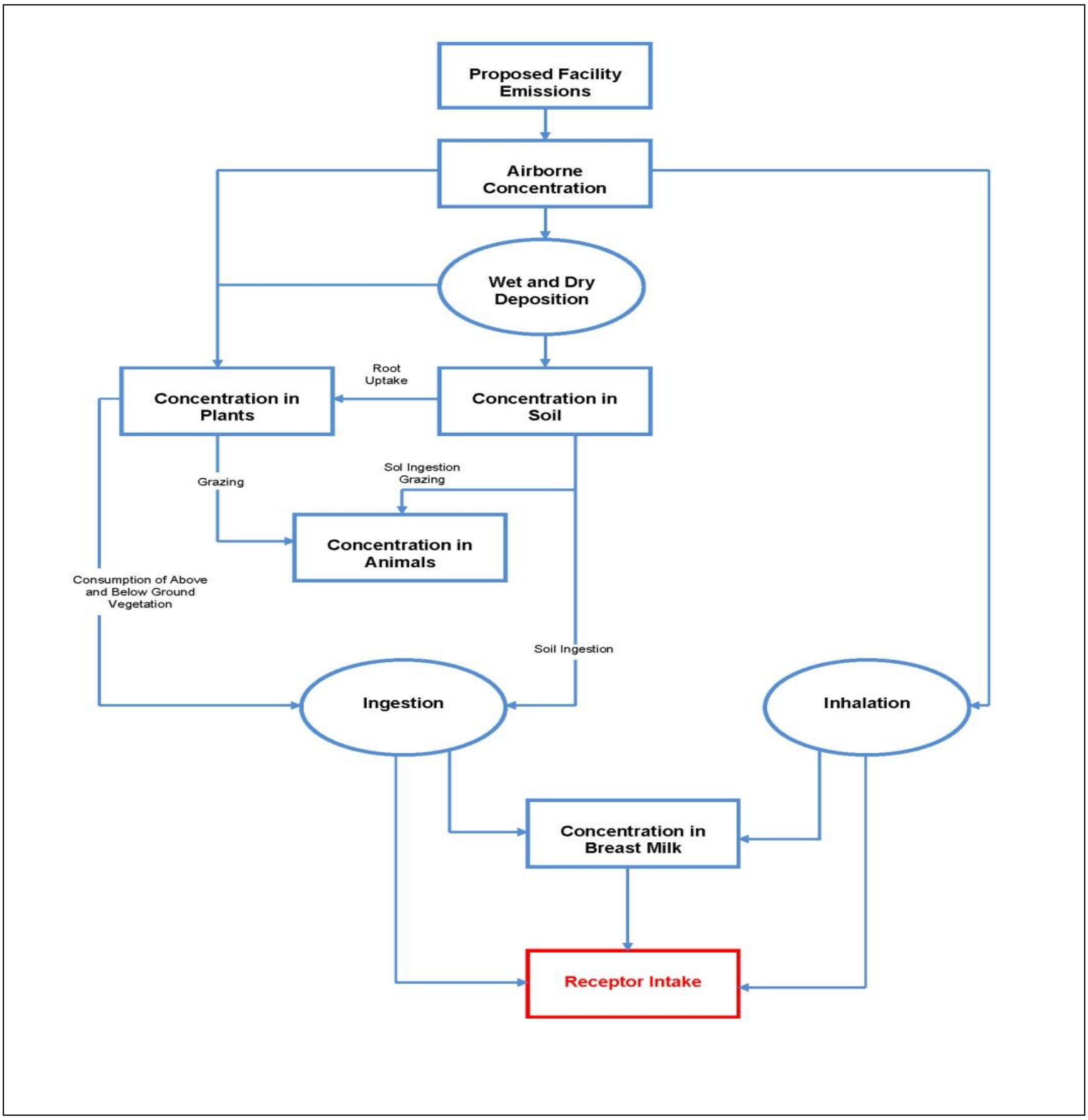

Figure 1: Exposure Scenario

5.3 Identification of Sensitive Receptor Locations

As described above, the detailed dispersion modelling report describes that the potential air quality impact of the proposed plant was assessed over an area of 10km radius from the Application site. The receptor grid spacing used was: 100m across a 5km radius circular grid and 200m across the 5km – 10km radius (13696 data points).

