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Riverside Resource Recovery Facility

Riverside Resource Recovery Limited

Dioxins Pathway Intake Assessment

ENGINEERING CONSULTING

Document approval

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1 Introduction

Fichtner Consulting Engineers Ltd (Fichtner) has been engaged by Riverside Resource Recovery Limited (RRRL) to undertake a Dioxins Pathway Intake Assessment (DPIA) to support the environmental permit (EP) variation application for the Riverside Resource Recovery Facility (the Facility).

RRRL is proposing the following changes to the EP:

- Increasing the processing capacity of the Facility from 785,000 tonnes per annum to 850,000 tonnes per annum; and
- Reducing the emission limit values (ELVs) within the EP to align with the upper range of the relevant BAT-AELs (Best Available Techniques-Associated Emissions Levels) for an 'existing facility' as defined within the WI BREF (Waste Incineration Best Available Techniques Reference document).

This DPIA has been undertaken to assess the effect of the Facility operating at the proposed annual throughput and ELVs.

The WI BREF was published by the European Integrated Pollution Prevention and Control (IPPC) Bureau in December 2019. The WI BREF has introduced BAT-AELs which are more stringent than those currently set out in the IED for some pollutants, including dioxins, furans and dioxin-like poly-chlorinated biphenyls (PCBs). We have assumed that the emissions from the Facility will comply with the upper range of the relevant BAT-AELs set out in the WI BREF for existing plants.

The advice from health specialists such as the Health Protection Agency 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 dioxin releases.

For most substances released from the Facility, the most significant effects on human health will arise by inhalation. The Air Quality Assessment Levels (AQALs) outlined within the Air Quality Assessment (AQA) 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 AQALs, then the pollutant is unlikely to have an adverse effect on human health.

For dioxins, furans, and dioxin-like PCBs, which accumulate in the environment, inhalation is only one of the potential exposure routes. Therefore, other exposure routes are considered within 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 of RCE-13 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. 128 106-115

[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. "Risk of congenital anomalies near municipal waste incinerators in England and Scotland: Retrospective population-based cohort study". Environment International (Parkes et al).

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 emission limits stated in the IED. Neither paper found any evidence of an association of waste incineration facilities with the health outcomes considered. Given that the Facility will actually operate to tighter standards, as RRRL is applying for the upper range of the relevant BAT-AELs from the WI 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 has been undertaken and is presented in 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. The Facility will meet the upper range of the relevant BAT-AELs outlined in the WI BREF for an existing plant. Limits have been set 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 the impact of inhalation of these pollutants on human health is presented in the AQA. 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 5.0, "IRAP"). In addition, a review of published literature on the health effects of energy recovery facilities has been undertaken and is presented in section 2.

The consented but not yet constructed Riverside Energy Park (REP) will be located on adjacent land to the west of the Facility. The EP for REP includes the emission limits for a 'New Facility' as defined in the Waste Incineration BREF for dioxins, furans and dioxin-like PCBs. Therefore, the contribution from REP has also been considered within this assessment.

3.2 Chemicals of Potential Concern (COPC)

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

- PCDD/Fs (dioxins and furans, individual congeners); and
- Dioxin-like PCBs;

This assessment considers 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 Environment Agency'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. A cumulative assessment which includes the contribution from REP has also been undertaken.

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"(Environment Agency 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 8.

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

The IRAP software, 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.

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 Table 2, and have been included in the IRAP-h model.

Table 2: Pathways Considered

Pathway	Residential	Agricultural	Allotment
Direct inhalation	Yes	Yes	Yes
Ingestion of soil	Yes	Yes	Yes
Ingestion of home-grown produce	Yes	Yes	Yes
Ingestion of drinking water	Yes	Yes	Yes
Ingestion of eggs from home-grown chickens	-	Yes	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		

Some households may keep chickens and consume the eggs. The impact on these households is considered to be between the impact at an agricultural receptor and a standard resident receptor. The approach used within this assessment considers an agricultural receptor at the point of maximum impact as a worst case assumption.

As shown in Table 2, the pathway from the ingestion of mother's milk in infants is considered within this 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

[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.

