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Dioxin Pathway Intake Assessment

ENGINEERING CONSULTING

Document approval

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Document revision record

Revision no	Date	Details of revisions	Prepared by	Checked by
0	19/07/2024	First Issue	RSF	SMN

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1 Introduction

Fichtner Consulting Engineers Ltd (Fichtner) has been engaged by Indaver to undertake a Dioxin Pathway Intake Assessment to support the application for a variation to the Environmental Permit (EP) for the Rivenhall Integrated Waste Management Facility (IWMF). The IWMF includes a Combined Heat and Power (CHP) Plant (herein referred to as the CHP Plant) which comprises two waste incineration lines.

As the fuel combusted at the CHP Plant is sourced from waste, the limits on emissions to air are 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, and the Waste Incineration Best Available Techniques Reference document (BREF). This includes limits on emissions of dioxins and furans (collectively referred to as “dioxins” for the purpose of this assessment).

The advice from health specialists such as the UK Health Security Agency (formerly 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 CHP Plant, the most significant effects on human health will arise by inhalation. However, for dioxins and dioxin-like polychlorinated biphenyls (PCBs) which accumulate in the environment, inhalation is only one of the potential exposure routes. This is the only source within the IWMF which includes a limit on emissions to air of these substances.

For dioxins and dioxin-like PCBs the health assessment criteria are expressed as the total intake from ingestion and inhalation. Therefore, this assessment considers exposure routes other than just inhalation.

2 Literature review

The HPA, whose role was taken over by Public Health England (PHE) and more recently by the UK Health Security Agency, 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. 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 CHP Plant 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 will actually operate to tighter standards, as it will 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 CHP Plant of pollutants that accumulate in the environment would not be significant. Nonetheless, a quantitative assessment of the effect of emissions from the CHP Plant has been undertaken and is presented in the following sections.

3 Issue Identification

3.1 Issue

The key issue for consideration is the release of substances to atmosphere from the CHP Plant which have the potential to harm human health. Details of the dispersion modelling can be found in the Dispersion Modelling Assessment submitted with the EP application.

The CHP Plant will be required to meet the BAT AELs prescribed in the Waste Incineration BREF and the ELVs in the IED. 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. 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 5.1.1, "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 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.

The following table outlines the MDIs (the typical intake from existing background sources) and TDIs for dioxins.

Table 1: Assessment Criteria for Intake of Dioxins

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%	90.00%

Source: Contaminants in soil: updated collation of toxicology data and intake values for humans: dioxins, furans and dioxin-like PCBs, Environment Agency 2009[5].

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 CHP Plant is not significant.

[5] This document has been archived by the EA. The page detailing the TDI and MDI has been appended as Appendix C.

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[6]. This Protocol is a development of the approach defined by Her Majesties Inspectorate on Pollution (HMIP) in the UK in 1996[7], 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)	Yes	Yes

