Energy from Waste Combined Heat and Power Facility, North Yard, Devonport

Health Effects arising from Emissions of Metals and Organic Substances

April 2011

Prepared for

MVV Environment Devonport Ltd

Revision Schedule

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Glossary

The following terms and definitions relate to the meaning of these terms as used within this report.

Acute effects	An effect that occurs within a short time after exposure.
Average Daily Dose	The estimated mean dose received by an individual over the course of a day.
Averaging Time	A reference time period e.g. an average daily dose is reported for an averaging time of one day.
Bioaccumulation	The process by which chemicals are taken up into an organism either directly by exposure or indirectly through consumption of contaminated material. Concentrations can accumulate higher up the food chain to levels significantly higher than the original exposure concentration.
Carcinogenic Slope Factor	An upper bound on the increased cancer risk from a lifetime of oral (ingestion) exposure to a substance based on the dose-response relationship of the substance.
Chemicals of Potential Concern	Substances identified through the risk assessment process as being of concern to human health.
Chronic effects	An effect that occurs over a long time period or following a long period of exposure.
Congeners	Substances with molecules that share slightly different chemical structures.
Dioxins/Furans	This is the abbreviated name for a family of toxic substances that share a similar chemical structure and a common mechanism of toxic action. They include the congeners polychlorinated dibenzo dioxins (PCDDs) and polychlorinated dibenzo furans (PCDFs).
Dose	The USEPA define 'Dose' as, the amount of a substance available for interaction with metabolic processes or biologically significant receptors after crossing the exchange boundary of an organism. An equivalent definition is the amount of a substance taken up by an exposed individual following inhalation, ingestion or absorption across the skin.

Dose-response relationship	The relationship between the dose and the proportion of exposed individuals observed to demonstrate effects.
Emission	The substance or the mass of a substance emitted into the atmosphere.
Excess Lifetime Risk	The probability that an individual will develop cancer over a lifetime as a result of exposure to specific carcinogenic chemicals through multiple exposure pathways.
Exposure	The US EPA defines 'exposure' as, the condition of a chemical contacting the exchange boundary of an organism. A broader definition is, the amount of a substance inhaled, ingested or present at the skin surface.
Exposure (Direct)	Inhalation of air containing substances at predicted concentrations.
Exposure (Indirect)	Results from contact of human and ecological receptors with soil, plants or water bodies on which emitted chemicals have been deposited.
Exposure Duration	The length of time that a receptor is exposed via a specific pathway.
Exposure Frequency	This is the amount of time a receptor is exposed to COPCs by all pathways. The HHRAP assumes that receptors are exposed 350 days a year, with a 2 week period away from the relevant exposure location.
Exposure Pathway	This is the route that a chemical takes from its source, through the environment to the individual being exposed.
Exposure Scenario	The combination of relevant exposure pathways to which an individual receptor may be exposed to specific substances.
Hazard	An impact to human health by chemicals of potential concern.
Hazard Index	The total chronic hazard attributable to exposure to all COPCs through a single exposure pathway.
Hazard Quotient	The comparison of oral and inhalation exposure estimates to reference dose and reference concentration values.

Human Health Risk Assessment Protocol	A structured approach to quantifying the risks to human health associated with exposure to compounds of potential concern.
Ingestion	The act of eating or drinking a substance that may then result in the substance being taken up via the digestive system.
Inhalation	The act of breathing in a substance that may then result in the substance being taken up via the respiratory system.
Industrial Risk Assessment Program	A commercially available computer programme developed to calculate excess life time risk and hazard index values following the requirements from the 2005 U.S. EPA-OSW Human Health Risk Assessment Protocol.
Industrial Emissions Directive	A directive of the European Union, the requirements of which will replace requirements of the Waste Incineration Directive (WID) by 2013.
International Toxic Equivalent	This weighs the toxicity of the less toxic compounds as a fraction of the toxicity of a reference compound. In the case of dioxins the toxicity of each individual congener is weighted to 2,3,7,8-TCDD, which is given a reference value of 1.
Lifetime	In estimating the average lifetime exposure of individual receptors or populations to substances, a lifetime is taken to be 70 years.
Lipophilic	A substance is considered lipophilic if it is readily dissolved in fat-like solvents.
Media	For the purposes of this assessment, media are parts of the wider environment that a substance could be contained within. This includes soil, water, air, biota etc.
Metals	The 12 metals, in their elemental form or contained within compounds, for which emission limit values are defined within the Waste Incineration Directive.
Nitrogen Dioxide	A molecule composed of one nitrogen atom and two oxygen atoms, present in outdoor air as a gas.
Oxides of Nitrogen	A collective term for all gases composed of nitrogen and oxygen, including nitrogen dioxide.

Particulate Matter	A solid or liquid particle (a droplet) that in the context of this report is small enough to be suspended in air.
PM_{10}	Mass of particles per cubic metre of air passing through the inlet of a size selective sampler with a transmission efficiency of 50% at an aerodynamic diameter of 10 micrometres.
$PM_{2.5}$	Mass of particles per cubic metre of air passing through the inlet of a size selective sampler with a transmission efficiency of 50% at an aerodynamic diameter of 2.5 micrometres.
Pathway	A term used to represent a series of sequential physical or chemical actions by which a substance is transported from a source, through the environment to a receptor. Typically described using a label that relates to the mechanism that receptors are exposed by, e.g. inhalation pathway.
Polycyclic aromatic hydrocarbons	A group of several hundred chemically related persistent organic compounds of various chemical structures and toxicity. Benzo[a]pyrene is used in National air quality regulations as a marker species for reporting concentrations of PAH in ambient air.
Population	All individuals living within a defined area.
Receptor	For the purposes of the human health risk assessment a receptor is, a hypothetical individual potentially exposed to chemicals of potential concern emitted to the atmosphere from the facility in question.
Reference Concentration	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.
Reference Dose	A daily oral intake rate that is estimated to pose no appreciable risk of adverse health effects, even to sensitive populations, over a 70 year lifetime.
Risk	An estimation of the probability that an adverse health impact may occur as a result of exposure to chemicals in the amount and by the pathways identified.
Sulphur Dioxide	A molecule composed of one sulphur and two oxygen atoms, present in outdoor air as a gas.

Threshold	The dose or exposure level below which no appreciable effects on human health are observed.
Tolerable Daily Intake	A World Health Organisation definition of the dose of a substance that an individual could be exposed to on each day of an entire lifetime, at which appreciable health risks do not occur. See similar 'reference dose' term used by USEPA.
Unit Risk Factor	The upper bound excess lifetime cancer risk estimated to result from continuous exposure to a substance at a concentration of 1µgm^{-3} in air.
Waste Incineration Directive	A directive of the European Union that defines the minimum standard of environmental performance that must be achieved by installations burning waste or waste derived fuels.

Abbreviations

ADD	Average Daily Dose
COPC	Compound of Potential Concern
COT	Committee on Toxicology
CSF	Cancer Slope Factor
FSA	Food Standards Agency
HHRAP	Human Health Risk Assessment Protocol
HQ	Hazard Quotient
HI	Hazard Index
IED	Industrial Emissions Directive
IRAP	Industrial Risk Assessment Program
TEF	Toxic Equivalency Factor
PAH	Polycyclic aromatic hydrocarbon
PCDD	Polychlorinated di benzo(p)dioxin
PCDF	Polychlorinated di benzo furans
RfD	Reference Dose
RfC	Reference Concentration
SGV	Soil Guideline Values
TDS	Total Dietary Study
TDI	Tolerable Daily Intake
URF	Unit Risk Factor
US EPA	United States Environmental Protection Agency
WHO	World Health Organisation
WID	Waste Incineration Directive

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1 Introduction

1.1.1 URS/Scott Wilson have been appointed by MVV Environment Devonport Ltd to prepare an assessment of health effects arising from the emissions of metals and organic substances from the proposed Energy from Waste (EfW) Combined Heat and Power (CHP) facility located at North Yard, Devonport, Plymouth. The proposed EfW CHP facility will emit a mixture of substances, including particulate matter, oxides of nitrogen, sulphur dioxide, metals, polycyclic aromatic hydrocarbons (PAHs) and dioxin/furans into the atmosphere throughout the operational lifetime of the facility. The impact of the emissions from the proposed EfW CHP facility on ambient concentrations of air pollutants and the methods used to calculate the impacts have been reported in the Air Quality Dispersion Modelling Report[1]. The human health effects associated with the exposure of the local population to the predicted changes in atmospheric concentrations of particulate matter (PM_{10} and $PM_{2.5}$), nitrogen dioxide and sulphur dioxide has been quantified within another report[2].

1.1.2 This report quantifies the human health effects associated with the exposure of the local community, within 10km of the proposed EfW CHP facility, to the predicted change in atmospheric concentrations of metals, PAHs and dioxins/furans.

1.1.3 The Waste Incineration Directive (WID)[3] seeks to regulate the burning of waste, where waste is used as a fuel or is disposed of at a plant where energy generation or production is the main purpose. The directive defines operating conditions for the incineration process, emission monitoring requirements and limit values for emission of substances to air and water. At the present time the WID directive has been transposed into national legislation through the Environmental Permitting (England and Wales) Regulations 2010[4].

1.1.4 The Industrial Emissions Directive (IED) 2010/75/EU[5] entered into force on 7th January 2011 and incorporated a number of directives, including WID, into a single overall directive. All European Union member states are required to transpose this directive into national legislation within two years. The emission limit values and operating conditions specified within WID have been retained within the IED and will continue to be applied to waste incineration facilities.

1.1.5 The methodology for assessing the effects on human health from such facilities is based on the United States Environmental Protection Agency (US EPA) Human Health Risk Assessment Protocol (HHRAP)[6]. This provides a systematic and transparent protocol for undertaking site-specific risk assessments of human exposure to emissions from combustion facilities. The main steps within the HHRAP are:

- characterising the source of the hazard;
- identifying the relevant pathways via which receptors could be exposed;

[1] Scott Wilson (2011) Air Quality Technical Appendix 13.1,Energy from Waste Combined Heat and Power Facility, North Yard, Devonport: Environmental Statement Volume 3: Appendices
[2] Scott Wilson (2011) Appendix 18.1 Assessment of Health Effects from Exposure to Particulate Matter, Nitrogen Dioxide and Sulphur Dioxide, Energy from Waste Combined Heat and Power Facility, North Yard, Devonport: Environmental Statement Volume 3: Appendices
[3] European Commission (2000) Directive 2000/76/EC on the Incineration of Waste
[4] H.M. Government (2010) Environmental Permitting Regulations (England and Wales). SI 675, the Stationary Office
[5] European Union (2010) Directive 2010/75/EU on Industrial Emissions (integrated pollution prevention and control) (recast)
[6] US EPA (2005) Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, U.S. EPA Office of Solid Waste, September 2005

- calculating concentrations of COPCs in environmental media;
- calculating the magnitude of human exposure; and
- quantifying the risk of health effects.

1.1.6 This report applies the HHRAP methodology published by the US EPA to quantify the risks of human health effects from exposure to metals, PAHs and dioxins/furans, associated with the operation of the EfW CHP facility. The HHRAP encompasses more than a decade of research into the risk assessment of combustion facilities on the subject of hazard identification and health risks. No further review of the underpinning medical literature has been undertaken in support of this document.

1.1.7 The relationship between exposure to air pollutants, either singly or in combination, and the resulting effects on health remains a topic of active research. Although emissions from the installation's stack are initially in the form of airborne substances, inhalation is not the only relevant exposure pathway for some of the substances of concern. The HHRAP method adopts the source – pathway – receptor approach to assess exposure via all pathways.

1.1.8 Taking a generic example, where a stack is the source and the substance emitted into the atmosphere is a potential hazard to human health. The people that make up the population of the land surrounding the stack are receptors that may be exposed to a dose of the substance. The substance might move through the environment via a number of available pathways before the receptors are exposed to it. One pathway might be dispersion through the atmosphere followed by inhalation into the receptor's lungs. Another pathway might be deposition from the atmosphere onto the ground, followed by uptake into plants that are then eaten by livestock, which are then in turn consumed by receptors.

1.1.9 If a receptor was to live their entire life at a location where they breathed the substance at the highest airborne concentrations and they only ate locally grown food and drank local water from the location where the concentrations of the deposited substance were highest, then they would experience the maximum hypothetical level of exposure. This maximum level is described as 'hypothetical' because it is a conservative assumption and ignores the fact that most consumed food stuffs will be sourced from retail operations.

1.1.10 Within HHRAP the health impact on the entire exposed population is characterised using six types of receptors to represent hypothetical maximum exposure scenarios:

- the resident (adult) and resident's child;
- the farmer (adult) and farmer's child; and
- the fisher (adult) and fisher's child.

1.1.11 The receptor locations within the assessment have been chosen for each receptor type, based upon the predicted maximum concentrations from the air quality dispersion modelling assessment[1]. This enables the potential health effects for the exposed population to be quantified, based on the maximum dose that a representative receptor within the study area is likely to be exposed to.

1.1.12 The substances of potential concern (COPC) considered within this report have the potential to induce long term, chronic effects on human health at environmental concentrations. For some

of these substances there is no minimum concentration below which adverse health effects will not occur and it is therefore appropriate to consider the risk of effects occurring. The receptors considered in this assessment are representative of the maximum hypothetical lifetime risk of human health effects that members of the population would be exposed to. For the purposes of this assessment, risks are presented for lifetimes of 70 years duration for an adult receptor and 6 years duration for a child receptor. The assessment quantifies the risk for carcinogenic effects and for non-carcinogenic effects and reports these risks using internationally recognised metrics.

1.1.13 In this report the terminology used is of necessity technical and the meaning of the terms may differ from their use in conversational English. A glossary of the terms used is provided within this report.

2 Methodology

2.1 Introduction

2.1.1 This assessment considers the risk of effects on human health occurring within the local population when exposed to emissions to air from the proposed EfW CHP facility at North Yard, Devonport. The approach to this assessment is as follows:

- characterising the source of the hazard;
- identifying the relevant pathways via which receptors could be exposed;
- calculating concentrations of COPCs in environmental media;
- calculating the magnitude of human exposure; and
- quantifying the risk of health effects.

2.1.2 The hazard source consists of Compounds of Potential Concern (COPCs), which are substances emitted from waste treatment facilities at rates permitted under the Waste Incineration Directive. The hazard source has previously been quantified through a detailed dispersion modelling exercise that has reported on substances emitted and dispersed within the atmosphere, and the amount of COPCs deposited to ground[1].

2.1.3 The relevant exposure pathways are identified as either direct (inhalation) or indirect (ingestion of water, soil, vegetation and animal products contaminated through the food chain). The receptors are chosen based on the results of the maximum predicted concentrations from the dispersion modelling exercise and surrounding site specific conditions.

2.1.4 The level of exposure and dose to COPCs via each pathway can be calculated for each receptor once the source, exposure pathways and receptors have been quantified. Ultimately a total risk for carcinogenic and non-carcinogenic effects occurring in each of the receptors from the various different exposure scenarios is calculated.

2.1.5 The current and future land use, the location of water bodies and associated watersheds and any special population characteristics (e.g. infants or elderly) are considered within the assessment of exposure to COPCs.

2.1.6 The risk of effects on human health arising from exposure to dioxins and furans, PAHs and metals emitted from the proposed EfW CHP facility are estimated for hypothetical scenarios, including that of an individual exposed for a lifetime to the effects of the highest airborne concentrations and consuming mostly locally grown food.

2.1.7 The methods outlined in the US EPA HHRAP have been encompassed into a commercially available risk assessment modelling tool called the Industrial Risk Assessment Program (IRAP) by Lakes Environmental Software. URS/Scott Wilson holds a user licence for the latest version of this software (4.0), which has been used to conduct the assessment of the risks to human health via the method outlined above.

2.1.8 HHRAP has been specifically developed to enable the estimation of the level of exposure received by the local population via the combination of potential exposure pathways in a

consistent and repeatable manner. HHRAP considers the fate and transport of substances through soil, water and biota (plant material) following deposition onto these surfaces. This is then used to calculate the potential uptake of these substances by the receptors via the relevant pathways.

