



**Ardley EfW  
Oxfordshire**

**Human Health Risk Assessment**

**Viridor Waste Ltd**

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## **VIRIDOR Ardley Energy from Waste Project – Human Health Risk Assessment**

### **1.0 INTRODUCTION**

#### **1.1 Health Impact Assessment**

Health Impact Assessment (HIA) enables a judgement to be made as to whether a policy or development project has significant effects on the health of the potentially impacted population and also seeks to quantify the extent of predicted outcomes. SLR has undertaken a 'prospective HIA' to predict the potential consequences of the proposed Energy from Waste (EfW) facility at Ardley using the best available tools and UK technical guidance.

HIA is a multi-faceted process and comprises 5 main steps:

- *Screening* that determines whether a project is worth subjecting to HIA
- *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.

This exercise is concerned with the risk assessment stage and is 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 Ardley EfW.

#### **1.2 Human Health Risk Assessment for Ardley EfW**

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 Ardley.

The objective of this assessment was to evaluate the potential risk for populations that may be 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:

1. Compounds that exist predominantly in the gaseous phase, e.g. SO<sub>x</sub>, NO<sub>x</sub> and other acid gases. The health effects of these compounds are due to 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. PM<sub>10</sub>) also induces health effects following inhalation and is assessed against an air quality standard.
2. Compounds that exist associated with the particulate phase, e.g. metals and non-volatile organic compounds such as dioxins. Exposure to these persistent compounds will predominantly arise from indirect pathways following deposition of particles from the air; such pathways include ingestion of soil,

dust and food grown in contaminated soil and to a lesser extent 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.

Predominantly gaseous phase contaminants are considered in Chapter 5 of the ES, Air Quality, and this section of the report details a HHRA of the persistent contaminants predominantly associated with the particulate phase that may accumulate in the environment and therefore result in long term exposure. 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.

This primary focus of this assessment of persistent contaminants is a 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 methodology is based on a worst-case scenario approach, using the maximum predicted contaminant concentrations and assessing these levels against soil criteria that are protective of the most sensitive receptors within exposed populations.

## **2.0 METHODOLOGY**

The principles of Health Impact Assessment has largely been developed from Environmental Impact Assessment (National Assembly for Wales, 2000). The human health risk assessment of Ardley EfW has therefore been undertaken following the UK Risk Assessment Framework specified 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 is based on a tiered approach, where the level of complexity and effort/cost generally increases with each tier as conservatism and uncertainty decrease.

The common Risk Assessment Framework is summarised below

- Tier 1: Risk Screening - This is concerned with the development of an outline conceptual model and establishing whether there is any potential for unacceptable risks and a 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), which generally requires more complex risk modelling tools and the generation of more detailed data to characterise the site and receptors under consideration

A staged approach to HHRA for an EfW facility following this framework is presented below.

### **2.1 Tier 1: Risk Screening and Development of Conceptual Model**

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:

#### **2.1.1 Source of hazard**

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 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.

### 2.1.2 Hazard

Stack emissions are potentially hazardous due to the discharge and dispersion of both gaseous phase and particulate contaminants that have properties hazardous to health, i.e. acid gases (NO<sub>x</sub> and SO<sub>x</sub>) that have acute respiratory effects and persistent pollutants such as metals and dioxins that have a range of chronic toxicological effects. Metals and dioxins specified by the WID are detailed in Section 2.1.6.

### 2.1.3 Transport mechanisms

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

**Table 1**  
**WID Emission Limit Values**

Pollutant	Emission Limits (mg/Nm <sup>3</sup> ) <sup>(a)</sup>		
	Daily average values	Half hourly averages	
		100 <sup>th</sup> Percentile	97 <sup>th</sup> Percentile
Particles	10	30	10
TOC	10	20	10
HCl	10	60	10
HF	1	4	2
SO <sub>2</sub>	50	200	50
NO <sub>x</sub>	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/Nm<sup>3</sup> of combustion gas for at least 95% of all measurements determined as 10 minute averages or 100 mg/Nm<sup>3</sup> 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).

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 as a result of

meteorological conditions. The most important meteorological parameters governing the atmospheric dispersion of pollutants are as follows:

- wind direction determines the broad transport of the emission and the sector of the compass into which the emission is dispersed;
- 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 stability<sup>1</sup> than Pasquill.

#### **2.1.4 Pathways**

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 and crops).

#### **2.1.5 Targets/receptors and exposure**

License and discharge conditions for ERfWs are summarised in Table 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 and at school who may be exposed over long periods of time to low levels of combustion gases and particulates. This exposure is quantified in subsequent tiers of the risk assessment process.

#### **2.1.6 Contaminants of concern**

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 pollutants and individual contaminants specified by the WID are listed below:

<sup>1</sup> 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  $H$ .

**Table 2  
Persistent Pollutants**

<b>Class</b>	<b>Pollutant</b>
Group I metals	Cadmium
	Thallium
Group II metals	Mercury
Group III metals	Antimony
	Arsenic
	Chromium
	Cobalt
	Copper
	Lead
	Manganese
	Nickel
	Vanadium
Dioxins (PCDDs)	2,3,7,8-TCDD
	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

All contaminants from the list above are included in HHRA for EfW emissions with the exception of antimony, cobalt and manganese which 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 (2002c). Antimony and manganese are considered to possess only moderate toxicity while cobalt is thought to be of low toxicity.

A toxicity equivalence (TEQ) approach was applied to the individual dioxins and furans which relates their toxicity to that of the most toxic and best studied compound, 2,3,7,8-TCDD. Further details of the TEQ approach for dioxins and the de novo derivation of assessment criteria are provided in Appendix A.

## **2.2 Tier 2: Generic Quantitative Risk Assessment**

Atmospheric concentrations of gaseous phase contaminants and particulate 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 human health risk assessment. HHRA 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 of the principles of the Contaminated Land Regime and associated technical guidance.

The Contaminated Land Regime has been developed to assess the health risks associated with different levels of contaminants in soil in order to make quantified assessment. 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.

## **2.3 Tier 3: Detailed Quantitative Risk Assessment**

Where the screening exercise indicates that exposure exceeds 50% of the assessment criteria, site-specific assessment is undertaken for specific receptor locations. DQRA entails the utilisation of more detailed data on contaminant transport and receptor characteristics. 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 incorporate additional exposure pathways such as incorporation of persistent contaminants into the food chain.

### 3.0 HUMAN HEALTH RISK ASSESSMENT

As described above, HHRA for persistent contaminants in stack emissions from the proposed Ardley EfW facility follows the UK Risk Assessment Framework and GQRA will compare predicted concentrations of contaminants in soil, following deposition of particulate matter and semi-volatile contaminants, to generic assessment criteria used in the health risk assessment of contaminated land. This assessment of indirect exposure following deposition of persistent contaminants is based on the methodology detailed by Macleod et al (2006) for modelling indirect human exposure to airborne air pollution control (APC) residues<sup>2</sup> released from landfills. The approach detailed by Macloed et al employs air dispersion modelling to estimate dust emissions and predict deposition at selected receptor locations.

#### 3.1 Risk Screening and Development of Conceptual Model

The development of a conceptual site model (CSM) is the first stage of the risk assessment. The CSM identifies the potential sources, critical pathways and receptors relevant to HHRA for the Ardley EfW.

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 in Section 2.1.6.

The potential air quality impact of the proposed plant was assessed over an area of 17km x 17km (SW corner NGR 446500, 217500) at a resolution of 500m. A more detailed 5km x 5km grid (SW corner NGR 451750, 223100) with a resolution of 100m was also applied. Discrete receptor points were also located at 50 nearby houses and other sensitive locations as detailed in Table 5-17 of the Air Quality chapter.

Sources, types of receptors, relevant pathways of exposure and their potential significance are detailed in Table 3 below.

**Table 3**  
**Conceptual Site Model**

<b>Sources</b>			
Gaseous and particulates emitted in exhaust gases from Ardley EfW stack			
Acid gases, e.g. NO <sub>x</sub> and SO <sub>x</sub>			
Particulates (as PM <sub>2.5</sub> and PM <sub>10</sub> )			
Persistent contaminants in vapour and particulate phase, e.g. metals and dioxins			
<b>Receptors</b>			
<b>Receptor</b>	<b>Location</b>	<b>Risk</b>	
Resident	Children and adults in downwind residential properties	High	
Commercial	Workers in downwind commercial properties.	Medium	
Schools	Children and teachers in downwind schools	Medium-High	
Farmer	Downwind farms	Medium	

<sup>2</sup> 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.

<b>Pathways</b>	<b>Pathway</b>	<b>Description</b>	<b>Risk</b>	
	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	
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		
<b>Pollutant linkages</b>	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 in 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. 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 but will be mitigated to some extent by rainfall and washing produce. CLEA UK model assumes root vegetables are unpeeled		
	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		
<b>Overall risk assessment</b>	<b>MEDIUM-HIGH</b>			

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 UK model. Dietary exposure from the consumption of home-grown plants that have taken up 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 UK model, nor are uptake

of contaminants into herd animals following consumption of foliage affected by deposition and exposure of babies via breast milk. However, a number of conservative assumptions are built into the quantitative risk assessment, which will provide an indication of the relative magnitude of the risk posed by emissions from the EfW facility and DQRA will be triggered if predicted soil concentrations exceed 50% of GAC.

The approach detailed by Macloed et al (2006) employs air dispersion modelling to estimate dust emissions and predict deposition at selected receptor locations.

Similar to the assessment of particulates emitted from an EfW facility, key pollutant linkages identified by Macloed 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)

## **3.2 Generic Quantitative Risk Assessment**

### **3.2.1 Methodology**

GQRA for Ardley EfW entails the comparison of predicted concentrations of contaminants in soil, following deposition of particulate matter and semi-volatile contaminants, to generic assessment criteria used in the health risk assessment of contaminated land.

The GQRA for the Ardley EfW follows the methodology described by Macloed 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 UK model. A number of assumptions in the methodology of Macloed 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 private garden and where the family consumes its own homegrown produce.

The 'CLEA' model used by Macloed et al was replaced by the Environment Agency with the updated 'CLEA UK' model in October 2005. CLEA UK incorporated a number of modifications to CLEA but the two models are broadly similar and the basic principles are summarised below.

### **3.2.2 The CLEA UK Model**

The CLEA UK model estimates the intake of contaminants from soil humans from various exposure pathways (Environment Agency, 2005). 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 HCVs<sup>3</sup>) which are considered to be either tolerable or representative of minimal risk.

CLEA UK is a probabilistic model in that certain input parameters (such as bodyweight and amount of vegetables that are consumed) are represented by a range rather than a single value. UK policy is that the 95<sup>th</sup> percentile of the ADE is compared 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' 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.

Generic assessment criteria have been published by the Environment Agency and Defra for selected contaminants and these are known as soil guideline values (SGVs). SGVs combine both authoritative science and policy judgements. With the exception of lead, which uses another model, the published SGVs have been derived using the CLEA UK model.

For those contaminants for which published SGVs are not available SLR has generated GAC following the approaches recommended in CLR reports 9 & 10 (Defra & Environment Agency, 2002a,b) and associated material (e.g. CLEA briefing notes 1-4). Physico-chemical input parameters used to populate the CLEA UK model were selected from Environment Agency/Defra publications, where available, and authoritative data sources<sup>4</sup>.

It should be noted that a number of exposure pathways relevant to this HIA cannot be assessed using the CLEA UK model; these include:

<sup>3</sup> 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.

<sup>4</sup> 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

These pathways are potentially significant but would only be considered by DQRA if generic risk assessment indicated a potential risk from any of the contaminants (i.e. soil concentrations were greater than 50% of GAC).

Wet, dry and vapour deposition of contaminants to soil is estimated using AEROMOD, 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.

### 3.2.3 Site specific AEROMOD dispersion of particulates & semi-volatile contaminants

The methodology for the prediction of deposition rates for particulates and semi-volatile contaminants is presented in Air Quality chapter of the ES. A summary of the calculated deposition rates in downwind receptor locations is provided below.