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

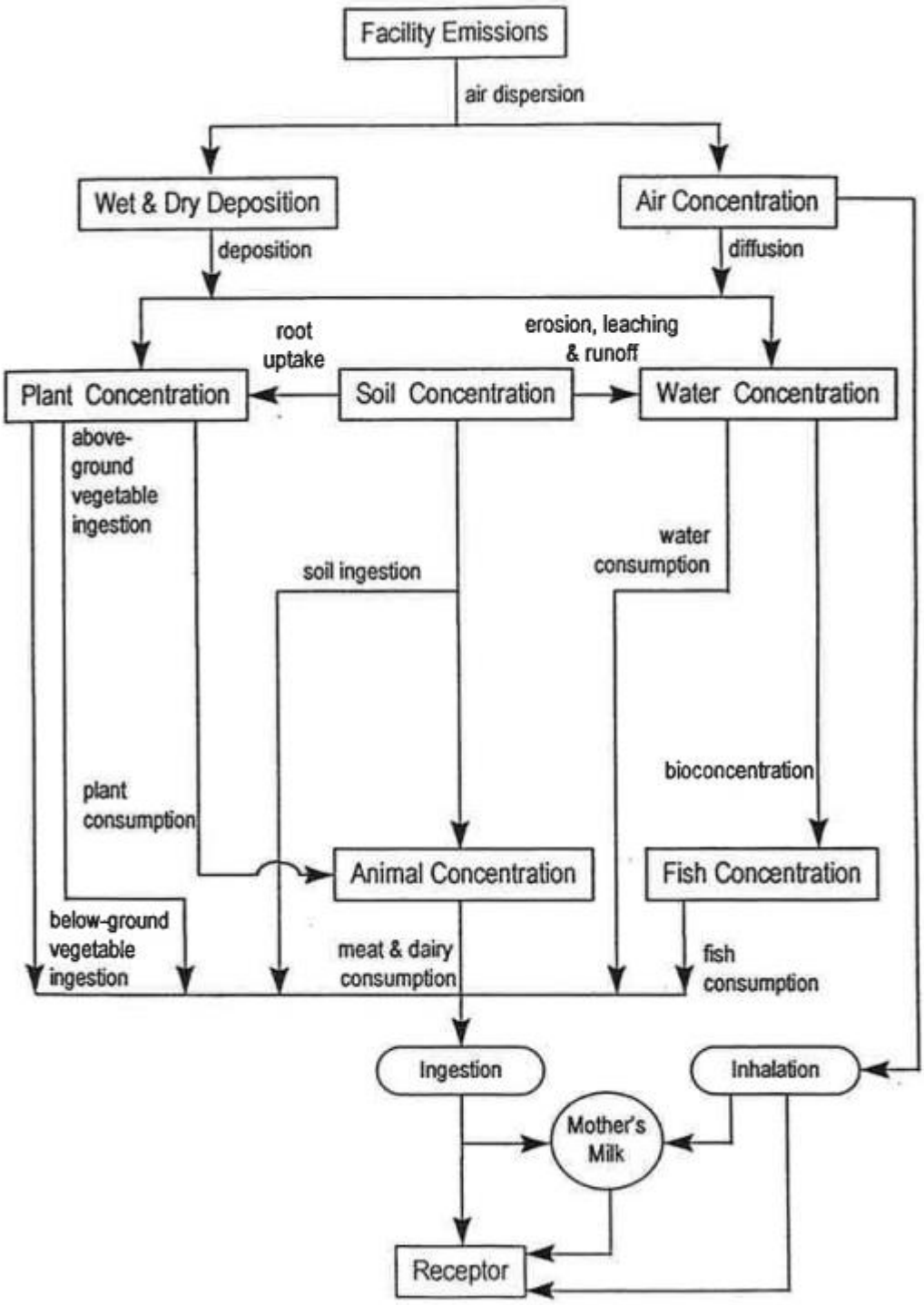
As shown in Figure 1, the pathway from the ingestion of mother's milk in infants is considered within the assessment. The IRAP model calculates the amount of dioxins entering the mother's milk and being passed on to the infants. IRAP does not include data on individual PCBs, but it does include data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

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

[7] 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. Therefore, a safety factor of 1.5 has been applied to the dioxin and dioxin-like PCBs emission rate when considering the impact of the intake via mother's milk. 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, and fish consumption 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[8] 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 an 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[9] 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[10] 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.

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

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

[10] 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. Whilst game fishing potentially takes place in the area around the IWMF, no game fishing lakes have been identified within the modelling domain, and the likelihood of persons sourcing a large proportion of their diet from a game fishery is very low. In addition, the accumulation of pollutants in river systems is not of significant concern, as any pollutants will be washed downstream rather than accumulating, and accumulation in estuaries and seas will be diluted by tidal action. Therefore, the fish consumption pathway has been excluded from this assessment.

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. Agricultural receptors represent a farm holding or area land of horticultural interest. Receptors have been defined as 'agricultural' if they are attached to, or surrounded by, agricultural land.

The specific receptors identified in the Dispersion Modelling Assessment have been considered in this assessment, with the exception of those receptors not representative of areas of long term exposure, e.g. the footpath (D18 to D35). An additional receptor has been included at the point of maximum impact, in which lies in an arable field (i.e. is in agricultural use, but not used for grazing animals). This point has been included as both a residential and fully agricultural receptor type to demonstrate the theoretical maximum impact of the CHP Plant The sensitive receptors assessed are listed in Table 3. Reference should be made to Appendix B which shows the location of these receptors with respect to the CHP Plant.