2.1.9 Within HHRAP the receptors chosen are classified as either a resident, farmer or fisher receptor types. It is also necessary to distinguish between an adult and child receptor as children are considered to be at a greater risk of experiencing health effects from a specified dose due to their lower body weights. The farmer receptor is assumed to consume proportionally more locally grown food than a resident. This means that these receptors are at a greater risk of eating food contaminated by emissions from the source. A fisher receptor type is utilised where there is the potential for the consumption of locally caught fish from water bodies affected by emissions from the source to constitute the main source of protein within the receptors diet. For resident type receptors it is assumed that they are home gardeners within an urban area and as such consume locally grown produce with some incidental ingestion of soil. All receptors types are assumed to be present at the same location all year apart from a 2 week holiday period (350 days).

2.1.10 The air quality dispersion modelling assessment[1] generates output files that are imported into the IRAP model to calculate concentrations of COPCs within each exposure pathway that are ultimately taken up by human receptors. In order to perform this calculation IRAP requires the following input parameters;

- Physical and chemical properties of COPCs
- Site specific information e.g. precipitation rate, wind speed
- Information for each receptor type e.g. body weight, consumption rates of food, exposure rates

2.1.11 The HHRAP default values that have been incorporated within IRAP are used for the majority of input values, as discussed in the following sections.

2.2 Hazard Source

2.2.1 The proposed EfW CHP facility will process an estimated 245,000 tonnes per annum of industrial, commercial and municipal solid waste using combined heat and power technology. The waste material will be combusted and the heat generated is then used to generate steam. This steam will drive a turbine and produce electricity for use at the facility, to supply Devonport Dockyard and Her Majesty's Naval Base (HMNB) and for export to the national grid. Steam will also be extracted from the turbine and fed into the Devonport Dockyard and HMNB steam network to be used for heating purposes.

2.2.2 Throughout its operational lifetime the proposed EfW CHP facility will emit a number of different substances into the atmosphere via a stack, which are referred to in this assessment as Compounds of Potential Concern (COPCs). The WID specifies plant operating conditions (e.g. temperature and residence times) as well as emission limit values, which represent an upper limit on the permitted concentrations of COPCs that can be emitted from the EfW CHP facility. The emission limits used within in this assessment, as specified in the WID and retained within the IED, are set out in Table 2.1.

Table 2.1 Daily averaged emission limit values in the WID

Pollutant	Emission Limit value (mg/m^3)	Averaging period
Total Dust	10	Daily mean
Gaseous and vaporous organic substances, expressed as total organic carbon	10	Daily mean
Hydrogen Chloride (HCl)	10	Daily mean
Hydrogen Fluoride (HF)	1	Daily mean
Sulphur Dioxide (SO_2)	50	Daily mean
Nitrogen monoxide (NO) and nitrogen dioxide (NO_2), expressed as nitrogen dioxide for existing incineration plants with a nominal capacity exceeding 6 tonnes per hour or new incineration plants	200	Daily mean
Carbon Monoxide (CO)	50	Daily mean
Cadmium (Cd) and Thallium (Tl)	Total 0.05	All average values over the sampling period 30 minutes to 8 hours
Mercury (Hg)	0.05	
Antimony (Sb), Arsenic (As), Lead (Pb), Chromium (Cr), Cobalt (Co), Copper (Cu), Manganese (Mn), Nickel (Ni) and Vanadium (V)	Total 0.5	
Dioxins	0.1 ng I-TEQ / Nm^3	CEN method, sample period 6 to 8 hours

Compounds of Potential Concern (COPCs)

2.2.3 The COPCs of relevance to this assessment are permitted emissions under WID (shown in Table 2.1). Specific physical and chemical information on these substances is included within the US EPA HHRAP COPC companion database for the assessment of long term health effects. The particular substances considered with regards to the assessment of their effects on human health are listed below:

- Polychlorinated di benzo(p)dioxins/furans (PCDD/F) as individual congeners;
- Benzo(a)pyrene;
- Antimony (Sb);
- Arsenic (As);
- Cadmium (Cd);
- Chromium (Cr), trivalent and hexavalent;
- Mercury (Hg);
- Lead (Pb); and
- Nickel (Ni).

2.2.4 Benzo(a)pyrene has been included in the list of COPCs to represent all polycyclic aromatic hydrocarbons (PAHs) emissions within this assessment. Although no emissions limits are specified under WID, monitoring of these substances is required under the directive.

2.2.5 The 2005 HHRAP excluded thallium (Tl) by virtue of there being no reference dose, reference concentration or cancer slope factors available for thallium. This is contrast to the draft 1998 HHRAP which did include compound specific parameter values for thallium in Appendix A of the draft 1998 US EPA HHRAP[7]. The physical and chemical properties of thallium are well known and it has been considered appropriate to include thallium in the list of COPCs for the assessment of any human health effects. Therefore, the 1998 US EPA HHRAP[7] reference data has been used to assess the risk to human health associated with exposure of the local population to thallium.

Emission Concentrations

2.2.6 The emission concentrations of the COPCs considered in this assessment have been reported in the air quality dispersion modelling assessment[1]. The WID places limit values on the emissions of substances in the short term i.e. daily average values, which have been used as a conservative assumption within this assessment of long term health effects.

2.2.7 The individual emissions concentrations and rates for each of the inorganic COPCs are shown in Table 2.2 below. Some of the metals with specified emission limits in the WID do no not pose a risk to human health in the long term and have not been included within the HHRAP e.g. cobalt, copper, manganese and vanadium. These metals have therefore been excluded from this assessment of the risks to human health.

Table 2.2 Emission concentrations and rates of metals used for the human health risk assessment

Metal Group defined in WID	Pollutant	Emission Concentration[(a)] (mg Nm^{-3})	Emission Rate (g s^{-1})
Group 1	Cadmium	0.05	0.0028
	Thallium	0.05	0.0028
Group 2	Mercury	0.05	0.0028
Group 3	Antimony	0.5	0.028
	Arsenic	0.003	0.00017
	Total Chromium	0.033	0.0018
	Chromium (vi)	0.00069	0.000039
	Lead	0.5	0.028
	Nickel	0.136	0.0076

(a) Emission concentrations for individual metals have been set at the group WID limit value apart from arsenic, nickel and chromium, which are set based upon the Environment Agency's Interim guidance on metals for waste incineration. Within this guidance note chromium has been assumed to be 97.9% Cr(iii) and 2.1% Cr(vi).

2.2.8 The concentration of mercury has been adjusted in order to take account of the loss of mercury to the global cycle. The default values within IRAP assume that 48% of total mercury is deposited as divalent mercury (mercuric chloride), 2% is deposited as elemental mercury and the rest being lost to the global cycle. IRAP assumes that the exposed population will only be

[7] US EPA (1998) Human Health Risk Assessment for Hazardous Waste Combustion Facilities, U.S. EPA Office of Solid Waste, Peer Review Draft, July 1998

exposed to elemental mercury through direct inhalation of the vapour phase whereas exposure to divalent mercury will occur via both direct and indirect inhalation of vapour and particle bound mercuric chloride. This leads to the following emission rates for elemental and divalent mercury:

- Elemental mercury at 5.59×10^{-6}
- Divalent mercury 1.34×10^{-3}

2.2.9 As stated above, benzo(a)pyrene has been included in the list of COPCs as representative of all polycyclic aromatic hydrocarbons (PAHs) with an emission concentration of 0.001 mg Nm^{-3} and an emission rate of 5.59×10^{-5} g s^{-1} as previously reported in the air quality dispersion modelling assessment[1].

2.2.10 Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are organic substances formed as a by-product of combustion processes and in the manufacture of certain chlorinated organic chemicals. PCDD/Fs have been classified as persistent organic pollutants (POPs) with a significant potential to bioaccumulate[8]. The basic structure of the dioxin family is composed of benzene rings interconnected by two oxygen atoms. The degree and position of the chlorination to the basic structure determines the type of the individual dioxin with 75 individual compounds being possible. Furans are of a similar structure but with a carbon atom replacing one of the chlorine atoms, yielding 125 individual furan compounds. Each individual compound is referred to as a congener and each has slightly different chemical and physical properties in the environment that are determined by the position and degree of chlorination within the molecule.

2.2.11 The assessment of the effect of PCDD/Fs on human health takes into account the affect of the different physical and chemical properties of the individual congeners on their behaviour in the environment. Individual congeners are used to conduct the assessment of the health risk from dioxins/furans. A standard PCDD/F emission profile for municipal waste incinerators has previously been derived by Her Majesty's Inspectorate of Pollution (HIMP)[9] and will be used to represent the congener emission profile in this assessment (Table 2.3). Toxic equivalency factors (TEF) are used to express the toxicities of the different PCDD/Fs in relation to the most toxic dioxin, 2,3,7,8-TCDD. These TEFs have been used to generate toxic equivalent emissions (I-TEQ) for each congener based upon the standard emissions profile. The total TEQ for all PCDD/Fs has been assumed to be no more than that of the maximum WID limit of 0.1 ng I-TEQ Nm^{-3}.

[8] WHO (2010) Dioxins and their effects on human health, Factsheet No. 225, May 2010
[9] DOE (1996) Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes Contract No. HMIP/CPR2/41/1/181

Table 2.3 Congener Profile for the proposed EfW CHP facility for all of the PCDD/Fs

Congener	Annual Mean Emission Concentration (ng Nm^{-3})	I-TEF (toxic equivalent factors)[(a)]	Annual Mean Emission (ng I-TEQ Nm^{-3})
2,3,7,8-TCDD	0.0031	1.0	0.0031
1,2,3,7,8-PeCDD	0.025	0.5	0.0125
1,2,3,4,7,8-HxCDD	0.029	0.1	0.0029
1,2,3,6,7,8-HxCDD	0.026	0.1	0.0026
1,2,3,7,8,9-HxCDD	0.021	0.1	0.0021
1,2,3,4,6,7,8-HpCDD	0.17	0.01	0.0017
OCDD	0.40	0.001	0.0004
2,3,7,8-TCDF	0.028	0.1	0.0028
2,3,4,7,8-PeCDF	0.054	0.5	0.027
1,2,3,7,8-PeCDF	0.028	0.05	0.0014
1,2,3,4,7,8-HxCDF	0.22	0.1	0.022
1,2,3,6,7,8-HxCDF	0.081	0.1	0.0081
1,2,3,7,8,9-HxCDF	0.0040	0.1	0.00040
2,3,4,6,7,8-HxCDF	0.087	0.1	0.0087
1,2,3,4,6,7,8-HpCDF	0.44	0.01	0.0044
1,2,3,4,7,8,9-HpCDF	0.040	0.01	0.00040
OCDF	0.4	0.001	0.0004
Total (ng I-TEQ m^{-3})			**0.1**

(a) TEF values obtained from Part 2 of Annex VI to the Industrial Emissions Directive 2010/75/EU[5]

2.2.12 The emissions rates used in the IRAP model for each of the PCDD/Fs are shown in Table 2.4. These rates have been calculated based upon percentage contribution of each congener to the total emission rates of all dioxin/furans at WID emission limits.

Table 2.4 Emission rates used in the IRAP model for all of the PCDD/Fs

Congener	Emission Rate (g s^{-1})
2,3,7,8-TCDD	1.7×10^{-10}
1,2,3,7,8-PeCDD	6.9×10^{-10}
1,2,3,4,7,8-HxCDD	1.6×10^{-10}
1,2,3,6,7,8-HxCDD	1.2×10^{-10}
1,2,3,7,8,9-HxCDD	1.4×10^{-10}
1,2,3,4,6,7,8-HpCDD	9.4×10^{-11}
OCDD	2.2×10^{-11}
2,3,7,8-TCDF	1.5×10^{-10}
2,3,4,7,8-PeCDF	1.5×10^{-9}
1,2,3,7,8-PeCDF	7.7×10^{-11}
1,2,3,4,7,8-HxCDF	1.2×10^{-9}
1,2,3,6,7,8-HxCDF	2.2×10^{-11}
1,2,3,7,8,9-HxCDF	4.5×10^{-10}
2,3,4,6,7,8-HxCDF	4.8×10^{-10}
1,2,3,4,6,7,8-HpCDF	2.4×10^{-10}
1,2,3,4,7,8,9-HpCDF	2.2×10^{-11}
OCDF	2.2×10^{-11}

Properties of COPCs

2.2.13 The HHRAP includes a database that defines the physical and chemical properties of 206 COPCs, as well as toxicity factors for each COPC. This database is the source of the default values within the IRAP model. The physical and chemical properties determine how each of the COPCs would move within the environment and the extent to which they would bioconcentrate in different foodstuffs (e.g. meat, fish, vegetation, soil and water). An example of the range of different properties used within IRAP is presented in Table 2.5. Data for lead and 2,3,7,8-TCDD is included in Table 2.5 to provide an illustration of the marked differences in the properties associated with organic and inorganic substances.

2.2.14 Toxicity benchmarks (e.g. reference dose/concentrations, slope factors, unit risk factors) with regards to human health effects are shown in Table 2.6 for all of the COPCs considered in this assessment. These values are provided in the HRRAP and used to determine the carcinogenic and non-carcinogenic risks associated with inhalation or ingestion exposure to each of the COPCs.

2.2.15 The Carcinogenic Slope Factor (CSF) and Unit Risk Factors (URF) for each COPC are used to calculate the carcinogenic risk from ingestion and inhalation respectively. The ingestion Reference Dose (RfD) and Inhalation Reference Concentration (RfC) are used to calculate the non-carcinogenic risk associated with exposure to each COPC. The detailed methodology for calculating the non-carcinogenic and carcinogenic risks to human health are provided in section 2.6 and 2.7.

Table 2.5 Example IRAP Input Parameters for Lead and 2,3,7,8-TCDD

Parameter Description	Symbol	Units	Lead	2,3,7,8-TCDD
Chemical abstract service number	CAS No.	-	7439-92-1	1746-01-6
Molecular weight	MW	g $mole^{-1}$	209.21	322.0
Melting point of chemical	T_m	K	603.15	578.7
Vapour pressure	V_p	atm	3.97×10^{-12}	1.97×10^{-12}
Aqueous solubility	S	mg L^{-1}	9580	1.93×10^{-5}
Henry's Law constant	H	atm-m^3 mol^{-1}	0.025	3.29×10^{-5}
Diffusivity of COPC in air	D_a	cm^2 s^{-1}	0.0772	0.104
Diffusivity of COPC in water	Dw	cm^2 s^{-1}	9.6×10^{-6}	5.6×10^{-6}
Octanol-water partition coefficient	K_ow	-	5.37	6,309,573
Organic carbon-water partition coefficient	K_oc	mL g^{-1}	0	3,890,451
Soil-water partition coefficient	Kd_s	mL g^{-1}	900	38,904
Suspended sediments/surface water partition coefficient	Kd_sw	L kg^{-1}	900	291,784
Bed sediment/sediment pore water partition coefficient	Kd_bs	mL g^{-1}	900	155,618
COPC loss constant due to biotic and abiotic degradation	K_s_g	a^{-1}	0	0.03
Fraction of COPC air concentration	f_v		0.007	0.664
Root concentration factor	RCF	mL g^{-1}	0	39,999
Plant-soil bioconcentration factor for below ground produce	br_root_veg	-	0.009	1.03
Plant-soil bioconcentration factor for lefy-vegetables	br_leafy_veg	-	0.0136	0.00455
Plant-soil bioconcentration factor for forage	br_forage	-	0.045	0.00455
COPC air-to-plant biotransfer factor for leafy vegetables	bv_leafy_veg	-	0	65,500
COPC air-to-plant biotransfer factor for forage	bv_forage	-	0	65,500
COPC biotransfer factor for milk	ba_milk	day kg^{-1}	0.00025	0.0055
COPC biotransfer factor for beef	ba_beef	day kg^{-1}	0.0003	0.026
COPC biotransfer factor for pork	ba_pork	day kg^{-1}	0	0.032
COPC biotransfer factor for chicken	ba_chicken	day kg^{-1}	0	0.019
Plant-soil bioconcentration factor for eggs	ba_egg	-	0	0.011
Fish bioconcentration factor	BCF_fish	L kg^{-1}	0.09	34,400
Fish bioaccumulation factor	BAF_fish	L kg^{-1}	0	0
Biota-sediment accumulation factor	BSAF_fish	-	0	0.09
Plant-soil bioconcentration factor for grain	br_grain	-	0.009	0.00455

