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Consulting Engineers Limited

Thameside Energy Recovery Facility

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Dioxin Pathway Intake Assessment

ENGINEERING CONSULTING

Document approval

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1 Introduction

Fichtner Consulting Engineers Ltd (Fichtner) has been engaged to undertake a Dioxin Pathway Intake Assessment to support the Environmental Permit (EP) variation application for the Thameside Energy Recovery Facility (herein referred to as the Facility). Full details of the proposed changes being applied for can be found in the Supporting Information document submitted with this application.

As the fuel combusted at the Facility will be sourced from waste, the limits on emissions to air set in the existing EP are be based on those outlined in Chapter IV and Annex VI of the Industrial Emissions Directive (IED) (2010/75/EU) for waste incineration and co-incineration plants. This includes limits on emissions of dioxins and furans (collectively referred to as “dioxins” for the purpose of this assessment).

The Waste Incineration Best Available Techniques Reference (BREF) document was published by the European Integrated Pollution Prevention and Control (IPPC) Bureau in December 2019. The Environment Agency (EA) will be required to implement conditions within all permits requiring operators to comply with the requirements set out in the BREF within four years of the publication date. This will include the Facility. The Waste Incineration BREF has introduced BAT-AELs (Best Available Techniques Associated Emission Levels) which are more stringent than those currently set out in the IED for some pollutants. This assessment has been carried out assuming that the Facility operates at the ELVs in the existing EP. However, a detailed discussion on the effect of the introduction of the BAT AELs has also been provided as part of this assessment.

The advice from health specialists such as the Health Protection Agency (HPA) is that the damage to health from emissions from incineration and co-incineration plants is likely to be very small, and probably not detectable. Nevertheless, the specific effects on human health of the Facility have been considered and are presented in this report. This includes a review of published literature on the health effects of energy recovery facilities, and a quantitative assessment of the effect of the Facility.

For most substances released from the Facility, the most significant effects on human health will arise by inhalation. However, for dioxins and dioxin-like PCBs which accumulate in the environment, inhalation is only one of the potential exposure routes.

A Dispersion Modelling Assessment (DMA) has been submitted with this application. The environmental quality standards (EQSs) referred to in the DMA have been set by the various authorities at a level which is considered to present minimum or zero risk to human health. It is widely accepted that, if the concentrations in the atmosphere are less than the EQSs, then the pollutant is unlikely to have an adverse effect on human health. For dioxins and dioxin-like PCBs no EQS has been set, and the health assessment criteria is expressed as the total intake from ingestion and inhalation. Therefore, this assessment considers exposure routes other than just inhalation.

The Facility is located adjacent to the Tilbury Green Power (TGP) Facility, which has a separate EP and is fired on waste wood. The combined impact of the operation of the Facility and the TGP Facility has been considered in this assessment.

2 Literature review

The HPA, whose role has now been taken over by Public Health England (PHE), published a note RCE-13 "The Impact on Health of Emissions to Air from Municipal Waste Incinerators", in 2009[1]. The summary states:

> *"While it is not possible to rule out adverse health effects from modern, well-regulated municipal waste incinerators with complete certainty, any potential damage to the health of those living close-by is likely to be very small, if detectable"*

PHE commissioned further research in 2012, while continuing to state that the conclusions of RCE-13 remain applicable. These studies were commissioned from the Small Area Health Statistics Unit, which is based at Imperial College London and Kings College London. The methodology and results of the studies have been published in a series of papers in scientific journals. The three most recent papers, known as Ghosh et al (2018)[2], Freni-Sterrantino et al (2019)[3] and Parkes et al (2019)[4], are the most relevant.

These studies considered whether living near a municipal waste incinerator (MWI) is linked with adverse reproductive and infant health outcomes. These outcomes were studied as they are considered more sensitive to the accumulation of pollutants in the environment than other potential markers such as lifetime cancer rates.

Ghosh et al (2018) concluded that:

> *"This large national study found no evidence for increased risk of a range of birth outcomes, including birth weight, preterm delivery and infant mortality, in relation to either MWI emissions or living near an MWI operating to the current EU waste incinerator regulations in Great Britain."*

Freni-Sterrantino et al (2019) concluded that:

> *"we did not find an association between the opening of a new MWI and changes in infant mortality trends or sex ratio at birth for 10 and 4 km buffers, using distance as proxy of exposure, after taking into account temporal trends in comparator areas and potential confounding factors."*

The objective of Parkes et al (2019) was as follows: *"To conduct a national investigation into the risk of congenital anomalies in babies born to mothers living within 10 km of an MWI associated with: i) modelled concentrations of PM_{10} as a proxy for MWI emissions more generally and; ii) proximity of residential postcode to nearest MWI, in areas in England and Scotland that are covered by a congenital anomaly register."* Under objective (i), which related congenital anomalies to modelled concentrations and so would be considered the more representative approach, the study

[1] https://www.gov.uk/government/publications/municipal-waste-incinerators-emissions-impact-on-health

[2] Ghosh RE, Freni Sterrantino A, Douglas P, Parkes B, Fecht D, de Hoogh K, Fuller G, Gulliver J, Font A, Smith RB, Blangiardo M, Elliott P, Toledano MB, Hansell AL. (2018) Fetal growth, stillbirth, infant mortality and other birth outcomes near UK municipal waste incinerators; retrospective population based cohort and case-control study. Environment International.

[3] Freni-Sterrantino, A; Ghosh, RE; Fecht, D; Toledano, MB; Elliott, P; Hansell, AL; Blangiardo, M. (2019) Bayesian spatial modelling for quasi-experimental designs: An interrupted time series study of the opening of Municipal Waste Incinerators in relation to infant mortality and sex ratio. Environment International.