**Table 4**  
**Deposition rate**

Class	Pollutant	Deposition (5 yr, $\mu\text{g}/\text{m}^2$ )		
		Average	Min	Max
Group I	Cadmium	2.7	0.30	15
	Thallium	2.7	0.30	15
Group II	Mercury (total)	85	5.9	530
	Hg(2+) vap	74	4.7	470
	Hg(0) vap	0.20	0.020	1.3
	Hg partic	11	1.2	57
Group III	Antimony	6.2	0.69	34
	Arsenic	5.9	0.65	32
	Chromium	6.3	0.70	34
	Cobalt	5.9	0.65	32
	Copper	5.9	0.65	32
	Lead	5.9	0.65	32
	Manganese	6.5	0.72	36
	Nickel	6.2	0.69	34
	Vanadium	6.2	0.69	34
Deposition (5 yr, fg TEQ/m <sup>2</sup> )				
Dioxins	Dioxins (total)	10,000	11,000	56,000
	particulate	9,800	1,100	53,000
	2378 TCDD gas	230	19	1,500
	2378 TCDF gas	180	15	1,200

Air dispersion modelling for predicted emissions from the Ardley 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 6 years used. A single value, the maximum deposition rate, is used in 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 UK to assess risks to children from exposure to soil contamination. Deposition over a six-year period is calculated below in Table 5.

**Table 5**  
**Calculation of 6-year deposition rate**

		<b>Max Deposition (5 yr, µg/m<sup>2</sup>)</b>	<b>Deposition Rate (µg/m<sup>2</sup>/yr)</b>	<b>Max Deposition (6 yr, µg/m<sup>2</sup>)</b>
Group I	Cadmium	15	2.9	18
	Thallium	15	2.9	18
Group II	Mercury (total)	530	110	640
Group III	Antimony	34	6.8	41
	Arsenic	32	6.4	39
	Chromium	34	6.9	41
	Cobalt	32	6.4	39
	Copper	32	6.4	39
	Lead	32	6.4	39
	Manganese	36	7.1	43
	Nickel	34	6.8	41
	Vanadium	34	6.8	41
	Dioxins	Dioxins (total TEQ)	5.6 x10 <sup>-5</sup>	1.1 x10 <sup>-5</sup>

### 3.3 Risk Characterisation

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 UK. For the purpose of a 'worst case scenario' HHRA the maximum deposition rate calculated for any specific receptor was selected for each contaminant.

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 kg/m<sup>3</sup>) and that there was no onward mobilisation of contaminated dusts or degradation of organic contaminants such as dioxins.

As described previously, for a worst case scenario, the critical receptor at the most impacted location is assumed to be a female child aged 0-6 years of age; this is the most sensitive receptor specified for the HHRA of contaminated land in CLR 10 (Defra and Environment Agency, 2002b). Generic assessment criteria 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 6.

**Table 6**  
**Generic Quantitative Risk Assessment**

Contaminant	Max Deposition (6 yr, µg/m <sup>2</sup> )	Conc in top 0.1m (mg/kg)	SGV/GAC (mg/kg)	Hazard quotient	Time to exceedance (year)
Cadmium	18	1.1 x10 <sup>-4</sup>	1 (SGV 3)	1.1 x10 <sup>-4</sup>	54,000
Thallium	18	1.1 x10 <sup>-4</sup>	0.85 (SLR)	1.3 x10 <sup>-4</sup>	46,000
Mercury (total)	640	4.0 x10 <sup>-3</sup>	8 (SGV 5)	5.0 x10 <sup>-4</sup>	12,000
Arsenic	39	2.4 x10 <sup>-4</sup>	20 (SGV 1)	1.2 x10 <sup>-5</sup>	500,000
Chromium	41	2.6 x10 <sup>-4</sup>	130 (SGV 4)	2.0 x10 <sup>-6</sup>	3,000,000
Copper	39	2.4 x10 <sup>-4</sup>	220 (SLR)	1.1 x10 <sup>-6</sup>	5,000,000
Lead	39	2.4 x10 <sup>-4</sup>	450 (SGV 10)	5.4 x10 <sup>-7</sup>	11,000,000
Nickel	41	2.5 x10 <sup>-4</sup>	50 (SGV 7)	5.1 x10 <sup>-6</sup>	1,200,000
Vanadium	41	2.5 x10 <sup>-4</sup>	250 (SLR)	1.0 x10 <sup>-6</sup>	5,900,000
Dioxins (total)	5.5 x10 <sup>-5</sup> µg TEQ/kg	4.2 x10 <sup>-10</sup> mg TEQ/kg	2.6 x10 <sup>-5</sup> (SLR)	1.6 x10 <sup>-5</sup>	380,000

All predicted soil concentrations for the metals are extremely low and for all metals except mercury are <1 µg/kg; these concentrations are considerably less than UK soil background concentrations (e.g. 10-30 mg/kg lead<sup>5</sup>, 10 mg/kg arsenic<sup>6</sup>, 0.12 mg/kg mercury<sup>7</sup>). 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 5.4 x10<sup>-7</sup> to 5.0 x10<sup>-4</sup>. These results indicate an absence of risk to human health from the deposition of metal contaminants and even based on the conservative assumptions in this risk assessment methodology it would be thousands of years before GAC were exceeded at the most impacted receptor location, much longer than the anticipated operational lifetime of the facility.

The dioxin concentration in soil at the receptor location with the highest deposition rate was calculated as 4.2 x10<sup>-4</sup> 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). The maximum estimated concentration of dioxin in soil resulting from the deposition of particulates in emissions from operation of the EfW facility is also negligible in comparison with the GAC derived for 2,3,7,8-TCDD (HQ = 1.6 x10<sup>-5</sup>) which indicates an absence of health risk from this class of contaminants also.

<sup>5</sup> Macloed et al (2006)

<sup>6</sup> Defra and Environment Agency (2002d)

<sup>7</sup> SSLRC (2000)

#### **4.0 CONCLUSIONS**

A conceptual site model was constructed for the Ardley EfW that identified the potential sources of pollution, receptors and relevant pathways of exposure. Exposure to persistent pollutants such as heavy metals and dioxins/furans was estimated from particle phase and vapour deposition to soil.

A conservative worst 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 persistent contaminants (i.e. metals and dioxins). Predicted soil concentrations were compared to generic assessment criteria generated by the CLEA UK model, used to assess human health risks deriving from contaminated land

Results from generic quantitative risk assessment indicate that indirect, long-term exposure to all persistent 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 (~2,000) between the highest predicted soil concentrations and soil assessment criteria that are protective of the most sensitive human receptors.

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# **APPENDIX A**

## **Technical Data for HHRA Generic Assessment Criteria**

## **VIRIDOR Ardley EfW**

### **APPENDIX A**

#### **Contaminants of Concern – Derivation of Generic Assessment Criteria**

Where a published Soil Guideline Value (SGV) is not available for the contaminants of concern SLR has derived de novo Generic Assessment Criteria (GACs) to assess the risk from deposited particulate material. GACs have been derived using the beta version of the CLEA UK model (released by the Environment Agency in November 2005).

SLR GACs have been generated following the approaches recommended in CLR reports 9 & 10 (EA/Defra, 2002a,b) and associated material (CLEA briefing notes and CLEA UK Handbook) and are therefore based on health criteria values (HCVs) selected following the hierarchy of preferred sources described in CLR 9, detailed below:

1. Recommendations by authoritative bodies in the UK, e.g. reviews by the Committees on Toxicity, Mutagenicity and Carcinogenicity of Chemicals in Food, Consumer Products and the Environment (COT, COM and COC)
2. Source documents produced by the European Commission's committees such as the Scientific Committee on Food (SCF) or Risk Assessment Reports (RARs) produced under Existing Substances Regulations
3. Guidelines such as tolerable daily intakes (TDIs) produced by international authoritative organisations such as the World Health Organisation's 'Guidelines for Drinking Water Quality'
4. Reports and guidelines prepared by other national organisations such as the USEPA's Integrated Risk Information System (IRIS)
5. Reports produced by authoritative organisations but for different purposes, e.g. oral TDIs or Index Doses may be derived using information obtained from air quality standards
6. Occupational exposure levels, considered on a case-by-case basis using expert judgement and appropriately extrapolated to protect the general public.

Physico-chemical input parameters for the CLEA UK model were selected from Environment Agency/Defra publications, where available, and authoritative data sources<sup>1</sup>.

Generation of GACs for individual contaminants of concern and selection of the source data input to the CLEA model are detailed below. CLEA UK record sheets detailing the model input and output for calculation of the GACs are presented in Appendix B.

<sup>1</sup> For example, CRC Handbook of Chemistry and Physics, IUPAC-NIST Solubility Series and US Environmental Protection Agency databases.

## 1.0 THALLIUM

Thallium is an acute poison that can be lethal at low doses with effects on the gastrointestinal tract, cardiovascular and nervous systems. Long-term low dose exposure leads to similar but milder symptoms. The reproductive system appears to be susceptible to the toxic effects of thallium. Human toxicological data following long term low-level exposure are scarce as there are very few data on the effects of chronic occupational exposure to thallium.

The selected HCV for oral exposure ( $TDI_{oral}$ ) is based on the USEPA oral reference dose (RfD) for thallium sulphate reported in IRIS<sup>2</sup>. The RfD is based on a 90-day study in rats which determined a no observed adverse effect level (NOAEL) of 0.25 mg/kg bw/day for increases in the incidence of alopecia, lacrimation and exophthalmos, together with increased serum aspartate aminotransferase, lactate dehydrogenase and sodium levels and decreased glucose levels. The RfD was derived by application of an uncertainty factor of 3000 (including 10 for subchronic to chronic extrapolation, 10 for intraspecies extrapolation, and 10 to account for species variability) plus a factor of 3 to account for lack of reproductive and chronic toxicity data. This gives an oral RfD of  $8 \times 10^{-5}$  mg/kg bw/day for thallium sulphate which is corrected to  $6.5 \times 10^{-5}$  mg/kg bw/day thallium as it assumed that thallium is responsible for the compound's observed toxicity.

The selected HCV for inhalation exposure ( $TDI_{inh}$ ) is based on 1% of the Workplace Exposure Limit (WEL) of 0.1 mg/m<sup>3</sup> detailed in the 2007 Edition of HSE's EH40 document<sup>3</sup>. A  $TDI_{inh}$  of  $3.0 \times 10^{-4}$  mg/kg bw/day is derived based on the assumption that a 70 kg adult inhales 20m<sup>3</sup> of air daily (Defra and Environment Agency, 2002a).

The main route of exposure to thallium is from food and an estimated mean daily intake (MDI) of thallium from food of 2 ug/day is reported in the HSDB profile of thallium compounds<sup>4</sup>. The same source reports an estimated daily thallium intake from air for a 70 kg adult of 3.4 ng.

HCVs, MDI and physico-chemical data input to the CLEA-UK model for thallium are detailed in Table 1 below with reference to the source of the data.

**Table 1**  
**CLEA UK input data – Thallium**

Parameter	Value	Reference
$TDI_{oral}$	0.065 µg/kg bw/d	USEPA IRIS
$TDI_{inh}$	0.3 µg/kg bw/d	1% WEL (EH40, HSE 2007)
$MDI_{oral}$	2 µg/d	HSDB
$MDI_{inh}$	$3.4 \times 10^{-3}$ µg/d	HSDB
Aqueous solubility	$3.9 \times 10^4$ mg/L	WHO EHC 182 <sup>5</sup>
Kd	19 L/kg	ATSDR, 1992 <sup>6</sup>

<sup>2</sup> IRIS *Integrated risk assessment system, thallium sulfate*, US Environmental Protection Agency. Available [June 2008] at <http://www.epa.gov/iris/subst/0111.htm>

<sup>3</sup> <http://www.hse.gov.uk/coshh/table1.pdf>

<sup>4</sup> Hazardous Substances Data Bank [Thallium compounds] available June 2008 at <http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB>

<sup>5</sup> IPCS (1996) Thallium. Environmental Health Criteria Document No 182. International Programme on Chemical Safety, WHO, Geneva.

<b>Parameter</b>	<b>Value</b>	<b>Reference</b>
Dermal absorption factor	0.1	CLEA Briefing Note 1 <sup>7</sup>
Soil:plant CF (leafy)	0.004	Baes et al. 1984 <sup>8</sup>
Soil:plant CF (root)	0.004	Baes et al. 1984

The CLEA UK model calculates a generic assessment criteria of 0.85 mg/kg thallium in soil for a 'residential with plant uptake' exposure scenario. CLEA record sheets for calculation of the thallium GAC are provided in Appendix B.