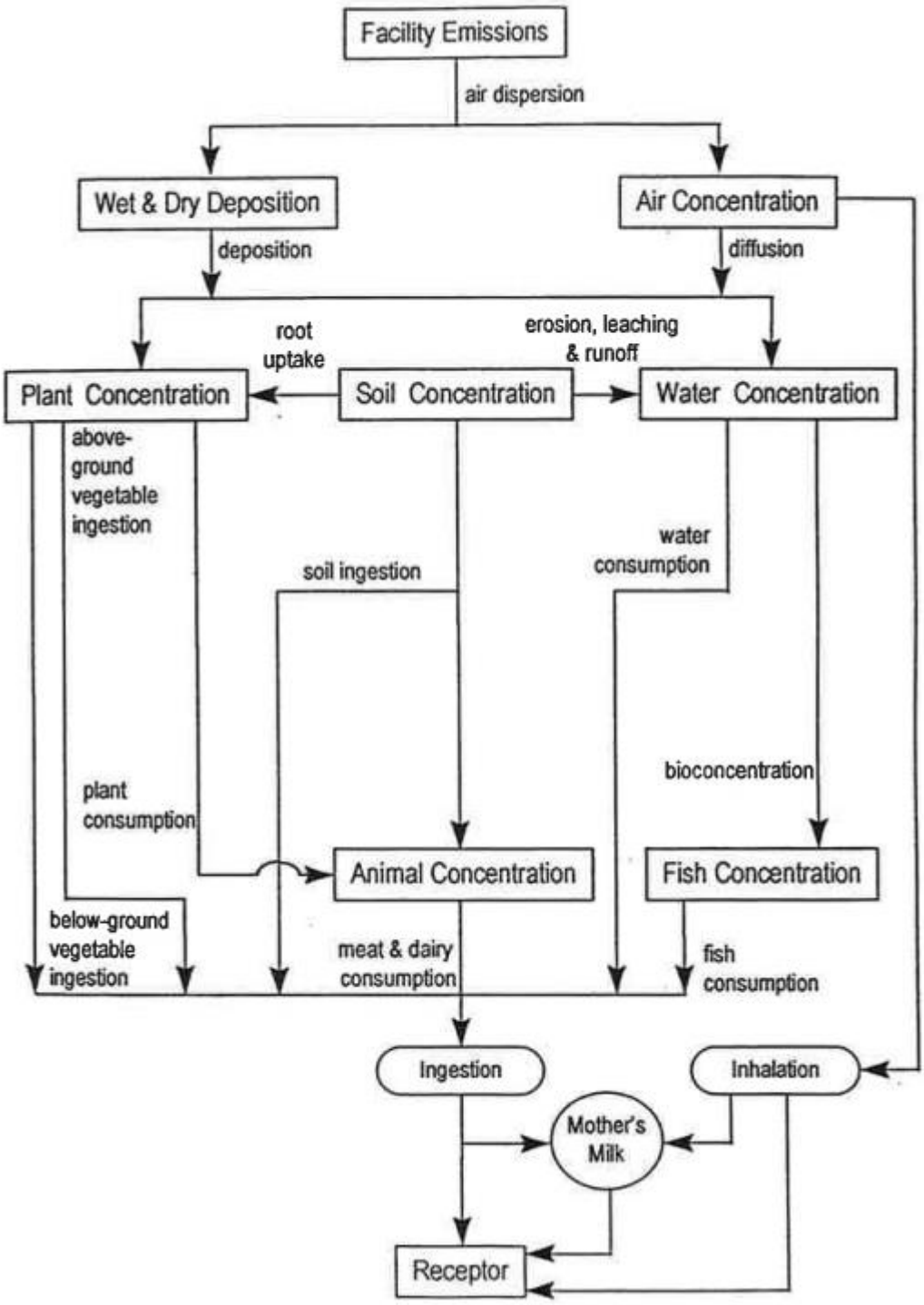
data for take-up and accumulation rates within the food chain for two groups of PCBs, known as Aroclor 1254 and Aroclor 1016. IRAP does not include these when determining the intake via mother's milk. The WI BREF includes the upper range of the relevant BAT-AELs, as follows:

