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Lostock Sustainable Energy Plant

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Appendix E3 - Human Health Risk Assessment

ENGINEERING CONSULTING

Document approval

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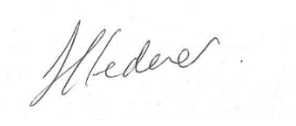
Prepared by:	Hannah Lederer		Environmental Scientist	13/07/2021
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Contents

1 Introduction

Lostock Sustainable Energy Plant Limited (LSEP Ltd) was granted an Environmental Permit (EP) for a waste incineration facility (referred to as the 'Facility') at Lostock Gralam, Northwich (Ref: EPR/WP3934AK). The EP was originally granted on 16 December 2013 and has since been subject to a single variation to include for a number of additional EWC codes.

Fichtner Consulting Engineers Limited (Fichtner) has been engaged to undertake a Human Health Risk Assessment (HHRA) to support the application for the variation to the Environmental Permit to increase the annual throughput of LSEP to 728,000 tonnes per annum. This HHRA has been carried out to determine the impact of the total throughput not just the increase in throughput. Hereafter throughout this report, all references to LSEP refer to the scheme as proposed, with the increase in throughput, unless otherwise specified.

1.1 Background

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

The Waste Incineration BREF was published by the European Integrated Pollution Prevention and Control (IPPC) Bureau in December 2019. The BREF has introduced BAT-AELs (BAT Associated Emission Levels) which are more stringent than those currently set out in the IED for some pollutants. It has been agreed with the Environment Agency as part of the pre-application discussions for the Environmental Permit variation application that the LSEP will need to meet the requirements of the BREF for an existing plant. Therefore, it has been assumed that the emissions from the LSEP would comply with the BAT-AELs set out in the BREF for existing plants.

The advice from health specialists such as the Health Protection Agency (HPA) (now Public Health England) 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 LSEP 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 LSEP.

For most substances released from the LSEP, 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.

The Air Quality Assessment Levels (AQALs) referred to in the Air Quality Analysis within Appendix E of the permit application 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 and dioxin-like PCBs the health assessment criterion is 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 has now been taken over by Public Health England (PHE), published a note RCE-13 "The Impact on Health of Emissions to Air from Municipal Waste Incinerators", in 2009[1]. The summary states:

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

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

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

Ghosh et al (2018) concluded that:

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

Freni-Sterrantino et al (2019) concluded that:

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

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

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

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

[3] Freni-Sterrantino, A; Ghosh, RE; Fecht, D; Toledano, MB; Elliott, P; Hansell, AL; Blangiardo, M. (2019) Bayesian spatial modelling for quasi-experimental designs: An interrupted time series study of the opening of Municipal Waste Incinerators in relation to infant mortality and sex ratio. Environment International. 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 LSEP and operating to the current standards of the IED. Neither paper found any evidence of an association of waste incineration facilities with the health outcomes considered. Given that the LSEP would actually operate to tighter standards, as it would use 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 LSEP of pollutants that accumulate in the environment would not be significant. Nonetheless, a quantitative assessment of the effect of emissions from the LSEP 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 LSEP which have the potential to harm human health. Details of dispersion modelling can be found in Appendix E2 [Process Emissions Modelling]. There are no other local sources which include emissions of dioxins or dioxin-like PCBs.

The LSEP will be designed to meet the BAT-AELs outlined in the Waste Incineration BREF. 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 Appendix E2 [Process Emissions Modelling]. However, dioxins and dioxin-like PCBs can accumulate in the environment, which means that inhalation is only one of the potential exposure routes. Therefore, impacts cannot be evaluated in terms of their effects on human health by simply reference to ambient air quality standards. An assessment needs to be made of the overall human exposure to the substances by the local population and the risk that this exposure causes. 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.

3.2 Chemicals of Potential Concern (COPC)

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

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

This risk assessment investigates the potential for long term health effect of these COPCs through other routes than just inhalation. The impact of all other pollutants released from the LSEP have been assessed against the AQALs for the protection of human health which are based on atmospheric concentrations of pollutants as the main pathway is via 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 USEPA's approach. However, the Environment Agency recommends that the results be assessed using the UK's approach, which is explained 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 Proposed Development, the predicted intake of a substance due to emissions from the LSEP is added to the MDI and compared with the TDI.

