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INTRODUCTION

Health Impact Assessment

- 16.1 This Technical Appendix presents a Human Health Risk Assessment (HHRA) for a proposed energy generation facility at New England Quarry near Plymouth, Devon. The site lies to the immediate south of the A38 dual carriageway.
- 16.2 Viridor is proposing to develop an Energy from Waste (EfW) facility that would treat 275,000 tonnes of residual non-hazardous waste per annum.
- 16.3 HHRA has been conducted to satisfy the requirement for a Health Impact Assessment (HIA) that enables a judgement to be made as to whether a policy or development project has significant potential effects on the health of the local population and also seeks to quantify the extent of predicted outcomes. SLR has undertaken a 'prospective HIA' to predict the potential effects on health arising from the operation of the proposed EfW facility at the New England site in using the best available tools and UK technical guidance.
- 16.4 HIA is a multi-faceted process and comprises 5 main steps:
- Screening that determines whether a HIA is necessary for the proposed development
 - Scoping that identifies potential hazards (or benefits) and sets the terms of reference for the assessment process.
 - Risk Assessment that involves characterising the nature and magnitude of health risks associated with a project.
 - Decision making involves considering the outcome of the risk assessment and the various options going forward.
 - Implementation and monitoring involves actions to implement the decision(s) and to observe their consequences.
- 16.5 This report present the results of a risk assessment dedicated to considering the nature and magnitude of potential health effects arising from the trace levels of persistent chemicals associated with particulate matter in stack emissions from the proposed EfW facility at the New England site.

Human Health Risk Assessment for New England EfW

- 16.6 A Human Health Risk Assessment (HHRA) was undertaken in order to estimate the potential level of risk posed to health by emissions generated by operation of the proposed EfW facility at the New England site.
- 16.7 The objective of this assessment was to evaluate the potential risk to the health of the local population in the event that they are exposed to emissions from the proposed EfW facility. The dominant source of emissions is assumed to be the discharge of residual combustion products

from the stack. Compounds released in stack emissions are divided into two classes, these are:

- 16.8 Compounds that exist predominantly in the gaseous phase, e.g. sulphur and nitrogen oxides (SO_x and NO_x) and other acid gases. The health effects of these compounds arise from exposure via the inhalation pathway and are most likely to be of an acute nature. Concentrations of these compounds in air are assessed by comparison to air quality standards. Fine particulate matter (i.e. particles with a mean aerodynamic diameter of <10 µm, i.e. PM₁₀) also induce health effects following inhalation and are assessed against an air quality standard.
- 16.9 Compounds that exist associated with the particulate phase, e.g. metals and non-volatile organic compounds such as dioxins. Exposure to these compounds will predominantly arise from indirect pathways following deposition of particles from the air. Such pathways include direct contact with shallow soils, ingestion of soil, dust and food grown in contaminated soil and ingestion of foods, such as meat or milk, derived from animals that have grazed on pastures or fodder that has been contaminated by aerial deposition of dust containing pollutants. Since these compounds tend to be persistent in the environment a long-term human health impact is plausible depending on site use;
- 16.10 In general, gaseous phase contaminants are evaluated as part of an Air Quality Assessment. This report presents a detailed HHRA of the persistent contaminants predominantly associated with the particulate phase that may accumulate in the environment and therefore result in long term exposure risks. Potential exposure to these contaminants is evaluated for indirect pathways such as ingestion of soil and soil-derived dust and incorporation into the food chain.
- 16.11 The purpose of this assessment of persistent contaminants is to undertake a site-specific screening exercise based on available UK guidance and models adapted to the purpose of a Human Health Risk Assessment (HHRA) for an EfW facility. The assessment is based upon adopting a worst-case scenario approach, using the maximum predicted contaminant concentrations in air, soil and other media and assessing the resultant exposure against criteria that are protective of the most sensitive receptors within exposed populations.

METHODOLOGY

- 16.12 The principles of Health Impact Assessment have largely been developed from Environmental Impact Assessment¹. The Human Health Risk Assessment of the New England EfW has therefore been undertaken following the UK Risk Assessment Framework outlined in the DETR (2000) publication 'Guidelines for Environmental Risk Assessment and Management' which is recommended for use in all public domain risk assessments. The UK Risk Assessment Framework follows a tiered approach, where the level of complexity and effort/cost generally increases with each tier as conservatism and uncertainty decrease.
- 16.13 The common Risk Assessment Framework is summarised below
- Tier 1: Risk Screening - This comprises the development of an outline conceptual model, to establish whether there is the potential for unacceptable risks to be present and evaluates the need for further assessment.
 - Tier 2: Generic Quantitative Risk Assessment (GQRA) - This Tier uses the conceptual model and generic assessment criteria (GAC), if available, to identify potentially unacceptable risks.
 - Tier 3: Detailed Quantitative Risk Assessment (DQRA) - This Tier involves development of site-specific assessment criteria (SSAC) or the quantification of exposure for a specific location or scenario(s) not addressed by GQRA. DQRA generally requires more complex risk modelling tools and the generation of more detailed data to characterise the site and receptors under consideration
- 16.14 A staged approach to HHRA for an EfW facility following this framework is presented below.

Tier 1: Risk Screening and Development of Conceptual Site Model

- 16.15 The risk assessment process utilises the source-pathway-receptor concept in the development of a conceptual site model and identifies sources of potential contaminants, receptors that could be at risk and the contaminant transport and exposure pathways that might result in the receptors being directly or indirectly exposed. A potential 'pollutant linkage', requiring further assessment, is identified where all three components are present. Information on a generic EfW facility pertinent to qualitative risk assessment and the development of a conceptual site model is detailed below:

¹ National Assembly for Wales (2000) Developing Health Impact Assessment in Wales. Published by the Health Promotion Division, National Assembly for Wales, Cardiff. ISBN 0 7504 2374 9

Source of hazard

- 16.16 The source of the hazard is the incineration of municipal solid waste, which results in combustion emissions from the EfW plant being released via a stack. The constituents of the stack emissions vary due to the composition of waste incinerated and the combustion process (residence time / temperature etc). Emission levels are defined by the Waste Incineration Directive (WID) which prescribes methodologies and standards in order to minimise/abate effects on the environment and health; specifications of the WID include the following:
- operating conditions, including gas temperatures and residence times (e.g. 850°C / 2 seconds);
 - emission limit values for a range of substance to air and water including dioxins; and,
 - emission monitoring requirements.

Hazard

- 16.17 Stack emissions are potentially hazardous due to the discharge and dispersion of both gaseous phase and particulate contaminants that have properties hazardous to health; for example acid gases (NO_x and SO_x) have acute respiratory effects; and persistent pollutants such as metals and dioxins have a range of chronic toxicological effects. Metals and dioxins specified by the WID are detailed below.

Transport mechanisms

- 16.18 The WID sets out emission limit values for emissions to air as detailed in Table 16-1 below; these emission limits would be set as Environmental Permit Conditions by the Environment Agency as part of the permitting process.

Table 16-1

WID Emission Limit Values

Pollutant	Emission Limits (mg/Nm ³) ^(a)		
	Daily average values	Half hourly averages	
		100 th Percentile	97 th Percentile
Particles	10	30	10
TOC	10	20	10
HCl	10	60	10
HF	1	4	2
SO ₂	50	200	50
NOx	200	400	200
CO (b)	50	150	100
Group 1 metals (c)		0.05	
Group 2 metals (d)		0.05	
Group 3 metals (e)		0.5	
Dioxins and furans		0.0000001 (f)	

Notes:

(a) Concentrations referenced to temperature 273 K, pressure 101.3 kPa, 11% oxygen, dry gas.

(b) 150 mg/Nm³ of combustion gas for at least 95% of all measurements determined as 10 minute averages or 100 mg/Nm³ of combustion gas of all measurements determined as half-hourly average values taken in any 24 hour period.

(c) Cadmium (Cd) and thallium (Tl)

(d) Mercury (Hg)

(e) Antimony (Sb), arsenic (As), lead (Pb), chromium (Cr), cobalt (Co), copper (Cu), manganese (Mn), nickel (Ni), and vanadium (V).