Table 2.6 Toxicity factors obtained from the HHRAP for the COPCs in this assessment

COPC	Ingestion Reference Dose	Inhalation Reference Concentration	Ingestion Carcinogenic Slope Factor	Inhalation Unit Risk Factor
Symbol	RfD	RfC	Ing_csf	Inh_URF
Units	(mg kg^{-1} d^{-1})	(mg m^{-3})	(mg kg^{-1} d^{-1})$^{-1}$	(µg m^{-3})$^{-1}$
Metals				
Antimony	0.0004	0.0014	0	0
Arsenic	0.0003	3.0×10^{-5}	1.5	0.0043
Cadmium	0.0004	0.0002	0.38	0.0018
Chromium (iii)	1.5	5.3	0	0
Chromium (vi)	0.0030	8.0×10^{-6}	0	0.012
Lead	0.000429	0.0015	0.0085	1.2×10^{-5}
Nickel	0.02	0.0002	0	0.00024
Thallium[(a)]	0.00008	0.00028	0	0
Elemental mercury	8.57×10^{-5}	0.0003	0	0
Mercuric chloride	0.0003	0.0011	0	0
Methyl mercury	0.0001	0.00035	0	0
PAHs				
Benzo(a)pyrene	0	0	7.3	0.0011
PCDDs				
2,3,7,8-TCDD	1×10^{-9}	0	150,000	0
1,2,3,7,8-PeCDD	0	0	0	0
1,2,3,4,7,8-HxCDD	0	0	0	0
1,2,3,6,7,8-HxCDD	0	0	6,200	1.3
1,2,3,7,8,9-HxCDD	0	0	6,200	1.3
1,2,3,4,6,7,8-HpCDD	0	0	0	0
OCDD	0	0	0	0
PCDFs				
2,3,7,8-TCDF	0	0	0	0
2,3,4,7,8-PeCDF	0	0	0	0
1,2,3,7,8-PeCDF	0	0	0	0
1,2,3,4,7,8-HxCDF	0	0	0	0
1,2,3,6,7,8-HxCDF	0	0	0	0
1,2,3,7,8,9-HxCDF	0	0	0	0
2,3,4,6,7,8-HxCDF	0	0	0	0
1,2,3,4,6,7,8-HpCDF	0	0	0	0
1,2,3,4,7,8,9-HpCDF	0	0	0	0
OCDF	0	0	0	0

(a) Reference dose for Thallium sourced from the 1998 US EPA HHRA Protocol[7]

Dispersion Modelling

2.2.16 The results of the air quality dispersion modelling assessment[1] have been generated through use of the air dispersion modelling software ADMS 4.2. Ground level concentrations and deposition rates have been generated using the model parameter values e.g. emission rates, building heights, terrain data, as detailed within the air quality assessment.

2.2.17 IRAP imports the dispersion model output files generated by the US EPA ISC or ISC-AERMOD dispersion models. The output files generated by ADMS 4.2 therefore require reformatting, before the information can be imported into IRAP.

2.2.18 This assessment of the risks to human health has been carried out utilising the concentration predictions reported in the Air Quality Dispersion Modelling Report[1] using ADMS. In addition to airborne concentrations of the COPCs, the human health risk assessment requires predictions of the following parameters, which have been made reported in the Air Quality Dispersion Modelling Report[1]:

- Airborne concentrations of vapour, particle and particle bound substances emitted;
- Wet deposition rates of vapour, particle and particle bound substances; and
- Dry deposition rates of particle and particle bound substances

2.2.19 The proposed EfW CHP facility will be equipped with fabric filters, which will mean the dominant size fraction of particles will be 1-2 µm in diameter and below. For particles of this size range a dry deposition velocity of 0.01 ms^{-1} has been used in the modelling to calculate dry deposition rates. Whereas a dry deposition velocity of 0.005 ms^{-1} has been used to calculate dry deposition rates for gaseous phase substances. Wet deposition rates have been calculated for both particulate and gaseous substances in ADMS using values for the washout coefficients A and B of 0.0001 and 0.64 respectively.

2.2.20 The results from the air quality assessment that are relevant to this assessment of the risks to human health are presented in Table 2.7 with all set up parameters used for the dispersion modelling presented in the air quality dispersion modelling assessment[1].

2.2.21 The points of maximum airborne concentration, dry deposition and wet deposition rates are represented by the relevant receptor locations as discussed in section 2.4 and shown on Figure 2.1. Note that the point of maximum wet deposition is heavily influenced by the assumed washout mechanism, which is very localised, hence the location of the point of maximum wet deposition rate in close proximity to the source.

Table 2.7 Maximum annual average concentrations and deposition rates associated with the EfW CHP facility

Pollutant	Annual Average Concentrations [a]	Vapour Dry Deposition Rate [b]	Particle Dry Deposition Rate [b]	Wet Deposition Rate [b]
Metals	***(µg m^{-3})***	***(mg m^{-2} $year^{-1}$)***	***(mg m^{-2} $year^{-1}$)***	***(mg m^{-2} yea r^{-1})***
Antimony	0.0063	1.973	0.986	76.0
Arsenic	3.8×10^{-5}	0.012	0.006	0.456
Cadmium	0.00063	0.099	0.197	7.60
Chromium III	0.00041	0.130	0.065	5.02
Chromium Vi	9.0×10^{-6}	0.003	0.001	0.105
Lead	0.0063	1.973	0.986	76.0
Nickel	0.0017	0.537	0.268	20.7
Thallium	0.00063	0.197	0.099	7.60
Elemental Mercury	1.3×10^{-6}	0.0004	0.0002	0.015
Mercuric Chloride	0.0003	0.095	0.047	3.65
PAHs				
Benzo(a)pyrene	1.3×10^{-5}	0.004	0.002	0.152
PCDD/Fs	***(fg m^{-3})***	***(ng m^{-2} $year^{-1}$)***	***(ng m^{-2} $year^{-1}$)***	***(ng m^{-2} $year^{-1}$)***
2,3,7,8-TCDD	0.038	0.012	0.006	0.467
1,2,3,7,8-PeCDD	0.155	0.049	0.024	1.88
1,2,3,4,7,8-HxCDD	0.036	0.011	0.006	0.437
1,2,3,6,7,8-HxCDD	0.026	0.008	0.004	0.316
1,2,3,7,8,9-HxCDD	0.032	0.010	0.005	0.392
1,2,3,4,6,7,8-HpCDD	0.022	0.007	0.003	0.256
OCDD	0.005	0.002	0.001	0.060
2,3,7,8-TCDF	0.035	0.011	0.005	0.422
2,3,4,7,8-PeCDF	0.335	0.106	0.053	4.07
1,2,3,7,8-PeCDF	0.017	0.005	0.003	0.211
1,2,3,4,7,8-HxCDF	0.272	0.086	0.043	3.32
1,2,3,6,7,8-HxCDF	0.005	0.002	0.001	0.060
1,2,3,7,8,9-HxCDF	0.100	0.032	0.016	1.22
2,3,4,6,7,8-HxCDF	0.108	0.034	0.017	1.31
1,2,3,4,6,7,8-HpCDF	0.055	0.017	0.009	0.663
1,2,3,4,7,8,9-HpCDF	0.005	0.002	0.001	0.060
OCDF	0.005	0.002	0.001	0.060

(a) Where 1 $\mu g m^{-3}$ is equal to 1×10^{-6} g m^{-3} and 1 fg m^{-3} is equal to 1×10^{-15} g m^{-3}
(b) Where 1 mg m^{-2} yr^{-1} is equal to 1×10^{-3} g m^{-2} yr^{-1} and 1 ng m^{-2} yr^{-1} is equal to 1×10^{-9} g m^{-2} yr^{-1}

2.3 Exposure Pathways

2.3.1 The local environment and site specific parameters within the study area will define the route that emissions could potentially take and lead to exposure at the relevant receptors. In order to calculate COPC specific exposure rates for each exposure pathway being considered some of the following information may be required:

- The COPC concentration in each media as calculated in Section 2.2 above;
- Consumption rates of receptors in each media;
- Receptor body weight; and
- The frequency and duration of exposure.

2.3.2 In any given situation, regardless of site specific circumstances, two primary pathways exist where human receptors could be exposed to COPCs. These are defined as being either direct or indirect exposure pathways. The direct exposure pathway occurs via the inhalation of vapour and particulate matter emissions of COPCs from the source. Whereas, there are numerous potential indirect exposure pathways, as listed below:

- Ingestion of vegetation and animal products contaminated with emissions from the proposed EfW CHP facility;
- Ingestion of locally grown or locally caught food (including vegetables, animals and fish);
- Ingestion of drinking water from surface water sources;
- Incidental ingestion of soil;
- Dermal (skin) contact with contaminated soil and water;
- Ingestion of breast milk.

2.3.3 Exposure via the ingestion pathways can occur over a period of time and should also be expressed in terms of body weight of the receptor. The body weight of a receptor is defined by the US EPA as being 70 kg for an adult and 15 kg as a child with an exposure duration of 30 years for an adult and 6 years for a child. For each exposure pathway the daily intake is defined as the dose per body weight. This highlights the importance of considering the child scenario, as for the same dose at a lower body weight the daily intake can be significantly higher.

2.3.4 Plants and animals could be exposed to COPCs via deposition or direct uptake from the air. Subsequent consumption of these plants and animals via the food chain could lead to human receptors being exposed. Information on the diet of the particular receptors (type and quantity of food consumed) is used to predict the total daily intake of COPCs via the ingestion (food) pathway. Food not produced in the local vicinity will not be contaminated by COPCs and therefore only food produced and consumed at the receptor location is considered relevant in the calculation of exposure via this pathway.

2.3.5 The dermal contact exposure pathway can be disregarded from most assessments of the effects on the human health of the local population unless there are site specific requirements for its inclusion. Exposure via this pathway will occur infrequently and coupled with low dermal absorption factors will lead to a low total dose being experienced over the lifetime of an individual human receptor. Dermal contact via aquatic pathways e.g. swimming and fishing, is not considered a significant pathway for similar reasons.

2.3.6 The HHRAP considers the ingestion of drinking water from a groundwater source as an insignificant exposure pathway from facilities similar to the proposed EfW CHP facility. Surface water bodies used as a drinking water source and their associated water shed should be identified within the study area. If such water bodies exist then the exposure via drinking water from surface water sources should be included within the assessment.

2.3.7 The IRAP model requires certain site specific parameters relating to the local area with which to model the fate and transport of the COPCs via each exposure pathway. The default values within IRAP and contained within the HHRAP have been used to represent the following site specific parameters (as shown in Annex B):

- 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).

2.3.8 The following site specific parameters, relating to surface conditions, are requires to be defined by the user in IRAP and have been included in this assessment as follows:

- Annual mean precipitation of 98.7 cm yr^{-1} (based on 2009 meteorological data obtained at the Plymouth Mountbatten meteorological station);
- Annual average evapotranspiration rate of 69.1 cm yr^{-1} (assumed to be 70% of annual mean precipitation);
- Mean annual irrigation of 0.0 cm a^{-1} i.e. no irrigation;

- Average annual runoff of 9.87 cm a^{-1} (assumed to be 10% of total precipitation);
- An average annual wind velocity of 5.3 ms^{-1} (obtained from 2009 meteorological data obtained at the Plymouth Mountbatten meteorological station); and
- The time period over which emissions would be deposited is assumed to be 30 years (the typical operation period for a facility of this specification).

Study Specific Exposure Pathways

2.3.9 Based on the local environment surrounding the proposed EfW CHP facility the potential significance of all the exposure pathways, identified above, has been assessed. This has identified that the exposure pathways relevant to this assessment are as follows:

- Inhalation;
- Ingestion of locally grown food and locally reared animal products e.g. milk and eggs
- Incidental ingestion of soil
- Ingestion of breast milk

2.3.10 For exposure to occur via ingestion of drinking water there must be a source of drinking water on the surface in the local area that is affected by the emissions from the EfW CHP facility. Within a 10 km radius of the proposed EfW CHP facility the surface waters are predominantly saline and do not represent a source of drinking water. Receptors obtain drinking water from sources located beyond the study area. This exposure pathway is not considered relevant in this assessment of human health effects.

2.3.11 Plymouth Sound, the River Tamar Estuary and its surrounding tributaries are potential sources of locally caught fish although the fish species most likely to be eaten are not generally continuously resident within the estuary. In this area the water bodies are tidal in nature with a tidal range of between 1.5 m and 6 m. The tidal area of the estuary is extensive. It represents a significant movement of water on a diurnal basis with regular influxes of saline water, leading to a well mixed body of water.

2.3.12 The HHRAP specifies that dilution effects due to tidal influences in estuarine water bodies are not considered when calculating COPC concentrations in water and sediments. Therefore the inclusion of surface water bodies in any assessment of health effects from the consumption of locally caught fish will not take into account the dilution effects due to the tidal influences on the River Tamar Estuary, resulting in gross over estimates of risks to health for this pathway. In addition, the local population can be considered to fit the urban resident type for whom fish caught within the estuary would not represent the main source of protein in their diet. For these reasons it has been considered appropriate to exclude the ingestion of locally caught fish as an exposure pathway in this assessment of health effects.

2.3.13 Based upon the local environment surrounding the proposed EfW CHP facility the following exposure pathways have been considered within this assessment with regards to ingestion.

- Soil (incidental);
- home grown produce (fruits and vegetables);

- home grown beef;
- home grown pork;
- home grown chicken;
- milk from home reared cows;
- eggs from home reared chickens; and
- breast milk.

2.3.14 The inclusion of all food groups within this assessment has conservatively assumed that there is both arable and pastoral land in addition to locally grown produce and animals within the vicinity of the proposed EfW CHP facility. The ingestion of home reared meat is only considered for farmers and the families of farmers.

2.4 Receptors

2.4.1 The HHRAP defines three generic hypothetical receptor types for use within the human health risk assessment process. The receptor types are a hypothetical adult and/or child Resident, Farmer and Fisher.

2.4.2 The hypothetical farmer receptor is included where a member of the farming family could be exposed to COPCs. A proportion of the farmer's diet is assumed to come from home grown produce that are affected by emissions from the facility. The hypothetical resident receptor is included in the assessment where exposure could occur in an urban or non-farm rural setting. The hypothetical fisher receptor is included within the assessment where locally caught fish is the main source of protein in the receptors diet in an urban or non-farm, rural setting.

2.4.3 The impacts reported in the Air Quality Dispersion Modelling Report[1] are used within the IRAP model to predict the location of maximum concentration and deposition rates for each particular land use type. The land use of the local area is then identified and used to define the number and location of each of the relevant hypothetical receptor types e.g. a resident receptor within a residential area.

2.4.4 For each hypothetical type of receptor and within each particular land use, up to three locations are selected based on the maximum predicted airborne concentration (both long term and short term), maximum predicted dry deposition rate and maximum predicted wet deposition rate. It is not uncommon for some of these maxima points to be co-located, resulting in less than three receptor locations actually being selected.

2.4.5 The calculated total exposure to each COPC via each pathway requires the use of specific information for each receptor type. The default values within the HHRAP have been used to represent the following receptor specific parameters (as shown in Annex C):

- Food (meat, dairy products, fish and vegetables), water and soil consumption rates for each receptor type. However, only Fishers are assumed to consume locally caught 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.

Study Specific Receptors

2.4.6 The proposed EfW CHP facility is to be located on land currently in the north east of HMNB Devonport, Plymouth. The land use of the surrounding area is predominantly residential and industrial with a large portion currently in industrial use by HMNB Devonport, to the south of the proposed development site.

2.4.7 To the north and north west of the site lies the residential area of Barne Barton. This area of housing is at a higher elevation than the proposed development site. There are further residential properties within City of Plymouth to the east, north east and south east of the site at Weston Mill, St. Budeaux, King's Tamerton, Camel's Head, North Prospect and Keyham. Plymouth City Centre lies approximately 5 km to the south east of the site. The residential areas of Saltash and Torpoint are located to the north west and south west respectively, on the western side of the River Tamar estuary. There are also the separate residential areas of Plympton and Plymstock located to the east and south east of the City of Plymouth.

2.4.8 Five residential areas have been selected to represent the potential for residential receptor exposure to emissions from the proposed EfW CHP facility:

- City of Plymouth;
- Saltash;
- Torpoint;
- Plympton and
- Plymstock.