[4] Parkes B, Hansell A.L., Ghosh R.E, Douglas P., Fecht D., Wellesley D., Kurinczuk J.J., Rankin J., de Hoogh K., Fuller G.W, Elliot P., and Toledano M.B. (2019) Risk of congenital anomalies near municipal waste incinerators in England and Scotland: Retrospective population-based cohort study. Environment International.

found no association with congenital abnormalities. Under objective (ii), there was a small excess risk, but the paper's authors note that this may be due to residual confounding.

The Imperial College website includes Frequently Asked Questions on this study. One of these is "Does the study show that MWIs are causing increased congenital anomalies in populations living nearby?" The answer is as follows.

> *"No. The study does not say that the small excess risks associated with congenital heart disease and genital anomalies in proximity to MWIs are caused by those MWIs, as these results may be explained by residual confounding factors i.e., other influences which it was not possible to take into account in the study. This possible explanation is supported further by the fact that the study found no increased risk in congenital anomalies due to exposure to emissions from incinerators."*

These three recent papers consider facilities in the UK, operating under the same regulatory regime which would apply to the Facility and operating to the current standards of the IED. The papers found no conclusive evidence of an association of waste incineration facilities with the health outcomes considered. Given that the Facility would actually operate to tighter standards, as it would be subject to the reduced emissions limits from the Waste Incineration BREF, the conclusions are directly relevant and support PHE's position statement that "*any potential damage to the health of those living close-by is likely to be very small, if detectable*".

Therefore, it can be concluded that the effect of emissions from the Facility of pollutants that accumulate in the environment would not be significant. Nonetheless, a quantitative assessment of the effect of emissions from the Facility has been undertaken and is presented within this report.

3 Issue Identification

3.1 Issue

The key issue for consideration is the release of substances to atmosphere from the Facility which have the potential to harm human health. Details of the dispersion modelling can be found in the DMA submitted with this application.

The Facility has an EP to operate which includes limits for pollutants known to be produced during the combustion of municipal waste which have the potential to impact upon the local environment either on human health or ecological receptors. An assessment of the impact of inhalation of these pollutants on human health is presented in the DMA submitted with the EP application. However, dioxins and dioxin-like PCBs can accumulate in the environment, which means that inhalation is only one of the potential exposure routes. The health assessment criterion is expressed as the total intake from ingestion and inhalation. Pathway modelling considering the intake from inhalation and ingestion has been carried out using the software “Industrial Risk Assessment Program-Human Health” (IRAP-h View – Version 5.1, “IRAP”). In addition, a review of published literature on the health effects of energy recovery facilities has been undertaken.

3.2 Chemicals of Potential Concern (COPC)

The following substances have been considered COPCs for the purpose of this assessment:

- PCDD/Fs (individual congeners), i.e., dioxins; and
- Dioxin-like PCBs;

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

4 Assessment Criteria

IRAP calculates the total exposure through each of the different pathways so that a dose from inhalation and ingestion can be calculated for each receptor. By default, these doses are then used to calculate a cancer risk, using the United States Environment Protection Agency's (USEPA)'s approach. However, this assessment applies the approach set out in the EA's document "Human Health Toxicological Assessment of Contaminants in Soil", ref SC050021 (2009).

For the COPCs considered, which have a threshold level for toxicity, a Tolerable Daily Intake (TDI) is defined. This is "an estimate of the amount of a contaminant, expressed on a bodyweight basis, which can be ingested daily over a lifetime without appreciable health risk." A Mean Daily Intake (MDI) is also defined, which is the typical intake from background sources (including dietary intake) across the UK. In order to assess the impact of the Facility, the predicted intake of a substance due to emissions from the Facility is added to the MDI and compared with the TDI.

The following table outlines the MDIs (the typical intake from existing background sources) and TDIs for dioxins and dioxin-like PCBs. These figures are defined in the "Contaminants in soil: updated collation of toxicology data and intake values for humans: dioxins, furans and dioxin-like PCBs" (EA 2009).

Table 1: Intake of Dioxins and Dioxin-Like PCBs

Item	Units	Intake	
		70 kg adult	20 kg child
Tolerable Daily Intake (TDI)	pg WHO-TEQ/kg bw/day	2.0	
Mean Daily Intake (MDI)	pg WHO-TEQ/kg bw/day	0.7	1.8
	% of TDI	35.00%	90.65%

To allow comparison with the TDI for dioxins, intake values for each dioxin are multiplied by a factor known as the WHO-TEF. A full list of the WHO-TEF values for each dioxin is provided in Table 7.

The TDI has been set at a level which can be ingested daily over a lifetime without appreciable health risk. Therefore, if the total exposure is less than the TDI, it can be concluded that the impact of the Facility is not significant.

5 Conceptual Site Model

5.1 Conceptual site model

IRAP, created by Lakes Environmental, is based on the USEPA Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities[5]. This Protocol is a development of the approach defined by Her Majesties Inspectorate on Pollution (HMIP) in the UK in 1996[6], taking account of further research since that date. The exposure pathways included in the IRAP model are shown in Table 2.

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

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

The pathways through which inhalation and ingestion occur and the receptors that have been considered to be impacted via each pathway are shown in the table below.

Table 2: Pathways Considered

Pathway	Residential	Agricultural
Direct inhalation	Yes	Yes
Ingestion of soil	Yes	Yes
Ingestion of home-grown produce	Yes	Yes
Ingestion of drinking water	Yes	Yes
Ingestion of eggs from home-grown chickens	-	Yes
Ingestion of home-grown poultry	-	Yes
Ingestion of home-grown beef	-	Yes
Ingestion of home-grown pork	-	Yes
Ingestion of home-grown milk	-	Yes
Ingestion of breast milk (infants only)	Infants only	

Some households may keep chickens and consume eggs and potentially the birds. The impact on these households is slightly higher than at a standard resident receptor, but much lower than at an agricultural receptor. The approach used considers an agricultural receptor at the point of maximum impact as a worst case assumption.