(f). The emission limit value refers to the total concentration of dioxins and furans calculated using the concept of toxic equivalence (TEQ).

16.19 The transport of pollutants from the EfW stack into the surrounding environment occurs as a result of the dispersion and dilution of the stack plume due to meteorological conditions. The most important meteorological parameters governing the atmospheric dispersion of pollutants are as follows:

16.20 wind direction determines the broad transport of the emission and the sector of the compass into which the emission is dispersed;

16.21 wind speed will affect ground level concentrations of emissions by increasing the initial dilution of pollutants in the emission; and atmospheric stability, which is a measure of the turbulence, particularly of the vertical motions present. Advanced dispersion models use Monin-Obukhov lengths - a more advanced method of determining stability² than Pasquill.

16.22 Detailed atmospheric dispersion modelling has been undertaken with due consideration to relevant guidance^{3,4}. A number of commercially available

² Defined as: 'the height over the ground, where mechanically produced (by vertical shear) turbulence is in balance with the dissipative effect of negative buoyancy, thus where Richardson number equals to 1.' Essentially it is a more quantitative method of estimating stability than the previously used Pasquill Stability Classes. It requires two quantities not routinely measured by national meteorological networks: the friction velocity u and flux of sensible heat H .

³ Air Dispersion modelling report requirements (for detailed air dispersion modelling). AQMAU, Environment Agency (not dated).

dispersion models are able to predict ground level concentrations arising from emissions to atmosphere from elevated point sources such as the New England EfW facility. For this assessment the AERMOD GIS PRIME model⁵ has been applied.

- 16.23 The AERMOD dispersion modelling program is widely used and accepted by the Environment Agency in the UK for undertaking such assessments and its predictions have been validated against real-time monitoring data by the USEPA⁶. It is therefore considered a suitable model for this assessment. Further details of the dispersion model input data are presented in Appendix A.

Pathways

- 16.24 Atmospheric transport of gases and particulates is the main potential pathway for identified hazards reaching a nearby human receptor. Exposure to gaseous contaminants will occur by direct inhalation. Exposure to particulate phase contaminants will primarily occur via indirect pathways following deposition to soil; these pathways include ingestion of soil and soil-derived dust and uptake of contaminants from soil into the food-chain (via home-grown produce, crops and animal products).

Targets/receptors and exposure

- 16.25 License and discharge conditions for ERfWs are summarised in Table 2-1 previously. Down wind 'receptor locations' may include residential areas, schools, businesses, allotments and farms. The most sensitive human receptors are generally considered to be young children in residential areas or regularly attending allotments who may be exposed over long periods of time to low levels of combustion gases and particulates and may also consume significant quantities of locally produced food. This exposure is quantified in subsequent tiers of the risk assessment process.

Contaminants of concern

- 16.26 Emissions of dioxins, furans and metals are governed under the Waste Incineration Directive, which sets emission concentrations for 3 groups of metals and for total dioxin/furan releases. Classes of persistent pollutants and individual contaminants specified by the WID are listed in Table 16-2 below:

⁴ Guidelines for the Preparation of Dispersion Modelling Assessment for Compliance with Regulatory Requirements – an update to the 1995 Royal Meteorological Society guidance. UK Atmospheric Dispersion Modelling Committee (ADMLC), Version 1.4, 2004.

⁵ Software used: BREEZE AERMOD GIS Pro, v6.

⁶ AERMOD: Latest Features and Evaluation Results. USEPA Report: EPA-454/R-03-003, June 2003, (http://www.epa.gov/scram001/dispersion_prefrec.htm#aermod)

Table 16-2
Persistent Pollutants

Class	Pollutant
Group I metals	Cadmium
	Thallium
Group II metals	Mercury
Group III metals	Antimony
	Arsenic
	Chromium
	Cobalt
	Copper
	Lead
	Manganese
	Nickel
	Vanadium
	Dioxins (PCDDs)
1,2,3,7,8-PeCDD	
1,2,3,4,7,8-HxCDD	
1,2,3,7,8,9-HxCDD	
1,2,3,6,7,8-HxCDD	
1,2,3,4,6,7,8-HpCDD	
OCDD	
Furans (PCDFs)	2,3,7,8-TCDF
	1,2,3,7,8-PeCDF
	2,3,4,7,8-PeCDF
	1,2,3,4,7,8-HxCDF
	1,2,3,7,8,9-HxCDF
	1,2,3,6,7,8-HxCDF
	2,3,4,6,7,8-HxCDF
	1,2,3,4,6,7,8-HpCDF
	1,2,3,4,7,8,9-HpCDD
	OCDF

16.27 All contaminants from the list above have been included in the HHRA for EfW emissions from the proposed New England facility with the exception of antimony, cobalt and manganese. These contaminants are not considered by quantitative risk assessment due to their absence from the list of priority contaminants for land contamination published by Defra and the Environment Agency (2002). Antimony and manganese are considered

to possess only moderate toxicity while cobalt is thought to be of low toxicity.

Tier 2: Generic Quantitative Risk Assessment (GQRA)

- 16.28 Atmospheric concentrations of gaseous phase contaminants and particulates are assessed against published air quality objectives and standards (contained within the Air Quality Standard Regulations 2007). There is no specific UK technical guidance for the assessment of indirect exposure to particulate emissions from combustion processes or a modelling tool capable of assessing this scenario in compliance with UK approaches to HHRA. HHRA of metals has therefore been undertaken using air dispersion modelling, the calculation of resultant soil concentrations following deposition of particulates over an extended time period and comparison of these concentrations to generic assessment criteria (GACs) based on the principles of the Contaminated Land Regime and associated technical guidance.
- 16.29 This assessment of indirect exposure following deposition of metals is based on the methodology detailed by Macleod et al (2006) for modelling indirect human exposure to airborne air pollution control (APC) residues⁷ released from landfills. The approach detailed by Macleod et al. employs air dispersion modelling to estimate dust emissions and predict deposition at selected receptor locations.
- 16.30 The guidance developed as part of the Contaminated Land Regime provides a methodology for the quantitative assessment of the potential health risks associated with the presence of different levels of contaminants in soil. Quantification is achieved using the CLEA model which has been produced by the Environment Agency to estimate exposure to contaminants in soil. CLEA has been used to derive the published soil guideline values (SGVs) that are available for a limited number of contaminants and the model can also be used to derive de novo assessment criteria that can also be used in generic quantitative risk assessment.

Tier 3: Detailed Quantitative Risk Assessment (DQRA)

- 16.31 DQRA entails the utilisation of more detailed data on contaminant transport and receptor characteristics to assess potential health risks. In addition to refinement of some of the assumptions used in deriving GACs it may also be necessary to use more complex exposure models that include additional exposure pathways such as incorporation of persistent contaminants into the food chain.
- 16.32 DQRA of dioxin emissions follows HMIP methodology presented in the 'Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes' (HMIP, 1996). Human exposure via a number of pathways has been estimated using algorithms presented in Appendix A and Annex C of the HMIP guidance to calculate deposition rates, environmental fate and

⁷ APC residues are the by-products of cleaning up flue gases from high temperature installations such as EfW plants; they comprise fine ash and the lime and finely divided carbon used to clean exhaust gases.

transport and intake by human receptors. HMIP exposure assessment methodology has been supplemented by use of the Environment Agency's CLEA model (v1.05) and dioxin risk assessment worksheet, which are used for the assessment of contaminated land. This model has been used to estimate exposure resulting from the deposition to soil of particulate matter containing dioxins and also includes assessment of the dermal exposure pathway which is not addressed by the HMIP methodology.

- 16.33 The overall risk posed by dioxin emissions from the New England EfW has been assessed by comparison of 'worst case scenario' intake to recommended health criteria values.