<sup>6</sup> Kd for clay mineral. ATSDR (1992) Toxicological profile for thallium. Agency for Toxic Substances and Disease Registry, Atlanta

<sup>7</sup> CLEA Briefing Note 1. Update on the Dermal Exposure Pathway. Environment Agency, March 2004

<sup>8</sup> Baes CF III, Sharp RD, Sjoreen AL & Shor RW (1984) A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture. ORNL-5786. Oak Ridge National Laboratory, Tennessee

## 2.0 COPPER

Copper is an essential element for humans and is considered to be only moderately toxic following oral exposure with the main effect being gastrointestinal disturbance. Copper may also induce allergic contact dermatitis in susceptible individuals following dermal contact.

The selected  $TDI_{oral}$  of 160  $\mu\text{g}/\text{kg}$  bw/d is based on the 'safe upper level for daily consumption over a lifetime' recommended by the FSA Expert Group on Vitamins and Minerals (EVM, 2003)<sup>9</sup>. This was based on a 13 week rat study which observed a NOAEL of 16 mg/kg bw/day for damage to the fore-stomach, kidney and liver. An overall uncertainty factor of 100 was applied (10 each for both inter- and intra-species variability). The EVM stated that this was consistent with small scale human studies where up to 10 mg/day were without adverse effects.

The  $TDI_{inh}$  is based on 1% of the Workplace Exposure Limit (WEL) of 1  $\text{mg}/\text{m}^3$  (8 hr TWA for copper dust) detailed in the 2007 Edition of EH40<sup>10</sup>. A  $TDI_{inh}$  of  $2.9 \times 10^{-3}$  mg/kg bw/day is derived based on the assumption that a 70 kg adult inhales  $20\text{m}^3$  of air daily.

$MDI_{oral}$  of 3.4 mg/day is based on average exposure from food (1.4 mg/day) and water (2 mg/day) described by the EVM and UK Drinking Water Inspectorate, respectively. An  $MDI_{inh}$  of 3.8  $\mu\text{g}/\text{d}$  is based on an average suburban air level of 190  $\text{ng}/\text{m}^3$  in the USA in the 1970s detailed in HSDB<sup>11</sup>, assuming that an adult inhales  $20\text{m}^3$  of air daily.

HCVs, MDI and physico-chemical data input to the CLEA-UK model for copper are detailed in Table 2 below with reference to the source of the data.

**Table 2**  
**CLEA UK input data – Copper**

Parameter	Value	Reference
$TDI_{oral}$	160 $\mu\text{g}/\text{kg}$ bw/d	EVM, 2003
$TDI_{inh}$	2.9 $\mu\text{g}/\text{kg}$ bw/d	1% WEL (EH40, HSE 2007)
$MDI_{oral}$	3400 $\mu\text{g}/\text{d}$	EVM (2003) & DWi (2007 <sup>12</sup> )
$MDI_{inh}$	3.8 $\mu\text{g}/\text{d}$	HSDB
Aqueous solubility	$1.27 \times 10^5$ mg/L	WHO <sup>13</sup> ( $\text{CuSO}_4$ solubility)
Kd	2120 L/kg	RIVM 711701023 <sup>14</sup>

<sup>9</sup> EVM (2003) Expert Group on Vitamins and Minerals, "Safe Upper Levels for Vitamins and Minerals", Food Standards Agency, May 2003, ISBN 1 904 026 11 7.

<sup>10</sup> <http://www.hse.gov.uk/coshh/table1.pdf>

<sup>11</sup> Hazardous Substances Data Bank [Copper compounds] available June 2008 at <http://toxnet.nlm.nih.gov/cgi-bin/sis/search/f?./temp/~aoLttv:2>

<sup>12</sup> DWI (2008) Drinking Water 2007, The Drinking Water Inspectorate 17th Annual Report, <http://www.dwi.gov.uk/pubs/annrep07/CIR07%20Drinking%20water%20in%20England%20and%20Wales.pdf>

<sup>13</sup> WHO Guidelines for Drinking Water Quality (2004) Background document for copper, [http://www.who.int/water\\_sanitation\\_health/dwq/chemicals/copper.pdf](http://www.who.int/water_sanitation_health/dwq/chemicals/copper.pdf)

<sup>14</sup> RIVM (2001) Technical Evaluation of the Intervention Values for Soil/Sediment and Groundwater. Netherlands National Institute of Public Health and the Environment. RIVM Report 711701 23

<b>Parameter</b>	<b>Value</b>	<b>Reference</b>
Dermal absorption factor	0.1	CLEA Briefing Note 1
Soil:plant CF (leafy)	0.4	Baes et al. 1984
Soil:plant CF (root)	0.4	Baes et al. 1984

The CLEA UK model calculates a generic assessment criteria of 220 mg/kg copper in soil for a 'residential with plant uptake' exposure scenario. CLEA record sheets for calculation of the copper GAC are provided in Appendix B.

### 3.0 VANADIUM

The key features of vanadium's hazard profile are respiratory toxicity following inhalation (in humans and animals) and bronchiolar/alveolar tumours following chronic exposure in rodents. Vanadium is less toxic by the oral route, with gastrointestinal disturbance reported in humans, and equivocal data on adverse effects on kidneys, spleen, lungs and blood pressure and reproductive/developmental toxicity in rats and mice. The evidence from the carcinogenicity and mutagenicity data suggests that if vanadium were regarded as a suspected human carcinogen, then it would be via a non-genotoxic mechanism and thus likely to have a threshold. It is therefore appropriate to set tolerable daily intakes (TDIs) for vanadium.

The selected  $TDI_{oral}$  for vanadium is based on the USEPA<sup>15</sup> RfD of 9 µg/kg bw/day for vanadium pentoxide (equivalent to 5 µg/kg bw/day vanadium). The RfD is based on the unpublished work of Stokinger *et al* (1953)<sup>16</sup> which established a NOAEL of 0.89 mg/kg bw/day for decreased hair cystine in rats. An uncertainty factor of 100 was applied (10 each for both intra- and inter-species variability) to derive the RfD of 9 µg/kg bw/day vanadium pentoxide. It should be noted that the USEPA has expressed low confidence in the RfD due to the lack of details in the reference study and the scarcity of data available on vanadium pentoxide. However, this is the only currently available guideline for long-term oral exposure to vanadium and is supported by the EFSA (2004)<sup>17</sup> observation that the lowest dose reported to cause adverse effects in humans is approximately 200 µg vanadium /kg bw/day.

The selected  $TDI_{inh}$  is derived from the air quality guideline of 1 µg/m<sup>3</sup> recommended by the WHO (2000)<sup>18</sup>. This air quality guideline is based upon consideration of long-term human occupational data that indicate a LOAEL of 20 µg/m<sup>3</sup> for chronic upper respiratory tract symptoms. As minimal adverse effects were observed on the upper respiratory tract at this level, a protection factor of 20 was chosen for environmental exposure. It was thought that exposure to vanadium at levels below 1 µg/m<sup>3</sup> was unlikely to have adverse health effects. Assuming that a 70 kg adult inhales about 20 m<sup>3</sup> of air daily, the WHO air quality guideline value equates to a  $TDI_{inh}$  of 0.3 µg/kg bw/day.

The EFSA (2004) stated that the intake of vanadium from normal food is estimated to be of the order of 10-20 µg/day and the upper value from this range (20 µg/day) is taken here as the mean daily intake from food in the UK. The IPCS (1988)<sup>19</sup> noted that vanadium concentrations in drinking water were generally less than 10 µg/L with an average of about 5 µg/L. An adult drinking 2 L daily would therefore ingest about 10

<sup>15</sup> USEPA (1996). Vanadium Pentoxide. United States Environmental Protection Agency. IRIS Database - Integrated Risk Information System. Viewed on-line at <http://www.epa.gov/iris/subst/0125.htm> [July 2008]

<sup>16</sup> Stokinger HE, Wagner WD, Mountain JT, Stocksill FR, Dobrogorski OJ and Keenan RG (1953). Unpublished results. Division of Occupational Health, Cincinnati, OH. (Cited in: Patty's Industrial Hygiene and Toxicology, 3rd ed., 1981, and described in USEPA, 1996).

<sup>17</sup> EFSA (2004) European Food Safety Authority. Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Vanadium. The EFSA Journal **33** 1-22

<sup>18</sup> WHO (2000). World Health Organization. Air quality guidelines for Europe, Second Edition. WHO Regional Publications, European Series No.91. Copenhagen: WHO Regional Office for Europe.

<sup>19</sup> IPCS (1988) International Programme on Chemical Safety. Vanadium, Environmental Health Criteria 81. World Health Organization, Geneva.

µg of vanadium via drinking water. The mean daily intake for an adult from food and water combined is therefore about 30 µg.

Recent air quality results for the UK indicate that values in central London have fallen from 20 ng/m<sup>3</sup> in 1980/81 to 4 ng/m<sup>3</sup> in 2002 (Defra, 2004)<sup>20</sup>; levels in other UK cities range from 1-3 ng/m<sup>3</sup>. A 70 kg adult inhaling 20 m<sup>3</sup> daily at the current level in central London would therefore have a daily inhalation intake of vanadium of about 80 ng.

HCVs, MDI and physico-chemical data input to the CLEA-UK model for vanadium are detailed in Table 3 below with references to the source of the data.

**Table 3**  
**CLEA UK input data – Vanadium**

<b>Parameter</b>	<b>Value</b>	<b>Reference</b>
TDI <sub>oral</sub>	5 µg/kg bw/d	USEPA IRIS
TDI <sub>inh</sub>	0.29 µg/kg bw/d	WHO (2000)
MDI <sub>oral</sub>	30 µg/d	EFSA (2004) & IPCS (1988)
MDI <sub>inh</sub>	0.08 µg/d	Defra (2004)
Aqueous solubility	8.82 x10 <sup>5</sup> mg/L	IPCS CICAD <sup>21</sup>
Kd	1000 L/kg	USEPA EcoSSL <sup>22</sup>
Dermal absorption factor	0.1	CLEA Briefing Note 1
Soil:plant CF (leafy)	0.00485	USEPA EcoSSL
Soil:plant CF (root)	0.00485	USEPA EcoSSL

The CLEA UK model calculates a generic assessment criteria of 250 mg/kg vanadium in soil for a 'residential with plant uptake' exposure scenario. CLEA record sheets for calculation of the vanadium GAC are provided in Appendix B.

<sup>20</sup> Defra (2004). Department for Environment, Food and Rural Affairs. E-Digest of Environmental Statistics. Available on-line at <http://www.defra.gov.uk/environment/statistics/index.htm>, September 2004.

<sup>21</sup> IPCS (2001) Vanadium Pentoxide and Other Inorganic Vanadium Compounds (Concise International Chemical Assessment Document 29), World Health Organisation, Geneva, Switzerland

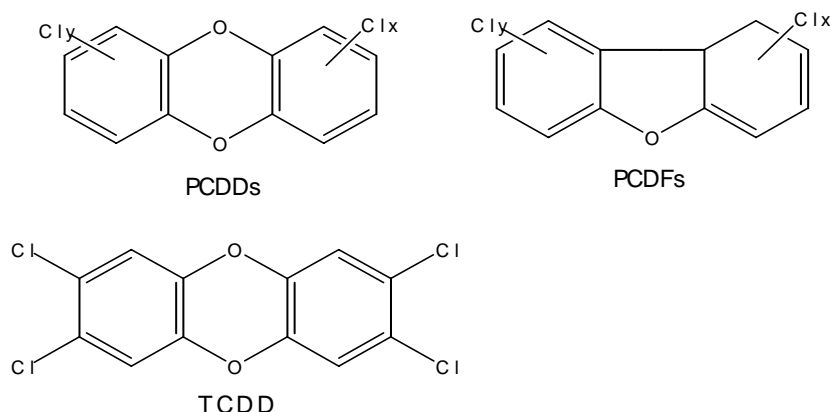
<sup>22</sup> USEPA (2007) Guidance for Developing Ecological Soil Screening Levels (Eco-SSLs), Attachment 4-1. Viewed at [http://www.epa.gov/ecotox/ecossl/pdf/ecossl\\_attachment\\_4-1.pdf](http://www.epa.gov/ecotox/ecossl/pdf/ecossl_attachment_4-1.pdf) July 2008

## 4.0 DIOXINS

Dioxins are chlorinated compounds generated as by-products of waste combustion and as trace contaminants during the synthesis of many organochlorine compounds. The compounds referred to here as dioxins are restricted to those having a structure and toxicity related to that of the parent compound, 2,3,7,8-tetrachloro-para-dibenzodioxin (TCDD), which is commonly known as dioxin. Polychloro-p-dibenzodioxins (PCDDs), such as TCDD, have the structure of two oxygen atoms combined with two chlorobenzene rings or a chlorobenzene and a benzene ring. Polychlorodibenzofurans (PCDFs, commonly known as furans) are related compounds and have one oxygen atom combined with two benzene rings. The presence of four chlorine atoms at positions, 2,3,7 and 8 appears to be responsible for the observed dioxin-like toxicity.