2.4.9 The land surrounding these residential areas is generally characterised by agricultural activities. Hypothetical farmer type receptors have been chosen to represent the rural areas to the north east, north west, south east and south west of the proposed development based on the predicted maximum concentration locations outside of urban areas.

2.4.10 The emissions from the proposed EfW CHP facility have been assessed for potential effects on human health at eleven hypothetical residential receptors and nine hypothetical famer receptors in the local vicinity. Both adult and child receptor types have been considered for each location. The selected hypothetical receptors and their locations are identified in Table 2.8 and shown on Figure 2.1 in Annex A.

2.4.11 The hypothetical resident and farmer receptor locations shown on Figure 2.1 in Annex A represent the location of maximum predicted impact of either air concentration (long term or short term), wet deposition or dry deposition in that particular land use defined area. All other locations within that particular land use defined area would be at a lower risk of experiencing

human health effects than the points of maximum impact, as they would have lower levels of exposure to COPCs.

Table 2.8 Hypothetical receptor type and locations used for the assessment of human health effects

Identifier	Hypothetical Receptor type	Location	Description of Maximum Impact	OS coordinates
PL1	Resident	Plymouth City	Air concentration (hourly)	245020, 57800
PL2	Resident		Air concentration (long term) and dry deposition rate	245000, 57890
PL3	Resident		Wet deposition rate	244900, 57580
PS1	Resident	Plymstock	Air concentration (hourly and long term) and dry deposition rate	248600, 53300
PS2	Resident		Wet deposition rate	251000, 53900
PT1	Resident	Plympton	Air concentration (hourly and long term), dry deposition and wet deposition rate	252400, 57300
RNE1	Farmer	Rural area to the north east of Plymouth between the River Tamar and River Plym	Air concentration (hourly)	245710, 60490
RNE2	Farmer		Air concentration (long term) and dry deposition rate	246035, 60555
RNE3	Farmer		Wet deposition rate	245580,60425
RNW1	Farmer	Rural area to the north west of Plymouth between the River Tamar and River Lynher	Air concentration (hourly)	242915, 57955
RNW2	Farmer		Air concentration (long term), dry deposition and wet deposition rate	242850, 57825
RSE1	Farmer	Rural area to the south east of Plymouth between the River Plym and the coast	Air concentration (hourly and long term) and dry deposition rate	250800, 55300
RSE2	Farmer		Wet deposition rate	252000, 56100
RSW1	Farmer	Rural area to the south west of Plymouth between the River Lynher and the sea	Air concentration (hourly)	243500, 56590
RSW2	Farmer		Air concentration (long term), dry deposition and wet deposition rate	242915, 57045
SA1	Resident	Saltash	Air concentration (hourly)	242980, 58345
SA2	Resident		Air concentration (long term), dry deposition and wet deposition rate	242850, 58150
TP1	Resident	Torpoint	Air concentration (hourly)	243760, 55485
TP2	Resident		Air concentration (long term) and dry deposition rate	242330, 55680
TP3	Resident		Wet deposition rate	243955, 55290

2.4.12 The receptor locations selected for use with this assessment of human health are hypothetical scenarios and are not necessarily representative of actual receptors within the local area. However, the hypothetical human health receptor locations in Table 2.8 and shown on Figure 2.1 in Annex A can be related to the actual receptors selected and used within the air quality dispersion modelling assessment[1]. The hypothetical receptor locations used within this assessment can be related to the nearest actual receptor locations within the local area reported within the air quality assessment, as shown in Table 2.9.

Table 2.9 Comparison of hypothetical to actual receptor locations

Hypothetical receptor location used in the assessment of health effects	Distance to EfW CHP facility (m)	Actual receptor location used in air quality assessment	Distance to EfW CHP facility (m)
PL1	348	R6	335
PL2	406	R32	420
PL3	116	R35	170
PS1	5697	R17	2,050
PS2	7188	R17	2,050
PT1	7606	R61	3,020
RNE1	3097	R65	1,430
RNE2	3263	R65	1,430
RNE3	2988	R65	1,430
RNW1	1919	R19	2,040
RNW2	1960	R19	2,040
RSE1	6410	R61	3,020
RSE2	7348	R61	3,020
RSW1	1729	R20	1,780
RSW2	1939	R20	1,780
SA1	1982	R19	2,040
SA2	2035	R19	2,040
TP1	2300	R21	2,400
TP2	3087	R57	3,050
TP3	2406	R58	2,470

2.5 Exposure Assessment for Metals and Dioxin/Furans

2.5.1 Various world government bodies have set target levels and guideline values for exposure to a variety of inorganic metals and dioxins/furans in soil and air. The Department for Environment, Food and Rural Affairs (Defra) has developed soil guideline values (SGVs) using the Contaminated Land Exposure Assessment (CLEA) model[10]. This model takes into account a number of exposure pathways including; ingestion of soil and contaminated vegetables and inhalation of dust and vapours, in order to generate limit values in soil that are set at a level for the protection for human health. The predicted soil concentrations of inorganic metals and dioxins/furans can be compared to these values to assess the effect on human health from the emissions of the proposed EfW CHP facility.

2.5.2 The latest UK Total Dietary Study (TDS) in 2006[11] and 2001[12] conducted by the Food Standards Agency provided an estimate of the total dietary intake of metals and dioxins/furans

[10] Environment Agency (2009) http://www.environment-agency.gov.uk/research/planning/33734.aspx - accessed on 25th August 2010
[11] FSA (2009) Measurement of the Concentrations of Metals and Other Elements from the 2006 UK Total Diet Study, Food Standards Agency January 2009
[12] FSA (2003) Dioxins and Dioxin-like PCBs in the UK Diet: 2001 Total Diet Study Samples, Food Standards Agency July 2003

for a range of receptors in a typical diet. The intake of metals and dioxins/furans attributed to the proposed EfW CHP facility can be compared to the intake experienced in a typical diet, as reported in the TDS, in order to assess the effect on human health.

2.5.3 A separate assessment of the contribution of Dioxins and Furans from the proposed EfW CHP facility to various food products has been made by comparison with the maximum levels specified by the European Commission[13]. The assessment within this report specifically reports results on dioxin and furan concentrations in milk and eggs, whereas food products are defined within the regulation as meat and meat products, fish, milk, eggs, oils and fats.

2.5.4 The World Health Organisation (WHO) and UK Committee on Toxicity (COT) have defined Tolerable Daily Intakes (TDI) for dioxins/furans of 1 to 4 pg I-TEQ kg-BW^{-1} d^{-1} and 2 pg I-TEQ kg-BW^{-1} d^{-1} respectively[14] [15]. The units of the TDI are defined as picogrammes of the International Toxic Equivalent per kilogram of bodyweight per day. The predicted lifetime daily intake of dioxins/furans at each receptor associated with the proposed EfW CHP facility have been compared to the above TDIs in order to assess the health risks over the lifetime of a single receptor.

2.5.5 An additional exposure pathway considered in this assessment is the infant exposure to dioxins and furans via the ingestion of their mother's breast milk. This pathway is of particular importance as dioxin like compounds are extremely lipophilic (fat soluble) and could bioaccumulate in breast milk. In addition, the lower infant body weight means they will experience a disproportionately higher impact than in an adult from the same initial exposure. The HHRAP reports a national (U.S.) average background exposure level of 60 pg TEQ kg^{-1} d^{-1} for all dioxins and furans in nursing infants. Predicted Average Daily Dose (ADD) associated with the proposed EfW CHP facility for each of the infant receptors is compared to this background exposure level in order to assess the impact on breast-fed infants from exposure to the sum of all dioxin/furans via ingestion of their mother's breast milk.

2.6 Method of Assessment for Non-Carcinogenic Effects

2.6.1 It is assumed that for most COPCs there is a threshold dose, below which no adverse effects will be observed. A reference dose is used to assess any potential health effects against exposure to COPCs exhibiting a threshold relationship. The reference dose (RfD) and reference concentration (RfC) represent a daily ingestion intake rate and a daily concentration in air respectively, at which there is no appreciable risk of adverse health effects. These reference values only identify the level below which effects are unlikely and they do not state anything about the risk for higher exposures. The reference dose and reference concentration for each COPC is provided in Table 2.6 above.

2.6.2 A Hazard Quotient (HQ) is used to assess the non-carcinogenic effects of emissions from the proposed EfW CHP facility on human health. This represents the potential to develop non-cancer health effects as a result of exposure to concentrations of COPCs. When assessing the level of exposure via the ingestion pathway the HQ is calculated as the Average Daily Dose (ADD) divided by the reference dose (RfD), as shown in equations (1) and (2) below.

[13] Commission Regulation 1881/2006, Setting of Maximum Levels for Certain Contaminants in Foodstuffs (19th December 2006)
[14] WHO (1998), Assessment of the Health Risk of Dioxins: Re-evaluation of the Tolerable Daily Intake (TDI), WHO Consultation, May 25-29 1998, Geneva, Switzerland
[15] COT (2001), Statement on the Tolerable Daily Intake for Dioxins and Dioxin like Polychlorinated Biphenyls, Committee on Toxicity, October 2001

$$HQ_{Ing,} = \frac{ADD_{Ing,}}{RfD_{Ing,}} \quad (1)$$

Where:

$$ADD_{Ing,} = \frac{I_{Ing,} \times ED \times EF}{AT \times 365} \quad (2)$$

2.6.3 Where: ADD_{Ing} = ingestion dose for the COPC; *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 (equal to *ED* for non-carcinogenic effects and 70 years for carcinogenic risks).

2.6.4 The HQ for the assessment of exposure via the inhalation pathway is calculated by dividing the exposure concentration by a reference concentration (RfC), as shown in equations (3) and (4) below.

$$HQ_{Inh} = \frac{EC}{RfC_{Inh}} \quad (3)$$

Where:

$$EC = \frac{C_a \times ED \times EF}{AT \times 365} \quad (4)$$

2.6.5 Where: *EC* is the exposure concentration of a COPC (μgm^{-3}), RfC_{Inh} is the reference concentration for a COPC (mgm^{-3}) and C_a is the concentration of the COPC in air.

2.6.6 If the daily intake is less than or equal to the reference dose, the hazard quotient would be less than or equal to 1 and this is considered to be a level that is protective of human health. A hazard quotient of greater than 1 would indicate the potential for non-carcinogenic human health effects.

2.6.7 A particular receptor has the potential to be exposed to multiple COPCs with non-carcinogenic effects. The total hazard quotient for all the COPCs exposed to a single receptor via one exposure pathway is defined by a Hazard Index (HI). The HI sums up all the individual hazard quotients from each COPC for a single pathway and assumes that the health effects from the emissions of the EfW CHP facility are additive.

2.6.8 In addition, a receptor could be exposed to the health effects of COPCs via numerous exposure pathways. The total hazard index is the sum of the individual hazard indices for each exposure pathway relevant to that receptor. This generates a total non-carcinogenic life-time risk for each individual receptor encompassing the exposure experienced via all COPCs and all relevant pathways.

2.7 Method of Assessment for Carcinogenic Effects

2.7.1 Carcinogenic risks associated with exposure to the emissions from the proposed EfW CHP facility are calculated in terms of the excess lifetime risk of developing cancer. For each of the individual COPCs, the US EPA has calculated a Carcinogenic Slope Factor (CSF) for the

ingestion exposure pathway and a Unit Risk Factor (URF) for the inhalation exposure pathway. The CSF represents an upper bound estimate of the carcinogenic risk for ingestion exposure to an individual COPC based on the does-response relationship. The URF represents a similar linear dose-response relationship albeit for concentrations in the air.

2.7.2 The probability that an individual will develop cancer over a lifetime (excess life-time risk) as a result of specific exposure to a certain carcinogenic COPC is calculated for the ingestion pathway using equation (5).

$$Risk_{Ing} = ADD_{Ing} \times CSF_{Ing} \quad (5)$$

2.7.3 Where ADD_{Ing} is the sum of the average daily dose from all ingestion exposure routes (mg/kg-day) and CSF is the cancer slope factor associated with ingestion exposure to a specific COPC $(mg/kg\text{-}day)^{-1}$.

2.7.4 The excess life-time risk of developing cancer associated with the inhalation of a specific COPC is calculated using equation (6).

$$Risk_{Inh} = EC \times URF_{Inh} \quad (6)$$

2.7.5 Where EC is the exposure concentration of a COPC (μgm^{-3}) and URF is the unit risk factor for inhalation exposure to a COPC (μgm^{-3}).

2.7.6 It is possible for a single receptor to be exposed to multiple COPCs within an individual pathway. Therefore the excess lifetime cancer risk for an exposure pathway is calculated as the sum of the cancer risks for individual COPCs for that pathway. Similarly a single receptor is at risk of being exposed to COPCs via multiple pathways. Therefore the total excess life time cancer risk for a single receptor is the sum of the total risk for all the individual exposure pathways relevant to that receptor.

2.8 Summary of Information

Inputs

2.8.1 The Chemicals of Potential Concern considered relevant to this assessment of human health effects on the local population exposed to emissions from the proposed EfW CHP facility, fall into the following three main classes: Dioxins/Furans; PAHs; and metals (including antimony, arsenic, cadmium, chromium (iii) & (vi), mercury, lead and nickel).

2.8.2 Table 2.10 shows the exposure scenarios for the each of the generic receptor types recommended by the HHRAP. An exposure scenario is defined as the relevant exposure pathways for each receptor at a specific location.

2.8.3 The study specific pathways and receptors discussed in sections 2.3 and 2.4 have been selected and considered relevant based upon Table 2.10. The ingestion of drinking water from surface water sources and the fisher exposure scenarios have not been evaluated in this assessment for the reasons outlined in section 2.3.

Table 2.10 Exposure scenarios recommended by the HHRAP[6] for each receptor type

Exposure Pathway	Recommended Exposure Scenario			
	Farmer	Farmer Child	Resident	Resident Child
Inhalation of vapour and particulates	✓	✓	✓	✓
Incidental ingestion of soil	✓	✓	✓	✓
Ingestion of home grown produce	✓	✓	✓	✓
Ingestion of home grown beef	✓	✓	×	×
Ingestion of milk from home grown cows	✓	✓	×	×
Ingestion of home grown chicken	✓	✓	a	a
Ingestion of eggs from home grown chickens	✓	✓	a	a
Ingestion of home grown pork	✓	✓	×	×
Ingestion of breast milk	b	×	b	×

(a) Site specific exposure setting characteristics (e.g. ponds on farm or presence of small livestock within residential areas) may warrant the consideration of this scenario, but has not been applied in this assessment.
(b) Infant exposure to dioxins/furans via the ingestion of their mothers breast milk is evaluated as a separate exposure pathway

Outputs

2.8.4 This assessment considers the effects on the human health of the local population when exposed to emissions from the proposed EfW CHP facility by using a number of different methods. The IRAP model calculates exposure concentrations and average daily doses experienced at each individual hypothetical receptor.

2.8.5 The exposure of receptors to metals and dioxin/furans from the proposed EfW CHP facility, via concentrations in soil and in the diet of the local population, is considered in this assessment by comparison to relevant standards and typical dietary values. The human health effect of the additional dioxin/furan concentrations associated with the emissions from the proposed EfW CHP facility are assessed by comparison with the TDI derived by the WHO and the UK COT. A separate exposure pathway is used to assess the infant exposure to dioxin/furans via the mother's breast milk by comparison to the US EPA background values.

2.8.6 In the assessment of the non-carcinogenic effects on human health a hazard quotient is calculated for each COPC for the ingestion and inhalation pathway by comparing the average dose received by a receptor to a reference dose, below which there is no appreciable risk of adverse health effects. A hazard index sums up the risk to human health experienced by a receptor to all the relevant COPCs via a single pathway and a total hazard index is calculated by combining the risks to all COPCs via all pathways.

2.8.7 Carcinogenic risk associated with exposure to the emissions from the proposed EfW CHP facility is calculated in terms of the excess lifetime risk of developing cancer at a single receptor for each COPC via the inhalation or ingestion pathway. This is done by multiplying the exposure concentration by a particular factor that takes into account the risk of developing

cancer based on the dose response relationship for that COPC. The excess lifetime cancer risk for an exposure pathway at a single receptor sums up the risk associated with the exposure to all the relevant COPCs. The total excess lifetime risk of developing cancer at a single receptor takes into account the risks associated with all the relevant COPCs via all the relevant pathways.