Table 3: BAT-AELs for Dioxins and Furans and Dioxins, Furans and Dioxin-Like PCBs

Plant Category	BAT-AEL (ng I-TEQ/Nm³)		Factor
	Dioxins and Furans	Dioxins, Furans, and Dioxin-Like PCBs	
Existing Plant	0.06	0.08	1.33
New Plant	0.04	0.06	1.50

For all intake pathways other than breast milk, IRAP considers dioxins and furans separately from dioxin-like PBCs. To account for the accumulation of dioxin-like PCBs in breast milk, the intake of dioxins and furans via breast milk has been increased by the factors above, i.e., by 1.33 for the Facility and 1.50 for REP. The impacts have then compared against the total TDI for dioxins, furans, and dioxin-like PCBs.

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 subsidence farmer in a subarea 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] have 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 this would be sourced wide-scale from 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 the Norton Fishery near Abridge, located approximately 18 km north 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 fishery is very low. It is possible (but unlikely) that game fishing takes place along rivers 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. 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 annual mean point of maximum impact of emissions from the Facility.

The receptor locations have been categorised as 'residential', 'allotment' or 'agricultural'. Residential receptors represent occupied residences within the study area. Agricultural receptors represent a farm holding or farmland. As shown in Table 2, 'residential' receptors already include the ingestion of home-grown produce. However, allotment owners might also keep chickens and consume the eggs. The consumption of home-grown poultry is considered negligible. Therefore, 'allotment' type receptors include the consumption of eggs from home-grown chickens. As a conservative measure, schools have been included as 'allotment' type receptors.

The specific receptors identified in the AQA that are within 2.5 km of the stack of the Facility have been considered in this assessment. The receptor naming convention from the AQA has been retained. In addition, a receptor has been assessed at the point of maximum impact, although this point is actually within the River Thames.

The area surrounding the Facility is mainly urban, with few allotments or areas of agricultural interest. The allotment located closest to the Facility (Gilbert Road Allotments) has been included as a receptor. Although no farms lie within 2.5 km of the stack, part of Rainham Marshes lies within this distance. This land may be managed by grazing by cattle or sheep, so the closest part of Rainham Marshes to the Facility has been included as an agricultural receptor within this assessment.

The sensitive receptors considered are listed in Table 4, and a plan showing the location of these receptors in relation to the Facility is presented in Appendix B.

Table 4: Sensitive Receptors

ID	Receptor Name	Location		Type of receptor
		X	Y	
MAX	Point of maximum impact	550625	180974	Agricultural / Allotment / Residential
R01	Harris Garrard Academy East	548447	179561	Allotment
R02	Harris Garrard Academy West	548203	179699	Allotment
R03	St Brides Close	547979	179883	Resident
R04	Jubilee Primary School	547366	180534	Allotment
R05	Cherbury Close	548054	181106	Resident
R06	Sunningdale Close	548067	181170	Resident
R18	Hart Way	550566	182761	Resident
R24	Lower Road	550668	178833	Resident
R25	Clydesdale Way	549736	179858	Resident
R26	Wallace Close	547786	180716	Resident
R27	Little Brights Road	549632	179716	Resident
R28	Norman Road	549598	179653	Resident
R29	Gilbert Road Allotments	549040	179170	Allotment

ID	Receptor Name	Location		Type of receptor
		X	Y	
R30	Rainham Marshes	551374	181274	Agricultural

7 IRAP Model Assumptions and Inputs

The following section details the user defined assumptions used within the IRAP-h model are presented in sections 7.1 to 7.6, and justifications for these assumptions are provided 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 above ground vegetables.

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

These are specified within the Environment Agency series of reports: "Contaminants in soil: updated collation of toxicology data and intake values for humans". 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 — 0.688kg/day

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.

Weather data was obtained for the period 2015 to 2019 from the London City Airport weather station, as used within the dispersion modelling. This provides the annual average precipitation which can be used to calculate the general IRAP-h input parameters, as presented in Table 5.