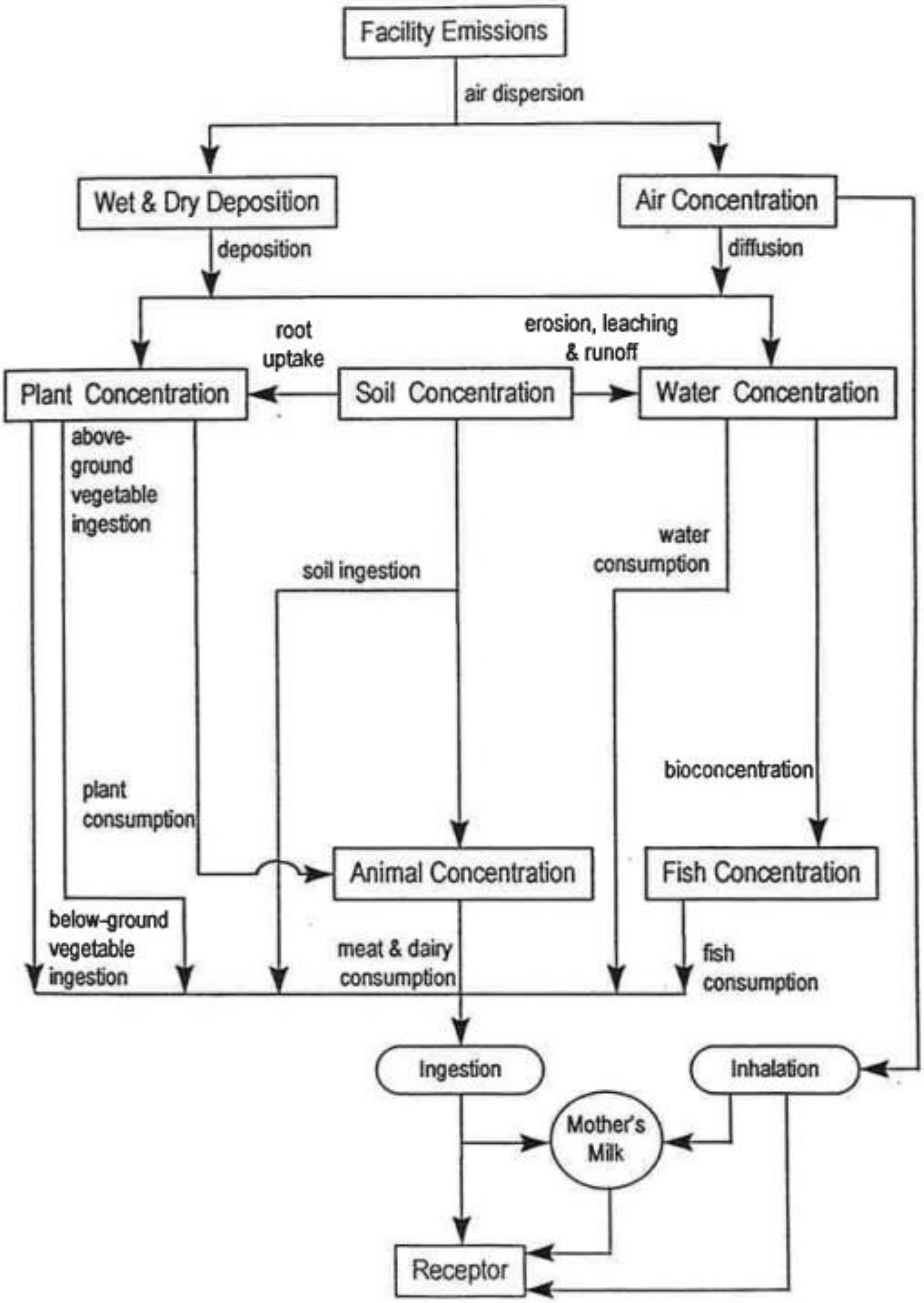
As shown in Figure 1, the pathway from the ingestion of mother's milk in infants is considered within the assessment. The IRAP model calculates the amount of dioxins entering the mother's milk and being passed on to the infants. IRAP does not include data on individual PCBs, but it does include data for take-up and accumulation rates within the food chain for two groups of PCBs,

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

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

known as Aroclor 1254 and Aroclor 1016. IRAP does not include these when determining the intake via mother's milk. A factor of 1.5 has been applied to the dioxin emission rate when considering the impact of the intake via mother's milk, to include the likely contribution from dioxin-like PCBs. The impacts are then compared against the TDI.

Figure 1: Conceptual Site Model – Exposure Pathways



5.2 Pathways excluded from assessment

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

5.2.1 Dermal absorption

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

The USEPA[7] provide an example from the risk assessment conducted for the Waste Technologies Inc. hazardous thermal treatment in East Liverpool, Ohio. This indicated that for an adult subsistence farmer in a sub-area with high exposures, the risk resulting from soil ingestion and dermal contact was 50-fold less than the risk from any other pathway and 300-fold less than the total estimated risk.

The HMIP document[8] provides a screening calculation using conservative assumptions, which states that the intake via dermal absorption is 30 times lower than the intake via inhalation, which is itself a minor contributor to the total risk.

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

5.2.2 Groundwater

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

The USEPA[9] has concluded that the build-up of dioxins in the aquifer over realistic travel times relevant to human exposure was predicted to be so small as to be essentially zero.

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

5.2.3 Surface water

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

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

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

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

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

5.2.4 Fish consumption

The consumption of locally caught fish has been excluded from the assessment. Whilst fish makes up a proportion of the UK diet, it is not likely that significant quantities of fish would be sourced from an area within close proximity to the Facility.