CONCEPTUAL SITE MODEL

Risk Screening and Development of Conceptual Model

- 16.34 As described above, HHRA for persistent contaminants in stack emissions from the proposed New England EfW facility follows the UK Risk Assessment Framework. The first tier of the framework is the development of a conceptual site model (CSM). The CSM identifies the potential sources, critical pathways and receptors relevant to risks to human health for the New England EfW.
- 16.35 For the purpose of the HHRA exhaust gases from the stack are assumed to be the only source of emission. Contaminants of concern are those described previously below.
- 16.36 Sources, types of receptors, relevant pathways of exposure and their potential significance are detailed in Table 16-3 below.

Table 16-3
Conceptual Site Model

Sources	Gaseous and particulates emitted in exhaust gases from the New England EfW stack Acid gases, e.g. NO _x and SO _x Particulates (as PM _{2.5} and PM ₁₀) Persistent contaminants in vapour and particulate phase, e.g. metals and dioxins		
Receptors	Receptor	Location	Risk
	Resident	Children and adults in downwind residential properties (may also tend allotments)	High
	Commercial	Workers in downwind commercial properties.	Medium
	Schools	Children and teachers in downwind schools	Medium-High
	Farmer	Downwind farms	Medium
Pathways	Pathway	Description	Risk
	Inhalation	Inhalation of gaseous contaminants	High
		Inhalation of vapour phase contaminants	
		Inhalation of particulates	
		Inhalation of airborne soil/dust following deposition	Low-Medium
	Incidental ingestion of soil/dust	Ingestion of soil and soil-derived dust, particularly by children	Medium-High
	Consumption of contaminated produce	Residents and allotment owners consuming produce grown in soil contaminated by wet and dry deposition. Assumes high proportion of food is home-grown	Medium
		Consumption of local fruit and vegetable crops affected by wet and dry deposition. This will be a variable proportion of diet	Low-Medium

HUMAN HEALTH 16

Pollutant linkages	Consumption of contaminated meat and dairy products	Consumption of locally produced eggs, dairy and meat products following bioaccumulation, particularly of dioxins, in local food chain.	Medium-High
	Ingestion of maternal breast milk	Ingestion of breast milk by local babies of mothers exposed to dioxins via ingestion of soil and local/home-grown produce.	High
	Inhalation	Inhalation of exhaust gases and particulates is a direct pathway and will pose a greater risk closer to the facility. It is of most concern for the very young and old and those suffering from respiratory conditions. However, scrubbing and filtration measures prior to exhaust via the stack will remove a significant proportion of the acid gases and particulates.	
	Ingestion of soil/dust	Ingestion of soil and soil-derived dust is an indirect pathway of particular concern for children who ingest the largest amounts (such as Pica children). This pathway will be of greatest significance at the location of deposition hot-spots.	
	Consumption of contaminated produce	Ingestion of fruit and vegetable (home-grown and local produce) affected by deposition is a potentially significant indirect pathway. This pathway is most likely to be significant for dioxins and furans which can bioaccumulate in the food-chain leading to elevated concentrations in food types with a higher fat content such as meat and dairy products.	
	Maternal breast milk	Indirect pathway of potential concern for local breast fed babies. Compounds such as dioxins can be stored in the fatty tissue of the mother following local exposure and released in breast milk. However, mother's exposure due to the EfW facility is likely to be insignificant compared to background intake from diet	
Overall risk assessment	Potentially complete linkages exist requiring further assessment		

- 16.37 Dermal exposure through skin contact with contaminated soil is considered to be of low significance but is included in the risk assessment as it is an exposure pathway considered by the CLEA v1.05 model. Dietary exposure from the consumption of home-grown plants that have taken up metal contaminants from soil is considered by the CLEA model based on soil:plant concentration factors for below and above ground crops (i.e. root vegetables and leafy vegetables). Plant uptake following wet and dry deposition to plant foliage is not considered by the CLEA model; nor are uptake of contaminants into herd animals following consumption of foliage affected by deposition and exposure of babies via breast milk. These pathways are assessed for dioxins using the HMIP methodology.

GENERIC QUANTITATIVE RISK ASSESSMENT (GQRA)

Methodology

- 16.38 GQRA of metals for the New England EfW entails the comparison of predicted concentrations of contaminants in soil, following deposition of particulate matter and vapour, to generic assessment criteria used in the health risk assessment of contaminated land.
- 16.39 The GQRA of metals for the New England EfW follows the methodology described by Macleod et al (2006) in that soil concentrations resulting from wet and dry deposition of particulates and vapour are compared to generic assessment criteria for soil generated using the CLEA v1.05 model. The approach detailed by Macleod et al (2006) employs air dispersion modelling to estimate dust emissions and predict deposition at selected receptor locations.
- 16.40 Similar to the assessment of particulates emitted from an EfW facility, key pollutant linkages identified by Macleod et al. were as follows:
- i. Inhalation of airborne dust (containing metals and dioxins) by local residents and workers (characterised as potentially high risk)
 - ii. Incidental ingestion of soil/dust (containing metals and dioxins) by local residents and particularly children (characterised as potentially high risk)
 - iii. Ingestion of maternal breast milk (containing dioxins) by local breast-fed babies (characterised as potentially high risk)
 - iv. Consumption of contaminated produce (containing dioxins and metals) by local residents consuming home-grown produce such as fruit and vegetables (characterised as potentially medium risk)
 - v. Consumption of contaminated dairy and meat products (containing dioxins) by consumers of locally produced dairy and meat products (characterised as potentially high risk)
- 16.41 A number of assumptions in the methodology of Macleod et al (2006), which are also utilised in this assessment are summarised below:
- i. It is assumed that particulate deposition occurs at a constant annual rate and that deposited dust is mixed evenly into the top 0.1m of soil;
 - ii. A 6-year deposition period is assumed with the concentration at the end of the sixth year being assumed to have been present from the start. This is a conservative assumption and consistent with the exposure duration used in the CLEA model to assess risks to children from exposure to soil contamination;
 - iii. Local onward mobilisation of contaminated dusts is assumed to be negligible compared to the primary flux to the receptor location and the relative contribution from other exposure pathways such as ingestion;

- iv. The most sensitive receptor and standard land-use is considered to be a female child aged 0-6 in a residential setting with a private garden and where the family consumes its own home-grown produce.
- 16.42 The 'CLEA' model used by Macleod et al (2006) was replaced by the Environment Agency with the updated 'CLEA v1.05' model in 2009. CLEA v1.05 incorporated a number of modifications to CLEA but the two models are broadly similar and the basic principles are summarised below.

The CLEA v1.05 Model

- 16.43 The CLEA v1.05 model estimates the intake of contaminants from soil by humans from various exposure pathways (Environment Agency, 2009a, b). This is achieved by combining information about contaminant properties, soil, site and building characteristics, modelling approaches for predicting fate and transport of contaminants and information about receptor characteristics and behaviour. The estimated intake of a contaminant (average daily exposure or ADE) is compared with appropriate toxicological benchmarks (health criteria values or HCVs⁸) which are considered to be either tolerable or representative of minimal risk.
- 16.44 CLEA v1.05 estimates ADE for various exposure scenarios and compares this to the HCV as a basis for establishing generic assessment criteria (GAC). GAC are derived for a set of standard of assumptions relating to land-use, receptor behaviour, site, building and soil characteristics and are used to assess whether the soil concentration of a particular contaminant poses a significant risk to human health resulting from a particular land use (e.g. 'residential', 'allotment' or 'commercial'). These values represent 'intervention values', which indicate to an assessor that soil contaminant concentrations above this level could pose an unacceptable risk to the health of site users and that further investigation and/or remediation is required.
- 16.45 GAC have been published by the Environment Agency for selected contaminants and these are known as soil guideline values (SGVs). SGVs combine both authoritative science and policy judgements. For those contaminants for which published SGVs are not available SLR has generated GAC following the approaches recommended in Science Reports SR2 and SR3 (Environment Agency, 2009a, c). Physico-chemical input parameters used to populate the CLEA v1.05 model were selected from Environment Agency publications, where available (e.g. SR7, Environment Agency 2009d), and authoritative data sources⁹.
- 16.46 It should be noted that a number of exposure pathways relevant to this HIA cannot be assessed using the CLEA v1.05 model; these include:

⁸ For threshold effect contaminants a 'tolerable daily soil intake' (TDSI) is derived by subtracting 'mean daily intake' (MDI) derived from background intake from the prescribed 'tolerable daily intake' (TDI). For non-threshold effect contaminants an 'index dose' (ID) is specified that represents an acceptable level of lifetime risk from a specific source of exposure, i.e. background exposure is not taken into account.