3 Results

3.1 Exposure Assessment

Metals

3.1.1 The maximum additional contribution to soil concentrations associated with the emissions of arsenic, cadmium, mercury, nickel and lead from the proposed EfW CHP facility, predicted at the resident and farmer receptors at the point of maximum impact in the study area are presented in Table 3.1 below. Values are also presented for the hypothetical resident SA1 receptor as it is nearest resident type receptor to the maximally impacted farmer receptor in the study area.

Table 3.1 Maximum contribution to trace metal concentrations in soil associated with the proposed EfW CHP facility for the resident and farmer receptor located at the point of maximum impact in the study area

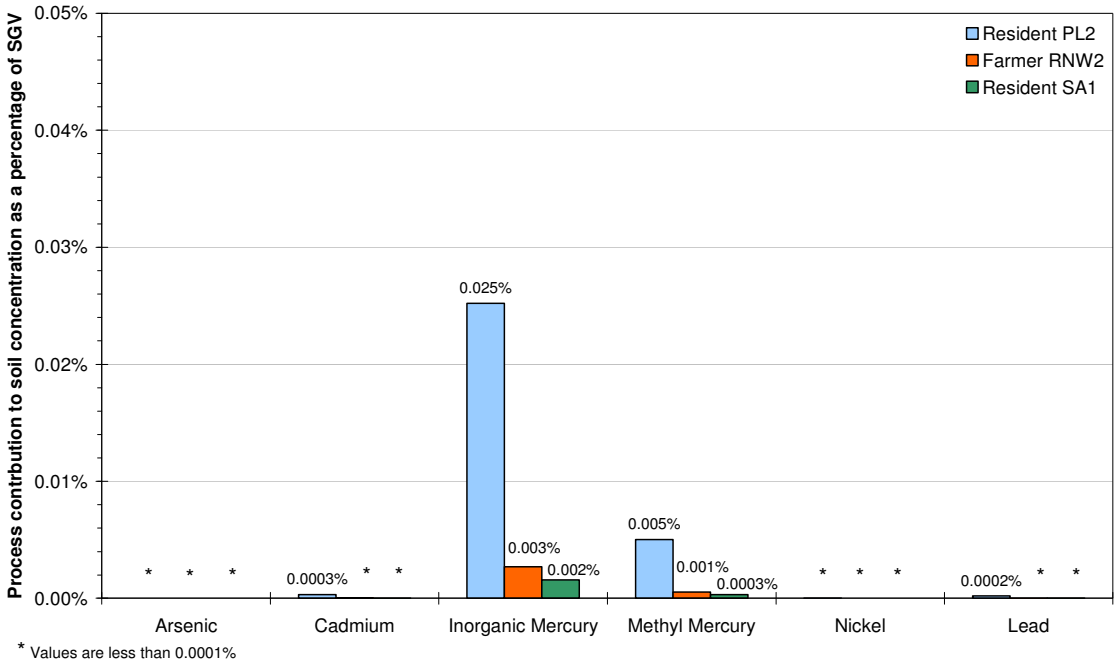
Metal	Resident PL2 (mg kg^{-1})	Farmer RNW2 (mg kg^{-1})	Resident SA1 (mg kg^{-1})	Soil Guideline Value (mg kg^{-1}) [(a)]
Arsenic	5.31×10^{-9}	5.70×10^{-10}	3.29×10^{-10}	32
Cadmium	5.68×10^{-6}	6.10×10^{-7}	3.52×10^{-7}	1.8
Inorganic Mercury	2.02×10^{-2}	2.17×10^{-3}	1.25×10^{-3}	80
Methyl Mercury	4.03×10^{-4}	4.33×10^{-5}	2.50×10^{-5}	8
Nickel	1.66×10^{-5}	1.78×10^{-6}	1.03×10^{-6}	130
Lead	8.45×10^{-4}	9.08×10^{-5}	5.24×10^{-5}	450

(a) Most stringent SGV used for each substance

3.1.2 A comparison of the predicted contribution to the soil concentrations associated with the proposed EfW CHP facility for each metal as a percentage of the most stringent SGV is presented in Figure 3.1.

3.1.3 The highest contribution to soil concentrations are predicted for inorganic mercury at the resident PL2 location, as it is nearer the EfW CHP facility and located in the downwind direction. The largest additional contribution to soil concentrations at the hypothetical farmer RNW2 receptor are also from inorganic mercury but are a factor of ten less than those predicted for the hypothetical resident PL2 receptor. Contributions to the concentrations of inorganic mercury are predicted to be less than 0.025% of the SGV at the resident PL2 location. All other predicted contributions to soil concentrations for arsenic, cadmium, methyl mercury, nickel and lead are less than 0.01% of the relevant SGV.

Figure 3.1 Predicted Maximum Contribution to Metal Concentrations in Soil as a Percentage of the Most Stringent SGV for Receptors Located at the Point of Maximum Impact



3.1.4 The predicted dietary intake of metals associated with the emissions from the proposed EfW CHP facility for the resident and farmer receptor types located at the point of maximum impact in the study area are shown in Table 3.2 below. The typical dietary intake of these substances obtained from the UK TDS in 2006[11] has been provided in Table 3.2 for comparison purposes.

Table 3.2 Dietary intake of metals associated with the proposed EfW CHP facility for the resident and farmer receptors located at the points of maximum impact

Metal	Resident PL2 (µg kg-BW^{-1} d^{-1})	Farmer RNW2 (µg kg-BW^{-1} d^{-1})	Resident SA1 (µg kg-BW^{-1} d^{-1})	UK TDS Intake (µg kg-BW^{-1} d^{-1}) [(a)]
Arsenic	3.75×10^{-5}	1.06×10^{-5}	2.22×10^{-6}	1.65 - 1.68
Cadmium	6.24×10^{-4}	1.03×10^{-4}	3.69×10^{-5}	1.4×10^{-1} - 1.7×10^{-1}
Chromium (b)	4.91×10^{-4}	6.38×10^{-4}	2.93×10^{-5}	2.8×10^{-1} - 3.7×10^{-1}
Lead	6.25×10^{-3}	2.18×10^{-3}	3.70×10^{-4}	9.0×10^{-2} - 1.0×10^{-1}
Mercury (c)	4.72×10^{-4}	1.80×10^{-4}	2.92×10^{-5}	2.0×10^{-2} - 5.0×10^{-2}
Nickel	1.69×10^{-3}	1.93×10^{-3}	1.00×10^{-4}	1.49 - 1.63
Thallium	6.68×10^{-4}	2.38×10^{-3}	3.96×10^{-5}	1.1×10^{-2} - 1.2×10^{-2}

(a) Mean exposure for an adult
(b) Total chromium (trivalent and hexavalent)
(c) Total mercury (organic and inorganic)

3.1.5 The hypothetical resident receptor location (PL2) would experience a greater impact on dietary intake of any metal emitted from the EfW CHP facility, than would be experienced at any other location within the City of Plymouth or the wider study area. The impact on dietary intake varies in magnitude for each metal. The largest absolute change in dietary intake is predicted for the metal lead. This location is representative of a hypothetical resident receptor whose main exposure pathway is via the ingestion of home grown fruits and vegetables and some incidental ingestion of soil. The dietary intake obtained from the TDS in 2006 is typical of intake rates of

metals for adults in the UK population that obtain the majority of their food from retail stores. The maximum predicted in take at this location within Plymouth (PL2), can be considered conservative as it ignores the fact that most consumed food stuffs will be sourced from retail operations in the vicinity and as such represents a robust assessment of the impact of emissions from the EfW CHP facility on daily intake rates.

3.1.6 The predicted maximum dietary intake for the hypothetical receptor scenarios can be compared to the typical dietary intake rates for each of the metals obtained from the UK TDS in 2006 list in Table 3.2. For example the predicted dietary intake of lead in the maximum exposed hypothetical resident type receptor in Plymouth (PL2) of 6.2 x 10^{-3} µg kg-BW^{-1} d^{-1} are markedly less than the equivalent typical dietary intake value of 9.0 x 10^{-2} – 1.0 x 10^{-1} µg kg-BW^{-1} d^{-1}. For mercury (both organic and inorganic) a dietary intake of 4.72 x 10^{-4} µg kg-BW^{-1} d^{-1} was predicted at the maximally impacted hypothetical resident type receptor in Plymouth (PL2), while a typical dietary intake value of 2.0 x 10^{-2} - 5.0 x 10^{-2} µg kg-BW^{-1} d^{-1} was obtained from the UK TDS in 2006.

3.1.7 The maximum exposed hypothetical farmer type receptor (RNW2) would experience a greater impact on the dietary intake rate of each metal emitted from the EfW CHP facility, than would be experienced at any other rural location within the study area. This receptor is a hypothetical receptor location and conservatively assumes that a significant proportion of the farmer's diet comes from home grown/reared food and animal produce. At this location the predicted maximum dietary intake of lead for the hypothetical farmer RNW2 receptor scenario of 2.18 x 10^{-3} µg kg-BW^{-1} d^{-1} is less than the typical dietary value of 9.0 x 10^{-2} - 1.0 x 10^{-1} µg kg-BW^{-1} d^{-1} from the UK TDS. The predicted dietary intake of mercury (both organic and inorganic) of 1.80 x 10^{-4} µg kg-BW^{-1} d^{-1} can be compared to the typical dietary values of 2.0 x 10^{-2} - 5.0 x 10^{-2} µg kg-BW^{-1} d^{-1} obtained from the UK TDS in 2006.

3.1.8 In practise the maximum impact on dietary intake of all metals at farmer type receptors would fall between the hypothetical scenario represented by RNW2 and the nearby hypothetical urban resident scenario for Saltash (SA1). The greater the proportion of shop bought food in the household diet of these receptors the closer the dietary intake values for these metals would be to the typical values presented in the UK TDS.

Dioxins/Furans

3.1.9 The maximum additional contribution to soil concentrations associated with the emissions of dioxins/furans from the proposed EfW CHP facility, predicted at the resident and farmer receptors located at the point of maximum impact in the study area, are presented in Table 3.3 below.

Table 3.3 Maximum contributions to soil concentrations of dioxins/furans associated with the proposed EfW CHP facility for the resident and farmer receptors located at the point of maximum impact in the study area

COPC	Resident PL2 (µg kg^{-1})	Farmer RNW2 (µg kg^{-1})	Resident SA1 (µg kg^{-1})	Soil Guideline Value (µg kg^{-1}) [(a)]
Total PCDD/PCDF	3.56 x 10^{-4}	3.83 x 10^{-5}	2.21 x 10^{-5}	8

3.1.10 The largest additional contribution of dioxins and furans to soil concentrations associated with the proposed EfW CHP facility is predicted to occur at the hypothetical resident PL2 scenario. This additional contribution to soil concentrations represents 0.0044% of the Soil Guideline Value for total dioxins and furans. All other additional contributions of dioxins and furans to the

soil concentration at the other hypothetical farmer and resident receptor locations are predicted to be below 0.001% of the Soil Guideline Value. The additional contribution of the proposed EfW CHP facility to the concentrations of dioxins and furans in milk and eggs at the maximally impacted farmer receptors in each of the rural areas considered in this assessment are shown in Table 3.4.

Table 3.4 Predicted contributions to dioxin/furan concentrations in milk and eggs associated with the proposed EfW CHP facility for the maximally impacted farmer receptors in each of the rural areas considered in this assessment

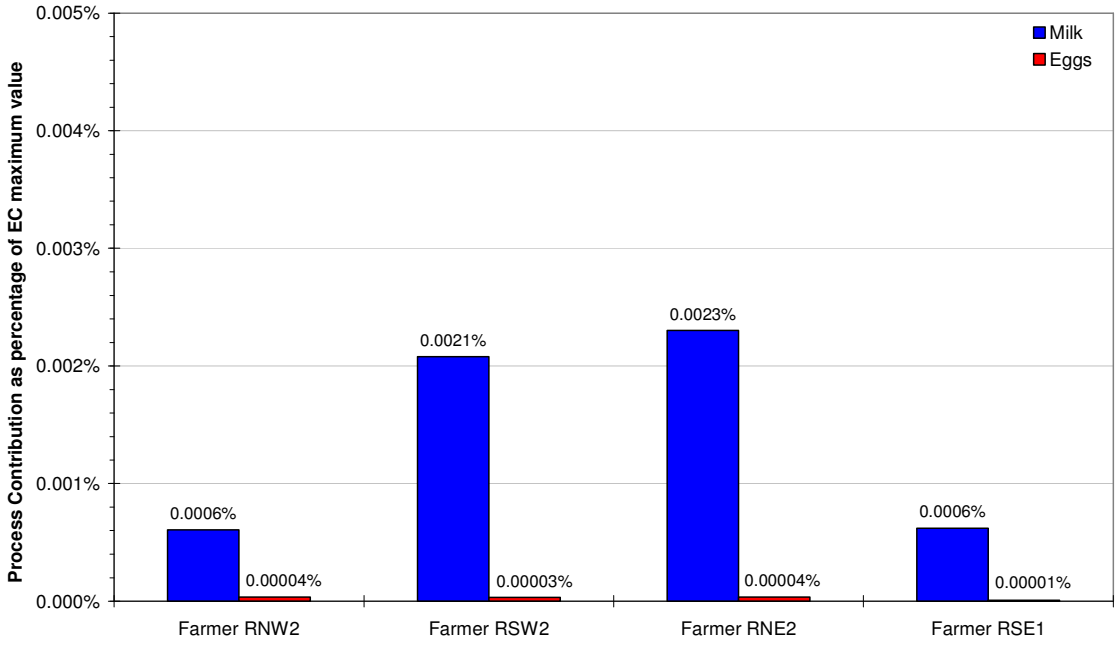
Farmer Receptor	Concentration in Milk [(a)] (pg I-TEQ g^{-1} fat)	Concentration in Eggs [(b)] (pg I-TEQ g^{-1} fat)
Farmer RNW2	1.82×10^{-5}	1.10×10^{-6}
Farmer RSW2	6.24×10^{-5}	9.53×10^{-7}
Farmer RNE2	6.90×10^{-5}	1.08×10^{-6}
Farmer RSE1	1.86×10^{-5}	2.73×10^{-7}
Maximum European Level[13]	3	3

(a) Assuming a fat content of milk of 3% (b) Assuming a fat content of eggs of 12%

3.1.11 A comparison of the predicted additional dioxin/furan concentrations in milk and eggs associated with the proposed EfW CHP facility as a percentage of the maximum European levels[13] is presented in Figure 3.2.

3.1.12 The largest additional contribution to the concentration of dioxins and furans in milk associated with the EfW CHP facility occurs in the hypothetical farmer RNE2 scenario. This largest additional concentration represents less than 0.0025% of the maximum European level[13]. The largest additional contribution to the concentration of dioxin and furans in eggs is predicted to occur in the hypothetical farmer RNW2 scenario, which represents less than 0.001% of the maximum permitted European level.