A review of the local waterbodies has been undertaken to see if there are any game fishing lakes in the local area[10]. The closest game fishing lake is at the Buckland Fishery near Cliffe, located approximately 11 km east of the Facility. Due to the distance from the Facility, it is considered that the impact at the fishery will be imperceptible. In addition, the likelihood of persons sources a large proportion of their diet from a trout fishery is very low. Game fishing may also take place along rivers and coasts in the local area. However, the accumulation of pollutants in river systems is not of significant concern, as any pollutants will be washed downstream rather than accumulating, and the extremely small quantity of pollutants deposited in seawater would be diluted by the action of tides and currents. Therefore, the fish consumption pathway has been excluded from this assessment.

[10] Locations Map, http://www.fisharound.net/where-to-fish/locations-map

6 Sensitive Receptors

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

For the purposes of this assessment, receptor locations have been categorised as 'residential' or 'agricultural'. Residential receptors represent a known place of residence that is occupied within the study area and includes local schools. Agricultural receptors represent a farm holding or area of cultivated or grazing land.

The DMA submitted with this application considered the impact at specific receptor locations and the point of maximum impact. For the purposes of this assessment, the point of maximum impact has been included as both an agricultural and residential receptor to assess the theoretical maximum impact of the Facility. In addition, a number of receptors locations in the closest areas of farmland to the Facility have also been included. The sensitive receptors assessed are listed in Table 3. Reference should be made to Figure 2 contained in Appendix B which shows the location of these receptors with respect to the Facility.

Table 3: Sensitive Receptors

ID	Receptor Name	Location		Type of Receptor
		X	Y	
MAX	Point of maximum impact	562675	177825	Agricultural / Residential
R1	Pier Lodge Day Nursery	561813	177297	Residential
R2	Curzon Drive	562023	177386	Residential
R3	Elm Rd	562180	177530	Residential
R4	Cherry Tree Close	562512	177661	Residential
R5	Jubilee Garden	562814	177565	Residential
R6	Dock Rd Flats	563336	176677	Residential
R7	St Marys Close	562608	177865	Residential
R8	Thameside Primary School	562369	177570	Residential
R9	Belmont Castle Academy	560535	178135	Residential
R10	Farmland 1	563298	178037	Agricultural
R11	Farmland 2	563479	178170	Agricultural
R12	Farmland 3	563328	178236	Agricultural
R13	Farmland 4	563220	178298	Agricultural

7 IRAP Model Assumptions and Inputs

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

7.1 Concentrations in soil

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

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

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

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

7.2 Concentrations in plants

The concentrations in plants are determined by considering direct deposition and air-to-plant transfer for above ground produce, and root uptake for above ground and below ground produce.

The calculation takes account of the different types of plant. For example, uptake of substances through the roots will differ for below ground and above ground vegetables, and deposition onto plants will be more significant for vegetables which grow above ground.

7.3 Concentrations in animals

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

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

7.4 Concentrations in humans

7.4.1 Intake via inhalation

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

- adults – 20 m^3/day; and
- children – 7.2 m^3/day.

These are as specified within the EA's document "Human Health Toxicological Assessment of Contaminants in Soil". The calculation also takes account of time spent outside, since most people spend most of their time indoors.

7.4.2 Intake via soil ingestion

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

7.4.3 Ingestion of food

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

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

7.4.4 Breast milk ingestion

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

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

The assumptions used were:

- Exposure duration of infant to breast milk 1 year
- Proportion of ingested dioxin that is stored in fat 0.9
- Proportion of mother's weight that is stored in fat 0.3
- Fraction of fat in breast milk 0.04
- Fraction of ingested contaminant that is absorbed 0.9
- Half-life of dioxins in adults 2,555 days
- Ingestion rate of breast milk 0.688 kg/day
- Factor on total dioxin intake to account for dioxin-like PCBs 1.5

7.5 Estimation of COPC concentration in media

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

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

Meteorological data was obtained for the period 2014 to 2018 from the Gravesend Broadness weather station, which is the meteorological data that was used for the dispersion modelling in the

DMA. For full details of the meteorological data reference should be made the DMA. This provides the annual average precipitation which can be used to calculate the general IRAP-h input parameters, as presented in Table 4.

Table 4: Site-Specific Properties

Input Variable	Assumption	Value (cm/year)
Annual average evapotranspiration	70% of annual average precipitation	49.05
Annual average irrigation	0% of annual average precipitation	0.00
Annual average precipitation	100% of annual average precipitation	70.07
Annual average runoff	10% of annual average precipitation	7.01

The average wind speed was taken as 4.0 m/s, calculated from the average of the five years of weather data from Gravesend Broadness.

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

Table 5: Deposition Assumptions

Deposition Phase	Dry Deposition Velocities (m/s)	Ratio Dry deposition to Wet deposition	
		Dry Deposition	Wet Deposition
Vapour	0.005	1.0	2.0
Particle	0.010	1.0	2.0
Bound particle	0.010	1.0	2.0

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

7.6 Modelled emissions

For the purpose of this assessment, it is assumed that the Facility and the TGP Facility operates at the ELV dioxins for its entire operational life. In reality, the Facility and the TGP Facility will be shut down for periods of maintenance and will typically operate below the emission limits prescribed in the permits.