⁹ E.g. CRC Handbook of Chemistry and Physics, IUPAC-NIST Solubility Series and US Environmental Protection Agency.

- deposition of particulates on the leaves of fruit and vegetables
- secondary uptake into the food chain
- ingestion of breast milk by breast-fed babies

16.47 These pathways are potentially significant for dioxins and have been considered by DQRA using HMIP methodology, which is discussed in Section 5.

Site specific AERMOD dispersion of particulates & semi-volatile contaminants

16.48 Wet, dry and vapour deposition of contaminants to soil was estimated using AERMOD, a USEPA air dispersion model designed to predict pollutant concentrations from continuous point and area sources. This enables the concentrations of windblown particulates and contaminant concentrations at nearby human receptor locations to be estimated.

16.49 Detailed methodology for the prediction of deposition rates for particulates and semi-volatile contaminants is presented in Appendix A detailing the air quality assessment. A summary of the deposition rates calculated by AERMOD at downwind receptor locations is provided in Table 16-4 below.

Table 16-4

Deposition rate

Class	Pollutant	Deposition (5 yr, µg/m ²)		
		Average	Min	Max
Group I	Cadmium	5	0.9	67
	Thallium	5	0.9	67
Group II	Mercury (particulate)	18	3.5	261
	Hg(2+) vap	181	27	4484
	Hg(0) vap	4.6 x10 ⁻²	1.0 x10 ⁻²	1.0
Group III	Antimony	11	2.1	153
	Arsenic	10	2	147
	Chromium	11	2.1	155
	Cobalt	10	2	147
	Copper	10	2	147
	Lead	10	2	147
	Manganese	12	2.2	160
	Nickel	11	2.1	153
Vanadium	11	2.1	153	

16.50 Air dispersion modelling for predicted emissions from the New England EfW indicates a variable rate of deposition for the downwind receptor locations that have been assessed due to variations in the meteorological data set over the 5 years used. A single value, the maximum deposition rate, is used in this generic quantitative risk assessment as a conservative worst case scenario. Deposition over a 6-year period was calculated for each

contaminant for use in the HHRA as this is the exposure duration used in CLEA v1.05 to assess risks to children from exposure to soil contamination. Deposition over a six-year period is calculated below in Table 16-5.

Table 16-5

Calculation of 6-year deposition rate

		Max Deposition (5 yr, $\mu\text{g}/\text{m}^2$)	Deposition Rate ($\mu\text{g}/\text{m}^2/\text{yr}$)	Max Deposition (6 yr, $\mu\text{g}/\text{m}^2$)
Group I	Cadmium	67	13	80
	Thallium	67	13	80
Group II	Mercury (partic)	261	52	313
	Hg(2+) vap	4484	897	5380
	Hg(0) vap	1.0	2.0×10^{-1}	1.2
Group III	Antimony	153	31	184
	Arsenic	147	29	176
	Chromium	155	31	186
	Cobalt	147	29	176
	Copper	147	29	176
	Lead	147	29	176
	Manganese	160	32	191
	Nickel	153	31	184
	Vanadium	153	31	184

Risk Characterisation

- 16.51 GQRA was undertaken by comparing the calculated soil concentration at the most impacted receptor location to generic assessment criteria (GAC) comprised of SGVs and in-house screening criteria derived by SLR using CLEA v1.05. For the purpose of a 'worst case scenario' HHRA the maximum deposition rate calculated for any specific receptor was selected for each contaminant. The model assumes a generic CLEA sandy loam soil, however the model is not sensitive to soil organic matter given the compounds being assessed.
- 16.52 Soil concentrations after 6 years were calculated by assuming that deposited particulate matter would be mixed evenly in the top 0.1m of the soil (density $1600 \text{ kg}/\text{m}^3$) and that there was no onward mobilisation of contaminated dusts.
- 16.53 As described previously, for a worst case scenario, the critical receptor at the most impacted location is assumed to a female child aged 0-6 years of age; this is the most sensitive receptor specified for the HHRA of contaminated land in the Environment Agency's CLEA Report (Environment Agency, 2009a). GAC for soil that are protective of this receptor are compared to worst case soil concentration calculated for the most impacted location. Results of the generic risk assessment are presented in Table 16-6.

Table 16-6

Generic Quantitative Risk Assessment

Contaminant	Max Deposition (6 yr, µg/m ²)	Conc in top 0.1m (mg/kg)	SGV/GAC ¹⁰ (mg/kg)	Hazard quotient	Time to exceedance (year)
Cadmium	80	5.0 x10 ⁻⁴	1.8 (Allot SGV)	2.8 x10 ⁻⁴	21,000
Thallium	80	5.0 x10 ⁻⁴	2.7 (Resi GAC)	1.9 x10 ⁻⁴	32,000
Mercury (partic)	313	2.0 x10 ⁻³	80 (Allot SGV)	2.4 x10 ⁻⁵	245,000
Hg(2+) vap	5380	3.4 x10 ⁻²	80 (Allot SGV)	4.2 x10 ⁻⁴	14,275
Hg(0) vap	1.2	7.5 x10 ⁻⁶	1 (Resi SGV)	7.5 x10 ⁻⁶	800,000
Arsenic	176	1.1 x10 ⁻³	32 (Resi SGV)	3.4 x10 ⁻⁵	174,000
Chromium	186	1.2 x10 ⁻³	7.8 (Allot GAC)	1.5 x10 ⁻⁴	40,200
Copper	176	1.1 x10 ⁻³	524 (Allot GAC)	2.1 x10 ⁻⁶	2,850,000
Lead	176	1.1 x10 ⁻³	250 (Allot GAC)	4.4 x10 ⁻⁶	1,360,000
Nickel	184	1.1 x10 ⁻³	130 (Resi SGV)	8.8 x10 ⁻⁶	680,000
Vanadium	184	1.1 x10 ⁻³	18 (Allot GAC)	6.4 x10 ⁻⁵	94,000

16.54 All predicted soil concentrations for the metals are extremely low; these concentrations are considerably less than UK soil background concentrations (e.g. 10-30 mg/kg lead¹¹, 10 mg/kg arsenic¹², 0.12 mg/kg mercury¹³). All estimated maximum concentrations of metals in soil resulting from the deposition of particulates in emissions from operation of the EfW facility are negligible in comparison with the relevant GAC, with hazard quotients (HQ = soil conc / GAC) ranging from 2.1 x10⁻⁶ to 2.8 x10⁻⁴. These results indicate an absence of risk to human health from the deposition of metal contaminants. Despite the conservative assumptions in this risk assessment methodology it would be thousands of years before GAC were exceeded at the most impacted receptor location; this is much longer than the anticipated operational lifetime of the facility.

Detailed Quantitative Risk Assessment (DQRA)

16.55 The Environment Agency has published SGVs and risk assessment worksheets for dioxins in soil but a number of exposure pathways relevant to dioxins are not considered by the CLEA model. This HHRA has therefore adopted a DQRA approach to the risk assessment of dioxin emissions based on HMIP Methodology, adapted to current UK practice and

¹⁰ The lowest SGV/GAC is selected as this corresponds to the most sensitive land use scenario. Taken from Environment Agency (2009e-h) and GAC derived by SLR using CLEA v1.05 (see Appendix A for details)

¹¹ Macleod et al (2006)

¹² Environment Agency (2009e)

¹³ SSLRC (2000)

incorporating exposure estimates generated by the Environment Agency SGVs/worksheets.