Figure 3.2 Predicted Additional Dioxin/Furan Concentrations in Milk and Eggs as a Percentage of the Maximum European Permitted Levels at the Maximally Impacted Farmer Receptors



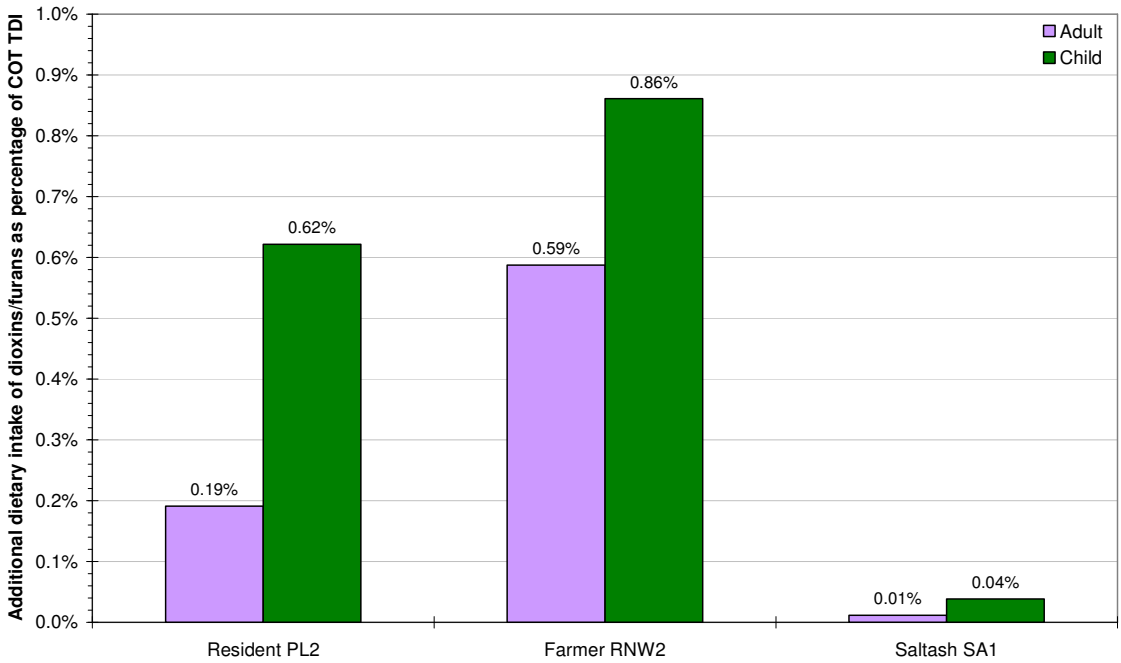
3.1.13 The average daily intake of dioxins and furans associated with the proposed EfW CHP facility over the lifetime of the resident and farmer receptors, located at the point of maximum impact in the study area, is shown in Table 3.5. These values are presented along with the WHO and COT tolerable daily intake values for comparison purposes.

Table 3.5 Average daily intake of dioxins/furans associated with the proposed EfW CHP facility for the adult and child of each resident and farmer receptor, located at the point of maximum impact in the study area

Receptor	Adult (pg I-TEQ kg-BW^{-1} d^{-1})	Child (pg I-TEQ kg-BW^{-1} d^{-1})
Resident PL2	0.0038	0.0124
Farmer RNW2	0.0117	0.0172
Resident SA1	0.0002	0.0008
COT TDI[15]	2 pg I-TEQ kg-BW^{-1} d^{-1}	
WHO TDI[14]	1 to 4 pg I-TEQ kg-BW^{-1} d^{-1}	

3.1.14 The predicted average daily intake of dioxins and furans have been directly compared as a percentage of the COT TDI value, as shown in Figure 3.3.

Figure 3.3 Predicted Daily Intake of Dioxin/Furan for Receptors Located at the Point of Maximum Impact as a percentage Tolerable Daily Intake

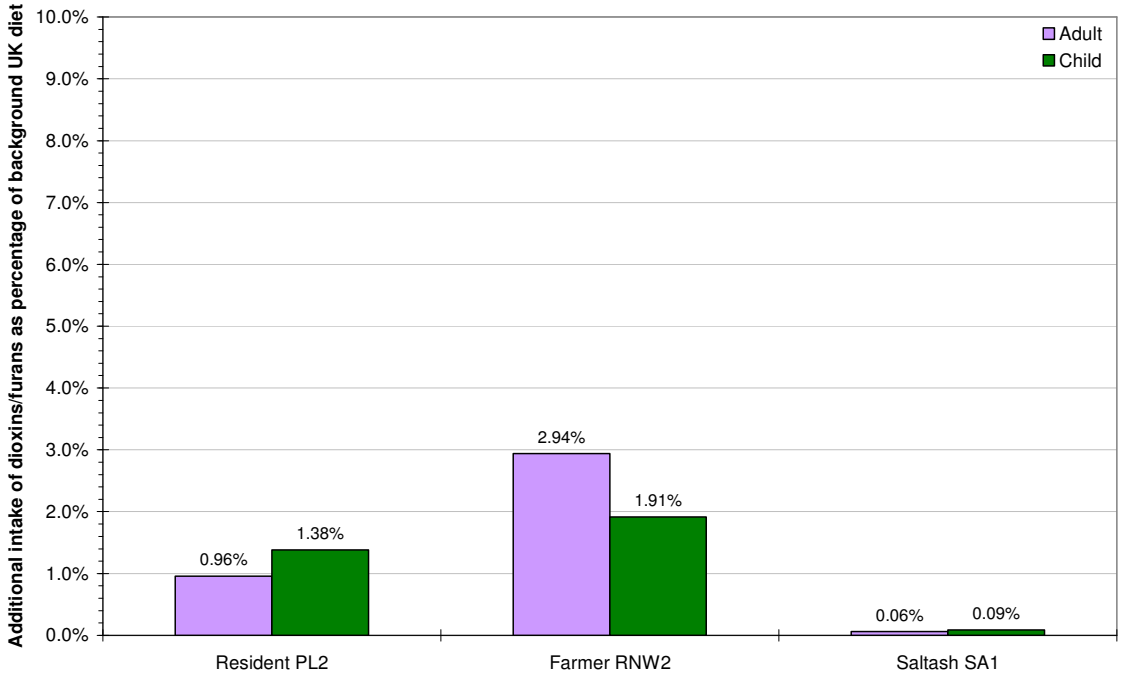


3.1.15 The total dioxins and furans associated with the EfW CHP facility across all hypothetical receptor scenarios are predicted to contribute less than 1.0% of the COT TDI value. The hypothetical farmer's child receptor type (RNW2) is predicted to experience an impact that equates to 0.86% of the COT TDI value and the impact at all other child or adult receptors within the study area would be smaller in magnitude. The daily intake predicted at RNW2 for the Farmer's child receptor type is approximately 100 times smaller than the COT TDI value and the lower range value of the WHO TDI.

3.1.16 The daily intake of dioxins and furans in the hypothetical resident PL2 scenario for the child receptor type is predicted to contribute a maximum of 0.62% of the COT TDI. The other hypothetical resident SA1 scenario is predicted to contribute less than 0.1% to the COT TDI for both the adult and child receptor types. SA1 is the nearest resident type receptor to the farmer RNW2 receptor and predicted impacts are more than a factor of 10 less, for this receptor type that does not consume home grown meat.

3.1.17 The predicted average daily intake of dioxins and furans associated with the proposed EfW CHP facility over the lifetime of the same receptors identified above can also be compared to the typical dietary intake of these substances, as obtained from the UK TDS undertaken in 2001. The predicted intake of dioxins and furans as a percentage of the typical UK dietary intake is presented in Figure 3.4.

Figure 3.4 Predicted Daily Intake of Dioxin/Furan for Receptors Located at the Point of Maximum Impact as a Percentage of typical UK Dietary Values



3.1.18 The most recently available data from the FSA have shown that dioxin and furan levels in the UK diet are declining. The analysis of the 2001 TDS samples for dioxin and furan concentrations have reported average daily intakes for adults and children (aged 4 – 6 years) of 0.4 and 0.9 pg kg-BW^{-1} day^{-1} respectively. This is a decrease from the 1997 values of 0.9 and 2.1 pg kg-BW^{-1} day^{-1} for an adult and child respectively.

3.1.19 The predicted dietary intake of dioxins and furans associated with the proposed EfW CHP facility represents less than 3% of the 2001 UK dietary values for all hypothetical receptor scenarios. The largest contributions to the typical dietary values are predicted to occur in the hypothetical farmer RNW2 scenario for the adult receptor type. The largest contribution to the typical dietary values of 1.36% and 0.96% is predicted to occur at the hypothetical PL2 location for the child and adult receptor types respectively. At the nearest hypothetical resident receptor (SA1) to Farmer RNW2, the contribution from the operation of the facility represents less than 0.1 % of the typical UK dietary intake values. This is a significant reduction in values for a

similar geographical location and reflects the conservative nature of the assessment for impacts on the rural community.

3.1.20 The predicted additional average daily dose of dioxins/furans associated with the proposed EfW CHP facility experienced by infants via their mother's breast milk for the resident and farmer receptors types located at the point of maximum impact in the study area, is shown in Table 3.6.

Table 3.6 Additional average daily dose of dioxins/furans associated with the proposed EfW CHP facility for infants via exposure from their mother's breast milk at the resident and farmer receptors types located at the point of maximum impact in the study area

Receptor	Additional Average daily dose from breast feeding (pg I-TEQ kg^{-1} d^{-1})
Resident PL2	0.042
Farmer RNW2	0.139
Resident SA1	0.003
US EPA Criteria	60
WHO Criteria	1 to 4
UK COT Criteria	2

3.1.21 The largest additional average daily dose (ADD) in an infant from breast feeding is predicted to occur in the hypothetical farmer RNW2 scenario, which represents less than 0.25% of the US EPA criteria value and less than 7% of the UK COT value. The corresponding additional ADD predicted in the hypothetical resident PL2 and SA1 scenario are approximately a factor of 10 and 100 respectively less than the ADD predicted in the farmer RNW2 scenario.The farmer receptor scenarios are assumed to consume locally grown and reared animal products, which are the most significant exposure route for dioxins and furans, whereas the resident scenario assumes a more varied and predominantly non local source for its diet. The predicted additional ADDs for farmer receptor scenarios are therefore larger than those for resident scenarios as exposure to dixoins/furans mainly occurs through the food chain.

3.1.22 The predicted additional ADD for all the hypothetical receptor scenarios are at least a factor of 10 below both the COT TDI value and the lower range of the WHO TDI value. The duration of exposure via the breast fed infant pathway to these additional ADD values is short, with the ADD over the lifetime of an individual significantly lower and similar to the values presented in Table 3.5.

3.2 Assessment of Non-Carcinogenic Effects

Non-Carcinogenic Effects by Receptor Type

3.2.1 The exposure concentrations experienced at the most sensitive receptors from emissions of each COPC associated with the proposed EfW CHP facility via inhalation and ingestion, represented by exposure concentrations and average daily doses respectively, are presented in Tables 3.7 to 3.9. The individual HQs, calculated for each COPC for each receptor using the method in section 2.6 by dividing the predicted exposure concentrations by reference concentrations, are also presented in the same tables below. In addition, the HI for each exposure pathway for all the COPCs along with the total HI for that receptor has been calculated.

Table 3.7 Summary of the exposure experienced by the resident PL2 child receptor for each COPC via inhalation and ingestion

COPC	Exposure Concentration (µg m^{-3}) Inhalation	Average daily dose (mg kg^{-1} day^{-1}) Ingestion	Hazard Quotient (HQ)	
			Inhalation	Ingestion
Antimony	6.26×10^{-3}	1.65×10^{-10}	4.29×10^{-3}	6.17×10^{-6}
Arsenic	3.75×10^{-5}	5.34×10^{-9}	1.20×10^{-3}	2.88×10^{-4}
Cadmium	6.26×10^{-4}	1.50×10^{-6}	3.00×10^{-3}	3.60×10^{-3}
Total Chromium	4.13×10^{-4}	7.78×10^{-8}	7.47×10^{-8}	8.30×10^{-7}
Chromium (VI)	8.67×10^{-6}	1.65×10^{-9}	1.04×10^{-3}	8.79×10^{-6}
Lead	6.26×10^{-3}	8.91×10^{-7}	4.00×10^{-3}	3.37×10^{-2}
Mercuric Chloride	4.50×10^{-5}	7.42×10^{-8}	3.92×10^{-5}	3.83×10^{-3}
Methyl mercury	-	4.26×10^{-9}	-	6.65×10^{-4}
Nickel	1.70×10^{-3}	2.41×10^{-7}	8.16×10^{-3}	1.95×10^{-4}
Thallium	6.26×10^{-4}	2.54×10^{-6}	2.14×10^{-3}	3.04×10^{-2}
2,3,7,8-TCDD	3.84×10^{-11}	1.67×10^{-14}	-	2.55×10^{-2}
HI for Exposure Pathway			0.024	0.073
Total Hazard Index			0.097	

Table 3.8 Summary of the exposure experienced by the Farmer RNW2 child receptor for each COPC via inhalation and ingestion

COPC	Exposure Concentration (µg m^{-3}) Inhalation	Average daily dose (mg kg^{-1} day^{-1}) Ingestion	Hazard Quotient (HQ)	
			Inhalation	Ingestion
Antimony	6.54×10^{-4}	3.60×10^{-10}	4.48×10^{-4}	8.63×10^{-7}
Arsenic	3.92×10^{-6}	1.86×10^{-8}	1.25×10^{-4}	5.95×10^{-5}
Cadmium	6.54×10^{-5}	2.39×10^{-7}	3.13×10^{-4}	5.72×10^{-4}
Total Chromium	4.31×10^{-5}	1.02×10^{-6}	7.80×10^{-9}	6.52×10^{-7}
Chromium (VI)	9.06×10^{-7}	2.16×10^{-8}	1.09×10^{-4}	6.90×10^{-6}
Lead	6.54×10^{-4}	4.26×10^{-6}	4.18×10^{-4}	9.51×10^{-3}
Mercuric Chloride	4.70×10^{-6}	3.23×10^{-7}	4.10×10^{-6}	1.03×10^{-3}
Methyl mercury	-	1.48×10^{-8}	-	1.42×10^{-4}
Nickel	1.78×10^{-4}	2.94×10^{-6}	8.52×10^{-4}	1.41×10^{-4}
Thallium	6.54×10^{-5}	2.78×10^{-6}	2.24×10^{-4}	3.33×10^{-2}
2,3,7,8-TCDD	4.02×10^{-12}	5.23×10^{-13}	-	5.02×10^{-4}
HI for Exposure Pathway			0.002	0.045
Total Hazard Index			0.048	

Table 3.9 Summary of the exposure experienced by the resident SA1 child receptor for each COPC via inhalation and ingestion

COPC	Exposure Concentration ($\mu g\ m^{-3}$) Inhalation	Average daily dose ($mg\ kg^{-1}\ day^{-1}$) Ingestion	Hazard Quotient (HQ)	
			Inhalation	Ingestion
Antimony	3.64×10^{-4}	1.65×10^{-10}	2.49×10^{-4}	3.95×10^{-7}
Arsenic	2.19×10^{-6}	5.34×10^{-9}	6.99×10^{-5}	1.71×10^{-5}
Cadmium	3.64×10^{-5}	8.88×10^{-8}	1.75×10^{-4}	2.13×10^{-4}
Total Chromium	2.40×10^{-5}	7.78×10^{-8}	4.35×10^{-9}	4.97×10^{-8}
Chromium (VI)	5.05×10^{-7}	1.65×10^{-9}	6.05×10^{-5}	5.26×10^{-7}
Lead	3.64×10^{-4}	8.91×10^{-7}	2.33×10^{-4}	1.99×10^{-3}
Mercuric Chloride	2.62×10^{-6}	7.42×10^{-8}	2.28×10^{-6}	2.37×10^{-4}
Methyl mercury	-	4.26×10^{-9}	-	4.09×10^{-5}
Nickel	9.91×10^{-5}	2.41×10^{-7}	4.75×10^{-4}	1.16×10^{-5}
Thallium	3.64×10^{-5}	1.53×10^{-7}	1.25×10^{-4}	1.84×10^{-3}
2,3,7,8-TCDD	2.24×10^{-12}	1.67×10^{-14}	-	1.60×10^{-5}
HI for Exposure Pathway			0.0014	0.0044
Total Hazard Index			0.006	

3.2.2 The HQs is a comparison of the predicted oral and inhalation exposure estimates to the reference dose and concentration values. A total Hazard Index value of 1 or less represents a level of exposure below which no appreciable risk of adverse health effects, even to sensitive populations, over a 70 year time period would occur.

3.2.3 The largest HQs for the inhalation pathway are predicted for nickel in the resident and farmer hypothetical receptor scenarios, which represent approximately 30-40% of the total HI for that pathway. Larger HQs are predicted for the child type of receptor for both the farmer and resident receptor scenarios. The ingestion HQ for lead is predicted to be the largest for the child resident PL2 receptor scenario out of all the hypothetical receptor scenarios and represents approximately 45% of the total HI for that exposure pathway. In the other resident and farmer hypothetical scenarios for the child receptor type, the largest HQ is predicted for lead and thallium respectively, which represent approximately 45% and 75% of the total respectively. For the majority of COPCs the HQs predicted at the resident SA1 receptor are approximately a factor of between 5 to 10 less than those at the nearby farmer RNW2 receptor type.