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

Table 6: COPC Emissions Modelled

COPC	Split of congeners for a release of 1 ng I-TEQ/Nm^3 (1)	Emission conc. (ng/Nm^3) (2)	Emission rate (ng/s) (3)	
			Facility	TGP Facility
Sum I-TEQ dioxins (4)	-	0.1 ng	0.1 ng	-
2,3,7,8-TCDD	0.031	0.0031	0.218	0.139
1,2,3,7,8-PeCDD	0.245	0.0245	1.726	1.102

COPC	Split of congeners for a release of 1 ng I-TEQ/Nm³[(1)]	Emission conc. (ng/Nm³)[(2)]	Emission rate (ng/s) [(3)]	
			Facility	TGP Facility
1,2,3,4,7,8-HxCDD	0.287	0.0287	2.022	1.291
1,2,3,6,7,8-HxCDD	0.258	0.0258	1.817	1.161
1,2,3,7,8,9-HxCDD	0.205	0.0205	1.444	0.922
1,2,3,4,6,7,8-HpCDD	1.704	0.1703	12.002	7.665
OCDD	4.042	0.4041	28.470	18.183
2,3,7,8-TCDF	0.277	0.0277	1.951	1.246
1,2,3,7,8-PCDF	0.277	0.0277	1.951	1.246
2,3,4,7,8-PCDF	0.535	0.0535	3.768	2.407
1,2,3,4,7,8-HxCDF	2.179	0.2178	15.348	9.802
1,2,3,6,7,8-HxCDF	0.807	0.0807	5.684	3.630
1,2,3,7,8,9-HxCDF	0.042	0.0042	0.296	0.189
2,3,4,6,7,8-HxCDF	0.871	0.0871	6.135	3.918
1,2,3,4,6,7,8-HpCDF	4.395	0.4394	30.957	19.771
1,2,3,4,7,8,9-HpCDF	0.429	0.0429	3.022	1.930
OCDF	3.566	0.3565	25.118	16.042
Total dioxins	20.150	2.0143	141.929	90.644
Dioxin-like PCBs	-	0.0092	0.648	0.414

Notes:

(1) Split of the congeners taken from Table 7.2a from the HMIP document.

(2) All emissions are expressed at reference conditions of dry gas, 273.15K, 11% oxygen (Facility), 6% oxygen (TGP Facility).

(3) Emission release rate calculated by multiplying the normalised volumetric flow rate by the emission concentration.

A number of points should be noted for the two groups of COPCs:

1. Dioxins

The split of the different dioxins and furans is based on split of congeners for a release of 1 ng I-TEQ/Nm³ as presented in in Table 6. This data is taken from Table 7.2a from the HMIP document "Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes".

To determine the emission rates, this split of the different dioxins has been multiplied by normalised volumetric flow rate to determine the release rate of each congener.

2. Dioxin-like PCBs

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

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

- Maximum = 9.2×10^{-3} ng[TEQ]/m³
- Mean = 2.6×10^{-3} ng[TEQ]/m³
- Minimum = 5.6×10^{-5} ng[TEQ]/m³

For the purpose of this assessment, the maximum monitored dioxin-like PCB concentration has been used which has been converted to an emission rate using the volumetric flow. Under the IED, waste biomass plants (co-incinerators) have the same emission limit for dioxins as waste incineration plants (0.1 ng/Nm³) but expressed at 6% reference oxygen content. As the measures to abate dioxins and dioxin-like PCBs are the same for incinerators and co-incinerators, it is considered appropriate to model emissions of PCBs from TGP (a co-incinerator) at 9.2×10^{-3} ng[TEQ]/m³ (6% reference oxygen content).

The IRAP software, and the HHRAP database which underpins it, does not include any data on individual PCBs, but it does include data for take-up and accumulation rates within the food chain for two groups of PCBs, known as Aroclor 1254 and Aroclor 1016. Each Aroclor is based on a fixed composition of PCBs. Since we are not aware of any data on the specification of PCBs within incinerator or co-incinerator emissions, as a worst-case assumption it has been assumed that PCB emissions consist entirely of each of the two Aroclor compositions and the maximum impact of either composition has been presented.

As shown in Table 1, the MDI and TDI for dioxins and dioxin-like PCBs is given in pg WHO-TEQ/kg bw/day. However, the split of congeners presented in Table 6, which are used to calculate the release rate of each dioxin, are based on the I-TEFs listed in Annex VI Part II of the IED. To determine the total intake TEQ for comparison with the TDI, the output of the IRAP model has been multiplied by the relevant WHO-TEFs. The I-TEFs and WHO-TEFs are shown in Table 7.

Table 7: Toxic Equivalency Factors for Dioxins and Furans

Congener	IED I-TEQ Multiplier	2005 WHO-TEF Multiplier
2,3,7,8-TCDD	1	1
1,2,3,7,8-PeCDD	0.5	1
1,2,3,4,7,8-HxCDD	0.1	0.1
1,2,3,6,7,8-HxCDD	0.1	0.1
1,2,3,7,8,9-HxCDD	0.1	0.1
1,2,3,4,6,7,8-HpCDD	0.01	0.01
OCDD	0.001	0.0003
2,3,7,8-TCDF	0.1	0.1
1,2,3,7,8-PCDF	0.5	0.3
2,3,4,7,8-PCDF	0.05	0.03
1,2,3,4,7,8-HxCDF	0.1	0.1
1,2,3,6,7,8-HxCDF	0.1	0.1
1,2,3,7,8,9-HxCDF	0.1	0.1
2,3,4,6,7,8-HxCDF	0.1	0.1
1,2,3,4,6,7,8-HpCDF	0.01	0.01

Congener	IED I-TEQ Multiplier	2005 WHO-TEF Multiplier
1,2,3,4,7,8,9-HpCDF	0.01	0.01
OCDF	0.001	0.0003

Source: Contaminants in soil: updated collation of toxicological data and intake values for humans, Dioxins, furans and dioxin-like PCBs (Science report: SC050021/TOX 12), EA, 2009

8 Results

8.1 Assessment against TDI - point of maximum impact

The following tables present the impact of emissions of dioxins and dioxin-like PCBs from the Facility at the point of maximum impact of emissions from the Facility for an 'agricultural' receptor. As explained in section 2, this receptor type assumes the direct inhalation, and ingestion from soil, drinking water, and home-grown eggs and meat, beef, pork, and milk. This assumes that the person lives at the point of maximum impact and consumes home-grown produce etc. This is considered to be a worst-case scenario. Reference should be made to the Figure 2 contained in Appendix B for the location of the point in relation to the Facility.