HMIP Methodology for assessment of dioxins in food chain and breast milk

- 16.56 Intake from additional exposure pathways such as inhalation of airborne dioxins, secondary uptake into the food chain and ingestion of breast milk by breast-fed babies has been calculated using the intake algorithms and exposure parameters specified by HMIP (Annexes C16-17 and Annex E, respectively).
- 16.57 The approach defined within the HMIP guidance comprises of four sections (detailed in section 4.1 of the HMIP guidance) as follows:
1. *Measure or estimate emissions from the EfW facility.*
 2. *Model the fate and transport of the emissions through the atmosphere and through soil, water and biota following deposition onto land. Estimate concentrations of the emitted chemicals in the environmental media at the point of exposure.*
 3. *Calculate uptake of the emitted chemicals into humans coming into contact with the affected media.*
 4. *Assess the significance of the absorbed dose in terms of a likely health impact.*

Deposition of Emissions

- 16.58 The HMIP approach for deposition (HMIP Section 4.5) is based on partitioning the predicted ground level concentrations of each congener (modelled as a vapour) between the particulate and vapour phase. This approach assumes that both particles and vapours disperse in the same way and does not allow for depletion.
- 16.59 Whilst deposition algorithms within advanced dispersion models (such as AERMOD) allow for the modelling of deposition of particles and vapours from the point of release, and also account for depletion, these are not compatible with the HMIP approach for uptake assessment and therefore the HMIP approach to quantifying deposition has been applied.

Calculation of Media Concentrations

- 16.60 Concentrations of dioxins in the upper 1cm, 10cm and 15cm of soil are calculated according to the equations detailed in Annex C1 of the HMIP guidance; these assume that particulate deposition occurs at a constant annual rate and that deposited particulates are mixed evenly, with a constant proportion being lost due to physico-chemical processes, as defined by the soil loss constant detailed in HMIP Annex C2.
- 16.61 Exposure to the resultant concentrations of dioxins in garden soil and soil-derived indoor dust are calculated within the most recent version of the Environment Agency's CLEA model for the assessment of contaminated land (Environment Agency, 2009a,b). Concentrations of dioxins in home-

- grown produce at residential receptor locations have also been calculated using the CLEA model.
- 16.62 Concentration of dioxins in other environmental media are not considered within the CLEA model and have been calculated using the algorithms detailed in Annex C of the HMIP guidance and listed below:
- Plant crops grown in the local area (HMIP Annexes C6-C10);
 - Dioxin concentrations in animal tissue resulting from bioaccumulation in the food chain (Annexes C12-C14); &
 - Maternal intake and concentration in breast milk (Annex C17).
- 16.63 Concentrations of dioxins in soil, and subsequently crops and animal tissue, were calculated for a worst case scenario using the maximum concentrations of dioxins in air calculated by air dispersion modelling, i.e. at the point of maximum ground level impact from EfW emissions.
- 16.64 Concentrations of dioxins in drinking water and fish were not assessed as no drinking water abstractions from surface water were identified in the vicinity of the EfW and exposure via other pathways arising from surface water are deemed to be relatively insignificant compared to exposure from soil and diet.

Calculation of Human Intake

- 16.65 The dioxin intake rates resulting from exposure to the environmental media were calculated for the worst case exposed receptors as detailed in the HMIP guidance
- 16.66 Intake resulting from exposure to dioxins in soil via direct ingestion, dermal contact and consumption of home-grown produce is estimated using the latest version of the Environment Agency's CLEA model (v1.05 released in September 2009). Exposure of an individual receptor is calculated by CLEA based on a set of standard assumptions relating to land-use, receptor behaviour, site, building and soil characteristics.
- 16.67 Intake from additional exposure pathways such as inhalation of airborne dioxins, secondary uptake into the food chain and ingestion of breast milk by breast-fed babies has been calculated using the intake algorithms and exposure parameters specified by HMIP (Annexes C16-17 and Annex E, respectively).

Effect on Human Health

- 16.68 The relative contribution of individual congeners to the overall toxicity of a mixture of dioxins is calculated by the use of toxicity equivalence factors (TEFs). It is generally acknowledged that the toxicity of individual dioxins is mediated by the same mechanism of action with the dioxin 2,3,7,8-TCDD being the most potent and best studied congener so TEFs define potency in relation to 2,3,7,8-TCDD.
- 16.69 The WHO European Centre for Environment and Health and the International Program on Chemical Safety have developed a set of criteria for TEF calculations for the relevant dioxin and furan congeners (Van den Berg et al., 2006). These TEFs have since been endorsed by the UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment COT for use in UK assessments of dioxin exposure. The

potential dioxin-like activity contributed by each congener is determined by multiplying the concentration of the congener by its WHO-designated TEF to yield the dioxin toxic equivalent (TEQ) for that congener. The net TEQ is the sum of the individual TEQs for each dioxin or dioxin-like compound.

WHO TEFs for dioxins and furans are detailed in Table 16-7 below.

Table 16-7
WHO TEFs for Dioxins (Van den Berg et al, 2006)

	Congener	WHO TEF
Dioxins (PCDDs)	2,3,7,8-TCDD	1
	1,2,3,7,8-PeCDD	1
	1,2,3,4,7,8-HxCDD	0.1
	1,2,3,7,8,9-HxCDD	0.1
	1,2,3,6,7,8-HxCDD	0.1
	1,2,3,4,6,7,8-HpCDD	0.01
	OCDD	0.0003
Furans (PCDFs)	2,3,7,8-TCDF	0.1
	1,2,3,7,8-PeCDF	0.03
	2,3,4,7,8-PeCDF	0.3
	1,2,3,4,7,8-HxCDF	0.1
	1,2,3,7,8,9-HxCDF	0.1
	1,2,3,6,7,8-HxCDF	0.1
	2,3,4,6,7,8-HxCDF	0.1
	1,2,3,4,6,7,8-HpCDF	0.01
	1,2,3,4,7,8,9-HpCDD	0.01
	OCDF	0.0003

16.70 Intake values resulting from all exposure pathways considered are combined to generate an overall 'hypothetical maximum exposure' intake for each receptor, the receptors considered in this assessment are:

- breast-fed infant;
- child resident;
- adult resident;
- adult farmer; and
- farmer's child.

16.71 The potential health risk posed by the intake of environmental media containing dioxins, (expressed as toxicity equivalence/TEQ) is assessed by comparison to health criteria values (HCVs) recommended by the UK COT and the WHO.

- 16.72 Background intake to dioxins is significant due to their persistence and bioaccumulative nature; where possible this is assessed using region-specific data, or by using the 'mean daily intake' (MDI) value specified by Defra and the Environment Agency.

Dispersion Modelling

- 16.73 The dispersion modelling identifies the location of maximum ground level concentration (greatest impact), and also predicts the ground level concentrations at a number of sensitive discrete receptors in the vicinity of the Installation. The highest annual average (from the 5-years of meteorological data) predicted ground level concentration in femto-grams per cubic metre (10-15 g/m³) of each congener at the location of greatest impact are presented below (Table 16-8) along with data for the average and minimum predicted impacts.

Table 16-8 Predicted Congener Ground Level Concentrations (fg/m³)

Compound	Minimum Predicted Impact	Average Predicted Impact	Maximum Predicted Impact
2,3,7,8 –TCDD	0	0.001745	0.01573
1,2,3,7,8–PeCDD	0	0.005583	0.05033
1,2,3,4,7,8–Hx	0	0.005583	0.05033
1,2,3,6,7,8–	0	0.012911	0.11639
1,2,3,7,8,9–	0	0.011166	0.10066
1,2,3,4,6,7,8–Hp	0	0.076417	0.68887
OCCD	0	0.120382	1.08519
2,3,7,8-TCDF	0	0.025123	0.22648
1,2,3,7,8-PeCDF	0	0.017447	0.15727
2,3,4,7,8-PeCDF	0	0.024077	0.21704
1,2,3,4,7,8-	0	0.028612	0.25793
1,2,3,6,7,8-	0	0.020587	0.18559
1,2,3,7,8,9-	0	0.004537	0.0409
2,3,4,6,7,8-	0	0.023029	0.2076
1,2,3,4,6,7,8-Hp	0	0.054433	0.4907
1,2,3,4,7,8,9-Hp	0	0.008374	0.07549
OCDF	0	0.031404	0.28309
TOTAL	0	0.471	4.25

- 16.74 From these data it is evident that the peak predicted ground level impact is nearly ten times greater than the average predicted impact generated by air dispersion modelling. The risk assessment uses data from the location of maximum predicted impact to give the worst case exposure.