Table 5: Ground Type Dependent Properties

Input Variable	Assumption	Value (cm/year)
Annual average evapotranspiration	70% of annual average precipitation	47.88
Annual average irrigation	0% of annual average precipitation	0.00
Annual average precipitation	100% of annual average precipitation	68.40
Annual average runoff	10% of annual average precipitation	6.84

The average wind speed was taken as 4.41 m/s, calculated from the average of the five years of weather data used in the dispersion modelling.

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

Table 6: 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
Note: The above deposition velocities have been agreed with the UK Environment Agency for all IRAP based assessments where modelling of specific deposition of pollutants is not undertaken. These are considered to be conservative.			

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

7.6 Modelled emissions

For the purpose of this assessment, it is assumed that the Facility and REP operate at the upper range of the relevant BAT-AELs within the WI BREF for their entire operational life. In reality both Facilities will be shut down for periods of maintenance and will typically operate below the emission limits prescribed in the relevant permit.

The following table presents the emissions rates of each COPC modelled and the associated ELVs which have been used to derive the emission rate.

Table 7: COPC Emissions Modelled

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	REP	Facility	REP
Sum I-TEQ	-	0.06	0.04	-	-
2,3,7,8-TCDD	0.031	0.0019	0.0012	0.297	0.148
1,2,3,7,8-PeCDD	0.245	0.0147	0.0098	2.351	1.167

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	REP	Facility	REP
1,2,3,4,7,8-HxCDD	0.287	0.0172	0.0115	2.754	1.367
1,2,3,6,7,8-HxCDD	0.258	0.0155	0.0103	2.475	1.228
1,2,3,7,8,9-HxCDD	0.205	0.0123	0.0082	1.967	0.976
1,2,3,4,6,7,8-HpCDD	1.704	0.1022	0.0681	16.349	8.114
OCDD	4.042	0.2424	0.1616	38.780	19.246
2,3,7,8-TCDF	0.277	0.0166	0.0111	2.658	1.319
1,2,3,7,8-PCDF	0.277	0.0166	0.0111	2.658	1.319
2,3,4,7,8-PCDF	0.535	0.0321	0.0214	5.133	2.547
1,2,3,4,7,8-HxCDF	2.179	0.1307	0.0871	20.906	10.376
1,2,3,6,7,8-HxCDF	0.807	0.0484	0.0323	7.743	3.843
1,2,3,7,8,9-HxCDF	0.042	0.0025	0.0017	0.403	0.200
2,3,4,6,7,8-HxCDF	0.871	0.0522	0.0348	8.357	4.147
1,2,3,4,6,7,8-HpCDF	4.395	0.2636	0.1757	42.167	20.927
1,2,3,4,7,8,9-HpCDF	0.429	0.0257	0.0172	4.116	2.043
OCDF	3.566	0.2139	0.1426	34.213	16.980
Total dioxins	20.150	0.0019	0.8057	193.33	95.946
Dioxin-like PCBs	-	0.0092	0.0092	1.472	1.096

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, 11% oxygen, 273.15K.
(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 7. 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 Environment Agency 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 x 10^{-3} ng[TEQ]/m³
- Minimum = 5.6 x 10^{-5} ng[TEQ]/m³

To ensure that this assessment is conservative, the maximum monitored PCB concentration has been used. The maximum concentration has been converted to an emission rate using the volumetric flow.

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. We are not aware of any data on the specification of PCBs within incinerator or co-incinerator emissions; therefore, as a worst-case assumption we have 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 shown in Table 7 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-h model has been multiplied by the relevant WHO-TEFs. The I-TEFs and WHO-TEFs are shown in Table 8.

Table 8: 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
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), Environment Agency, 2009

8 Results

8.1 Assessment against TDI - point of maximum impact

The following table presents the impact of emissions of dioxins and dioxin-like PCBs from the Facility and from REP at the point of maximum impact of emissions from the Facility. As explained in section 4, the 'agricultural' 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 a very worst-case scenario, especially given that the point on maximum impact is located within the River Thames, as shown in the plan showing the location of these receptors in relation to the Facility in Appendix B.