As detailed in section 1, emissions from the TGP Facility have been included in the model to determine whether the operation of the two facilities will cause any exceedance of the TDI. The results presented for the TGP Facility are the contribution at the point of maximum impact from the Facility.

Table 8: Impact Analysis – Dioxins and Dioxin-Like PCBs – Point of Maximum Impact

Receptor Type	MDI (% of TDI)	Process Contribution (% of TDI)		Overall (% of TDI)
		Facility	TGP Facility	
Adult				
Agricultural	35.00%	2.28%	1.22%	38.50%
Residential	35.00%	0.05%	0.03%	35.08%
Child				
Agricultural	90.65%	3.25%	1.74%	95.64%
Residential	90.65%	0.18%	0.10%	90.93%

The TDI is an estimate of the amount of a contaminant, expressed on a bodyweight basis, which can be ingested daily over a lifetime without appreciable health risk. As shown in Table 8, at the point of maximum impact the overall intake (including the contribution from existing dietary intake and the contribution from TGP Facility) is less than the TDI for dioxins and dioxin-like PCBs. Therefore, there would not be an appreciable health risk based on the emission of these pollutants.

8.2 Breast milk exposure

The total accumulation of dioxins in an infant resulting from emissions from the Facility is presented in Table 9.

Table 9: Breast Milk Impact Analysis – Point of Maximum Impact

Receptor Type	Process Contribution (% of TDI)		Overall (% of TDI)
	Facility	TGP Facility	
Infant breast milk intake			
Agricultural	19.75%	10.50%	30.24%C
Residential	0.37%	0.20%	0.57%

There are no ingestion pathways besides breast milk ingestion for an infant receptor. As the process contribution is less than the TDI, it is considered that the Facility will not increase the health risks from the accumulation of dioxins and dioxin-like PCBs in infants significantly.

8.3 Maximum impact at a receptor

The following tables outline the impact of emissions from the Facility at the most affected receptor (i.e., the receptor with the greatest impact from ingestion and inhalation of emissions from the Facility) (R10 – Farmland 1, noting that this location is not inhabited but has been selected to represent the closest areas of farmland to the Facility).

Table 10: Impact Analysis – Dioxins and Dioxin-Like PCBs – Maximum Impacted Receptor

Receptor Type	MDI (% of TDI)	Process Contribution (% of TDI)		Overall (% of TDI)
		Facility	TGP Facility	
Adult				
Agricultural	35.00%	2.08%	1.12%	38.19%
Child				
Agricultural	90.65%	2.96%	1.59%	95.20%

As shown, for the most impacted receptor the contribution from the Facility is less than 2% of the TDI, and the overall intake (including the contribution from existing dietary intake and the contribution from TGP Facility) is less than the TDI for dioxins and dioxin-like PCBs. Therefore, there would not be an appreciable health risk based on the emission of these pollutants.

In addition, the total accumulation of dioxins in an infant, resulting from emissions from the Facility considering the breast milk pathway and based on an adult agricultural receptor at R10 feeding an infant, is 0.359 pg WHO-TEQ / kg-bw / day which is 17.96% of the TDI, and the contribution from TGP is 9.62% for a total of 27.58% of the TDI. Therefore, as the process contribution is less than the TDI, it is considered that the Facility will not increase the health risks from the accumulation of dioxins in infants significantly.

Detailed results for all identified receptor locations are presented in Appendix A.

8.4 Uncertainty and sensitivity analysis

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

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

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

The IRAP software, and the HHRAP database which underpins it, does not include any data on individual PCBs, but it does include data for take-up and accumulation rates within the food chain for two groups of PCBs, known as Aroclor 1254 and Aroclor 1016. Each Aroclor is based on a fixed composition of PCBs. Since we are not aware of any data on the specification of PCBs within incinerator or co-incinerator emissions, as a worst-case assumption it has been assumed that PCB emissions consist entirely of each of the two Aroclor compositions and the maximum impact of either composition has been presented.

IRAP does not include these Aroclors (which are being used as a proxy for dioxin-like PCBs) when determining the intake via mother's milk. Therefore, a safety factor of 1.5 has been applied to dioxins and dioxin-like PCBs on when considering the impact of the intake via mother's milk.

8.5 Upset process conditions

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

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

Article 47 continues with:

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

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

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

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

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

In relation to the magnitude of dioxin emissions during plant start-up and shutdown, research has been undertaken by AEA Technology on behalf of the EA[11]. Whilst elevated emissions of dioxins (within one order of magnitude) were found during shutdown and start-up phases where the fuel was not fully established in the combustion chamber, the report concluded that:

---

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

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

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

8.6 Implementation of the BREF

The Waste Incineration BREF and the European Union Commission Implementing Decision (EU) 2019/2010 dated 12 November 2019 (establishing the best available techniques (BAT) conclusions, under Directive 2010/75/EU of the European Parliament and of the Council, for waste incineration) were published in December 2019. For the purpose of this assessment, it has been assumed that the IED emission limits will apply as they are the current limits provided in the EP for the Facility. No allowance has been made to the future reduction in emission limits which will be applied as part of the implementation of the Waste Incineration BREF.

Whilst the Facility was granted an EP prior to December 2019, it is expected that the EA will consider the Facility as a new Facility as construction has not yet commenced. It is expected that long-term emission limits from the Waste Incineration BREF will be implemented in accordance with the upper end of the BAT-AEL ranges for a 'new' facility. The expected emission limit values relevant to this assessment are provided in Table 11.