The structural formula of TCDD and representative structures of PCDDs and PCDFs are shown in Figure 1.

**Figure 1. Representative structures of PCDDs and PCDFs and the structural formula of TCDD.**



Dioxins have been demonstrated to accumulate and be very persistent in the human body, with measured half-lives of up to 11 years. Extensive research has demonstrated that dioxin and dioxin-like compounds can cause a diverse array of toxic effects in animal systems, including disruption of the reproductive and immune systems, developmental effects such as birth defects and altered sexual development and cancer. Although many of these effects have not been seen in humans, it appears that dioxins do increase the risk of cancer in exposed individuals, but much remains unknown about the health effects of these compounds.

### *The TEF approach*

Studies in animals have indicated that the effects of dioxin and dioxin-like compounds are mediated by a specific cytoplasmic receptor protein, the Ah receptor. The 2,3,7,8-TCDD or dioxin molecule has the greatest affinity for the Ah receptor and is therefore considered to be the most potent synthetic Ah receptor ligand. All other dioxin-like compounds with effects mediated by the same receptor protein, albeit with lower potency, are considered to be functionally equivalent to dioxin hence the term 'dioxin-like'; their potencies relative to TCDD can be characterised by Toxic Equivalency Factors (TEFs).

The TEF values calculated for a single congener can vary by several orders of magnitude depending on species, tissue, and the model chosen. Nevertheless, because of the potential value of the TEF approach in simplifying calculations of the risk associated with measured concentrations of these compounds in the environment and in human tissue, 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 PCDD, PCDF and PCB congeners (Van den Berg *et al.*, 2006)<sup>23</sup>. These TEFs have been endorsed by most international bodies concerned with environmental health and safety and are recommended for use by the UK Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT). 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 4 below.

**Table 4**  
**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

The published Defra/EA toxicological report for contaminated land assessment (TOX 12, Defra/EA 2003)<sup>24</sup> contains recommendations for oral daily intake of dioxins and

<sup>23</sup> Van den Berg M et al (2006) The 2005 World Health Organization Re-evaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-like Compounds. Toxicological Sciences Advance Access, July 2006.

<sup>24</sup> Defra and Environment Agency (2003) Contaminants in Soil: Collation of Toxicological Data and Intake Values for Humans. Dioxins, Furans and Dioxin-like PCBs. R&D Publication TOX12

dioxin-like compounds and these are:  $TDI_{oral}$  2  $\mu\text{g}/\text{kg}$  bw/day;  $MDI_{oral}$  126  $\mu\text{g}/\text{day}$ . Intake via inhalation is assumed to be negligible in comparison to oral intake and no expert group has derived an inhalation or dermal TDI for dioxins or dioxin-like compounds.

HCVs, MDI and physico-chemical data input to the CLEA-UK model for derivation of a GAC for 2,3,7,8-TCDD (TEF = 1 and therefore applicable to overall dioxin TEQ concentrations) are detailed in Table 5 below with reference to the source of the data.

**Table 5**  
**CLEA UK input data – 2,3,7,8 TCDD**

Parameter	Value	Reference
$TDI_{oral}$	$2.0 \times 10^{-6} \mu\text{g}/\text{kg}$ bw/d	TOX 12 (Defra/EA, 2003)
$TDI_{inh}$	-	
$MDI_{oral}$	$1.26 \times 10^{-4} \mu\text{g}/\text{d}$	TOX 12 (Defra/EA, 2003)
$MDI_{inh}$	-	
Henry's Law constant	$1.35 \times 10^{-3}$	Lancaster Uni Database <sup>25</sup>
Air diffusion coefficient	$1.0 \times 10^{-6} \text{m}^2/\text{s}$	Generic low value
Water diffusion coefficient	$1.0 \times 10^{-10} \text{m}^2/\text{s}$	Generic low value
Vapour pressure	$2.5 \times 10^{-7} \text{Pa}$	Lancaster Uni Database
Aqueous solubility	$2.0 \times 10^{-4} \text{mg}/\text{L}$	SRC PhysProp Database <sup>26</sup>
Koc	6.66	Lancaster Uni Database
Kd	6.8	Lancaster Uni Database
Relative molecular mass	322	SRC PhysProp Database
Dermal absorption factor	0.1	CLEA Briefing Note 1
Soil:plant CF (leafy)	$2.7 \times 10^{-3}$	PCDD/Fs, Rideout & Teschke (2004) <sup>27</sup>
Soil:plant CF (root)	$4.2 \times 10^{-3}$	PCDD/Fs, Rideout & Teschke (2004)

The CLEA UK model calculates a generic assessment criteria of  $2.6 \times 10^{-5} \text{mg}/\text{kg}$  dioxin TEQ in soil for a 'residential with plant uptake' exposure scenario. CLEA record sheets for calculation of the dioxin GAC are provided in Appendix B.

<sup>25</sup> Lancaster University Research Database. Polychlorinated Dibenzo Dioxins and Furans (PCDD/Fs). Viewed online at <http://www.lec.lancs.ac.uk/ccm/research/database/4.html> July 2008

<sup>26</sup> SRC PhysProp Database[2,3,7,8-Tetrachlorodibenzo-p-dioxin] Viewed online at <http://esc.syrres.com/interkow/webprop.exe?CAS=1746-01-6> July 2008

<sup>27</sup> Rideout K & Teschke K (2004) Potential for increased human food-borne exposure to PCDD/F when recycling sewage sludge on agricultural land. Environmental Health Perspectives 2004;112:959-969

# **APPENDIX B**

## **CLEA UK Record Sheets**

**CLEA UK MODEL 2005 VERSION Version 1.0**

<b>Simulation Date:</b>	05/08/2008
<b>Type of simulation:</b>	Generic

<b>Company Name:</b>	SLR Consulting
<b>Person running Simulation:</b>	ES
<b>Contact Number:</b>	01484 860521
<b>Site Name:</b>	HIA GACs
<b>Site Address:</b>	Generic

Chemical	HCV <sub>oral</sub> compared with which exposure route/s?			HCV <sub>inhal</sub> compared with which exposure route/s?			Assessment Criteria (mg.kg <sup>-1</sup> )					Site Specific Soil Concentration (mg.kg <sup>-1</sup> dry weight soil)	ADE/HCV (dimensionless)	
	oral	dermal	inhal	oral	dermal	inhal	oral & dermal (using HCV <sub>oral</sub> )	20% rule applied?	inhalation (using HCV <sub>inhalation</sub> )	20% rule applied?	integrated		oral & dermal	inhalation
2,3,7,8-TCDD	Yes	Yes	No	No	No	No	2.62E-05	Yes		Yes			1.00E+00	
Copper_SLR	Yes	Yes	No	No	No	Yes	2.16E+02	Yes	4.57E+05	Yes	2.16E+02		1.00E+00	1.00E+00
Thallium_SLR	Yes	Yes	No	No	No	Yes	8.51E-01	Yes	2.83E+05	Yes	8.51E-01		1.00E+00	1.00E+00
Vanadium_SLR	Yes	Yes	No	No	No	Yes	2.56E+02	No	2.27E+05	No	2.56E+02		1.00E+00	1.00E+00

**Land-use selected:** residential with plant uptake

Land-use Parameters		Age Class																	
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
Exposure Frequency (day.yr <sup>-1</sup> )	direct soil and dust ingestion	1.80E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02												
	consumption of home grown produce	2.50E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02												
	consumption of soil attached to home grown produce	2.50E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02												
	skin contact, indoors	1.80E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02												
	skin contact, outdoors	6.50E+01	1.30E+02	1.30E+02	1.30E+02	1.30E+02	1.30E+02												
	inhalation of dust and vapours, indoors	3.65E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02												
	inhalation of dust and vapours, outdoors	3.65E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02	3.65E+02												
Respiration Frequency (hr.day <sup>-1</sup> )	active, indoors	2.00E+00	3.00E+00	3.00E+00	3.00E+00	3.00E+00	3.00E+00												
	active, outdoors	1.00E+00	2.00E+00	2.00E+00	3.00E+00	3.00E+00	2.00E+00												
	passive, indoors	2.00E+01	1.80E+01	1.80E+01	1.80E+01	1.80E+01	1.60E+01												
	passive, outdoors	1.00E+00	1.00E+00	1.00E+00	0.00E+00	0.00E+00	0.00E+00												
Soil Ingestion Rate (mg.day <sup>-1</sup> )	soil ingestion rate																		
		Calculated	Calculated	Calculated	Calculated	Calculated	Calculated												
Soil-skin adherence factor (mg.cm <sup>-2</sup> )	indoor	6.00E-02	6.00E-02	6.00E-02	6.00E-02	6.00E-02	6.00E-02												
	outdoor	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00	1.00E+00												
Exposed skin fraction (dimensionless)	indoor maximum exposed skin fraction	3.20E-01	3.30E-01	3.20E-01	3.50E-01	3.50E-01	3.30E-01												
	outdoor maximum exposed skin fraction	2.60E-01	2.60E-01	2.50E-01	2.80E-01	2.80E-01	2.60E-01												

**Receptor selected:** female (UK)

Start Age Class	End Age Class	Exposure duration (years)	Averaging Time (years)
1	6	6	6

## BUILDING PARAMETERS

Building type selected:		Residential - typical house
Building Parameters	Units	Input Value
height of living/working space above ground	cm	4.80E+02
height of cellar space below ground	cm	0.00E+00
enclosed space floor length	cm	6.40E+02
enclosed space floor width	cm	6.40E+02
foundation or slab thickness	cm	1.50E+01
living/working space air exchange rate	hr <sup>-1</sup>	5.00E-01
pressure differential between soil and enclosed space	g.cm <sup>-2</sup>	3.00E+01
floor-wall seam crack width	cm	2.00E-01
fixed crack to total area ratio	unitless	1.25E-03
volumetric flow rate of soil gas entering the building	cm <sup>3</sup> .s <sup>-1</sup>	3.95E+01
volumetric building ventilation rate from indoor to outdoor air	cm <sup>3</sup> .s <sup>-1</sup>	2.73E+04

## SOIL CHARACTERISTICS

Soil type selected:		sandy - UK
Soil parameter	Units	Input value
grainsize	cm	5.00E-02
total porosity	cm <sup>3</sup> cm <sup>-3</sup>	4.60E-01
air-filled porosity	cm <sup>3</sup> cm <sup>-3</sup>	3.10E-01
water-filled porosity	cm <sup>3</sup> cm <sup>-3</sup>	1.50E-01
dry bulk density	g cm <sup>-3</sup>	1.60E+00
enrichment factor	dimensionless	6.00E+00
soil pH	dimensionless	7.00E+00
fraction of organic carbon	dimensionless	5.80E-03
van Genuchten shape parameter	dimensionless	3.47E-01
residual water content	cm <sup>3</sup> cm <sup>-3</sup>	3.00E-02
saturated hydraulic conductivity	cm.s <sup>-1</sup>	6.47E-03
ambient soil/water temperature	K	2.83E+02
intrinsic soil permeability	cm <sup>2</sup>	8.63E-08

## SITE PARAMETERS

Site Parameter	Units	Input Value
Area of source-zone	cm <sup>2</sup>	2.25E+06
Depth below ground to source zone	cm	1.15E+02
Equivalent threshold wind speed (7m)	m.s <sup>-1</sup>	1.13E+01
Fraction of soil in building dust	dimensionless	7.50E-01
Fraction of the site with hard or vegetative cover	dimensionless	5.00E-01
Mean annual windspeed (10m)	m.s <sup>-1</sup>	4.69E+00
Normalised annual average concentration of dust particles	kg.m <sup>-3</sup> per g.m <sup>-2</sup> .s <sup>-1</sup>	1.10E-02
Tracked back soil adjustment factor	dimensionless	1.00E+00
Width of contaminated zone in direction of prevailing wind	cm	1.50E+03
Wind speed distribution function	dimensionless	1.94E-01
Wind speed in mixing zone (1-2m)	m.s <sup>-1</sup>	3.00E+00