3.2.4 Contributions to the hazard index for the ingestion exposure pathway are also predicted for cadmium and mercuric chloride. Antimony and lead are predicted to provide a contribution to the HI for the inhalation exposure pathway for each hypothetical receptor scenario.

Non-Carcinogenic Effects by Pathway

3.2.5 The HIs calculated for each exposure pathway, which takes into account the HQs for exposure to all COPCs via this pathway, for the most sensitive receptors are shown in Table 3.10.

Table 3.10 Summary of the Hazard Indices for each exposure pathway for the most sensitive receptors

Pathway	HI for Resident PL2 Child Receptor	HI for Farmer RNW2 Child Receptor	HI for Resident SA1 Child Receptor
Inhalation	0.024	0.002	0.001
Ingestion of above ground vegetables	0.061	0.009	0.004
Ingestion of beef	-	0.010	-
Ingestion of chicken	-	0.000002	-
Ingestion of eggs	-	0.000002	-
Ingestion of milk	-	0.025	-
Ingestion of pork	-	0.000006	-
Ingestion of soil	0.012	0.001	0.0007
Total Hazard Index	0.097	0.048	0.006

3.2.6 The total HI for the hypothetical resident PL2 child receptor scenario is approximately a factor of 10 larger than that of the resident SA1 child receptor scenario, located in Saltash and 2 times larger than that of the farmer RNW2 child. For both the hypothetical child resident receptor scenarios the largest non-carcinogenic risk occurs via the ingestion of above ground vegetables pathways, which represents approximately 60% of the total HI. The ingestion of food products and in particular the ingestion of milk is predicted to be the largest non-carcinogenic pathway risk for the hypothetical child farmer receptor scenario, which represents more than 50% of the total HI.

3.2.7 The total HI for the farmer RNW2 is approximately a factor of 10 larger than the nearby resident SA1 type receptor indicating that the extra risk for the farmer type of receptor occurs via the ingestion of locally grown vegetable products and locally reared animal products. The relative contribution of each pathway to the total hazard index value are consistent with experience in most studies. None of the total hazard index values determined in this study represent a significant effect.

Summary of Non-Carcinogenic Effects

3.2.8 The total Hazard Index for each receptor, which takes into account the cumulative risk for each COPC via each pathway, calculated by IRAP is presented in Table 3.11.

Table 3.11 Summary of the total Hazard Index for each receptor type

Receptor Name	Receptor Type	Total Hazard Index (HI)
PL1	Adult Resident	0.040
PL1	Child Resident	0.077
PL2	Adult Resident	0.051
PL2	Child Resident	**0.097**
PL3	Adult Resident	0.009
PL3	Child Resident	0.029
PS1	Adult Resident	0.001
PS1	Child Resident	0.002
PS2	Adult Resident	0.001
PS2	Child Resident	0.002
PT1	Adult Resident	0.002
PT1	Child Resident	0.003
SA1	Adult Resident	0.003
SA1	Child Resident	0.006
SA2	Adult Resident	0.004
SA2	Child Resident	0.007
TP1	Adult Resident	0.001
TP1	Child Resident	0.003
TP2	Adult Resident	0.002
TP2	Child Resident	0.003
TP3	Adult Resident	0.001
TP3	Child Resident	0.003
RNE1	Adult Farmer	0.034
RNE1	Child Farmer	0.044
RNE2	Adult Farmer	0.035
RNE2	Child Farmer	0.044
RNE3	Adult Farmer	0.034
RNE3	Child Farmer	0.044
RNW1	Adult Farmer	0.034
RNW1	Child Farmer	0.043
RNW2	Adult Farmer	0.037
RNW2	Child Farmer	**0.048**
RSE1	Adult Farmer	0.010
RSE1	Child Farmer	0.012
RSE2	Adult Farmer	0.009
RSE2	Child Farmer	0.012
RSW1	Adult Farmer	0.021
RSW1	Child Farmer	0.027
RSW2	Adult Farmer	0.032
RSW2	Child Farmer	0.041
Criterion		1.0

3.2.9 All of the Total Hazard Indices presented in Table 3.11 for each of the individual hypothetical receptor scenarios represent values that are at least one order of magnitude lower than the reference dose at which there is an appreciable risk of health effects occurring over a 70 year lifetime..

3.2.10 The hypothetical child resident type receptor (PL2), which is located in the Plymouth urban area at the point of maximum deposition. The total Hazard Index at PL2 of 0.097 is approximately an order of magnitude lower than the reference dose (HI value of 1.0), at which there is an appreciable risk of health effects occurring over the lifetime of an individual. The risk of the operation of the EfW CHP facility resulting in non-carcinogenic health effects at locations within the City of Plymouth is low near the point of maximum impact and decreases to very low with distance from the facility.

3.2.11 Outside of the City of Plymouth, the total Hazard Index for the hypothetical farmer's child type receptors (RNW2) of 0.048, is a factor of approximately 20 less than the reference dose (HI value of 1.0) at which there is an appreciable risk of health effects occurring over the lifetime of an individual. This represents the impact on a maximum exposed hypothetical member of the rural community. The total Hazard Index for the maximum exposed hypothetical resident type receptor in Saltash (SA1) is 0.003 and a factor of approximately 10 less than that of the rural receptor RNW2. The risk of the operation of the EfW CHP facility resulting in non-carcinogenic health effects at any rural location or urban area outside the City of Plymouth is very low.

3.3 Assessment of Carcinogenic Effects

Carcinogenic Effects for each COPC

3.3.1 The exposure concentrations experienced at the most sensitive receptors from the emissions of each COPC associated with the proposed EfW CHP facility via inhalation and ingestion, represented by exposure concentrations and average daily doses respectively, are presented in Tables 3.12 to 3.14. The individual lifetime risk of developing cancer are also presented in the same tables below and are calculated for each COPC at each receptor using the method in section 2.7 by multiplying the predicted exposure concentrations by the relevant carcinogenic risk factor for inhalation and ingestion. In addition, the excess lifetime cancer risk for each exposure pathway encompassing all the COPCs and the total excess lifetime cancer risk for that receptor has been calculated.

Table 3.12 Summary of the exposure experienced by the resident PL2 adult receptor for each COPC via inhalation and ingestion

COPC	Exposure Concentration ($\mu g\ m^{-3}$) Inhalation	Average daily dose ($mg\ kg^{-1}\ day^{-1}$) Ingestion	Lifetime Cancer Risk	
			Inhalation	Ingestion
Arsenic	3.75×10^{-5}	3.75×10^{-8}	6.63×10^{-8}	2.31×10^{-8}
Benzo(a)pyrene	1.25×10^{-5}	1.62×10^{-8}	5.66×10^{-9}	4.85×10^{-8}
Cadmium	6.26×10^{-4}	6.24×10^{-7}	4.63×10^{-7}	9.74×10^{-8}
Chromium (VI)	8.67×10^{-6}	1.03×10^{-8}	4.28×10^{-8}	-
Lead	6.26×10^{-3}	6.25×10^{-6}	3.09×10^{-8}	2.18×10^{-8}
Nickel	1.70×10^{-3}	1.69×10^{-6}	1.68×10^{-7}	-
Total dioxins/furans	-	2.72×10^{-12}	-	6.02×10^{-8}
Total Lifetime Risk for Exposure Pathway			7.76×10^{-7}	2.51×10^{-7}
Total Lifetime Risk for receptor			1.03×10^{-6}	

Table 3.13 Summary of the exposure experienced by the farmer RNW2 adult receptor for each COPC via inhalation and ingestion

COPC	Exposure Concentration ($\mu g\ m^{-3}$) Inhalation	Average daily dose ($mg\ kg^{-1}\ day^{-1}$) Ingestion	Lifetime Cancer Risk	
			Inhalation	Ingestion
Arsenic	3.92×10^{-6}	1.06×10^{-8}	9.24×10^{-9}	8.75×10^{-9}
Benzo(a)pyrene	1.31×10^{-6}	1.68×10^{-7}	7.88×10^{-10}	6.72×10^{-7}
Cadmium	6.54×10^{-5}	1.03×10^{-7}	6.45×10^{-8}	2.14×10^{-8}
Chromium (VI)	9.06×10^{-7}	1.34×10^{-8}	5.95×10^{-9}	-
Lead	6.54×10^{-4}	2.18×10^{-6}	4.30×10^{-9}	1.02×10^{-8}
Nickel	1.78×10^{-4}	1.93×10^{-6}	2.34×10^{-8}	-
Total dioxins/furans	-	1.08×10^{-11}	-	3.61×10^{-7}
Total Lifetime Risk for Exposure Pathway			1.08×10^{-7}	1.07×10^{-6}
Total Lifetime Risk for receptor			1.18×10^{-6}	

Table 3.14 Summary of the exposure experienced by the resident SA1 adult receptor for each COPC via inhalation and ingestion

COPC	Exposure Concentration ($\mu g\ m^{-3}$) Inhalation	Average daily dose ($mg\ kg^{-1}\ day^{-1}$) Ingestion	Lifetime Cancer Risk	
			Inhalation	Ingestion
Arsenic	2.19×10^{-6}	2.22×10^{-9}	3.86×10^{-9}	1.37×10^{-9}
Benzo(a)pyrene	7.28×10^{-7}	9.87×10^{-10}	3.29×10^{-10}	2.96×10^{-9}
Cadmium	3.64×10^{-5}	2.90×10^{-8}	2.69×10^{-8}	5.77×10^{-9}
Chromium (VI)	5.05×10^{-7}	6.14×10^{-10}	2.49×10^{-9}	-
Lead	3.64×10^{-4}	3.70×10^{-7}	1.80×10^{-9}	1.29×10^{-9}
Nickel	9.91×10^{-5}	1.00×10^{-7}	9.77×10^{-9}	-
Total dioxins/furans	-	1.70×10^{-13}	-	3.70×10^{-9}
Total Lifetime Risk for Exposure Pathway			4.52×10^{-8}	1.51×10^{-8}
Total Lifetime Risk for receptor			6.03×10^{-8}	

3.3.2 The largest predicted lifetime cancer risk via the inhalation exposure pathway is for cadmium for the hypothetical resident and farmer receptor scenarios. Exposure via inhalation of cadmium represents approximately 60% of the total lifetime cancer risk for exposure to all COPCs via the inhalation pathway for each hypothetical receptor.

3.3.3 For the hypothetical farmer receptor scenarios the largest contribution to the lifetime cancer risk via the ingestion exposure pathway is predicted to occur for the total dioxins/furans and benzo[a]pyrene. Taken together the exposure via ingestion to total dioxins/furans and benzo[a]pyrene represents over 90% of the total lifetime cancer risk via the ingestion pathway for the hypothetical farmer receptor type. The largest contribution to the lifetime cancer risk via the ingestion exposure pathway for the hypothetical resident receptor types is predicted to occur for cadmium. Exposure to cadmium via the ingestion pathway at these locations represents approximately 40% of the total lifetime cancer risk for this receptor via this pathway with contributions also occurring for total dioxins/furans and benzo[a]pyrene.

Carcinogenic Effects for each Pathway

3.3.4 The total lifetime cancer risks calculated for each exposure pathway, which takes into account the risk for exposure to all COPCs via this pathway, for the most sensitive receptors are shown in Table 3.15.

Table 3.15 Summary of the total lifetime cancer risk for each exposure pathway for the most sensitive receptors

Pathway	Lifetime Risk for Resident PL2 adult Receptor	Lifetime Risk for Farmer RNW2 adult Receptor	Lifetime Risk for Resident SA1 adult Receptor
Inhalation	7.76×10^{-7}	1.08×10^{-7}	4.52×10^{-8}
Ingestion of above ground vegetables	2.44×10^{-7}	4.91×10^{-8}	1.46×10^{-8}
Ingestion of beef	-	2.27×10^{-7}	-
Ingestion of chicken	-	2.66×10^{-10}	-
Ingestion of drinking water	-	-	-
Ingestion of eggs	-	1.73×10^{-10}	-
Ingestion of fish	-	-	-
Ingestion of milk	-	7.82×10^{-7}	-
Ingestion of pork	-	1.26×10^{-8}	-
Ingestion of soil	7.34×10^{-9}	1.12×10^{-9}	4.58×10^{-10}
Total Lifetime Risk	1.03×10^{-6}	1.18×10^{-6}	6.03×10^{-8}

NB – Where an exposure pathway has been left blank it has not been considered appropriate to calculate a HI for this exposure pathway in that receptor scenario.

3.3.5 The total lifetime cancer risk for the hypothetical farmer RNW2 receptor scenario is only slightly larger than that of the resident PL2 receptor and approximately 20 times larger than that of the nearest resident SA1 receptor. For both the hypothetical resident receptors the largest risk to carcinogenic health effects occurs via the inhalation exposure pathway. The inhalation exposure pathway represents approximately 75% of the total carcinogenic risk via all pathways for these receptors.

3.3.6 The ingestion of food products and in particular the ingestion of milk is predicted to be the exposure pathway with the largest risk of carcinogenic effects for the hypothetical farmer receptor scenario. This exposure pathway represents approximately 66% of the total overall carcinogenic risk via all ingestion exposure pathways for the farmer RNW2 receptor scenario.

Summary of Carcinogenic Effects

3.3.7 The total lifetime cancer risk for each receptor, which takes into account the cumulative risk for each COPC via each pathway, calculated by IRAP is presented in Table 3.16.

Table 3.16 Summary of the total lifetime cancer risk for each receptor type

Receptor Name	Receptor Type	Total Hazard Index (HI)
PL1	Adult Resident	8.04×10^{-7}
PL1	Child Resident	2.26×10^{-7}
PL2	Adult Resident	**1.03×10^{-6}**
PL2	Child Resident	2.85×10^{-7}
PL3	Adult Resident	1.17×10^{-7}
PL3	Child Resident	6.57×10^{-8}
PS1	Adult Resident	2.55×10^{-8}
PS1	Child Resident	6.97×10^{-9}
PS2	Adult Resident	2.24×10^{-8}
PS2	Child Resident	6.20×10^{-9}
PT1	Adult Resident	3.16×10^{-8}
PT1	Child Resident	8.83×10^{-9}
SA1	Adult Resident	6.03×10^{-8}
SA1	Child Resident	1.69×10^{-8}
SA2	Adult Resident	7.74×10^{-8}
SA2	Child Resident	2.16×10^{-8}
TP1	Adult Resident	2.73×10^{-8}
TP1	Child Resident	7.53×10^{-9}
TP2	Adult Resident	3.15×10^{-8}
TP2	Child Resident	8.68E-09
TP3	Adult Resident	2.64×10^{-8}
TP3	Child Resident	7.38×10^{-9}
RNE1	Adult Farmer	1.09×10^{-6}
RNE1	Child Farmer	2.32×10^{-7}
RNE2	Adult Farmer	1.10×10^{-6}
RNE2	Child Farmer	2.35×10^{-7}
RNE3	Adult Farmer	1.09×10^{-6}
RNE3	Child Farmer	2.33×10^{-7}
RNW1	Adult Farmer	1.07×10^{-6}
RNW1	Child Farmer	2.28×10^{-7}
RNW2	Adult Farmer	**1.18×10^{-6}**
RNW2	Child Farmer	2.52×10^{-7}
RSE1	Adult Farmer	3.05×10^{-7}
RSE1	Child Farmer	6.49×10^{-8}
RSE2	Adult Farmer	2.90×10^{-7}
RSE2	Child Farmer	6.19×10^{-8}
RSW1	Adult Farmer	6.63×10^{-7}
RSW1	Child Farmer	1.41×10^{-7}
RSW2	Adult Farmer	1.00×10^{-6}
RSW2	Child Farmer	2.14×10^{-7}
Criteria		1×10^{-5}

3.3.8 The largest carcinogenic risk is predicted for the hypothetical resident PL2 adult and farmer RNW2 adult receptor scenarios. The additional total **lifetime** (70 year period) carcinogenic risks to health at these hypothetical receptors associated with the proposed EfW CHP facility are 1.03 x 10^{-6} for the resident PL2 and 1.18 x 10^{-6} for the farmer RNW2. The additional total lifetime carcinogenic risk at the resident receptor SA1 nearest to the farmer RNW2 is 6.03 x 10^{-8}. Expressing these values in terms of a probabilistic risk estimate of developing cancer over the lifetime of an individual, results in a 1 in 970,874, 1 in 847,458 and a 1 in 16,583,747 probability of developing cancer for the resident PL2, farmer RNW2 and resident SA1 receptors respectively. These risks of developing cancer over the lifetime of an individual are significantly smaller than the 1 x 10^{-5} (1 in 100,000) lifetime risk of developing cancer considered acceptable by the US EPA.