Table 3: Sensitive Receptors

ID	Receptor name	Location		Type of receptor
		X	Y	
Max	Max	582877	220665	Agricultural / Residential
R1	Sheepcotes Farm (Hanger No. 1)	581565	220328	Resident
R2	Allshot's Farm (Scrap Yard)	582893	220458	Resident
R3	Haywards	583236	221163	Resident
R4	Herons Farm	582443	221378	Resident
R5	Gosling Farm	582443	221378	Resident
R6	Curd Hall Farm	583262	221708	Resident
R7	Church (adjacent to Bradwell Hall)	581832	222158	Resident
R8	Bradwell Hall	581838	222319	Agricultural
R9	Rolphs Farmhouse	580676	220513	Agricultural
R10	Silver End / Bower Hall / Fossill Hall	581287	219731	Agricultural
R11	Rivenhall Pl/ Hall	581861	219104	Agricultural
R12	Parkgate Farm / Watchpall Cottages	582337	219195	Agricultural
R13	Ford Farm / Rivenhall Cottage	582698	218598	Agricultural
R14	Porter's Farm	583391	219242	Agricultural
R15	Unknown Building	583131	219462	Resident
R16	Bumby Hall / The Lodge / Polish Site (light industry)	582947	220115	Resident
R17	Green Pastures Bungalow	581249	221176	Resident
R18	Deeks Cottage	582873	221255	Resident

ID	Receptor name	Location		Type of receptor
		X	Y	
R19	Gosling Cottage / Barn	581508	221305	Resident
R20	Felix Hall / The Close House / Park Farm	584578	219574	Agricultural
R21	Glazenwood House	579980	222134	Resident
R22	Bradwell Hall	580570	222802	Agricultural
R23	Perry Green Farm	580899	221973	Agricultural
R24	The Granary / Porter Farm / Rook Hall	584106	218964	Agricultural
R25	Grange Farm	584888	222222	Agricultural
R26	Coggeshall	585070	222839	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 CHP Plant is taken as 30 years; and
- the soil mixing depth is taken as 2 cm in general and 20 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 – 20 m^3/day; and
- children – 7.2 m^3/day.

These are as specified within the Environment Agency'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
- Safety factor on total dioxin intake to account for 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.

Weather data from the Andrewsfield meteorological recording station has been obtained for the period 2018 to 2022, as used within the Dispersion Modelling Assessment. This has been 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	41.58
Annual average irrigation	0% of annual average precipitation	0
Annual average precipitation	100% of annual average precipitation	59.40
Annual average runoff	10% of annual average precipitation	5.94

The average wind speed was taken as 4.03 m/s, calculated from the average of the five years of weather data.

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 Dispersion Modelling Assessment.

As set out in the Dispersion Modelling Assessment the proposed phasing of the IWMF would mean that not all of the building would be constructed at the same time. The modelling showed that the greatest impact was predicted to occur if only the CHP Plant is operating and only the building for the CHP Plant has been constructed (i.e. not the full build out of the IWMF). As such this assessment has focussed on the impact for Phase 1 – i.e. only the CHP Plant. Predicted impacts with the other aspects of the IWMF are predicted to be lower due to the increased dispersion of combining the emissions from the other sources. None of the other sources would include emissions of dioxins or dioxin-like PCBs it is considered appropriate to base this assessment on the scenario with only the CHP Plant operating and only the build out for this aspect of the IWMF. The impact of the full build out will be lower.