Exposure Pathways

Chemical	Oral						Dermal						Inhalation										background exposure (oral)	background exposure (inhalation)
	direct soil and soil derived indoor dust ingestion		consumption of site-grown vegetables		consumption of soil attached to site-grown vegetables		skin contact with soil-derived indoor dust		skin contact with soil		inhalation of soil derived indoor dust		inhalation of soil dust		inhalation of vapours indoors		inhalation of vapour's outdoors		vapour model calculated values		dispersion factor for ambient air		ADE Mean (ug.kg-1 bw.day-1)	ADE Mean (ug.kg-1 bw.day-1)
	ADE % Contribution	ADE Mean (ug.kg-1 bw.day-1)	ADE % Contribution	ADE Mean (ug.kg-1 bw.day-1)	ADE % Contribution	ADE Mean (ug.kg-1 bw.day-1)	ADE % Contribution	ADE Mean (ug.kg-1 bw.day-1)	ADE % Contribution	ADE Mean (ug.kg-1 bw.day-1)	ADE % Contribution	ADE Mean (ug.kg-1 bw.day-1)	ADE % Contribution	ADE Mean (ug.kg-1 bw.day-1)	ADE % Contribution	ADE Mean (ug.kg-1 bw.day-1)	ADE % Contribution	ADE Mean (ug.kg-1 bw.day-1)	soil air diffusion coefficient (cm <sup>2</sup> .s <sup>-1</sup> )	soil-vapour to indoor air attenuation coefficient (dimensionless)	young child (cm.s-1)	older child (cm.s-1)	adult (cm.s-1)	ADE Mean (ug.kg-1 bw.day-1)
2,3,7,8-TCDD	7.05E+01	1.76E-07	8.51E+00	2.13E-08	5.91E+00	1.48E-08	2.07E+00	5.18E-09	1.30E+01	3.26E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	9.63E-04	1.31E-04	8.10E+00	N/A	N/A	0.00E+00	0.00E+00
Copper_SLR	7.40E+00	1.45E+00	9.04E+01	1.77E+01	6.20E-01	1.22E-01	2.18E-01	4.27E-02	1.37E+00	2.68E-01	1.04E-03	2.05E-04	3.53E-04	6.92E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.10E+00	N/A	N/A	0.00E+00	0.00E+00
Thallium_SLR	7.04E+01	5.73E-03	8.60E+00	6.99E-04	5.90E+00	4.80E-04	2.07E+00	1.68E-04	1.30E+01	1.06E-03	1.66E-03	1.35E-07	5.60E-04	4.55E-08	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.10E+00	N/A	N/A	0.00E+00	0.00E+00
Vanadium_SLR	6.91E+01	1.72E+00	1.02E+01	2.55E-01	5.79E+00	1.44E-01	2.03E+00	5.06E-02	1.28E+01	3.18E-01	9.75E-03	2.43E-04	3.30E-03	8.20E-05	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.10E+00	N/A	N/A	1.04E+00	1.99E-03

## HEALTH CRITERIA VALUES

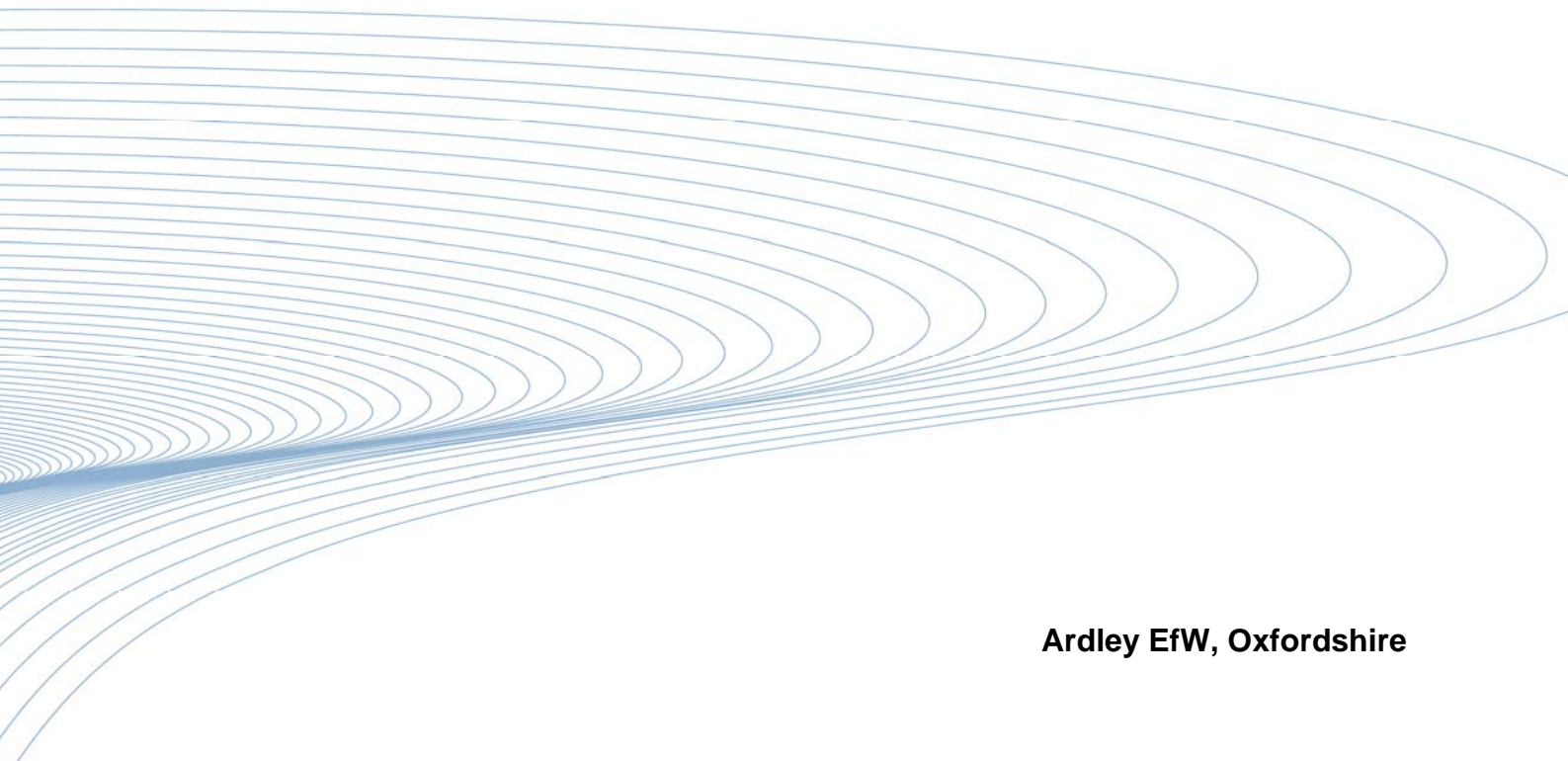
Chemical	TDI (ug.kg-1 bw.day-1)		ID (ug.kg-1 bw.day-1)		MDI (µg day-1)	
	oral	inhalation	oral	inhalation	oral	inhalation
2,3,7,8-TCDD	4.00E-07	2.00E-01	none	none	0.00E+00	0.00E+00
Copper_SLR	3.20E+01	5.80E-01	none	none	0.00E+00	0.00E+00
Thallium_SLR	1.30E-02	6.00E-02	none	none	0.00E+00	0.00E+00
Vanadium_SLR	5.00E+00	2.90E-01	none	none	3.00E+01	8.00E-02

**PHYSICO-CHEMICAL PROPERTIES**

Chemical	Henry's Law Constant (dimensionless)	Reference Temperature (K)	Henry's Law Constant (atm.m <sup>3</sup> .mol <sup>-1</sup> )	Reference Temperature (K)	Henry's Law Constant at ambient temperature (dimensionless)	Enthalpy of vaporisation at boiling point (cal.mol <sup>-1</sup> )	Boiling Point (K)	Critical Temperature (K)	Air diffusion coefficient (m <sup>2</sup> .s <sup>-1</sup> )	Water diffusion coefficient (m <sup>2</sup> .s <sup>-1</sup> )	Relative atomic (molecular) mass (g - mol)	Vapour pressure at 10°C (Pa)	Aqueous solubility at 10°C (mg.L <sup>-1</sup> )	organic carbon - water distribution coefficient, log K <sub>oc</sub> (log[cm <sup>3</sup> .water.g <sup>-1</sup> .oc])	octanol-water distribution coefficient log <sub>10</sub> log K <sub>ow</sub> (dimensionless)	soil-water distribution coefficient, K <sub>d</sub> (cm <sup>3</sup> . water.g <sup>-1</sup> - soil)	Total soil concentration to pore water concentration ratio (cm <sup>3</sup> .water.g-soil)	dermal absorption fraction (dimensionless)	Type of soil-to-plant concentration factor, dry or fresh weight? (leafy) (dimensionless)	Type of soil-to-plant concentration factor, dry or fresh weight? (root) (dimensionless)	soil-to-plant concentration factor (leafy) (ug.g <sup>-1</sup> FW plant over ug.cm <sup>-3</sup> soil solution)	soil-to-plant concentration factor (root) (ug.g <sup>-1</sup> FW plant over ug.cm <sup>-3</sup> soil solution)	dust Enrichment Factors used?
2,3,7,8-TCDD	3.34E+00	2.98E+02	3.34E+00	2.98E+02	1.35E-03	N/A	7.20E+02	N/A	1.00E-06	1.00E-10	3.22E+02	2.50E-07	2.00E-04	6.66E+00	6.80E+00	2.65E+04	2.65E+04	1.00E-01	dw	dw	2.70E-03	4.20E-03	Yes
Copper_SLR	N/A	N/A	N/A	N/A	0.00E+00	N/A	N/A	N/A	N/A	N/A	N/A	N/A	1.27E+05	N/A	N/A	2.12E+03	2.12E+03	1.00E-01	dw	dw	4.00E-01	4.00E-01	Yes
Thallium_SLR	N/A	N/A	N/A	N/A	0.00E+00	N/A	N/A	N/A	N/A	N/A	N/A	N/A	3.94E+04	N/A	N/A	1.90E+01	1.90E+01	1.00E-01	dw	dw	4.00E-03	4.00E-03	No
Vanadium_SLR	N/A	N/A	N/A	N/A	0.00E+00	N/A	N/A	N/A	N/A	N/A	N/A	N/A	8.82E+04	N/A	N/A	1.00E+03	1.00E+03	1.00E-01	dw	dw	4.85E-03	4.85E-03	Yes

**MEDIA CONCENTRATIONS**

Chemical	Distribution of chemical in soil						Distribution in Non-soil Media													Boundary Limits					
	mg.g <sup>-1</sup>			%			mg.m <sup>-3</sup> - air						mg.g <sup>-1</sup> FW plant matter							mg.L <sup>-1</sup>	mg.L <sup>-1</sup>	% max	mg.m <sup>-3</sup>	mg.m <sup>-3</sup>	% max
	total soil concentration	soil sorbed concentration	dissolved concentration	vapour concentration	soil sorbed concentration	dissolved concentration	vapour concentration	indoor vapour air concentration	outdoor vapour air concentration for Young Child	outdoor vapour air concentration for Older Child	outdoor vapour air concentration for Adults	outdoor vapour air concentration	outdoor dust air concentration	indoor dust air concentration	concentration in Brussels sprouts	concentration in cabbage	concentration in carrot	concentration in leafy salad	concentration in onion, leek, shallots	concentration in potatoes	pore water concentration		maximum aqueous solubility	ambient source vapour concentration	
2,3,7,8-TCDD	2.62E-08	2.62E-08	9.26E-14	2.58E-16	1.00E+02	3.54E-04	9.87E-07	1.75E-13	1.23E-15	N/A	N/A	1.99E-14	1.49E-14	6.72E-12	8.91E-12	1.07E-11	2.83E-12	1.72E-11	2.31E-11	9.88E-10	2.00E-04	4.94E-04	1.33E-09	3.42E-05	3.90E-03
Copper_SLR	2.16E-01	2.16E-01	9.54E-06	0.00E+00	1.00E+02	4.42E-03	0.00E+00	0.00E+00	0.00E+00	N/A	N/A	1.64E-07	1.23E-07	8.19E-03	1.09E-02	8.37E-03	3.45E-03	1.35E-02	1.81E-02	1.02E-01	1.27E+05	8.01E-05	0.00E+00	0.00E+00	N/A
Thallium_SLR	8.51E-04	8.51E-04	4.20E-06	0.00E+00	1.00E+02	4.93E-01	0.00E+00	0.00E+00	0.00E+00	N/A	N/A	6.46E-10	4.84E-10	3.23E-07	4.29E-07	3.30E-07	1.36E-07	5.31E-07	7.15E-07	4.48E-02	3.94E+04	1.14E-04	0.00E+00	0.00E+00	N/A
Vanadium_SLR	2.56E-01	2.56E-01	2.40E-05	0.00E+00	1.00E+02	9.38E-03	0.00E+00	0.00E+00	0.00E+00	N/A	N/A	1.94E-07	1.45E-07	1.18E-04	1.56E-04	1.20E-04	4.96E-05	1.93E-04	2.60E-04	2.56E-01	8.82E+04	2.90E-04	0.00E+00	0.00E+00	N/A



**Ardley EfW, Oxfordshire**

**Addendum Detailed Quantitative Risk Assessment - Dioxins**

**Viridor**

**February 2010**

**SLR Ref: 402.0036.00449**



solutions for today's environment

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## APPENDICES

Appendix A Dioxin Worksheets

## **1.0 INTRODUCTION**

### **1.1 Terms of Reference**

This report comprises an addendum to SLR's Human Health Risk Assessment report (Ref: 409-0036-00349) of October 2008. The latter was originally submitted in support of a Planning Application for an Energy from Waste (EfW) facility at Ardley, Oxfordshire and subsequently in support of an Environmental Permit (EP) application for the same scheme.