Table 9: Impact Analysis – Dioxins and Dioxin-Like PCBs – Point of Maximum Impact

Receptor Type	MDI (% of TDI)	Process Contribution (% of TDI)		Overall (% of TDI)
		Facility	REP	Cumulative
Adult				
Agricultural	35.00%	5.04%	2.71%	42.75%
Allotment	35.00%	0.16%	0.08%	35.24%
Residential	35.00%	0.11%	0.06%	35.18%
Child				
Agricultural	90.65%	7.12%	3.83%	**101.60%**
Allotment	90.65%	0.44%	0.24%	91.33%
Residential	90.65%	0.35%	0.19%	91.20%

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 9, for all receptor types, at the point of maximum impact, the overall intake (including the contribution from existing dietary intakes and the contribution from REP) is less than the TDI for dioxins and dioxin-like PCBs, except for an agricultural child receptor. Therefore, there would not be an appreciable health risk based on the emission of these pollutants for adult receptors.

For an agricultural child receptor, the overall intake slightly exceeds the TDI. The contribution from the Facility is predicted to be 7.12% of the TDI, and the contribution from REP is predicted to be 3.83% of the TDI. However, as detailed in section 6 the point of maximum impact lies within the River Thames. Therefore, there is no relevant exposure at this point, and is not an impact which would happen in reality. The maximum impact at receptor locations is discussed in section 8.3.

8.2 Breast milk exposure

The total accumulation of dioxins in an infant due to emissions from the Facility, considering the breast milk pathway and based on an adult agricultural receptor at the point of maximum impact feeding an infant, is summarised in Table 10.

Table 10: Breast Milk Exposure

Receptor Type	Process Contribution (% of TDI)		Overall (% of TDI)
	Facility	REP	Cumulative
Agricultural	38.41%	23.02%	61.43%
Allotment	0.98%	0.59%	1.56%
Residential	0.73%	0.44%	1.17%

The maximum intake for an infant being breast fed by an agricultural receptor at the point of maximum impact is predicted to be 38.41% of the TDI. This is only 0.98% and 0.73% of the TDI for an allotment and residential-type receptor respectively. The contribution from REP at the point of maximum impact of the Facility is 23.02% of the TDI for an infant being breast fed by an agricultural receptor, so the theoretical maximum cumulative intake is 61.43% of the TDI.

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

8.3 Maximum impact at receptors

The following tables outline the impact of emissions from the Facility at the most affected receptor of each type (i.e., the receptors with the greatest impact from ingestion and inhalation of emissions). This overall maximum impacted receptor (R30 – Rainham Marshes) has been classified as an agricultural receptor, which is conservative as it assumes that a significant proportion of the diet of the receptor is sourced from the receptor point assessed, including meat and milk products. In reality, this location is only intermittently grazed by sheep and cattle. Given that people in the UK tend to source their diet from a wide geographical area, very little intake will occur due to any accumulation of dioxins at this receptor location.

Table 11: Dioxins and Dioxin-Like PCBs Impact – Maximum Impacted Receptors

Receptor Type	MDI (% of TDI)	Process Contribution (% of TDI)		Overall (% of TDI)
		Facility	REP	Cumulative
Adult				
Agricultural (R30)	35.00%	3.63%	1.93%	40.56%
Allotment (R04)	35.00%	0.03%	0.03%	35.06%
Residential (R26)	35.00%	0.03%	0.03%	35.05%
Child				
Agricultural (R30)	90.65%	5.13%	2.73%	98.51%
Allotment (R04)	90.65%	0.09%	0.07%	90.92%
Residential (R26)	90.65%	0.09%	0.08%	90.92%

For the maximum impacted receptor of each receptor type the overall impact (including the contribution from existing dietary intakes) 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.