7.6 Modelled emissions

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

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/Nm³[1]	Emission conc. (ng/Nm³)[2]	Emission rate (ng/s)[3]
Sum I-TEQ dioxins[4]	-	0.04 ng I-TEQ/Nm³	-
2,3,7,8-TCDD	0.031	0.0012	0.130
1,2,3,7,8-PeCDD	0.245	0.0098	1.025
1,2,3,4,7,8-HxCDD	0.287	0.0115	1.200
1,2,3,6,7,8-HxCDD	0.258	0.0103	1.079
1,2,3,7,8,9-HxCDD	0.205	0.0082	0.857
1,2,3,4,6,7,8-HpCDD	1.704	0.0681	7.127
OCDD	4.042	0.1616	16.906
2,3,7,8-TCDF	0.277	0.0111	1.159
1,2,3,7,8-PCDF	0.277	0.0111	1.159
2,3,4,7,8-PCDF	0.535	0.0214	2.238
1,2,3,4,7,8-HxCDF	2.179	0.0871	9.114
1,2,3,6,7,8-HxCDF	0.807	0.0323	3.375
1,2,3,7,8,9-HxCDF	0.042	0.0017	0.176
2,3,4,6,7,8-HxCDF	0.871	0.0348	3.643
1,2,3,4,6,7,8-HpCDF	4.395	0.1757	18.382
1,2,3,4,7,8,9-HpCDF	0.429	0.0172	1.794
OCDF	3.566	0.1426	14.915
Total dioxins	20.150	0.8057	84.279
Dioxin-like PCBs[5]	-	0.009	0.962

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.

(4) The Waste Incineration BREF includes an emission limit for dioxins of 0.04 ng I-TEQ/Nm³, or a combined limit of 0.06 ng WHO-TEQ/Nm³ for dioxins when dioxin-like PCBs are included. As this assessment considers dioxin-like PCBs separately, the lower limit of 0.04 ng I-TEQ/Nm³ for dioxins has been used.

(5) Refer to note 2 below this table.

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

For the purpose of this assessment, the maximum monitored PCB concentration has been used which 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. 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 is given in pg WHO-TEQ/kg bw/day. However, the split of congeners 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. Two sets of WHO-TEFs have been considered; those values published in 2005, and those published in 2022. The Environment Agency has not published any guidance relating to the publication of the 2022 WHO-TEFs. Correspondence with the Environment Agency has confirmed that the 2005 WHO-TEFs can continue being used for the main assessment, and a sensitivity analysis be undertaken using the 2022 WHO-TEFs (refer to section 8.4.1). The I-TEFs and WHO-TEFs are shown in Table 7.

Table 7: Toxic Equivalency Factors for Dioxins and Furans

Congener	IED I-TEF Multiplier(1)	2005 WHO-TEF Multiplier(1)	2022 WHO-TEF Multiplier(2)
Dioxins			
2,3,7,8-TCDD	1	1	1
1,2,3,7,8-PeCDD	0.5	1	0.4
1,2,3,4,7,8-HxCDD	0.1	0.1	0.09
1,2,3,6,7,8-HxCDD	0.1	0.1	0.07
1,2,3,7,8,9-HxCDD	0.1	0.1	0.05
1,2,3,4,6,7,8-HpCDD	0.01	0.01	0.05
OCDD	0.001	0.0003	0.001
Furans			
2,3,7,8-TCDF	0.1	0.1	0.07
1,2,3,7,8-PCDF	0.05	0.03	0.1

Congener	IED I-TEF Multiplier[(1)]	2005 WHO-TEF Multiplier[(1)]	2022 WHO-TEF Multiplier[(2)]
2,3,4,7,8-PCDF	0.5	0.3	0.01
1,2,3,4,7,8-HxCDF	0.1	0.1	0.3
1,2,3,6,7,8-HxCDF	0.1	0.1	0.09
1,2,3,7,8,9-HxCDF	0.1	0.1	0.2
2,3,4,6,7,8-HxCDF	0.1	0.1	0.1
1,2,3,4,6,7,8-HpCDF	0.01	0.01	0.02
1,2,3,4,7,8,9-HpCDF	0.01	0.01	0.1
OCDF	0.001	0.0003	0.002

Source: (1) 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. (2) The 2022 world health organization re-evaluation of human and mammalian toxic equivalency factors for polychlorinated dioxins, dibenzofurans and biphenyls, DeVito et al, 2023.

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 CHP Plant at the point of maximum impact of emissions from the CHP Plant 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 contained in Appendix B for the location of the point in relation to the IWMF.