Following the above a Schedule 5 Notice was received in relation to the Application. Question 13 (2.3) of this Notice commented that the application did not contain a full detailed human health risk assessment and that some exposure pathways that could be of significance were not included. Following correspondence with the Environment Agency it was determined that further assessment in relation to dioxins was required.

### **1.2 Health Impact Assessment**

This report presents an addendum Detailed Quantitative Risk Assessment (DQRA) in relation to the potential impact of dioxins on local human receptor groups. The report should be read in conjunction with SLR's report of October 2008 since the level of Generic Quantitative Risk Assessment (GQRA) presented in this report with respect to other persistent pollutants (i.e. heavy metals) is considered appropriate.

## 2.0 METHODOLOGY

The principles of Health Impact Assessment have largely been developed from Environmental Impact Assessment<sup>1</sup>. The Human Health Risk Assessment of the Ardley 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.

The 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.

Tiers 1 and 2 of this process were presented in SLR's HHRA report of October 2008. The following sections of this report present a Tier 3 DQRA.

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.

DQRA of dioxin emissions follows HMIP methodology presented in the 'Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes' (HMIP, 1996). This methodology has been selected above others on the basis that it is the only one consistent with current good practice for HHRA in the UK.

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 transport and intake by human receptors. HMIP exposure assessment methodology has been supplemented by use of the Environment Agency's CLEA model 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.

<sup>1</sup> 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

The overall risk posed by dioxin emissions from the proposed Ardley EfW plant has been assessed by comparison of 'worst case scenario' intake to recommended health criteria values.

### **3.0 DETAILED QUANTITATIVE RISK ASSESSMENT (DQRA)**

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

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

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).

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 Biomass 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.*

##### **3.1.1 Deposition of Emissions**

The HMIP approach for deposition 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.

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.

##### **3.1.2 Calculation of Media Concentrations**

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 physio-chemical processes, as defined by the soil loss constant detailed in HMIP Annex C2.

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.

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).

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 Biomass emissions.

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 proposed EfW plant and exposure via other pathways arising from surface water are insignificant compared to exposure from soil and diet.

### **3.1.3 Calculation of Human Intake**

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.

Intake resulting from exposure to dioxins in soil via direct ingestion, dermal contact and consumption of home-grown produce is estimated using the Environment Agency's CLEA model. 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.

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).

### **3.1.4 Effect on Human Health**

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.

The WHO European Centre for Environment and Health and the International Program on Chemical Safety has 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 3-1 below.

**Table 3-1  
WHO TEFs for Dioxins (Van den Berg et al, 2006)**

	<b>Congener</b>	<b>WHO TEF</b>
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

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.

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.

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.

### 3.2 Dispersion Modelling

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/m<sup>3</sup>) of each congener at the location of greatest impact are presented below along with data for the average and minimum predicted impacts.

**Table 3-2  
Predicted Congener Ground Level Concentrations (fg/m<sup>3</sup>)**

<b>Compound</b>	<b>Average Predicted Impact</b>	<b>Minimum Predicted Impact</b>	<b>Maximum Predicted Impact</b>
2,3,7,8 –TCDD	0.0042	0.0005	0.0243
1,2,3,7,8–PeCDD	0.0133	0.0015	0.0778
1,2,3,4,7,8–Hx CDD	0.0133	0.0015	0.0778
1,2,3,6,7,8– HxCDD	0.0308	0.0035	0.1798
1,2,3,7,8,9– HxCCD	0.0266	0.0030	0.1555
1,2,3,4,6,7,8–Hp	0.1823	0.0208	1.0644
OCCD	0.2871	0.0327	1.6767
2,3,7,8-TCDF	0.0599	0.0068	0.3499
1,2,3,7,8-PeCDF	0.0416	0.0047	0.2430
2,3,4,7,8-PeCDF	0.0574	0.0066	0.3354
1,2,3,4,7,8- HxCDF	0.0682	0.0078	0.3985
1,2,3,6,7,8- HxCDF	0.0491	0.0056	0.2867
1,2,3,7,8,9- HxCDF	0.0108	0.0012	0.0632
2,3,4,6,7,8- HxCDF	0.0549	0.0063	0.3208
1,2,3,4,6,7,8-Hp	0.1298	0.0148	0.7582
1,2,3,4,7,8,9-Hp	0.0200	0.0023	0.1166
OCDF	0.0749	0.0085	0.4374
<b>TOTAL</b>	<b>1.1243</b>	<b>0.1282</b>	<b>6.5660</b>

From these data it is evident that the peak predicted ground level impact is about six 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.

### 3.3 Results of Transport (Soil and Biota) Modelling

#### 3.3.1 Predicted soil concentration

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. Met office data (1971 – 2000 averages) was used for factors such as average rainfall and temperature etc.

**Table 3-3  
Predicted Congener Soil Concentrations**

Compound	Airborne conc. (fg/m <sup>3</sup> )	Total deposition (pg/m <sup>2</sup> /a)	Soil loss constant (a <sup>-1</sup> )	Soil conc. – 10cm depth (pg/kg)	Soil conc. – 10cm depth (ng TEQ/kg)
2,3,7,8 –TCCD	0.0243	0.9	0.21	0.080	8.0 x10 <sup>-5</sup>
1,2,3,7,8–PeCDD	0.0778	4.1	0.10	0.087	8.7 x10 <sup>-5</sup>
1,2,3,4,7,8–HxCDD	0.0778	4.5	0.07	0.051	5.1 x10 <sup>-6</sup>
1,2,3,6,7,8–HxCDD	0.1798	10.5	0.07	0.117	1.2 x10 <sup>-5</sup>
1,2,3,7,8,9–HxCDD	0.1555	9.1	0.07	0.101	1.0 x10 <sup>-5</sup>
1,2,3,4,6,7,8–Hp	1.0644	64	0.06	0.566	5.7 x10 <sup>-6</sup>
OCCD	1.6767	101	0.06	0.849	2.5 x10 <sup>-7</sup>
2,3,7,8-TCDF	0.3499	14.8	0.33	1.648	1.6 x10 <sup>-4</sup>
1,2,3,7,8-PeCDF	0.2430	13.9	0.15	0.353	1.1 x10 <sup>-5</sup>
2,3,4,7,8-PeCDF	0.3354	19.1	0.15	0.488	1.5 x10 <sup>-4</sup>
1,2,3,4,7,8-HxCDF	0.3985	24	0.09	0.311	3.1 x10 <sup>-5</sup>
1,2,3,6,7,8-HxCDF	0.2867	17.1	0.09	0.223	2.2 x10 <sup>-5</sup>
1,2,3,7,8,9-HxCDF	0.0632	3.8	0.07	0.038	3.8 x10 <sup>-6</sup>
2,3,4,6,7,8-HxCDF	0.3208	19	0.09	0.250	2.5 x10 <sup>-5</sup>
1,2,3,4,6,7,8-Hp	0.7582	46	0.06	0.435	4.4 x10 <sup>-6</sup>
1,2,3,4,7,8,9-Hp	0.1166	7.0	0.06	0.067	6.7 x10 <sup>-7</sup>

Compound	Airborne conc. (fg/m <sup>3</sup> )	Total deposition (pg/m <sup>2</sup> /a)	Soil loss constant (a <sup>-1</sup> )	Soil conc. – 10cm depth (pg/kg)	Soil conc. – 10cm depth (ng TEQ/kg)
OCDF	0.4374	26	0.06	0.228	6.8 x10 <sup>-8</sup>

The total dioxin concentration in soil at the receptor location with the highest deposition rate was calculated as 6.1 x10<sup>-4</sup> 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).

### 3.3.2 Predicted plant concentrations

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 3-4 below.

**Table 3-4  
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	5.1	10.1	5.7	5.1	7.2	7.5	5.1	19.0
1,2,3,7,8– PeCDD	10.14	51.0	14.4	10.14	27.3	30.0	10.14	125.2
1,2,3,4,7,8– HxCDD	9.8	80	17	9.8	39	44	9.8	207
1,2,3,6,7,8– HxCDD	23	205	42	23	99	111	23	537
1,2,3,7,8,9– HxCDD	19	178	36	19	86	96	19	465
1,2,3,4,6,7,8– Hp	25	1450	175	25	623	718	25	4037
OCCD	229	3465	568	229	1587	1801	229	9340
2,3,7,8-TCDF	86	258	104	86	158	169	86	571
1,2,3,7,8- PeCDF	6.7	291	36.4	6.7	126	145	6.7	805.9
2,3,4,7,8- PeCDF	9.2	341	44.0	9.2	148	170	9.2	943
1,2,3,4,7,8- HxCDF	4.5	704	78	4.5	298	344	4.5	1972
1,2,3,6,7,8- HxCDF	3.2	519	57.4	3.2	220	254	3.2	1456
1,2,3,7,8,9-	0.64	113.2	12.4	0.64	48	55	0.64	318

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)
HxCDF								
2,3,4,6,7,8- HxCDF	3.6	581	64	3.6	246	284	3.6	1629
1,2,3,4,6,7,8- Hp	3.4	1527	163	3.4	643	743	3.4	4292
1,2,3,4,7,8,9- Hp	0.52	248	26.5	0.52	104	120.8	0.52	697
OCDF	0.82	941	99	0.82	395	458	0.82	2648

The highest concentrations of dioxins were generally 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.

### 3.3.3 Predicted animal tissue concentrations

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 3-5 below.

**Table 3-5  
Predicted Animal Tissue Concentrations (pg/kg)**

Compound	Beef (pg/kg)	Offal (pg/kg)	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-TCCD	0.18	0.36	0.41	0.27	0.38	1.19	0.19	1.40	0.36	1.51
1,2,3,7,8-Pe	0.40	0.48	0.35	0.23	0.15	0.97	0.55	4.09	0.83	3.43
1,2,3,4,7,8-Hx	0.36	0.63	0.14	0.12	0.12	0.37	0.40	2.96	0.75	3.10
1,2,3,6,7,8-Hx	0.93	1.63	0.33	0.31	0.30	0.86	0.84	6.29	1.93	7.99
1,2,3,7,8,9-Hx	0.80	1.41	0.29	0.14	0.17	0.74	0.94	7.00	1.67	6.91
1,2,3,4,6,7,8-Hp	0.82	7.79	0.16	0.23	0.62	0.48	1.31	9.78	1.70	7.06
OCCD	0.47	5.58	0.08	0.10	0.71	0.19	4.00	29.80	0.97	4.00
2,3,7,8-TCDF	0.91	2.05	1.80	3.00	0.87	5.21	0.91	6.75	1.88	7.79
1,2,3,7,8-Pe	0.59	2.93	0.32	0.18	0.25	0.94	0.59	4.36	1.22	5.04
2,3,4,7,8-Pe	2.83	13.97	1.75	1.06	1.27	5.22	2.95	21.96	5.87	24.33
1,2,3,4,7,8-Hx	3.33	31.28	0.71	0.48	0.60	2.13	4.12	30.66	6.91	28.65
1,2,3,6,7,8-Hx	2.45	23.05	0.51	0.29	0.39	1.53	2.54	18.94	5.10	21.12
1,2,3,7,8,9-Hx	0.53	4.96	0.09	0.05	0.07	0.26	0.65	4.86	1.10	4.54
2,3,4,6,7,8-Hx	2.75	25.79	0.57	0.17	0.13	1.71	2.50	18.59	5.70	23.62
1,2,3,4,6,7,8-Hp	1.29	19.75	0.18	0.13	0.29	0.54	1.37	10.24	2.68	11.08

Compound	Beef (pg/kg)	Offal (pg/kg)	Prod (pg/kg)	Fats (pg/kg)	Pork (pg/kg)	Lamb (pg/kg)	Poultry (pg/kg)	Eggs (pg/kg)	Milk (pg/kg)	Dairy (pg/kg)
1,2,3,4,7,8,9-Hp	0.52	7.94	0.07	0.02	0.03	0.21	0.60	4.46	1.07	4.43
OCDF	0.26	1.84	0.03	0.03	0.07	0.09	0.29	2.16	0.55	2.27

Dioxins are highly lipophilic chemicals, which leads to their preferential accumulation in fatty tissue. The highest dioxin concentrations were therefore generally predicted for animal tissues and food types containing high levels of fats, e.g. dairy products, animal fats and offal.