Table 11: Expected Emission Limit Values (ELVs)

Parameter	Units	Periodic Limit
Dioxins & furans	ng I-TEQ /Nm^3	0.04
Dioxin & furan-like PCBs	ng WHO-TEQ/Nm^3	0.06

This means that the results presented are conservative as actual impacts in future will be lower.

9 Conclusions

This Dioxin Pathway Intake Assessment has been undertaken based on the following conservative assumptions:

- the Facility and the TGP Facility will operate continually at the ELV in the existing EPs for dioxins, and dioxin-like PCBs are emitted at a level no worse than the maximum measured at 24 MWI between 2008 and 2010 as advised by the EA; and
- the hypothetical maximum impacted receptor (an agricultural receptor at the point of maximum impact) only ingests food and drink sourced from the area with the maximum contribution from the Facility.

The results of the assessment show that, for the hypothetical maximum impacted receptor (an agricultural child receptor at the point of maximum impact of emissions from the Facility), the combined intake from the Facility, the TGP Facility, and the existing MDI intake of dioxins and dioxin-like PBCs via inhalation and ingestion is below the TDI. In addition, the ingestion of dioxins by an infant being breastfed by an agricultural receptor at the point of maximum impact of emissions from the Facility is less than the TDI. The impact at identified receptor locations is lower. Therefore, there would not be an appreciable health risk based on the emission of dioxins and dioxin-like PCBs. The implementation of the Waste Incineration BREF will mean that the emission limit for the Facility will be reduced in future. As such actual impacts are likely to be even lower

In conclusion, the impact of emissions of dioxins and dioxin-like PCBs from the Facility on human health is predicted to be not significant.

Appendices

A Detailed Results Tables

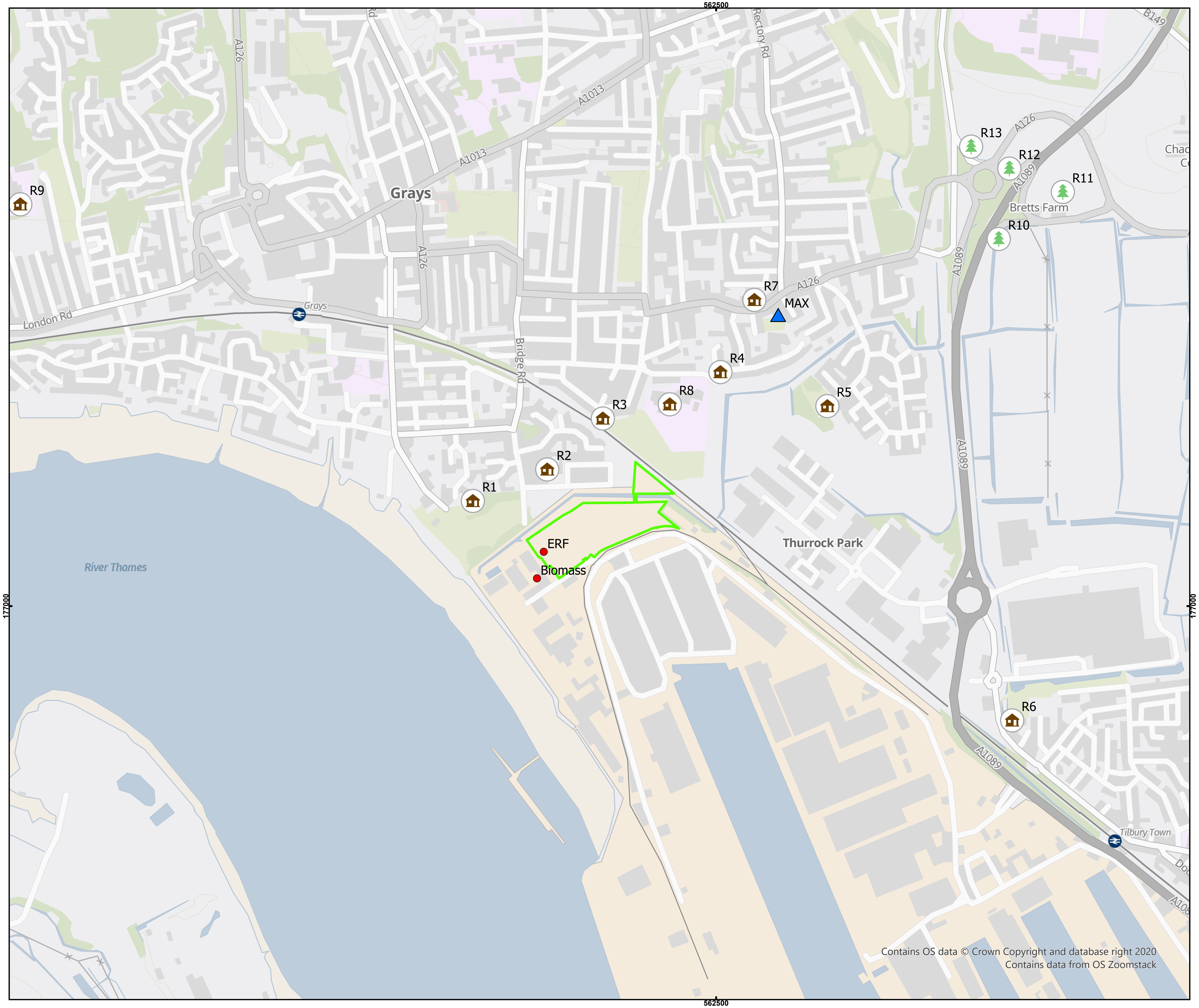
Table 12: Comparison with Total Dioxin and Dioxin-Like PCBs TDI Limits for Adult Receptors