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)
Adult			
Agricultural	35.00%	4.63%	39.63%
Residential	35.00%	0.11%	35.11%
Child			
Agricultural	90.00%	6.54%	96.54%
Residential	90.00%	0.34%	90.34%

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 impact (including the contribution from existing dietary intake) 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 CHP Plant, considering the breast milk pathway and based on an adult agricultural receptor at the point of maximum impact of emission from the CHP Plant feeding an infant, is 0.787 pg WHO-TEQ / kg-bw / day which is 39.33% of the TDI. For a residential-type receptor this is 0.016 pg WHO-TEQ / kg-bw / day, which is only 0.78% of the TDI.

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 in infants significantly.

8.3 Maximum impact at a receptor

The following table outlines the impact of emissions from the CHP Plant at the most affected receptor (i.e. the receptor with the greatest combined impact from ingestion and inhalation of emissions from the CHP Plant) (R25 – Grange Farm). This receptor 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, people in the UK tend to source their diet from a wide geographical area.

Table 9: Impact Analysis – Dioxins and Dioxin-Like PCBs – Maximum Impacted Receptor

Receptor type	MDI (% of TDI)	Process contribution (% of TDI)	Overall (% of TDI)
Adult			
Agricultural	35.00%	0.81%	35.81%
Child			
Agricultural	90.00%	1.15%	91.15%

As shown, for the most impacted receptor the overall impact (including the contribution from existing dietary intake) 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 CHP Plant considering the breast milk pathway and based on an adult agricultural receptor at R25 feeding an infant, is 0.138 pg WHO-TEQ / kg-bw / day which is 6.89% of the TDI. Therefore, as the process contribution is much less than the TDI, it is considered that the CHP Plant 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 CHP Plant.

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 the dioxin and dioxin-like PCBs emission rate when considering the impact of the intake via mother's milk.

8.4.1 Sensitivity analysis – WHO-TEFs

As detailed in Table 7 the output from the IRAP model for each congener has been multiplied by the appropriate WHO-TEF for comparison with the TDI. The WHO-TEFs were last updated in 2022, with the previous version being released in 2005. Correspondence with the Environment Agency has confirmed that the 2005 WHO-TEFs can continue being used for the main assessment, and a sensitivity analysis be undertaken using the 2022 WHO-TEFs.

The impact of the choice of WHO-TEFs on the results will depend on the split of congeners in the release and the intake pathways being assessed. As the CHP Plant is not yet operational the split of congeners is not known and the split of congeners from the HMIP document has been applied (refer to section 7.6). A comparison of the results at the point of maximum impact and the maximum impacted receptor is presented in Table 10.

Table 10: Sensitivity of Results to Choice of WHO-TEFs

Receptor ID	Receptor Type	Impact of emissions from CHP Plant (% of TDI)		
		2005 WHO-TEFs	2022 WHO-TEFs	% Change in impact
Max	Adult - agricultural	4.63%	5.35%	15.48%
	Child - agricultural	6.54%	7.59%	16.01%
	Infant - agricultural	39.33%	30.45%	-22.58%
	Adult - residential	0.11%	0.14%	31.59%
	Child - residential	0.34%	0.44%	31.43%
	Infant - residential	0.78%	0.68%	-13.01%
R25	Adult - agricultural	0.81%	0.94%	15.48%
	Child - agricultural	1.15%	1.33%	16.02%
	Infant - agricultural	6.89%	5.34%	-22.58%

As shown, the 2022 WHO-TEFs result in a higher impact than using the 2005 WHO-TEFs for all receptor types except infant receptors. However, applying the 2022 WHO-TEFs, for an agricultural child receptor at the point of maximum impact the impact of emissions from the CHP Plant is 7.59% of the TDI so the total intake is predicted to be 97.59% of the TDI. As this remains below the TDI, no significant effects are predicted based on the predicted impact using either the 2005 or 2022 WHO-TEFs.

9 Conclusions

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

- the CHP Plant will operate continually at the ELV for dioxins, i.e., at the maximum concentrations which it is expected that the CHP Plant 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 CHP Plant.