### 3.3.4 Predicted concentration in mothers milk

Dioxin concentrations in mothers' milk were calculated based on the mother's intake of dioxin from all exposure routes (i.e. diet, inhalation and from soil as calculated in the EA's dioxin worksheets and discussed in Section 3.4.1), 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 39.88 fg TEQ/kg.

## 3.4 Results of exposure/intake modelling

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.

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

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

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.

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.

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 (TEQ)) 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.

Average Daily Exposure predicted using the dioxin exposure worksheets for various receptors are detailed in Table 3-6 and Environment Agency Dioxin Exposure Worksheets used in this assessment are detailed in Appendix A.

**Table 3-6  
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	0.080	8.77E-07	8.32E-07	1.72E-07	2.29E-07
1,2,3,7,8–PeCDD	0.087	9.19E-07	9.06E-07	1.77E-07	2.39E-07
1,2,3,4,7,8–HxCDD	0.051	5.18E-08	4.96E-08	8.39E-09	1.20E-08
1,2,3,6,7,8–HxCDD	0.117	1.20E-07	1.15E-07	1.94E-08	2.77E-08
1,2,3,7,8,9–HxCDD	0.101	1.04E-07	9.92E-08	1.68E-08	2.40E-08
1,2,3,4,6,7,8–Hp	0.566	5.59E-08	5.06E-08	6.49E-09	1.05E-08
OCDD	0.849	2.49E-09	2.22E-09	2.58E-10	4.40E-10
2,3,7,8-TCDF	1.648	1.98E-06	1.66E-06	3.73E-07	4.90E-07
1,2,3,7,8-PeCDF	0.353	1.16E-07	1.07E-07	2.11E-08	2.86E-08
2,3,4,7,8-PeCDF	0.488	1.60E-06	1.48E-06	2.91E-07	3.95E-07
1,2,3,4,7,8-HxCDF	0.311	3.21E-07	2.98E-07	4.95E-08	7.16E-08
1,2,3,6,7,8-HxCDF	0.223	2.22E-07	2.15E-07	2.64E-08	4.23E-08
1,2,3,7,8,9-HxCDF	0.038	3.74E-08	3.61E-08	4.44E-09	7.12E-09
2,3,4,6,7,8-HxCDF	0.250	2.58E-07	2.40E-07	3.98E-08	5.76E-08
1,2,3,4,6,7,8-Hp	0.435	4.34E-08	3.91E-08	5.14E-09	8.25E-09
1,2,3,4,7,8,9-Hp	0.067	6.67E-09	6.01E-09	7.91E-10	1.27E-09
OCDF	0.228	6.74E-10	6.04E-10	7.39E-11	1.23E-10
<b>Total</b>	<b>5.893</b>	<b>6.72E-06</b>	<b>6.14E-06</b>	<b>1.21E-06</b>	<b>1.65E-06</b>

### 3.4.2 Dietary Intake

Dietary intake for the individual receptors was estimated on a TEQ basis based on plant and animal concentrations calculated in Section 3.3, 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 E3<sup>2</sup>. Dietary intakes resulting from consumption of the HMIP food types for each receptor are detailed in Table 3-7.

**Table 3-7  
Predicted Dietary Intake**

Food type	Dietary Intake (fg TEQ/kg bw/d)				
	Infant	Child	Adult	Farmer	Farm Child
Beef	0.25	0.19	0.10	0.30	0.55
Offal	0.35	0.18	0.17	0.50	0.54
Lamb	0.097	0.046	0.038	0.112	0.13
Pork	0.068	0.082	0.062	0.18	0.24
Poultry	0.105	0.052	0.050	0.15	0.15
Milk	24.14	3.87	1.47	4.42	11.60
Eggs	0.35	0.10	0.06	0.16	0.28
Dairy	10.34	3.22	1.97	6.06	9.90
Meat products	0.83	0.70	0.45	1.33	2.06
Fats, oil	0	0.77	0.40	1.19	2.26
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
<b>Total</b>	<b>37.88</b>	<b>9.86</b>	<b>5.53</b>	<b>16.04</b>	<b>29.19</b>

### 3.4.3 Intake due to inhalation

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 E1<sup>3</sup>. Predicted inhalation intake for each receptor is detailed below:

- Infant – 0.37 fg TEQ/kg bw/d
- Child – 0.24 fg TEQ/kg bw/d
- Adult – 0.08 fg TEQ/kg bw/d
- Farmer – 0.08 fg TEQ/kg bw/d
- Farmer's child – 0.24 fg TEQ/kg bw/d

<sup>2</sup> 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 to the average of 14.84 kg for CLEA Age Classes 2-6.

<sup>3</sup> 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

### 3.4.4 Intake for Breast-fed Infant

Based on exposure assumptions detailed in Environment Agency (2009a)<sup>4</sup> and HMIP Annex E6, intake for an infant from breast milk was calculated to be 0.1 pg TEQ/kg bw/d.

### 3.4.5 Total Intake

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

**Table 3-8  
Predicted Dioxin Intake**

Pathway	Intake (pg TEQ/kg bw/d)				
	Infant	Child	Adult	Farmer	Farm Child
CLEA pathways	6.72E-06	6.14E-06	1.21E-06	1.65E-06	6.14E-06
Diet	0.0379	0.0099	0.0055	0.0160	0.0292
Inhalation	3.69E-04	2.44E-04	8.06E-05	8.36E-05	2.44E-04
Breast milk	0.10				
<b>Total Intake</b>	<b>0.138</b>	<b>0.0101</b>	<b>0.0056</b>	<b>0.016</b>	<b>0.029</b>

Total predicted intake for worst case scenario exposure varies for the different receptor types, ranging from 0.0056 pg TEQ/kg bw/d for an adult resident to 0.138 pg TEQ/kg bw/d for a breast-fed infant. For all receptors, except the infant, dietary exposure (other than breast milk) is predicted to make the largest contribution to dioxin exposure.

### 3.4.6 Background intake

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 (0.39 pg/day for a 70 kg adult) which also indicates that exposure arising from operation of the EfW plant is likely to be insignificant compared to background sources.

## 3.5 Human health effects

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.

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 3-9 below.

<sup>4</sup> Infant bodyweight of 5.6 kg

**Table 3-9  
 Human Health Risk Assessment**

<b>Receptor</b>	<b>Maximum Predicted Intake (pg/kg bw/d)</b>	<b>COT TDI (pg/kg bw/d)</b>	<b>Hazard Quotient</b>
Infant	0.138	2	0.069
Child	0.010	2	0.005
Adult	0.006	2	0.003
Farmer	0.016	2	0.008
Farm Child	0.029	2	0.015

These results indicate an absence of risk to human health from dioxin emissions from the proposed Ardley EfW as the maximum predicted intake of dioxins for a residential receptor (i.e. child receptor in CLEA contaminated land guidance) is 200 times less than the UK recommended TDI.

The maximum intake predicted for a breast-fed infant (0.138 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)<sup>5</sup>. 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”.

All predicted intakes from the proposed Biomass facility are also significantly 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.

<sup>5</sup> 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<sup>-1</sup> bw day<sup>-1</sup> at 2–3 months and 19 pg WHO-TEQ kg<sup>-1</sup> bw day<sup>-1</sup> 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).

#### **4.0 CONCLUSIONS**

A conceptual site model has been developed for the proposed Ardley EfW plant 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.

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 model, which is used to assess human health risks deriving from contaminated land

Results from generic quantitative risk assessment for metals (see October 2008 report) 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 between the highest predicted soil concentrations and soil assessment criteria that are protective of the most sensitive human receptors.

For the assessment of human health risks arising from dioxin exposure due to airborne emissions from the proposed 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 proposed EfW facility.

It is therefore considered that the effect on health from the metal and dioxin emissions from the proposed Ardley EfW facility can be classified as highly unlikely to be significant.

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# **APPENDIX A**

## **Dioxin Worksheets**

**PCDDs, PCDFs and dioxin-like compounds worksheet for a user defined land use scenario**

This worksheet should be used only in conjunction with *Soil Guideline Values for dioxins, furans and dioxin-like PCBs in soil, Science Report SC050021 / Dioxins SGV*

For each congener/compound, enter the site-specific representative soil concentration and the calculated exposure factor from the CLEA software in the respective grey and yellow boxes. Enter the oral TDSI for the age classes considered in the green box. The Hazard Index is the ratio of the total soil exposure to the oral TDSI.

Substance	Soil concentration		Exposure factor		TEF	Average Daily Exposure	Contribution to ADE
	ng kg <sup>-1</sup> DW	x (	mg kg <sup>-1</sup> BW day <sup>-1</sup> per ng kg <sup>-1</sup> DW soil	pg kg <sup>-1</sup> BW day <sup>-1</sup> per ng kg <sup>-1</sup> DW soil			
2,3,7,8-TCDD	8.00E-05	x (	1.10E-11	1.10E-02	) x 1	= 8.77E-07	13.1
1,2,3,7,8-PeCDD	8.72E-05	x (	1.05E-11	1.05E-02	) x 1	= 9.19E-07	13.7
1,2,3,4,7,8-HxCDD	5.08E-05	x (	1.02E-11	1.02E-02	) x 0.1	= 5.18E-08	0.8
1,2,3,6,7,8-HxCDD	1.17E-04	x (	1.02E-11	1.02E-02	) x 0.1	= 1.20E-07	1.8
1,2,3,7,8,9-HxCDD	1.01E-04	x (	1.02E-11	1.02E-02	) x 0.1	= 1.04E-07	1.5
1,2,3,4,6,7,8-HpCDD	5.66E-04	x (	9.87E-12	9.87E-03	) x 0.01	= 5.59E-08	0.8
OCDD	8.49E-04	x (	9.78E-12	9.78E-03	) x 0.0003	= 2.49E-09	0.0
2,3,7,8-TCDF	1.65E-03	x (	1.20E-11	1.20E-02	) x 0.1	= 1.98E-06	29.5
1,2,3,7,8-PeCDF	3.53E-04	x (	1.09E-11	1.09E-02	) x 0.03	= 1.16E-07	1.7
2,3,4,7,8-PeCDF	4.88E-04	x (	1.09E-11	1.09E-02	) x 0.3	= 1.60E-06	23.8
1,2,3,4,7,8-HxCDF	3.11E-04	x (	1.03E-11	1.03E-02	) x 0.1	= 3.21E-07	4.8
1,2,3,7,8,9-HxCDF	2.23E-04	x (	9.96E-12	9.96E-03	) x 0.1	= 2.22E-07	3.3
1,2,3,6,7,8-HxCDF	3.76E-05	x (	9.96E-12	9.96E-03	) x 0.1	= 3.74E-08	0.6
2,3,4,6,7,8-HxCDF	2.50E-04	x (	1.03E-11	1.03E-02	) x 0.1	= 2.58E-07	3.8
1,2,3,4,6,7,8-HpCDF	4.35E-04	x (	9.96E-12	9.96E-03	) x 0.01	= 4.34E-08	0.6
1,2,3,4,7,8,9-HpCDF	6.70E-05	x (	9.96E-12	9.96E-03	) x 0.01	= 6.67E-09	0.1
OCDF	2.28E-04	x (	9.84E-12	9.84E-03	) x 0.0003	= 6.74E-10	0.0
PCB-77		x (			) x 0.0001	=	#VALUE!
PCB-81		x (			) x 0.0003	=	#VALUE!
PCB-126		x (			) x 0.1	=	#VALUE!
PCB-169		x (			) x 0.03	=	#VALUE!
PCB-105		x (			) x 0.00003	=	#VALUE!
PCB-114		x (			) x 0.00003	=	#VALUE!
PCB-118		x (			) x 0.00003	=	#VALUE!
PCB-123		x (			) x 0.00003	=	#VALUE!
PCB-156		x (			) x 0.00003	=	#VALUE!
PCB-157		x (			) x 0.00003	=	#VALUE!
PCB-167		x (			) x 0.00003	=	#VALUE!
PCB-189		x (			) x 0.00003	=	#VALUE!