Table 12: Breast Milk Exposure – Maximum Impacted Receptors

Receptor Type	Process Contribution (% of TDI)		Overall (% of TDI)
	Facility	REP	Cumulative
Agricultural (R30)	27.69%	16.40%	44.08%
Allotment (R04)	0.21%	0.18%	0.39%
Residential (R26)	0.18%	0.18%	0.36%

The maximum intake for an infant being breast fed by an agricultural receptor at the maximum impacted receptor (R30, Rainham Marshes) is predicted to be 27.69% of the TDI. This is only 0.21% and 0.18% of the TDI at the maximum impacted allotment and residential-type receptors respectively. The contribution from REP at R30 is 16.40% of the TDI for an infant being breast fed by an agricultural receptor, so the theoretical maximum cumulative intake at a receptor location is 44.08% of the TDI. However, this is based on consumption of contaminated produce, milk and meat from this receptor location. In reality, Rainham Marshes is only intermittently used for sheep and cattle grazing and therefore significant intake via these pathways is unlikely to occur in reality.

The predicted process contribution at the maximum impacted receptors is below the TDI, even under worst-case assumptions and including the effect of emissions from REP. Therefore, the impact of emissions from the Facility is negligible and it follows that the impact at the other residential receptors considered in the AQA will be even less.

Detailed results for all receptor locations shown in Table 4 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, residential, allotment and agricultural-type 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, we have assumed that PCBs 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 1.33 for the Facility and 1.50 for REP has been applied to dioxins and dioxin-like PCBs when considering the impact of the intake

via mother's milk. This aligns with the ELVs of 0.08 ng I-TEQ/Nm³ for the Facility and 0.06 ng I-TEQ/Nm³ for REP for dioxins and furans when dioxin-like PCBs are included.

9 Conclusions

This DPIA has been undertaken based on the following conservative assumptions:

- the Facility and REP will operate continually at the upper range of the relevant BAT-AELs as stated within the Waste Incineration BREF, i.e., at the maximum concentrations at which it is expected that the Facility and REP will be permitted to operate at; 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 an agricultural child receptor at the point of maximum impact, the combined intake from the Facility plus the contribution from REP and the existing MDI intake of dioxins and dioxin-like PBCs slightly exceeds the TDI. However, this point lies within the River Thames and is not an area of relevant exposure.

For an agricultural child receptor at the maximum impacted receptor, the combined intake from the Facility plus the contribution from REP 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 breast fed by an agricultural receptor at the maximum impacted receptor is less than the TDI. Assessing the impact at this receptor is highly conservative as it is an uninhabited point in Rainham Marshes selected to represent the occasional use of the land for grazing. The impact at all other identified receptor locations is even less. Therefore, there would not be an appreciable health risk based on the emission of these pollutants.