From the dispersion modelling results, 5 receptor locations were chosen for this HHRA assessment. The HHRA considers a hypothetical worst case scenario i.e., a Farmer receptor (child and adult) is present at the point of maximum (long term average) impact of the ERF emissions.

Table 5-2
Assessed Sensitive Receptor Locations

	Type	Receptor Description	Location (NGR) X	Location (NGR) Y
1	Farmer	Maximum GLC	331700	372000
2	Farmer	2nd Highest GLC	331600	372000
3	Farmer	3rd Highest GLC	331000	372300
4	Farmer	4th Highest GLC	331600	372100
5	Farmer	Maximum impact at Dees Estuary	330833.8	372326.6

5.4 Site Parameters for Estimating Media Concentrations

These parameters were specified for the study area as follows:

- annual average precipitation of 80 cm/annum (Met office 1980-2010 average for Colwyn Bay);
- annual average evapo-transpiration rate of 40 cm/annum (MAFF);
- annual average irrigation of 0 cm/annum (assumed not to be significant);
- annual average runoff of 20 cm/annum;
- an annual average wind velocity of 3.9m/s (from NWP Met Station);
- a time period over which deposition occurs of 30 years; and
- exposure period for adult of 70 years.

6.0 ASSESSMENT OF HAZARD AND RISK

The Total Cancer Risk, cumulative Hazard Index, dioxin/furan exposure, infant breast milk exposure, and lead exposure for the selected receptors are presented in the sections below, in accordance with the HHRAP.

6.1 Summary of Non-Carcinogenic Effects

The cumulative Hazard Index (HI) calculated by IRAP for emissions from the ERF for each of the receptors (adult and child) is presented in Table 6-1 below. The HI is the sum of all hazard quotients.

Table 6-1
Cumulative Hazard Index for all Receptors

No.	Type	Total Hazard Index
1	Farmer Adult	0.078
	Farmer Child	0.082
2	Farmer Adult	0.078
	Farmer Child	0.082
3	Farmer Adult	0.078
	Farmer Child	0.081
4	Farmer Adult	0.077
	Farmer Child	0.081
5	Farmer Adult	0.074
	Farmer Child	0.078

For the hypothetical worst case exposure scenario (i.e. this receptor does not exist), the total HI is 0.08 for a Farmer Child at the point of maximum deposition (Receptor 1). This is significantly below the unity (1.0).

6.2 Summary of Carcinogenic Effects

The total lifetime risk calculated by IRAP for emissions from the ERF Plant for each of the receptors (adult and child) is presented in Table 6-2.

Table 6-2
Total Lifetime Cancer Risk

No.	Type	Total Lifetime Cancer Risk
1	Farmer Adult	$1.20E^{-06}$
	Farmer Child	$2.41E^{-07}$
2	Farmer Adult	$1.20E^{-06}$
	Farmer Child	$2.40E^{-07}$
3	Farmer Adult	$1.18E^{-06}$
	Farmer Child	$2.38E^{-07}$
4	Farmer Adult	$1.19E^{-06}$
	Farmer Child	$2.36E^{-07}$
5	Farmer Adult	$1.13E^{-06}$
	Farmer Child	$2.26E^{-07}$

The total lifetime cancer risk is significantly less than the Government level of $1x10^{-5}$ (i.e. 1 in 100,000) considered to represent 'minimal risk' at all receptor locations.

The hypothetical worst case exposure scenario (i.e. this receptor does not exist), the Total Cancer Lifetime Risk is $1.2x10^{-6}$ (or 1.2 in a million) for a Farmer Adult at the point of maximum deposition (Receptor 1).

As the level of risk at all receptors is below 1 in 100,000 the potential impact of the Proposed development on Cancer Risk can be considered below the DEFRA minimal risk level.

6.3 Summary of Exposure to Dioxins and Furans

The Total Hazard Quotient for all dioxins and furans at each receptor is presented in Table 6-3 below. The model predicts that the increase in intake as a result of the process contribution is extremely small at 0.13% of mean daily intake (MDI). On this basis all receptors are well below the COT limit value of 2 pg I-TEQ/kg BW/day.