Receptor	Total Inhalation, (pg WHO-TEQ kg^{-1} bw day^{-1})		Total Ingestion, (pg WHO-TEQ kg^{-1} bw day^{-1})		Inhalation + Ingestion (pg WHO-TEQ kg^{-1} bw day^{-1})		Comparison (% of TDI)
	Facility	TGP	Facility	TGP	Facility	TGP	Overall
MDI (% of TDI)							**35.00%**
Point of maximum impact - agricultural	1.19E-04	6.59E-05	4.55E-02	6.59E-05	4.56E-02	6.59E-05	37.29%
Point of maximum impact - residential	1.19E-04	6.59E-05	9.02E-04	6.59E-05	1.02E-03	6.59E-05	35.05%
R1	7.85E-06	5.49E-06	5.94E-05	5.49E-06	6.73E-05	5.49E-06	35.00%
R2	1.34E-06	2.70E-06	1.02E-05	2.70E-06	1.15E-05	2.70E-06	35.00%
R3	9.76E-06	1.08E-05	7.40E-05	1.08E-05	8.37E-05	1.08E-05	35.00%
R4	7.44E-05	3.98E-05	5.63E-04	3.98E-05	6.38E-04	3.98E-05	35.03%
R5	9.94E-05	5.98E-05	7.53E-04	5.98E-05	8.52E-04	5.98E-05	35.05%
R6	4.19E-05	2.95E-05	3.17E-04	2.95E-05	3.59E-04	2.95E-05	35.02%
R7	1.08E-04	5.08E-05	8.21E-04	5.08E-05	9.29E-04	5.08E-05	35.05%
R8	4.29E-05	3.00E-05	3.25E-04	3.00E-05	3.68E-04	3.00E-05	35.02%
R9	1.87E-05	1.33E-05	1.42E-04	1.33E-05	1.61E-04	1.33E-05	35.01%
R10	1.08E-04	6.04E-05	4.14E-02	6.04E-05	4.15E-02	6.04E-05	37.08%
R11	9.46E-05	5.44E-05	3.62E-02	5.44E-05	3.63E-02	5.44E-05	36.82%
R12	9.24E-05	5.28E-05	3.53E-02	5.28E-05	3.54E-02	5.28E-05	36.77%
R13	9.66E-05	5.13E-05	3.69E-02	5.13E-05	3.70E-02	5.13E-05	36.85%

Table 13: Comparison with Total Dioxin and Dioxin-Like PCBs TDI Limits for Child Receptors

Receptor	Total Inhalation, (pg WHO-TEQ kg^{-1} bw day^{-1})		Total Ingestion, (pg WHO-TEQ kg^{-1} bw day^{-1})		Inhalation + Ingestion (pg WHO-TEQ kg^{-1} bw day^{-1})		Comparison (% of TDI)
	Facility	TGP	Facility	TGP	Facility	TGP	Overall
MDI (% of TDI)							**90.65%**
Point of maximum impact - agricultural	1.50E-04	8.30E-05	6.49E-02	8.30E-05	6.51E-02	8.30E-05	93.91%
Point of maximum impact - residential	1.50E-04	8.30E-05	3.46E-03	8.30E-05	3.61E-03	8.30E-05	90.83%
R1	9.90E-06	6.92E-06	2.28E-04	6.92E-06	2.38E-04	6.92E-06	90.66%
R2	1.69E-06	3.40E-06	3.90E-05	3.40E-06	4.07E-05	3.40E-06	90.65%
R3	1.23E-05	1.36E-05	2.84E-04	1.36E-05	2.96E-04	1.36E-05	90.67%
R4	9.37E-05	5.02E-05	2.16E-03	5.02E-05	2.25E-03	5.02E-05	90.77%
R5	1.25E-04	7.53E-05	2.88E-03	7.53E-05	3.01E-03	7.53E-05	90.80%
R6	5.28E-05	3.72E-05	1.22E-03	3.72E-05	1.27E-03	3.72E-05	90.72%
R7	1.37E-04	6.40E-05	3.15E-03	6.40E-05	3.28E-03	6.40E-05	90.82%
R8	5.40E-05	3.78E-05	1.25E-03	3.78E-05	1.30E-03	3.78E-05	90.72%
R9	2.36E-05	1.68E-05	5.44E-04	1.68E-05	5.67E-04	1.68E-05	90.68%
R10	1.36E-04	7.60E-05	5.90E-02	7.60E-05	5.92E-02	7.60E-05	93.61%
R11	1.19E-04	6.85E-05	5.16E-02	6.85E-05	5.17E-02	6.85E-05	93.24%
R12	1.16E-04	6.65E-05	5.04E-02	6.65E-05	5.05E-02	6.65E-05	93.18%
R13	1.22E-04	6.47E-05	5.27E-02	6.47E-05	5.28E-02	6.47E-05	93.29%

B Location of Sensitive Receptors



Legend

- Stack location
- Installation Site Boundary

Type of Receptor

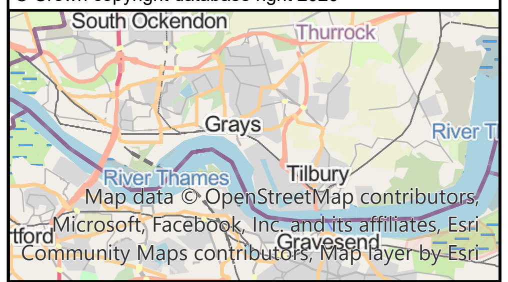
- Agricultural
- Agricultural / Residential
- Residential

Client:	Thameside Energy Recovery Facility Limited
Site:	Thameside Energy Recovery Facility
Project:	3265

Title:

Figure 2. Sensitive Receptors

Drawn by: Matt Clegg	Date: 23/06/2022



0 0.1 0.2 0.4 km

Scale: 1:10,000

N

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