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

Appendices

A Detailed Results Tables

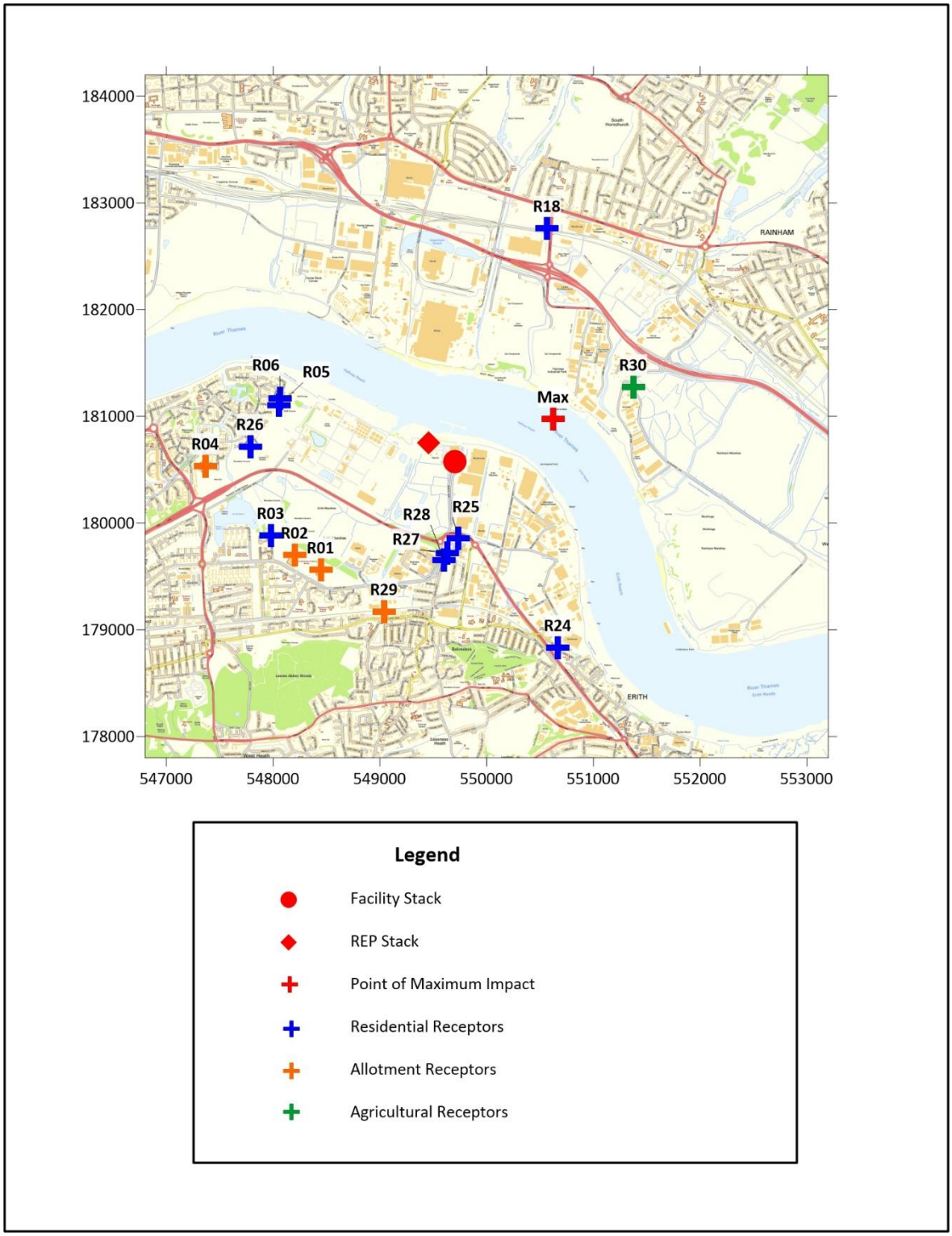
Table 13: Comparison with Total Dioxin and Dioxin-Like PCBs TDI Limits for Adult Receptors

Receptor	Total Inhalation, (pg WHO-TEQ kg^{-1} bw day^{-1})		Total Ingestion, (pg WHO-TEQ kg^{-1} bw day^{-1})		Total uptake, (pg WHO-TEQ kg^{-1} bw day^{-1})		Comparison (% of limit) - Cumulative
	Facility	REP	Facility	REP	Facility	REP	
MDI (% of TDI)							**35.00%**
Point of maximum impact - agricultural	2.75E-04	1.56E-04	1.00E-01	5.41E-02	1.01E-01	5.42E-02	42.750%
Point of maximum impact - allotment	2.75E-04	1.56E-04	2.83E-03	1.53E-03	3.11E-03	1.68E-03	35.240%
Point of maximum impact - residential	2.75E-04	1.56E-04	2.00E-03	1.08E-03	2.27E-03	1.24E-03	35.175%
R01 - Harris Garrard Academy East	3.43E-05	2.63E-05	3.53E-04	2.58E-04	3.87E-04	2.85E-04	35.034%
R02 - Harris Garrard Academy West	4.85E-05	2.91E-05	5.00E-04	2.86E-04	5.49E-04	3.15E-04	35.043%
R03 - St Brides Close	5.74E-05	4.54E-05	4.17E-04	3.15E-04	4.75E-04	3.60E-04	35.042%
R04 - Jubilee Primary School	5.78E-05	4.85E-05	5.96E-04	4.76E-04	6.54E-04	5.25E-04	35.059%
R05 - Cherbury Close	4.20E-05	4.87E-05	3.06E-04	3.38E-04	3.48E-04	3.86E-04	35.037%
R06 - Sunningdale Close	3.91E-05	4.40E-05	2.84E-04	3.05E-04	3.24E-04	3.49E-04	35.034%
R18 - Hart Way	4.54E-05	5.18E-05	3.30E-04	3.59E-04	3.76E-04	4.11E-04	35.039%
R24 - Lower Road	2.97E-05	2.16E-05	2.16E-04	1.50E-04	2.46E-04	1.72E-04	35.021%
R25 - Clydesdale Way	2.96E-05	4.03E-05	2.15E-04	2.79E-04	2.45E-04	3.20E-04	35.028%
R26 - Wallace Close	6.79E-05	6.48E-05	4.94E-04	4.49E-04	5.62E-04	5.14E-04	35.054%
R27 - Little Brights Road	2.88E-05	3.67E-05	2.10E-04	2.55E-04	2.39E-04	2.91E-04	35.026%
R28 - Norman Road	2.79E-05	3.45E-05	2.03E-04	2.39E-04	2.31E-04	2.74E-04	35.025%
R29 - Gilbert Road Allotments	2.61E-05	2.09E-05	2.69E-04	2.05E-04	2.95E-04	2.26E-04	35.026%
R30 - Rainham Marshes	1.98E-04	1.11E-04	7.24E-02	3.85E-02	7.26E-02	3.86E-02	40.563%