Table 6-3
Dioxin and Furan Hazard Index and Daily Intake

No.	Type	Process Contribution Intake (pg I-TEQ/kg(BW)/day)	% of MDI[(a)]	Hazard Quotient[(b)]
1	Farmer Adult	$9.3E^{-04}$	0.13%	$7.1E^{-04}$
	Farmer Child	$1.0E^{-03}$	0.10%	$1.0E^{-03}$
2	Farmer Adult	$9.3E^{-04}$	0.13%	$7.1E^{-04}$
	Farmer Child	$1.0E^{-03}$	0.10%	$1.0E^{-03}$
3	Farmer Adult	$9.2E^{-04}$	0.13%	$7.1E^{-04}$
	Farmer Child	$1.0E^{-03}$	0.10%	$1.0E^{-03}$
4	Farmer Adult	$9.1E^{-04}$	0.13%	$7.0E^{-04}$
	Farmer Child	$1.0E^{-03}$	0.10%	$1.0E^{-03}$
5	Farmer Adult	$8.8E^{-04}$	0.12%	$6.7E^{-04}$
	Farmer Child	$9.7E^{-04}$	0.10%	$9.7E^{-04}$

Table Note:
a) The MDI for dioxin is 0.7(Pg I-TEQ/kg(BW)/day) for adults and 1.0 (Pg I-TEQ/kg(BW)/day) for children
b)The limit value (RfD) for dioxin applied to calculate the Hazard Quotient is 1.3(Pg I-TEQ/kg(BW)/day) for adults and 1.0 (Pg I-TEQ/kg(BW)/day) for children.

6.4 Infant Breast Milk Exposure to Dioxins and Furans

The IRAP model calculates the Average Daily Dose (ADD) that would result from an adult receptor breast feeding an infant; a summary of the ADD (total sum of all PCDDs and PCDFs) for each of the infants of adult receptors considered for the assessment is presented in Table 6-4.

Table 6-4
Assessment of Infant ADD to Dioxins and Furans via Breast Milk

No.	Infant ADD [pg I-TEQ/kg BW/day]
1	0.25
2	0.25
3	0.25
4	0.25

No.	Infant ADD [pg I-TEQ/kg BW/day]
5	0.23

The values for all receptors are well below the COT limit value of 2 pg I-TEQ/kg BW/day.

6.5 Summary of Hazard and Risk

The findings of the assessment are that the predicted risks and hazards as a consequence of emissions from the proposed ERF plant are all within limits for the protection of human health as defined by the Environment Agency, NRW or US-EPA.

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

7.0 CLOSURE

This report has been prepared by SLR Consulting Limited with all reasonable skill, care and diligence, and taking account of the manpower and resources devoted to it by agreement with the client. Information reported herein is based on the interpretation of data collected and has been accepted in good faith as being accurate and valid.

This report is for the exclusive use of WTI UK Limited; no warranties or guarantees are expressed or should be inferred by any third parties. This report may not be relied upon by other parties without written consent from SLR.

SLR disclaims any responsibility to the client and others in respect of any matters outside the agreed scope of the work.

APPENDIX A

Congener Split Review

Table A-1
Congener Partitioning (% Contribution)

Source	**USEPA (Table 3-4)[23]**		**HMIP Table 7.2a[24].**	
Congener	Measured (ng/m^3)	% contribution	Measured (ng/m^3)	% contribution
2,3,7,8 –TCCD	0.0050	0.37%	0.0310	0.15%
1,2,3,7,8–PeCDD	0.0160	1.18%	0.2450	1.22%
1,2,3,4,7,8–Hx CDD	0.0160	1.18%	0.2870	1.42%
1,2,3,6,7,8–HxCDD	0.0370	2.74%	0.2580	1.28%
1,2,3,7,8,9–HxCCD	0.0320	2.37%	0.2050	1.02%
1,2,3,4,6,7,8–HpCCD	0.2190	16.21%	1.7040	8.46%
OCCD	0.3450	25.54%	4.0420	20.06%
2,3,7,8-TCDF	0.0720	5.33%	0.2770	1.37%
1,2,3,7,8-PeCDF	0.0500	3.70%	0.2770	1.37%
2,3,4,7,8-PeCDF	0.0690	5.11%	0.5350	2.66%
1,2,3,4,7,8-HxCDF	0.0820	6.07%	2.1790	10.81%
1,2,3,6,7,8-HxCDF	0.0590	4.37%	0.8070	4.00%
1,2,3,7,8,9-HxCDF	0.0130	0.96%	0.0420	0.21%
2,3,4,6,7,8-HxCDF	0.0660	4.89%	0.8710	4.32%
1,2,3,4,6,7,8-HpCDF	0.1560	11.55%	4.3950	21.81%
1,2,3,4,7,8,9-HpCDF	0.0240	1.78%	0.4290	2.13%
OCDF	0.0900	6.66%	3.5660	17.70%