The following table outlines the MDIs (the typical intake from existing background sources) and TDIs for dioxins and dioxin-like PCBs. These figures are defined in the "Contaminants in soil: updated collation of toxicology data and intake values for humans" series of toxicological reports, available from the Environment Agency's website.

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 6.

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 LSEP is negligible and the effect is not significant.

5 Conceptual Site Model

5.1 Conceptual site model

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

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

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

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

Table 2: Pathways Considered

Pathway	Residential	Agricultural	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	Yes
Ingestion of home-grown beef	-	Yes	-
Ingestion of home-grown pork	-	Yes	-
Ingestion of home-grown milk	-	Yes	-
Ingestion of breast milk (infants only)	Infants only		

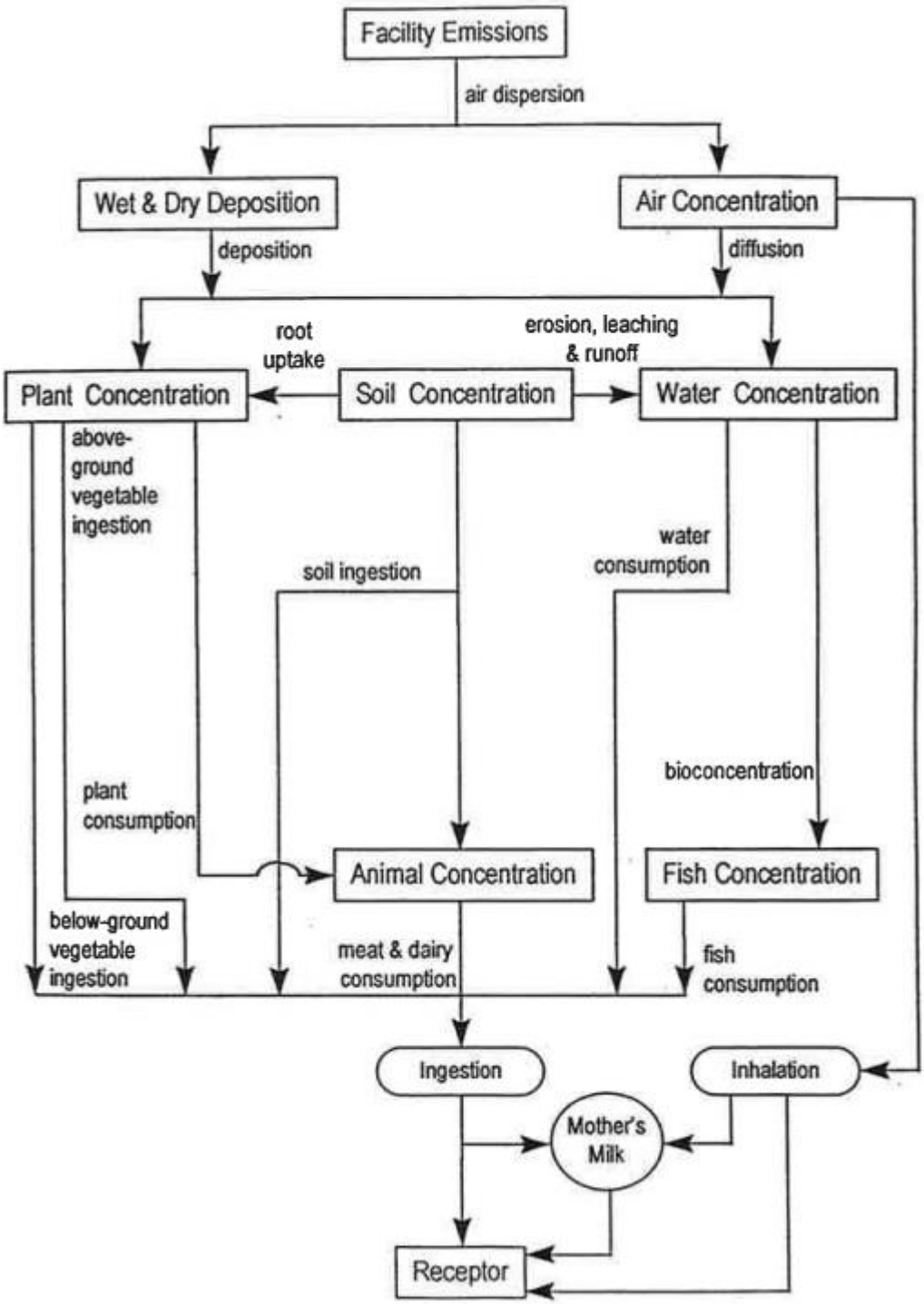
Some households may keep chickens and consume eggs and potentially the birds. The impact on these households is considered to be between the impact at an agricultural receptor and a standard resident receptor. To account for this the agricultural receptor in IRAP has been modified to exclude the ingestion of home-grown beef, pork and milk.