3.3.9 If these lifetime risks over a 70 year period are converted into annual risks of carcinogenic effects then the risk of developing cancer over a year becomes 1 in 67,961,180 for the resident PL2, 1 in 59,322,060 for the farmer RNW2 and 1 in 1,160,862,290 for the resident SA1. These probabilistic estimates of risk are significantly smaller than the annual risk of 1 x 10^{-6} (1 in 1,000,000), considered acceptable for industry within the UK[16].

3.4 Summary of Results

3.4.1 The assessment of health effects from exposure to metals and organic substances associated with the operation of the proposed EfW CHP facility reported the following:

- The contribution of emissions from the proposed EfW CHP facility to soil concentrations of each metal and the total dioxins/furans are low. The impacts represent an additional contribution of less than 0.025% of the respective soil guideline concentration values for metals and and less than 0.004% of the soil guideline concentration values for total dioxins/furans.

- A relatively low dietary intake of metals and dioxins/furans, when compared to the typical dietary intake values, is predicted to be associated with the proposed facility. The predicted dietary intake of lead in the hypothetical resident PL2 receptor scenario of 6.2 x 10^{-3} µg kg-BW^{-1} d^{-1} are markedly less than the equivalent typical UK dietary value of 9.0 x 10^{-2} – 1.0 x 10^{-1} µg kg-BW^{-1} d^{-1}. The dietary intake of total dioxins/furans is predicted to be <3% of typical UK dietary values, with the daily intake predicted to be <1% of the COT TDI value;

- A low additional exposure to total dioxins/furans of infants via their mother's breast milk is predicted. Additional daily intake values are predicted to be <0.25% of the US EPA criteria and <10% of the UK COT TDI value;

- The largest risk of non-carcinogenic health effects in the resident and farmer receptor scenarios associated with the EfW CHP facility are predicted at the hypothetical resident PL2 receptor and farmer RNW2 receptor. The nearest resident receptor type to the farmer RNW2 is predicted to have a total Hazard Index that is a factor of 10 lower. The total hazard indices for these hypothetical receptors locations are predicted to be at least a factor of 10 below the reference dose at which there is an appreciable risk of health effects occurring over a 70 year lifetime. The exposure pathways predicted to contain the largest risk to non-carcinogenic health effects are the ingestion

[16] CIWEM (2001) Risk Assessment for Environmental Professional, CIWEM Publication, December 2001

of above ground vegetables for the hypothetical resident receptor and the ingestion of milk for the hypothetical farmer receptor. Nickel, lead and thallium are predicted to be the COPCs with the largest risk of non-carcinogenic health effects via the inhalation and ingestion pathway.

- The hypothetical resident PL2 receptor and farmer RNW2 receptor are predicted to have the largest risk to carcinogenic health effects associated with the proposed EfW CHP facility. The total lifetime risk at these locations is a 1 in 970,874 and 1 in 847,458 risk of developing cancer over the entire lifetime of an individual respectively, which translates into an annual risk of 1 in 67,961,180 and 1 in 59,322,060 respectively. This is well within the acceptable annual risk of 1 in 1,000,000 for UK industrial operations. The largest risk of carcinogenic health effects is predicted to occur for cadmium via the inhalation exposure pathway in the hypothetical resident receptor scenarios. The ingestion of milk and ingestion of beef are predicted to be the exposure pathways with the largest risk of carcinogenic health effects in the hypothetical farmer receptor scenarios. Contributions of cadmium, benzo[a]pyrene and total dioxins/furans are predicted to be the COPCs with the largest risk of carcinogenic health effects via the ingestion pathway.

4 Conclusions

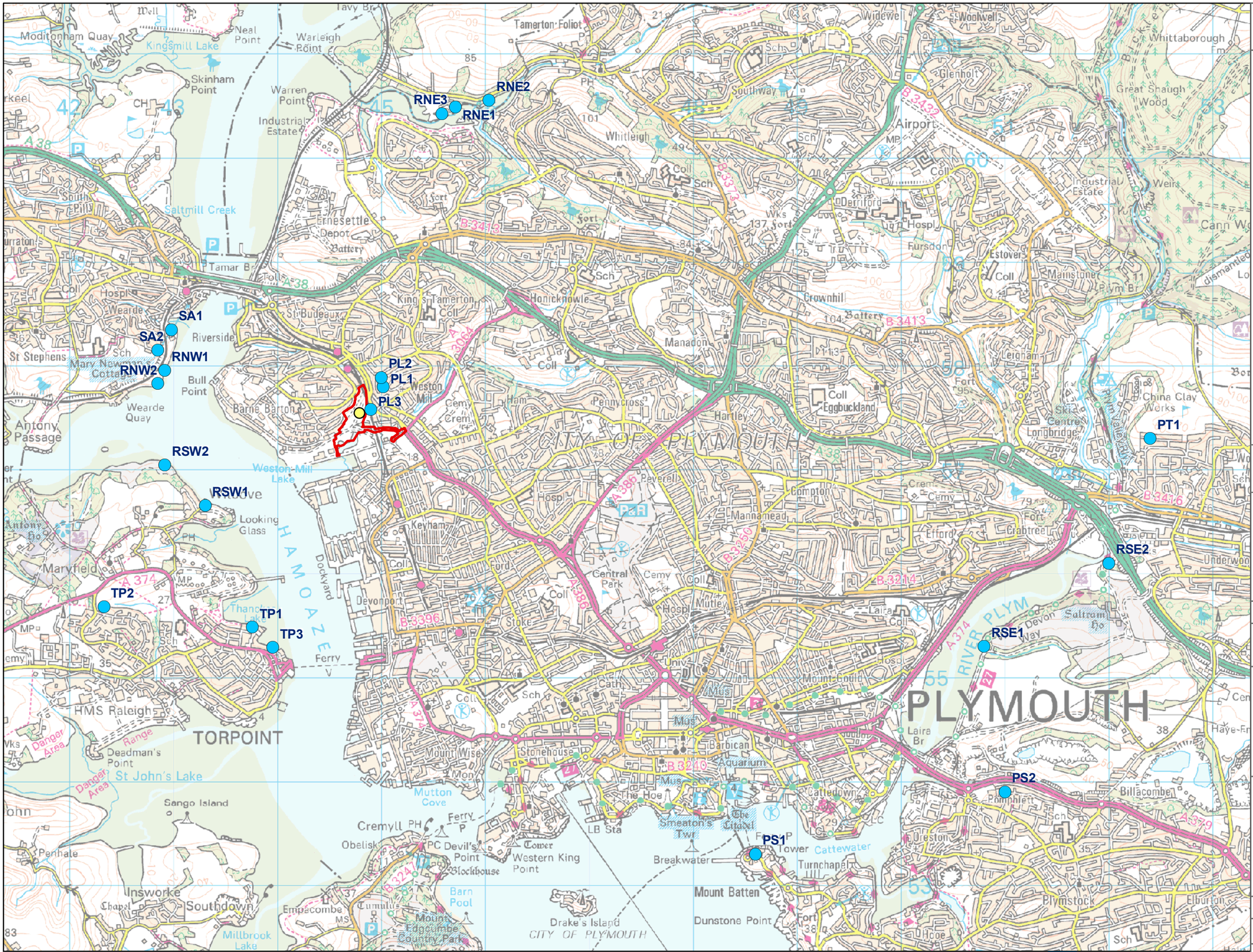
4.1.1 This assessment has quantified the risks to human health in the local population from exposure to various different chemicals of potential concern associated with the emissions of the proposed EfW CHP facility. The methodology used is consistent with the US EPA Human Health Risk Assessment Protocol[6]. The assessment has encompassed conservative assumptions regarding the exposure of a hypothetical individual receptor to the maximum concentrations of compounds of potential concern (COPCs) over the lifetime of an individual receptor and that a larger than average proportion of locally grown food is consumed. The COPCs emitted from the proposed EfW CHP facility have been identified, along with the exposure pathways of greatest concern and the potentially most sensitive hypothetical receptors within the vicinity. The commercially available human health risk assessment modelling tool IRAP and the results from the air dispersion modelling exercise, have been used to calculate exposure concentrations and the risk of health effects at the most sensitive hypothetical receptors via the inhalation and ingestion pathways.

4.1.2 This assessment of the health effects from metals and organic substances has shown that there is not a significant risk to human health associated with emissions from the proposed EfW CHP facility via the inhalation and ingestion exposure pathway. The annual carcinogenic risks at the most sensitive receptor locations are predicted to achieve the UK industry acceptable annual risk of 1 in 1,000,000. The total non-carcinogenic risks for all COPCs via all exposure pathways predicted concentrations significantly below the reference dose and reference concentrations, at which there is an appreciable risk of health effects occurring. A relatively low dietary intake of metals and dioxins/furans is predicted to be associated with the proposed facility, when compared to the typical UK dietary intake values.

4.1.3 The assessment methodology has deliberately used assumptions to generate scenarios that will lead to overestimations of the risk to human health. Such conservative assumptions include the duration and frequency of exposure to an individual i.e. they are assumed to live their entire lives in the area of maximum impact and that a significant portion of their diet is obtained from animal and vegetable products grown/reared in the local area where deposition occurs, whereas in reality it will originate from further afield. Taking into account the conservative nature of this assessment, it can be concluded with confidence that actual receptors within the City of Plymouth, or other communities would not be subject to a significant risk of carcinogenic and non-carcinogenic health effects from exposure to COPCs via the inhalation and ingestion pathways as a consequence of the proposed operation of the proposed EfW CHP facility.

Annex A

Figure 2.1 Human Health Risk Assessment Receptor Locations



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NOTES

- Human Health Receptor Locations
- Stack
- Site Boundary

Revision Details	By / Check	Date	Suffix

Drawing Status: FINAL

Job Title: EFW CHP FACILITY, NORTH YARD, DEVONPORT

Drawing Title: RECEPTOR LOCATIONS FOR HUMAN HEALTH RISK ASSESSMENT

Scale at A3	1:35,000		
Drawn: EW		Approved: GG	
Stage 1 Check: GG	Stage 2 Check: GG	Originated: 9511NM	Date: 14/04/11

Drawing Number: FIGURE 2.1 Rev

Contractor to SWDWP	Environmental & Planning Consultant	EPC Contractor	General Designer	Civil Works	Architect	Air Pollution Control	Boiler & Grate	Electrical & Control	Water Steam System
MVV Environment Devonport Ltd	Scott Wilson	MVV O&M	Envi Con Envi Con & Plant Engineering GmbH	KIER	SC	Lab	ideas full of energy! Standardkessel Baumgarte	SER www.ser.biz	Imtech

Annex B

Table B1 Default values within IRAP for selected site specific parameters used as part of the human health risk assessment

Parameter	Parameter Value	IRAP Symbol	Units
Soil dry bulk density	1.5	bd	g cm^{-3}
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 day^{-1}
Qty of grain eaten by CATTLE each day	0.47	beef_qp_grain	kg DW day^{-1}
Qty of silage eaten by CATTLE each day	2.5	beef_qp_silage	kg DW day^{-1}
Grain fraction grown on contam. soil eaten by CHICKEN	1.0	chicken_fi_grain	-
Qty of grain eaten by CHICKEN each day	0.2	chick_qp_grain	kg DW day^{-1}
Average annual evapotranspiration	69.09	e_v	cm yr^{-1}
Fish lipid content	0.07	f_lipid	-
Fraction of CHICKEN's diet that is soil	0.1	fd_chicken	-
Universal gas constant	8.205×10^{-5}	gas_r	atm-m^3 mol^{-1} K^{-1}
Average annual irrigation	0	i	cm yr^{-1}
Plant surface loss coefficient	18	kp	yr^{-1}
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^{-1}
Qty of grain eaten by MILK CATTLE each day	3.0	milk_qp_grain	kg DW d^{-1}
Qty of silage eaten by MILK CATTLE each day	4.1	milk_qp_silage	kg DW d^{-1}
Averaging time	1	milkfat_at	yr
Body weight of infant	9.4	milfat_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	day
Ingestion rate of breast milk	0.688	milkfat_ir_milk	kg day^{-1}
Viscosity of air corresponding to air temp.	1.81×10^{-04}	mu_a	g cm^{-1} s^{-1}

Parameter	Parameter Value	IRAP Symbol	Units
Average annual precipitation	98.7	p	cm yr^{-1}
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 day^{-1}
Qty of silage eaten by PIGS each day	1.4	pork_qp_silage	kg DW day^{-1}
Qty of soil eaten by CATTLE	0.5	qs_beef	kg day^{-1}
Qty of soil eaten by CHICKEN	0.022	qs_chick	kg day^{-1}
Qty of soil eaten by DAIRY CATTLE	0.4	qs_milk	kg day^{-1}
Qty of soil eaten by PIGS	0.37	qs_pork	kg day^{-1}
Average annual runoff	9.87	r	cm yr^{-1}
Density of air	$1.2E^{-3}$	rho_a	g cm^{-3}
Solids particle density	2.7	rho_s	g cm^{-3}
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^{-3}
Length of plant expos. to depos. - ABOVEGROUND	0.16	tp	year
Length of plant expos. to depos. - FORAGE	0.12	tp_forage	year
Length of plant expos. to depos. - SILAGE	0.16	tp_silage	year
Dry deposition velocity	0.5	vdv	cm s^{-1}
Dry deposition velocity for mercury	2.9	vdv_hg	cm s^{-1}
Wind velocity	5.3	w	m s^{-1}
Yield/standing crop biomass - edible portion ABOVEGROUND	2.24	yp	kg DW m^{-2}
Yield/standing crop biomass - edible portion FORAGE	0.24	yp_forage	kg DW m^{-2}
Yield/standing crop biomass - edible portion SILAGE	0.8	yp_silage	kg DW m^{-2}
Soil mixing zone depth	2.0	z	cm
Soil mixing depth for produce	2.0	z_p	Cm

Annex C

Table C1 Default values within IRAP for receptor specific parameters used as part of the human health risk assessment

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	year
Averaging time for noncarcinogens	30	6	40	6	30	6	year
Consumption rate of BEEF	0.0	0.0	0.00122	0.00075	0.0	0.0	Kg/kg-day FW
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-day FW
Consumption rate of ABOVEGROUND PRODUCE	0.00032	0.00077	0.00047	0.00113	0.00032	0.00077	Kg/kg-day FW
Consumption rate of BELOWGROUND PRODUCE	0.00014	0.00023	0.00017	0.00028	0.00014	0.00023	Kg/kg-day FW
Consumption rate of DRINKING WATER	1.4	0.67	1.4	0.67	1.4	0.67	L day^{-1}
Consumption rate of PROTECTED ABOVEGROUND PRODUCE	0.00061	0.0015	0.00064	0.00157	0.00061	0.0015	Kg/kg-day FW
Consumption rate of SOIL	0.0001	0.0002	0.0001	0.0002	0.0001	0.0002	kg day^{-1}
Exposure duration	30	6	40	6	30	6	year
Exposure frequency	350	350	350	350	350	350	day/year
Consumption rate of EGGS	0.0	0.0	0.00075	0.00054	0.0	0.0	Kg/kg-day FW
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	0.00125	0.00088	Kg/kg-day FW
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	year
Inhalation exposure frequency	350	350	350	350	350	350	day/year
Inhalation exposure time	24	24	24	24	24	24	hrs/day
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^3 hr^{-1}

Parameter Description	Adult Resident	Child Resident	Adult Farmer	Child Farmer	Adult Fisher	Child Fisher	Units
Consumption rate of MILK	0.0	0.0	0.01367	0.02268	0.0	0.0	Kg/kg-day FW
Consumption rate of PORK	0.0	0.0	0.00055	0.00042	0.0	0.0	Kg/kg-day FW
Time period at the beginning of combustion	0	0	0	0	0	0	year
Length of exposure duration	30	6	40	6	30	6	year