[23] U.S. EPA (Environmental Protection Agency). (2006) An inventory of sources and environmental releases of dioxin-like compounds in the United States for the years 1987, 1995, and 2000. National Center for Environmental Assessment, Washington, DC; EPA/600/P-03/002F. Available online at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=159286 .
[24] Her Majesty Inspectorate of Pollution (HMIP), 1996. Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes

global environmental solutions

AYLESBURY
7 Wornal Park, Menmarsh Road, Worminghall, Aylesbury, Buckinghamshire HP18 9PH
T: +44 (0)1844 337380

BELFAST
24 Ballynahinch Street, Hillsborough, Co. Down, BT26 6AW Northern Ireland
T: +44 (0)28 9268 9036

BRADFORD-ON-AVON
Treenwood House, Rowden Lane, Bradford-on-Avon, Wiltshire BA15 2AU
T: +44 (0)1225 309400

BRISTOL
Langford Lodge, 109 Pembroke Road, Clifton, Bristol BS8 3EU
T: +44 (0)117 9064280

CAMBRIDGE
8 Stow Court, Stow-cum-Quy, Cambridge CB25 9AS
T: + 44 (0)1223 813805

CARDIFF
Fulmar House, Beignon Close, Ocean Way, Cardiff CF24 5HF
T: +44 (0)29 20491010

CHELMSFORD
Unit 77, Waterhouse Business Centre, 2 Cromar Way, Chelmsford, Essex CM1 2QE
T: +44 (0)1245 392170

DUBLIN
7 Dundrum Business Park, Windy Arbour, Dundrum, Dublin 14 Ireland
T: + 353 (0)1 2964667

EDINBURGH
No. 4 The Roundal, Roddinglaw Business Park, Gogar, Edinburgh EH12 9DB
T: +44 (0)131 3356830

EXETER
69 Polsloe Road, Exeter EX1 2NF
T: + 44 (0)1392 490152

FARNBOROUGH
The Pavilion, 2 Sherborne Road, South Farnborough, Hampshire GU14 6JT
T: +44 (0)1252 515682

GLASGOW
4 Woodside Place, Charing Cross, Glasgow G3 7QF
T: +44 (0)141 3535037

HUDDERSFIELD
Westleigh House, Wakefield Road, Denby Dale, Huddersfield HD8 8QJ
T: +44 (0)1484 860521

LEEDS
Suite 1, Jason House, Kerry Hill, Horsforth, Leeds LS18 4JR
T: +44 (0)113 2580650

MAIDSTONE
19 Hollingworth Court, Turkey Mill, Maidstone, Kent ME14 5PP
T: +44 (0)1622 609242

NEWCASTLE UPON TYNE
Sailors Bethel, Horatio Street, Newcastle-upon-Tyne NE1 2PE
T: +44 (0)191 2611966

NOTTINGHAM
Aspect House, Aspect Business Park, Bennerley Road, Nottingham NG6 8WR
T: +44 (0)115 9647280

ST. ALBAN'S
White House Farm Barns, Gaddesden Row, Hertfordshire HP2 6HG
T: +44 (0)1582 840471

SHEFFIELD
STEP Business Centre, Wortley Road, Deepcar, Sheffield S36 2UH
T: +44 (0)114 2903628

SHREWSBURY
Mytton Mill, Forton Heath, Montford Bridge, Shrewsbury SY4 1HA
T: +44 (0)1743 850170

STAFFORD
8 Parker Court, Staffordshire Technology Park, Beaconside, Stafford ST18 0WP
T: +44 (0)1785 253331

WARRINGTON
Suite 9 Beech House, Padgate Business Park, Green Lane, Warrington WA1 4JN
T: +44 (0)1925 827218

WORCESTER
Suite 5, Brindley Court, Gresley Road, Shire Business Park, Worcester WR4 9FD
T: +44 (0)1905 751310

Energy | Waste Management | Planning & Development | Industry | Mining & Minerals | Infrastructure