As shown in Figure 1, the pathway from the ingestion of mother's milk in infants is considered within the assessment. This considers all dioxins and dioxin-like PCBs. The IRAP model calculates the amount of these COPCs entering the mother's milk and being passed on to the infants. The impacts are then compared against the TDI.

[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.

Figure 1: Conceptual Site Model – Exposure Pathways



5.2 Pathways excluded from assessment

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

5.2.1 Dermal absorption

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

The USEPA[7] provide an example from the risk assessment conducted for the Waste Technologies, Inc. hazardous thermal treatment in East Liverpool, Ohio. This indicated that for an adult subsistence farmer in a 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.

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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 LSEP.

A review of the local waterbodies has been undertaken to see if there are any game fishing lakes in the local area[10]. No game fisheries have been identified within 5 km of the LSEP. The closest site is the River Dane and Holmes Chapel which is 10 km to the south-east, and Mill Farm Trout Lakes is located 16 km to the north-east. Due to the distance between the LSEP and these sites it is considered that the impact at the identified fisheries would be imperceptible. The other fishing locations within 5 km of the LSEP are course fishing lakes and would not be a significant source of dietary fish. Therefore, this 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 LSEP, and at the point of maximum impact of annual mean emissions.

For the purposes of this assessment, receptor locations have been categorised as 'residential', 'agricultural' or 'allotment'. Residential receptors represent a known place of residence that is occupied within the study area. Agricultural receptors represent a farm holding where farmers may be expected to consume the livestock they rear here. Allotment receptors represent a land area of horticultural interest, where people may be expected to consume the vegetation grown here. Schools and nurseries have been described as 'allotment' as this provides a mid-way between 'agricultural' and 'residential'. As a conservative measures all schools have been included as 'allotment' type receptors.

The specific receptors identified in the Air Quality Analysis within Appendix E of the permit application have been considered in this assessment. In addition, a receptor has been assessed at the point of maximum impact and at the location of a known allotment approximately 780 m from the stack. These sensitive receptors are listed in Table 3. Reference should be made to Appendix B which shows the location of these receptors with respect to the LSEP and the assumed receptor type.

Table 3: Sensitive Receptors

ID	Receptor Name	Location		Type of Receptor
		X	Y	
Max	Point of maximum impact	368480	375160	Resident/ Agricultural / Allotment
R1	Works Lane	368206	374535	Resident
R2	Manchester Road 1	368368	374615	Resident
R3	Griffiths Road	368622	374676	Resident
R4	Arthur Street	369111	374754	Resident
R5	Station Road	369195	374655	Resident
R6	Lostock Hollow	369059	374205	Resident
R7	Birches Lane	369119	374030	Resident
R8	Birches Lane 2	369361	373864	Resident
R9	Village Close	369318	373603	Resident
R10	Cookes Lane	369064	373300	Resident
R11	Britannia Drive	368534	373024	Resident
R12	Cottage Close	368298	373564	Resident
R13	St. Johns Close	368125	373535	Resident
R14	Middlewich Road	367833	373465	Resident
R15	Birkenhead Street	367471	373707	Resident
R16	Bowden Drive	367267	373906	Resident
R17	Manchester Road 2	367609	374375	Resident