Table 14: 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})		Total uptake, (pg WHO-TEQ kg^{-1} bw day^{-1})		Comparison (% of limit) - Cumulative
	Facility	REP	Facility	REP	Facility	REP	
MDI (% of TDI)							**90.65%**
Point of maximum impact - agricultural	3.46E-04	1.96E-04	1.42E-01	7.64E-02	1.42E-01	7.66E-02	**101.603%**
Point of maximum impact - allotment	3.46E-04	1.96E-04	8.45E-03	4.58E-03	8.79E-03	4.77E-03	91.328%
Point of maximum impact - residential	3.46E-04	1.96E-04	6.72E-03	3.65E-03	7.07E-03	3.85E-03	91.196%
R01 - Harris Garrard Academy East	4.32E-05	3.32E-05	1.05E-03	7.74E-04	1.10E-03	8.07E-04	90.745%
R02 - Harris Garrard Academy West	6.11E-05	3.67E-05	1.49E-03	8.56E-04	1.55E-03	8.93E-04	90.772%
R03 - St Brides Close	7.23E-05	5.72E-05	1.40E-03	1.07E-03	1.48E-03	1.12E-03	90.780%
R04 - Jubilee Primary School	7.29E-05	6.11E-05	1.78E-03	1.43E-03	1.85E-03	1.49E-03	90.817%
R05 - Cherbury Close	5.30E-05	6.14E-05	1.03E-03	1.14E-03	1.08E-03	1.20E-03	90.764%
R06 - Sunningdale Close	4.93E-05	5.55E-05	9.57E-04	1.03E-03	1.01E-03	1.09E-03	90.755%
R18 - Hart Way	5.72E-05	6.53E-05	1.11E-03	1.22E-03	1.17E-03	1.28E-03	90.772%
R24 - Lower Road	3.74E-05	2.73E-05	7.27E-04	5.08E-04	7.64E-04	5.35E-04	90.715%
R25 - Clydesdale Way	3.73E-05	5.08E-05	7.24E-04	9.46E-04	7.62E-04	9.97E-04	90.738%
R26 - Wallace Close	8.56E-05	8.16E-05	1.66E-03	1.52E-03	1.75E-03	1.60E-03	90.817%
R27 - Little Brights Road	3.63E-05	4.63E-05	7.06E-04	8.62E-04	7.42E-04	9.08E-04	90.732%
R28 - Norman Road	3.52E-05	4.35E-05	6.84E-04	8.10E-04	7.19E-04	8.54E-04	90.729%
R29 - Gilbert Road Allotments	3.29E-05	2.63E-05	8.03E-04	6.13E-04	8.36E-04	6.40E-04	90.724%
R30 - Rainham Marshes	2.49E-04	1.40E-04	1.02E-01	5.44E-02	1.03E-01	5.45E-02	98.512%

B Location of Sensitive Receptors



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