Results of Transport (Soil and Biota) Modelling

Predicted soil concentration

- 16.75 Concentrations of individual congeners in soil were calculated based on the maximum airborne dioxin concentrations detailed above and predicted annual rates of wet and dry deposition. Soil loss is incorporated into this calculation to account for depletion due to volatilisation, leaching and surface runoff as shown in Table 16-9.

Table 16-9
Predicted Congener Soil Concentrations

Compound	Airborne conc (fg/m ³)	Total deposition (pg/m ² /a)	Soil loss constant (a ⁻¹)	Soil conc – 10cm depth (pg/kg)	Soil conc – 10cm depth (ng WHO-TEQ/kg)
2,3,7,8–TCCD	0.0157	0.6	0.21	0.053	5.3 x10 ⁻⁵
1,2,3,7,8–	0.0503	2.8	0.10	0.058	5.8 x10 ⁻⁵
1,2,3,4,7,8–Hx	0.0503	3.0	0.07	0.034	3.4 x10 ⁻⁶
1,2,3,6,7,8–	0.1164	7.0	0.07	0.079	7.9 x10 ⁻⁶
1,2,3,7,8,9–	0.1007	6.1	0.07	0.068	6.8 x10 ⁻⁶
1,2,3,4,6,7,8–Hp	0.6889	43	0.06	0.380	3.8 x10 ⁻⁶
OCCD	1.0852	68	0.06	0.570	1.7 x10 ⁻⁷
2,3,7,8-TCDF	0.2265	10.0	0.33	1.085	1.1 x10 ⁻⁴
1,2,3,7,8-PeCDF	0.1573	9.3	0.15	0.236	7.1 x10 ⁻⁶
2,3,4,7,8-PeCDF	0.2170	12.9	0.15	0.326	9.8 x10 ⁻⁵
1,2,3,4,7,8-	0.2579	16	0.09	0.209	2.1 x10 ⁻⁵
1,2,3,6,7,8-	0.1856	11.5	0.09	0.150	1.5 x10 ⁻⁵
1,2,3,7,8,9-	0.0409	2.5	0.07	0.025	2.5 x10 ⁻⁶
2,3,4,6,7,8-	0.2076	13	0.09	0.168	1.7 x10 ⁻⁵
1,2,3,4,6,7,8-Hp	0.4907	31	0.06	0.293	2.9 x10 ⁻⁶
1,2,3,4,7,8,9-Hp	0.0755	4.7	0.06	0.045	4.5 x10 ⁻⁷
OCDF	0.2831	18	0.06	0.153	4.6 x10 ⁻⁸

16.76 The total dioxin concentration in soil at the receptor location with the highest deposition rate was calculated as 4.0 x10⁻⁴ ng TEQ/kg which is considerably lower than typical concentrations of 1 to 100 ng TEQ/kg that have been measured in soil across Europe (EC/DETR, 1999).

Predicted plant concentrations

16.77 Concentrations of individual congeners were calculated in various plant types by summation of plant concentrations due to root uptake in above and below ground plants and direct deposition from air and air-to-plant transfer for above ground plants. Congener concentrations (fg/kg dry weight) in the different plant types considered by the HMIP guidance are detailed in Table 16-10 below.

Table 16-10
Predicted Congener Plant Concentrations

Compound	Potato (fg/kg)	Leafy Veg (fg/kg)	Legume (fg/kg)	Root Veg (fg/kg)	Fruit (fg/kg)	Fruiting Veg (fg/kg)	Cereal/ grain (fg/kg)	Forage (fg/kg)
2,3,7,8–TCCD	3.3	8.2	3.8	3.3	5.4	5.7	3.3	17.1
1,2,3,7,8–PeCDD	6.56	47.4	10.8	6.56	23.7	26.4	6.56	121.6
1,2,3,4,7,8–HxCDD	6.3	76	14	6.3	36	40	6.3	203
1,2,3,6,7,8–HxCDD	15	197	34	15	91	103	15	529

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1,2,3,7,8,9-HxCDD	13	171	29	13	79	89	13	458
1,2,3,4,6,7,8-Hp	16	1441	166	16	614	709	16	4028
OCCD	148	3384	487	148	1506	1720	148	9259
2,3,7,8-TCDF	55	228	74	55	128	139	55	541
1,2,3,7,8-PeCDF	4.3	288	34.1	4.3	123	142	4.3	803.5
2,3,4,7,8-PeCDF	6.0	338	40.8	6.0	145	167	6.0	940
1,2,3,4,7,8-HxCDF	2.9	702	76	2.9	296	343	2.9	1971
1,2,3,6,7,8-HxCDF	2.1	518	56.2	2.1	219	253	2.1	1455
1,2,3,7,8,9-HxCDF	0.41	113.0	12.2	0.41	48	55	0.41	317
2,3,4,6,7,8-HxCDF	2.4	580	63	2.4	245	283	2.4	1628
1,2,3,4,6,7,8-Hp	2.2	1525	162	2.2	641	742	2.2	4291
1,2,3,4,7,8,9-Hp	0.34	248	26.3	0.34	104	120.6	0.34	697
OCDF	0.53	941	99	0.53	395	457	0.53	2648

16.78 The highest concentrations of dioxins were predicted for above ground plants (e.g. vegetables, fruit and grass/forage) as the major contribution was predicted to arise from direct deposition of dioxins.

Predicted animal tissue concentrations

16.79 Congeners concentrations in animal tissues due to bioconcentration within the food chain were calculated based on the assumption that animals consumed grass (forage) or grain grown exclusively within the affected area (all of which is assumed to be affected by the maximum predicted airborne concentrations). Congener concentrations (fg/kg dry weight) in the different animal tissues and food types considered by the HMIP guidance are detailed in Table 16-11 below.

Table 16-11

Predicted Animal Tissue Concentrations (pg/kg)

Compound	Beef (pg/kg)	Offal (pg/kg)	Meat Prod (pg/kg)	Fats (pg/kg)	Pork (pg/kg)	Lamb (pg/kg)	Poultry (pg/kg)	Eggs (pg/kg)	Milk (pg/kg)	Dairy (pg/kg)
2,3,7,8 -TCDD	0.13	0.27	0.27	0.18	0.25	0.78	0.14	1.05	0.27	1.13
1,2,3,7,8-Pe	0.36	0.43	0.23	0.15	0.10	0.64	0.50	3.69	0.75	3.09
1,2,3,4,7,8-Hx	0.34	0.60	0.10	0.08	0.08	0.25	0.38	2.83	0.71	2.96
1,2,3,6,7,8-Hx	0.89	1.57	0.22	0.21	0.20	0.58	0.81	6.03	1.85	7.66
1,2,3,7,8,9-Hx	0.77	1.35	0.19	0.09	0.11	0.50	0.90	6.71	1.60	6.63
1,2,3,4,6,7,8-Hp	0.80	7.64	0.11	0.16	0.42	0.32	1.29	9.58	1.67	6.92
OCCD	0.46	5.47	0.05	0.06	0.47	0.13	3.92	29.20	0.95	3.92
2,3,7,8-TCDF	0.72	1.62	1.18	1.97	0.57	3.43	0.72	5.33	1.49	6.15
1,2,3,7,8-Pe	0.55	2.77	0.21	0.12	0.17	0.63	0.55	4.13	1.15	4.77
2,3,4,7,8-Pe	2.65	13.11	1.17	0.70	0.85	3.49	2.77	20.61	5.51	22.83
1,2,3,4,7,8-Hx	3.26	30.62	0.48	0.32	0.41	1.43	4.03	30.02	6.77	28.05
1,2,3,6,7,8-Hx	2.40	22.58	0.34	0.19	0.26	1.03	2.49	18.56	4.99	20.68
1,2,3,7,8,9-Hx	0.52	4.88	0.06	0.03	0.05	0.17	0.64	4.78	1.08	4.47
2,3,4,6,7,8-Hx	2.69	25.26	0.38	0.12	0.09	1.15	2.44	18.21	5.59	23.14
1,2,3,4,6,7,8-Hp	1.27	19.49	0.12	0.09	0.20	0.36	1.36	10.10	2.64	10.93
1,2,3,4,7,8,9-Hp	0.51	7.84	0.05	0.01	0.02	0.14	0.59	4.40	1.06	4.38
OCDF	0.26	1.82	0.02	0.02	0.05	0.06	0.29	2.13	0.54	2.24

16.80 Dioxins are highly lipophilic chemicals, which leads to their preferential accumulation in fatty tissue. The highest dioxin concentrations were

therefore predicted for animal tissues and food types containing high levels of fats, e.g. dairy products, animal fats and offal.