ID	Receptor Name	Location		Type of Receptor
		X	Y	
R18	Manchester Road 3	368026	374529	Resident
R19	Rudheath Senior Academy	367967	373347	Allotment
R20	Rudheath Primary Academy	368034	372783	Allotment
R21	Lostock Gralam Primary School	369205	374818	Allotment
R22	Wincham Community Primary School	368630	376327	Allotment
R23	Victoria Road Primary	366687	373822	Allotment
R24	Witton Church Walk Primary School	366340	373743	Allotment
R25	Leftwich community Primary School and County High School Leftwich	366499	371744	Allotment
R26	Victoria Infirmary	365510	373992	Resident
R27	Lostock Lodge Care Home	369801	375133	Resident
R28	Avandale Lodge Car Home	369110	374998	Resident
R29	Daneside Court Care Home	366121	373674	Resident
R30	Allotment	367890	374590	Allotment

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

These are as specified within the Environment Agency's 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 — 0.688 kg/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 2016 to 2020 from Manchester Airport weather station, as used within the air quality dispersion modelling. This provides the annual average precipitation which can be used to calculate the general IRAP-h input parameters, as presented in Table 4.

Table 4: Ground Type Dependent Properties

Input Variable	Assumption	Value (cm/year)
Annual average evapotranspiration	70% of annual average precipitation	58.88
Annual average irrigation	0% of annual average precipitation	0.00
Annual average precipitation	100% of annual average precipitation	84.12
Annual average runoff	10% of annual average precipitation	8.41

The average wind speed was taken as 3.48 m/s, calculated from the average of the five years of weather data from Manchester Airport.

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
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, to generate the inputs needed for the IRAP modelling. For details of the dispersion modelling methodology please refer to the Air Quality Analysis within Appendix E of the permit application.

7.6 Modelled emissions

For the purpose of this assessment it is assumed that the LSEP operates at the permitted ELVs for its entire operational life. In reality the LSEP 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 ELVs which have been used to derive the emission rate.

Table 6: COPC Emissions Modelled

COPC	Split of Congeners for a release of 1 ng I-TEQ/Nm^3(1)	I-TEFs for the congeners	Emission concentration (ng/Nm^3)(2)	Emission rate (ng/s)
Sum I-TEQ dioxin[s]	-	-	0.06 ng I-TEQ/Nm^3	-

COPC	Split of Congeners for a release of 1 ng I-TEQ/Nm³(1)	I-TEFs for the congeners	Emission concentration (ng/Nm³)(2)	Emission rate (ng/s)
2,3,7,8-TCDD	0.031	1	0.002	0.274
1,2,3,7,8-PeCDD	0.245	0.5	0.015	2.163
1,2,3,4,7,8-HxCDD	0.287	0.1	0.017	2.534
1,2,3,6,7,8-HxCDD	0.258	0.1	0.015	2.278
1,2,3,7,8,9-HxCDD	0.205	0.1	0.012	1.810
1,2,3,4,6,7,8-HpCDD	1.704	0.01	0.102	15.045
1,2,3,4,6,7,8,9-OctaCDD	4.042	0.001	0.242	35.687
2,3,7,8-TCDF	0.277	0.1	0.017	2.446
1,2,3,7,8-PCDF	0.277	0.05	0.017	2.446
2,3,4,7,8-PCDF	0.535	0.5	0.032	4.724
1,2,3,4,7,8-HxCDD	2.179	0.1	0.131	19.238
1,2,3,6,7,8-HxCDF	0.807	0.1	0.048	7.125
1,2,3,7,8,9-HxCDF	0.042	0.1	0.003	0.371
2,3,4,6,7,8-HxCDF	0.871	0.1	0.052	7.690
1,2,3,4,6,7,8-HpCDF	4.395	0.01	0.264	38.804
1,2,3,4,7,8,9-HpCDF	0.429	0.01	0.026	3.788
1,2,3,4,6,7,8,9-OctaCDF	3.566	0.001	0.214	31.484
Total	20.150	-	1.209	177.90
Dioxin-like PCBs	-	-	0.092	13.542

Notes:
(1) Split of the Congener 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

These are a group of similar halogenated organic compounds, which are generally found as a complex mixture. The toxicity of each compound is different and is generally expressed as a Toxic Equivalent Factor (TEF), which relates the toxicity of each individual compound to the toxicity of 2,3,7,8-TCDD, the most toxic dioxin. A full list of the TEF values for each dioxin is provided in Table 6. The total concentration is then expressed as a Toxic Equivalent (TEQ).