The impacts are based on Phase 1 of the IWMF – i.e. the operation of the CHP Plant without any further build out of the IWMF. The dispersion modelling assessment has demonstrated that this scenario results in the greatest impact, with the impact of the full build out of the IWMF lower. This is attributed to the increased dispersion as a result of combining the emissions from other sources (none of which include emissions of dioxins or dioxin-like PCBs), and the difference in the approach to modelling buildings given the site excavations.

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 CHP Plant), the combined intake from the CHP Plant and the existing MDI intake of dioxins and dioxin-like PCBs 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 point of maximum impact of emissions from the CHP Plant is below than the TDI. The impacts at identified receptor locations are lower. Therefore, there would not be an appreciable health risk based on the emission of dioxins and dioxin-like PCBs.

In conclusion, the impact of emissions of dioxins and dioxin-like PCBs from the CHP Plant on human health is predicted to be not significant even with the proposed phasing of the IWMF.

Appendices

A Detailed Results Tables

Table 11: 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 TDI)
MDI (% of TDI)				**35.00%**
Point of maximum impact - agricultural	2.65E-04	9.24E-02	9.27E-02	39.633%
Point of maximum impact - residential	2.65E-04	1.90E-03	2.17E-03	35.108%
R1	4.52E-05	3.24E-04	3.69E-04	35.018%
R2	1.54E-04	1.11E-03	1.26E-03	35.063%
R3	1.56E-04	1.12E-03	1.27E-03	35.064%
R4	3.18E-05	2.28E-04	2.60E-04	35.013%
R5	3.18E-05	2.28E-04	2.60E-04	35.013%
R6	7.48E-05	5.37E-04	6.11E-04	35.031%
R7	1.44E-05	1.03E-04	1.18E-04	35.006%
R8	1.26E-05	4.40E-03	4.42E-03	35.221%
R9	1.88E-05	6.56E-03	6.58E-03	35.329%
R10	3.87E-05	1.35E-02	1.35E-02	35.676%
R11	2.98E-05	1.04E-02	1.04E-02	35.521%
R12	3.28E-05	1.14E-02	1.14E-02	35.572%
R13	1.67E-05	5.80E-03	5.82E-03	35.291%
R14	2.26E-05	7.85E-03	7.88E-03	35.394%
R15	2.96E-05	2.13E-04	2.42E-04	35.012%
R16	7.30E-05	5.24E-04	5.97E-04	35.030%
R17	3.59E-05	2.57E-04	2.93E-04	35.015%
R18	1.19E-04	8.52E-04	9.70E-04	35.049%

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 TDI)
R19	3.91E-05	2.81E-04	3.20E-04	35.016%
R20	2.52E-05	8.77E-03	8.79E-03	35.440%
R21	1.74E-05	1.25E-04	1.42E-04	35.007%
R22	1.69E-05	5.88E-03	5.90E-03	35.295%
R23	2.30E-05	7.99E-03	8.02E-03	35.401%
R24	1.75E-05	6.09E-03	6.11E-03	35.305%
R25	4.65E-05	1.62E-02	1.62E-02	35.812%
R26	3.59E-05	1.25E-02	1.25E-02	35.626%

Table 12: 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 TDI)
MDI (% of TDI)				**90.00%**
Point of maximum impact - agricultural	3.34E-04	1.31E-01	1.31E-01	96.543%
Point of maximum impact - residential	3.34E-04	6.37E-03	6.71E-03	90.335%
R1	5.69E-05	1.09E-03	1.14E-03	90.057%
R2	1.94E-04	3.70E-03	3.89E-03	90.195%
R3	1.96E-04	3.74E-03	3.94E-03	90.197%
R4	4.01E-05	7.65E-04	8.05E-04	90.040%
R5	4.01E-05	7.65E-04	8.05E-04	90.040%
R6	9.42E-05	1.80E-03	1.89E-03	90.095%
R7	1.81E-05	3.46E-04	3.64E-04	90.018%
R8	1.59E-05	6.22E-03	6.24E-03	90.312%
R9	2.37E-05	9.27E-03	9.29E-03	90.465%
R10	4.87E-05	1.90E-02	1.91E-02	90.954%
R11	3.76E-05	1.47E-02	1.47E-02	90.736%
R12	4.13E-05	1.61E-02	1.62E-02	90.808%
R13	2.10E-05	8.20E-03	8.22E-03	90.411%
R14	2.84E-05	1.11E-02	1.11E-02	90.556%
R15	3.73E-05	7.12E-04	7.49E-04	90.037%
R16	9.20E-05	1.75E-03	1.85E-03	90.092%
R17	4.52E-05	8.62E-04	9.07E-04	90.045%
R18	1.50E-04	2.85E-03	3.00E-03	90.150%
R19	4.93E-05	9.39E-04	9.88E-04	90.049%