Predicted concentration in mothers milk

- 16.81 Dioxin concentrations in mothers' milk were calculated based on the mother's intake of dioxin from all exposure routes, half-life of dioxins in the body and the fraction of maternal intake that is bioavailable for transfer to breast milk. Using the algorithm detailed in HMIP Annex C17 the dioxin concentration in breast milk fat from a maximally exposed female was estimated to be 37 fg TEQ/kg.

Results of exposure/intake modelling

- 16.82 Human exposure to dioxins was estimated for a number of potential receptors considered as the worst case scenario affected by dioxins concentrations at the point of maximum ground level impact. Potential receptors considered in this assessment were:

- breast-fed infant;
- child resident;
- adult resident;
- farmer; and
- farmer's child.

- 16.83 Farm dwellers were assessed separately as the HMIP guidance considers them to consume a higher proportion of produce from the affected area.

Intake due to soil ingestion, dermal uptake and home-grown produce

- 16.84 Intake resulting from direct ingestion of soil and soil-derived dust, dermal exposure and consumption of home-grown produce was calculated using the latest version of the Environment Agency's CLEA model and worksheets provided for the calculation of dioxin exposure.

- 16.85 The Environment Agency has recommended soil guideline values (SGVs) in the form of total concentrations of dioxins, furans and dioxin-like PCBs. These SGVs are intended to be used for the generic quantitative risk of dioxin contaminated soil with the caveat that they are only applicable where the distribution of individual dioxin and PCB congeners matches the typical profile encountered in UK soils. The SGV report states that "this profile reflects input from general diffuse pollution over many years rather than contamination from a relatively recent or specific point source and assumes that there is no strong local or on-site signature..... and that soil aging and weathering has resulted in the integration of different sources terms and a relative enrichment of the more inert/immobile congeners". These issues, and the fact that the integrated SGVs include consideration of dioxin-like PCBs (not considered in this assessment), leads to the conclusion that it would be inappropriate to use the SGVs in this HHRA.

- 16.86 However, the dioxin risk assessment worksheets provided as an appendix to the SGV report are considered appropriate to use as they are intended to estimate the total exposure (adjusted for toxic equivalence) of each congener due to exposure pathways relevant to soil contamination. The

worksheets use an exposure factor (EF), estimated using the CLEA model, which is the sum of the average exposure from soil for all relevant pathways per unit concentration of the congener in soil according to standard land use scenarios

16.87 Average Daily Exposure predicted using the dioxin exposure worksheets for various receptors are detailed in Table 16-12 and Environment Agency Dioxin Exposure Worksheets used in this assessment are detailed in Appendix C.

Table 16-12

CLEA Predicted Average Daily Exposure

Compound	Soil Conc. (pg/kg)	ADE (pg TEQ/kg bw/d)			
		Infant	Child	Adult	Farmer
2,3,7,8 –TCDD	0.053	5.76×10^{-7}	5.47×10^{-7}	1.13×10^{-7}	1.50×10^{-7}
1,2,3,7,8–PeCDD	0.058	6.11×10^{-7}	6.01×10^{-7}	1.18×10^{-7}	1.59×10^{-7}
1,2,3,4,7,8–HxCDD	0.034	3.47×10^{-8}	3.32×10^{-8}	5.62×10^{-9}	8.04×10^{-9}
1,2,3,6,7,8–HxCDD	0.079	8.02×10^{-8}	7.69×10^{-8}	1.30×10^{-8}	1.86×10^{-8}
1,2,3,7,8,9–HxCDD	0.068	6.94×10^{-8}	6.65×10^{-8}	1.12×10^{-8}	1.61×10^{-8}
1,2,3,4,6,7,8–Hp	0.380	3.75×10^{-8}	3.40×10^{-8}	4.36×10^{-9}	7.07×10^{-9}
OCDD	0.570	1.67×10^{-9}	1.49×10^{-9}	1.74×10^{-10}	2.96×10^{-10}
2,3,7,8-TCDF	1.085	1.30×10^{-6}	1.10×10^{-6}	2.46×10^{-7}	3.23×10^{-7}
1,2,3,7,8-PeCDF	0.236	7.75×10^{-8}	7.17×10^{-8}	1.41×10^{-8}	1.91×10^{-8}
2,3,4,7,8-PeCDF	0.326	1.07×10^{-6}	9.89×10^{-7}	1.94×10^{-7}	2.64×10^{-7}
1,2,3,4,7,8-HxCDF	0.209	2.16×10^{-7}	2.00×10^{-7}	3.32×10^{-8}	4.81×10^{-8}
1,2,3,6,7,8-HxCDF	0.150	1.49×10^{-7}	1.44×10^{-7}	1.77×10^{-8}	2.84×10^{-8}
1,2,3,7,8,9-HxCDF	0.025	2.51×10^{-8}	2.42×10^{-8}	2.98×10^{-9}	4.78×10^{-9}
2,3,4,6,7,8-HxCDF	0.168	1.74×10^{-7}	1.61×10^{-7}	2.67×10^{-8}	3.87×10^{-8}
1,2,3,4,6,7,8-Hp	0.293	2.91×10^{-8}	2.62×10^{-8}	3.46×10^{-9}	5.54×10^{-9}
1,2,3,4,7,8,9-Hp	0.045	4.48×10^{-9}	4.04×10^{-9}	5.32×10^{-10}	8.53×10^{-10}
OCDF	0.153	4.53×10^{-10}	4.06×10^{-10}	4.97×10^{-11}	8.24×10^{-11}
Total		4.46×10^{-6}	4.08×10^{-6}	8.04×10^{-7}	1.09×10^{-6}

Dietary Intake

16.88 Dietary intake for the individual receptors was estimated on a TEQ basis based on plant and animal concentrations calculated in Section 5.3, mean food consumption rates specified in HMIP Annex E4 and the proportion of local produce consumed by residents and farmers (Annex E5). Intake is expressed on a bodyweight per day basis and receptor bodyweights, exposure frequency/duration and averaging times used in calculations were those detailed in HMIP Annex E314. Dietary intakes resulting from consumption of the HMIP food types for each receptor are detailed in Table 16-13.

¹⁴ NB. CLEA infant (Age Class 1) bodyweight of 5.6 kg has been used throughout the HHRA as this is more conservative than HMIP value of 8 kg. HMIP bodyweight values are used elsewhere as they are very close to CLEA values, e.g. HMIP (1996) specifies child bodyweight of 15 kg compared. average of 14.84 kg for CLEA Age Classes 2-6.