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". This data has been used in lieu of any specific data for the pyrolysis of plastics. This is considered conservative as

the pyrolysis process is expected to result in much lower dioxin formation than the combustion of municipal waste in an incinerator.

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. The output of the IRAP model is then multiplied by the relevant TEFs to determine the total intake TEQ for comparison with the TDI.

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.

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 emissions, as a worst-case assumption we have assumed that the PCBs are released in each of the two Aroclor compositions.

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 6 which are used to calculate the release rate of each dioxin are based on the I-TEFs listed in Annex VI Part II of the IED. To determine the total intake TEQ for comparison with the TDI, the output of the IRAP model has been multiplied by the relevant WHO-TEFs. The I-TEFs and WHO-TEFs are shown in Table 7.

Table 7: Toxic Equivalency Factors for Dioxins and Furans

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

Congener	IED I-TEQ Multiplier	2005 WHO-TEF Multiplier
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 tables present the impact of emissions of dioxins and dioxin-like PCBs from the LSEP at the point of maximum impact for each receptor type.

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%	1.87%	36.87%
Allotment	35.00%	0.07%	35.07%
Residential	35.00%	0.05%	35.05%
Child			
Agricultural	90.65%	2.58%	93.23%
Allotment	90.65%	0.19%	90.84%
Residential	90.65%	0.16%	90.81%

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, for the worst-case receptor 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.

8.2 Breast milk exposure

The total accumulation of dioxins in an infant, considering the breast milk pathway and based on an adult agricultural receptor at the point of maximum impact feeding an infant, is 0.238 pg WHO-TEQ / kg-bw / day which is 8.93% of the TDI. For a residential type receptor this is only 0.17% 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 operation of the LSEP will not increase the health risks from the accumulation of dioxins in infants significantly.

8.3 Maximum impact at a receptor

The following tables outline the impact of emissions from the LSEP at the most affected receptor (i.e. the receptor with the greatest impact from ingestion and inhalation of emissions) (R21 – Lostock Gralam Primary School). This conservatively has applied the allotment type receptor at this location, which is conservative as it assumes that a significant proportion of the diet of the receptor is sourced from the receptor point assessed. 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			
Allotment	35.00%	0.05%	35.05%
Child			
Allotment	90.65%	0.14%	90.79%

As shown, for the most impacted receptor 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.

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

8.4 Breast milk exposure

The total accumulation of dioxins and dioxin like PCBs in an infant, considering the breast milk pathway at R21 based on an adult allotment type receptor feeding an infant, is 0.004 pg WHO-TEQ / kg-bw / day which is 0.22% 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 operation of the LSEP will not increase the health risks from the accumulation of dioxins in infants significantly.

8.5 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 receptors have been assessed. The agricultural and allotment receptors are 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 LSEP.

9 Conclusions

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

- the LSEP will operate continually at the Waste Incineration BAT AEL for an existing plant, i.e. at the maximum concentrations which it is expected that the LSEP 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 LSEP.

The results of the assessment show that, for an agricultural child receptor at the point of maximum impact, the combined intake from the LSEP plus 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 point of maximum impact is less than the TDI. The impact at 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 LSEP on human health is predicted to be negligible and the effect is not significant.

Appendices

A Detailed Results Tables

Table 10: 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)
MDI (% of TDI)				**35.00%**
Max – Agricultural	1.84E-04	3.71E-02	3.73E-02	36.866%
Max - Allotment	1.84E-04	1.16E-03	1.34E-03	35.067%
Max - Residential	1.84E-04	7.89E-04	9.73E-04	35.049%
R1	3.96E-05	1.70E-04	2.09E-04	35.010%
R2	7.93E-05	3.40E-04	4.20E-04	35.021%
R3	1.14E-04	4.88E-04	6.01E-04	35.030%
R4	1.28E-04	5.51E-04	6.79E-04	35.034%
R5	1.27E-04	5.44E-04	6.71E-04	35.034%
R6	9.31E-05	3.99E-04	4.93E-04	35.025%
R7	1.11E-04	4.74E-04	5.85E-04	35.029%
R8	1.62E-04	6.94E-04	8.56E-04	35.043%
R9	1.49E-04	6.37E-04	7.85E-04	35.039%
R10	8.34E-05	3.58E-04	4.41E-04	35.022%
R11	3.71E-05	1.59E-04	1.96E-04	35.010%
R12	6.53E-06	2.80E-05	3.45E-05	35.002%
R13	1.30E-05	5.58E-05	6.88E-05	35.003%
R14	5.03E-05	2.16E-04	2.66E-04	35.013%
R15	6.68E-05	2.87E-04	3.53E-04	35.018%
R16	5.59E-05	2.40E-04	2.96E-04	35.015%
R17	3.36E-05	1.44E-04	1.78E-04	35.009%