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 TDI)
R20	3.17E-05	1.24E-02	1.24E-02	90.621%
R21	2.19E-05	4.19E-04	4.41E-04	90.022%
R22	2.13E-05	8.31E-03	8.33E-03	90.417%
R23	2.89E-05	1.13E-02	1.13E-02	90.566%
R24	2.20E-05	8.61E-03	8.63E-03	90.431%
R25	5.86E-05	2.29E-02	2.29E-02	91.147%
R26	4.52E-05	1.76E-02	1.77E-02	90.885%

B Location of Sensitive Receptors

Legend

CHP Plant Stack

Type

- Agricultural
- Agricultural / Residential
- Resident

Client:	Indaver
Site:	Rivenhall
Project:	3659

Title:

Figure 2. Dioxin Sensitive Receptors

Drawn by: ---	Date: 19/07/2024

0 0.2 0.4 0.8 km

N

Scale: 1:20,000

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C Data Source – Mean Daily Intake

congener, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD). The TEFs most recently agreed by the World Health Organization (WHO) in 2005 are recommended herein.

When assessing a dioxin-like compound, its toxic equivalence (TEQ) is its exposure dose multiplied by its TEF. The overall TEQ of exposure to a mixture of dioxin-like compounds is the sum of the TEQs for the individual compounds present. It is recommended that TEFs are not directly applied to concentrations of dioxin and dioxin-like compounds in soil to calculate TEQs; instead congener-specific equations should be used as far as possible to account for factors such as fate, transport and availability.

Health Criteria Values and risk assessment

The recommended oral tolerable daily intake (TDI_{oral}) of 2 pg WHO-TEQ kg^{-1} bodyweight (bw) is derived to protect against the induction of developmental effects in humans. As these are the most sensitive effects, the TDI is also expected to protect against all other toxic and carcinogenic effects

No authoritative assessments of the health risks posed by inhalation or dermal exposures to dioxins and dioxin-like PCBs were identified. There is no evidence to suggest that the toxicity of dioxins and dioxin-like PCBs is route-specific, or that there is any notable oral first-pass metabolism. The very limited data on absorption following inhalation suggest that pulmonary absorption may be greater than (in-feed) oral absorption. However, any potential difference is of negligible consequence to the risk assessment of dioxins and dioxin-like PCBs in soil, due to the overwhelming dominance of oral exposure pathways. Consequently, it is recommended that combined exposure from all pathways is compared against the TDI_{oral} in the derivation of SGVs.

Although information on dermal absorption is very limited, what is known suggests a low degree of absorption.

Mean daily intakes from non-soil sources

The adult oral mean daily intake (MDI_{oral}) of dioxins and dioxin-like PCBs from their presence in food and drinking-water is estimated to be about 49 pg WHO-TEQ day^{-1}.The adult inhalation mean daily intake (MDI_{inh}) from ambient air is estimated to be about 0.2 pg TEQ day^{-1}.

TDI and MDI values for dioxins, furans and dioxin-like PCBs

Parameter	Units	Oral (and inhalation combined)
MDI	pg WHO-TEQ day^{-1}	49
MDI for 70-kg adult	pg WHO-TEQ kg^{-1} bw day^{-1}	0.7
MDI for 20-kg child	pg WHO-TEQ kg^{-1} bw day^{-1}	1.8
TDI	**pg WHO-TEQ kg^{-1} bw day^{-1}**	**2**

[a] See Environment Agency (2009a) for details of MDI conversion factors.

Summary of changes to HCV recommendations

The TDI_{oral} of 2 pg WHO-TEQ kg^{-1} bw day^{-1} is the same as was recommended in the 2003 TOX report.

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