Table 16-13

Predicted Dietary Intake

Food type	Dietary Intake (fg TEQ/kg bw/d)				
	Infant	Child	Adult	Farmer	Farm Child
Beef	0.23	0.18	0.10	0.28	0.51
Offal	0.34	0.18	0.16	0.47	0.52
Lamb	0.065	0.030	0.025	0.074	0.09
Pork	0.045	0.055	0.041	0.12	0.16
Poultry	0.070	0.034	0.033	0.10	0.10
Milk	22.47	3.60	1.37	4.12	10.79
Eggs	0.23	0.07	0.04	0.11	0.19
Dairy	9.62	2.99	1.83	5.64	9.21
Meat products	0.77	0.65	0.42	1.23	1.92
Fats, oil	0	0.72	0.38	1.11	2.10
Potatoes	0.015	0.012	0.006	0.02	0.032
Leafy vegetables	0.41	0.31	0.46	0.98	0.67
Legumes	0.036	0.030	0.034	0.073	0.065
Root vegetables	0.007	0.002	0.002	0.004	0.004
Fruit	0.71	0.17	0.10	0.24	0.42
Fruiting vegetables	0.16	0.13	0.15	0.32	0.29
Cereals	0.006	0.001	0.001	0.002	0.004
Total	35	9.2	5.2	15	27

Intake due to inhalation

16.89 Intake due to inhalation of airborne dioxins was estimated based on the maximum predicted impact at ground level calculated in Section 3.1 and exposure parameters for each receptor detailed in HMIP Annex E115. Predicted inhalation intake for each receptor is detailed below:

- Infant – 0.24 fg TEQ/kg bw/d
- Child – 0.16 fg TEQ/kg bw/d
- Adult – 0.052 fg TEQ/kg bw/d
- Farmer – 0.054 fg TEQ/kg bw/d
- Farmer's child – 0.16 fg TEQ/kg bw/d

Intake for Breast-fed Infant

16.90 Based on exposure assumptions detailed in Environment Agency (2009a)¹⁶ and HMIP Annex E6, intake for an infant from breast milk was calculated to be 0.092 pg TEQ/kg bw/d.

Total Intake

16.91 The total intake resulting from dioxin emissions from the New England EfW installation was calculated for each receptor by combining the estimated intake from each exposure pathway.

¹⁵ Inhalation rates were taken from HMIP guidance rather than CLEA as they were higher (i.e. more conservative) and accounted for an indoor/outdoor split

¹⁶ Infant bodyweight of 5.6 kg

Table 16-14

Predicted Dioxin Intake

Pathway	Intake (pg TEQ/kg bw/d)				
	Infant	Child	Adult	Farmer	Farm Child
CLEA pathways	4.46×10^{-6}	4.08×10^{-6}	8.04×10^{-7}	1.09×10^{-6}	4.08×10^{-6}
Diet	0.0352	0.0092	0.0051	0.0149	0.0271
Inhalation	2.39×10^{-4}	1.58×10^{-4}	5.22×10^{-5}	5.41×10^{-5}	1.58×10^{-4}
Breast milk	0.09				
Total Intake	0.13	0.0093	0.0052	0.015	0.027

16.92 Total predicted intake for worst case scenario exposure varies for the different receptor types, ranging from 0.0052 pg TEQ/kg bw/d for an adult resident to 0.13 pg TEQ/kg bw/d for a breast-fed infant. For all receptors, except the infant, dietary exposure is predicted to make the largest contribution to dioxin exposure, accounting for approximately 99% of total intake.

Background intake

16.93 It is generally acknowledged that 90% of a persons' exposure to dioxins is likely to occur through their diet and the Environment Agency (2009i) estimate an adult mean daily intake of 49 pg TEQ, based on data from the Food Standards Agency, with food from animal origin being the dominant source. This intake is over 100 times higher than the predicted dietary intake for a theoretically maximum exposed adult resident (0.36 pg/day for a 70 kg adult) which also indicates that exposure arising from operation of the New England EfW is likely to be insignificant compared to background sources.

Human health effects

16.94 The potential health risk posed by the intake of dioxins, (expressed as TEQ) is assessed by comparison to a tolerable daily intake (TDI) of 2 pg/kg bw/day recommended by the UK COT and the World Health Organisation (WHO, 2002) TDI range of 1-4 pg/kg bw/day. WHO emphasised that the upper limit of the range (4 pg TEQ/kg bw/d) should be considered a maximum tolerable daily intake on a provisional basis, and the ultimate goal should be to reduce intakes to levels less than 1 pg TEQ /kg bw/day.

16.95 The theoretical maximum predicted intake for each receptor considered in this assessment is compared to the COT TDI to generate a hazard quotient (HQ = Intake/TDI) in Table 16-15 below.

Table 16-15

Human Health Risk Assessment

Receptor	Maximum Predicted Intake (pg/kg bw/d)	COT TDI (pg/kg bw/d)	Hazard Quotient
Infant	0.13	2	0.065
Child	0.0093	2	0.0047
Adult	0.0052	2	0.0026
Farmer	0.015	2	0.0075
Farm Child	0.027	2	0.014

- 16.96 These results indicate an absence of risk to human health from dioxin emissions from the proposed New England EfW as the maximum predicted intake of dioxins for a residential receptor (i.e. child receptor in CLEA contaminated land guidance) is approximately 200 times less than the UK recommended TDI.
- 16.97 The maximum intake predicted for a breast-fed infant (0.13 pg/kg bw/d) is under 10% of the COT TDI but this value is significantly lower than the MAFF estimated intakes of 170 pg/kg bw/d at 2 months and 39 pg/kg bw/d at 10 months for breast-feeding infants in 1993-94 (FSA, 2001)¹⁷. Despite the high intakes of dioxins experienced by nursing infants (about 100-fold those of an adult), the impact of breast feeding on infant body burden of dioxin is considered to be markedly less dramatic. FSA advise that “although dioxin intakes are higher than desirable for breast-fed babies, encouragement of breast-feeding should continue on the basis of overwhelming evidence of the benefit of human milk to the overall health and development of the baby”.
- 16.98 All predicted intakes from the proposed EfW facility are also lower than the WHO ‘target TDI’ of 1 pg/kg bw/d, confirming the absence of a risk to human health. This is despite the high degree of conservatism in this risk assessment which assumed that exposure resulted from the maximum predicted ground level concentration and all soils and plant crops (and farm animals subsequently feeding on these) were affected by this concentration.

¹⁷ A 2001-02 study of breast-fed infants in Yorkshire found that the upper bound mean daily intakes were estimated to be 72 pg WHO-TEQ kg⁻¹ bw day⁻¹ at 2–3 months and 19 pg WHO-TEQ kg⁻¹ bw day⁻¹ at 8–10 months, indicating that intakes of dioxins and PCBs by breast-fed infants may have dropped by over 50% since the 1993–1994 survey (Environment Agency, 2009i).

Conclusions

- 16.99 A conceptual site model has been developed for the New England Quarry EfW that has established potential sources of pollution, receptors and relevant pathways of potential exposure to persistent pollutants such as heavy metals and dioxins/furans from particle phase and vapour deposition to soil.
- 16.100 As potential linkages were identified an assessment of the risk these posed to human health was undertaken. A conservative worst case scenario was developed for a screening exercise in which it was assumed that the most sensitive receptor, a young female child, was present at the site location receiving the highest deposition rates of the metallic contaminants present in emissions from the facility. Predicted soil concentrations were compared to generic assessment criteria generated by the CLEA v1.05 model, which used to assess human health risks deriving from contaminated land.
- 16.101 Results from generic quantitative risk assessment for metals indicate that indirect, long-term exposure to all metal contaminants emitted from the proposed EfW facility and subsequently deposited to soil does not pose a health risk to downwind receptors. This conclusion is deemed to be robust as it is based on a worst case scenario and there is a large margin of safety (>3,500) between the highest predicted soil concentrations and soil assessment criteria that are protective of the most sensitive human receptors.
- 16.102 For the assessment of human health risks arising from dioxin exposure due to airborne emissions from the EfW facility estimates of individual congener levels in environmental media relevant to human exposure (e.g. soil, plant and animal foods and breast-milk) were used to predict theoretical maximum intakes for key receptors, including infants, children and farmers. The assessment demonstrated that the calculated intakes for these receptors were considerably lower than tolerable daily intakes recommended for the risk assessment of dioxins, thus demonstrating an absence of risk due to emissions from the New England EfW.
- 16.103 It is therefore considered that the effect on health from the metal and dioxin emissions from the New England EfW facility can be classified as highly unlikely to be significant.

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