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)
R18	3.13E-05	1.34E-04	1.65E-04	35.008%
R19	4.06E-05	2.56E-04	2.97E-04	35.015%
R20	3.38E-05	2.13E-04	2.47E-04	35.012%
R21	1.29E-04	8.11E-04	9.39E-04	35.047%
R22	1.27E-04	8.01E-04	9.29E-04	35.046%
R23	5.66E-05	3.57E-04	4.13E-04	35.021%
R24	5.48E-05	3.46E-04	4.01E-04	35.020%
R25	3.97E-05	2.50E-04	2.90E-04	35.015%
R26	3.32E-05	1.42E-04	1.75E-04	35.009%
R27	1.04E-04	4.45E-04	5.49E-04	35.027%
R28	1.29E-04	5.52E-04	6.81E-04	35.034%
R29	5.29E-05	2.27E-04	2.80E-04	35.014%
R30	3.08E-05	1.95E-04	2.25E-04	35.011%

Table 11: 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)
MDI (% of TDI)				**90.65%**
Max – Agriculture	2.32E-04	5.14E-02	5.16E-02	93.231%
Max - Allotment	2.32E-04	3.50E-03	3.73E-03	90.836%
Max - Resident	2.32E-04	2.88E-03	3.11E-03	90.805%
R1	4.99E-05	6.18E-04	6.68E-04	90.683%
R2	1.00E-04	1.24E-03	1.34E-03	90.717%
R3	1.43E-04	1.78E-03	1.92E-03	90.746%
R4	1.62E-04	2.01E-03	2.17E-03	90.758%
R5	1.60E-04	1.98E-03	2.14E-03	90.757%
R6	1.17E-04	1.46E-03	1.57E-03	90.729%
R7	1.39E-04	1.73E-03	1.87E-03	90.743%
R8	2.04E-04	2.53E-03	2.73E-03	90.787%
R9	1.87E-04	2.32E-03	2.51E-03	90.775%
R10	1.05E-04	1.30E-03	1.41E-03	90.720%
R11	4.67E-05	5.79E-04	6.26E-04	90.681%
R12	8.22E-06	1.02E-04	1.10E-04	90.656%
R13	1.64E-05	2.03E-04	2.20E-04	90.661%
R14	6.34E-05	7.86E-04	8.50E-04	90.692%
R15	8.42E-05	1.04E-03	1.13E-03	90.706%
R16	7.04E-05	8.74E-04	9.44E-04	90.697%
R17	4.23E-05	5.25E-04	5.67E-04	90.678%
R18	3.95E-05	4.88E-04	5.28E-04	90.676%

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)
R19	5.11E-05	7.71E-04	8.23E-04	90.691%
R20	4.26E-05	6.42E-04	6.85E-04	90.684%
R21	1.62E-04	2.44E-03	2.60E-03	90.780%
R22	1.60E-04	2.41E-03	2.57E-03	90.779%
R23	7.13E-05	1.07E-03	1.15E-03	90.707%
R24	6.90E-05	1.04E-03	1.11E-03	90.706%
R25	5.00E-05	7.54E-04	8.04E-04	90.690%
R26	4.18E-05	5.18E-04	5.60E-04	90.678%
R27	1.31E-04	1.62E-03	1.75E-03	90.738%
R28	1.62E-04	2.01E-03	2.17E-03	90.759%
R29	6.67E-05	8.27E-04	8.94E-04	90.695%
R30	3.88E-05	5.86E-04	6.25E-04	90.681%

B Location of Sensitive Receptors

Figure 2: Human Health Risk Assessment Sensitive Receptors

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