Total WHO-TEQ ADE	6.72E-06	pg WHO-TEQ kg <sup>-1</sup> BW day <sup>-1</sup>
TDSI	1.00E+00	pg WHO-TEQ kg <sup>-1</sup> BW day <sup>-1</sup>
Hazard Index	0.00	

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For each congener/compound, enter the site-specific representative soil concentration and the calculated exposure factor from the CLEA software in the respective grey and yellow boxes. Enter the oral TDSI for the age classes considered in the green box. The Hazard Index is the ratio of the total soil exposure to the oral TDSI.

Substance	Soil concentration		Exposure factor		TEF	Average Daily Exposure	Contribution to ADE
	ng kg <sup>-1</sup> DW	x (	mg kg <sup>-1</sup> BW day <sup>-1</sup> per ng kg <sup>-1</sup> DW soil	pg kg <sup>-1</sup> BW day <sup>-1</sup> per ng kg <sup>-1</sup> DW soil			
2,3,7,8-TCDD	8.00E-05	x (	1.04E-11	1.04E-02	) x 1	= 8.32E-07	13.6
1,2,3,7,8-PeCDD	8.72E-05	x (	1.04E-11	1.04E-02	) x 1	= 9.06E-07	14.8
1,2,3,4,7,8-HxCDD	5.08E-05	x (	9.78E-12	9.78E-03	) x 0.1	= 4.96E-08	0.8
1,2,3,6,7,8-HxCDD	1.17E-04	x (	9.78E-12	9.78E-03	) x 0.1	= 1.15E-07	1.9
1,2,3,7,8,9-HxCDD	1.01E-04	x (	9.78E-12	9.78E-03	) x 0.1	= 9.92E-08	1.6
1,2,3,4,6,7,8-HpCDD	5.66E-04	x (	8.94E-12	8.94E-03	) x 0.01	= 5.06E-08	0.8
OCDD	8.49E-04	x (	8.72E-12	8.72E-03	) x 0.0003	= 2.22E-09	0.0
2,3,7,8-TCDF	1.65E-03	x (	1.01E-11	1.01E-02	) x 0.1	= 1.66E-06	27.1
1,2,3,7,8-PeCDF	3.53E-04	x (	1.01E-11	1.01E-02	) x 0.03	= 1.07E-07	1.7
2,3,4,7,8-PeCDF	4.88E-04	x (	1.01E-11	1.01E-02	) x 0.3	= 1.48E-06	24.1
1,2,3,4,7,8-HxCDF	3.11E-04	x (	9.61E-12	9.61E-03	) x 0.1	= 2.98E-07	4.9
1,2,3,7,8,9-HxCDF	2.23E-04	x (	9.61E-12	9.61E-03	) x 0.1	= 2.15E-07	3.5
1,2,3,6,7,8-HxCDF	3.76E-05	x (	9.61E-12	9.61E-03	) x 0.1	= 3.61E-08	0.6
2,3,4,6,7,8-HxCDF	2.50E-04	x (	9.61E-12	9.61E-03	) x 0.1	= 2.40E-07	3.9
1,2,3,4,6,7,8-HpCDF	4.35E-04	x (	8.97E-12	8.97E-03	) x 0.01	= 3.91E-08	0.6
1,2,3,4,7,8,9-HpCDF	6.70E-05	x (	8.97E-12	8.97E-03	) x 0.01	= 6.01E-09	0.1
OCDF	2.28E-04	x (	8.82E-12	8.82E-03	) x 0.0003	= 6.04E-10	0.0
PCB-77		x (			) x 0.0001	=	#VALUE!
PCB-81		x (			) x 0.0003	=	#VALUE!
PCB-126		x (			) x 0.1	=	#VALUE!
PCB-169		x (			) x 0.03	=	#VALUE!
PCB-105		x (			) x 0.00003	=	#VALUE!
PCB-114		x (			) x 0.00003	=	#VALUE!
PCB-118		x (			) x 0.00003	=	#VALUE!
PCB-123		x (			) x 0.00003	=	#VALUE!
PCB-156		x (			) x 0.00003	=	#VALUE!
PCB-157		x (			) x 0.00003	=	#VALUE!
PCB-167		x (			) x 0.00003	=	#VALUE!
PCB-189		x (			) x 0.00003	=	#VALUE!

Total WHO-TEQ ADE	6.14E-06	pg WHO-TEQ kg <sup>-1</sup> BW day <sup>-1</sup>
TDSI	1.00E+00	pg WHO-TEQ kg <sup>-1</sup> BW day <sup>-1</sup>
Hazard Index	0.00	

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For each congener/compound, enter the site-specific representative soil concentration and the calculated exposure factor from the CLEA software in the respective grey and yellow boxes. Enter the oral TDSI for the age classes considered in the green box. The Hazard Index is the ratio of the total soil exposure to the oral TDSI.

Substance	Soil concentration		Exposure factor		TEF	Average Daily Exposure		Contribution to ADE	
	ng kg <sup>-1</sup> DW	x (	mg kg <sup>-1</sup> BW day <sup>-1</sup> per ng kg <sup>-1</sup> DW soil	pg kg <sup>-1</sup> BW day <sup>-1</sup> per ng kg <sup>-1</sup> DW soil		-	pg WHO-TEQ kg <sup>-1</sup> BW day <sup>-1</sup>	%	
2,3,7,8-TCDD	8.00E-05	x (	2.15E-12	2.15E-03	) x	1	=	1.72E-07	14.2
1,2,3,7,8-PeCDD	8.72E-05	x (	2.03E-12	2.03E-03	) x	1	=	1.77E-07	14.6
1,2,3,4,7,8-HxCDD	5.08E-05	x (	1.65E-12	1.65E-03	) x	0.1	=	8.39E-09	0.7
1,2,3,6,7,8-HxCDD	1.17E-04	x (	1.65E-12	1.65E-03	) x	0.1	=	1.94E-08	1.6
1,2,3,7,8,9-HxCDD	1.01E-04	x (	1.65E-12	1.65E-03	) x	0.1	=	1.68E-08	1.4
1,2,3,4,6,7,8-HpCDD	5.66E-04	x (	1.15E-12	1.15E-03	) x	0.01	=	6.49E-09	0.5
OCDD	8.49E-04	x (	1.01E-12	1.01E-03	) x	0.0003	=	2.58E-10	0.0
2,3,7,8-TCDF	1.65E-03	x (	2.27E-12	2.27E-03	) x	0.1	=	3.73E-07	30.8
1,2,3,7,8-PeCDF	3.53E-04	x (	1.99E-12	1.99E-03	) x	0.03	=	2.11E-08	1.7
2,3,4,7,8-PeCDF	4.88E-04	x (	1.99E-12	1.99E-03	) x	0.3	=	2.91E-07	24.0
1,2,3,4,7,8-HxCDF	3.11E-04	x (	1.59E-12	1.59E-03	) x	0.1	=	4.95E-08	4.1
1,2,3,7,8,9-HxCDF	2.23E-04	x (	1.18E-12	1.18E-03	) x	0.1	=	2.64E-08	2.2
1,2,3,6,7,8-HxCDF	3.76E-05	x (	1.18E-12	1.18E-03	) x	0.1	=	4.44E-09	0.4
2,3,4,6,7,8-HxCDF	2.50E-04	x (	1.59E-12	1.59E-03	) x	0.1	=	3.98E-08	3.3
1,2,3,4,6,7,8-HpCDF	4.35E-04	x (	1.18E-12	1.18E-03	) x	0.01	=	5.14E-09	0.4
1,2,3,4,7,8,9-HpCDF	6.70E-05	x (	1.18E-12	1.18E-03	) x	0.01	=	7.91E-10	0.1
OCDF	2.28E-04	x (	1.08E-12	1.08E-03	) x	0.0003	=	7.39E-11	0.0
PCB-77		x (			) x	0.0001	=		#VALUE!
PCB-81		x (			) x	0.0003	=		#VALUE!
PCB-126		x (			) x	0.1	=		#VALUE!
PCB-169		x (			) x	0.03	=		#VALUE!
PCB-105		x (			) x	0.00003	=		#VALUE!
PCB-114		x (			) x	0.00003	=		#VALUE!
PCB-118		x (			) x	0.00003	=		#VALUE!
PCB-123		x (			) x	0.00003	=		#VALUE!
PCB-156		x (			) x	0.00003	=		#VALUE!
PCB-157		x (			) x	0.00003	=		#VALUE!
PCB-167		x (			) x	0.00003	=		#VALUE!
PCB-189		x (			) x	0.00003	=		#VALUE!

Total WHO-TEQ ADE	1.21E-06	pg WHO-TEQ kg <sup>-1</sup> BW day <sup>-1</sup>
TDSI	1.00E+00	pg WHO-TEQ kg <sup>-1</sup> BW day <sup>-1</sup>
Hazard Index	0.00	

**PCDDs, PCDFs and dioxin-like compounds worksheet for a user defined land use scenario**

This worksheet should be used only in conjunction with *Soil Guideline Values for dioxins, furans and dioxin-like PCBs in soil, Science Report SC050021 / Dioxins SGV*

For each congener/compound, enter the site-specific representative soil concentration and the calculated exposure factor from the CLEA software in the respective grey and yellow boxes. Enter the oral TDSI for the age classes considered in the green box. The Hazard Index is the ratio of the total soil exposure to the oral TDSI.

Substance	Soil concentration		Exposure factor		TEF	Average Daily Exposure	Contribution to ADE
	ng kg <sup>-1</sup> DW	x (	mg kg <sup>-1</sup> BW day <sup>-1</sup> per ng kg <sup>-1</sup> DW soil	pg kg <sup>-1</sup> BW day <sup>-1</sup> per ng kg <sup>-1</sup> DW soil			
2,3,7,8-TCDD	8.00E-05	x (	2.86E-12	2.86E-03	) x 1 =	2.29E-07	13.9
1,2,3,7,8-PeCDD	8.72E-05	x (	2.75E-12	2.75E-03	) x 1 =	2.39E-07	14.6
1,2,3,4,7,8-HxCDD	5.08E-05	x (	2.36E-12	2.36E-03	) x 0.1 =	1.20E-08	0.7
1,2,3,6,7,8-HxCDD	1.17E-04	x (	2.36E-12	2.36E-03	) x 0.1 =	2.77E-08	1.7
1,2,3,7,8,9-HxCDD	1.01E-04	x (	2.36E-12	2.36E-03	) x 0.1 =	2.40E-08	1.5
1,2,3,4,6,7,8-HpCDD	5.66E-04	x (	1.86E-12	1.86E-03	) x 0.01 =	1.05E-08	0.6
OCDD	8.49E-04	x (	1.73E-12	1.73E-03	) x 0.0003 =	4.40E-10	0.0
2,3,7,8-TCDF	1.65E-03	x (	2.98E-12	2.98E-03	) x 0.1 =	4.90E-07	29.8
1,2,3,7,8-PeCDF	3.53E-04	x (	2.70E-12	2.70E-03	) x 0.03 =	2.86E-08	1.7
2,3,4,7,8-PeCDF	4.88E-04	x (	2.70E-12	2.70E-03	) x 0.3 =	3.95E-07	24.0
1,2,3,4,7,8-HxCDF	3.11E-04	x (	2.30E-12	2.30E-03	) x 0.1 =	7.16E-08	4.4
1,2,3,7,8,9-HxCDF	2.23E-04	x (	1.89E-12	1.89E-03	) x 0.1 =	4.23E-08	2.6
1,2,3,6,7,8-HxCDF	3.76E-05	x (	1.89E-12	1.89E-03	) x 0.1 =	7.12E-09	0.4
2,3,4,6,7,8-HxCDF	2.50E-04	x (	2.30E-12	2.30E-03	) x 0.1 =	5.76E-08	3.5
1,2,3,4,6,7,8-HpCDF	4.35E-04	x (	1.89E-12	1.89E-03	) x 0.01 =	8.25E-09	0.5
1,2,3,4,7,8,9-HpCDF	6.70E-05	x (	1.89E-12	1.89E-03	) x 0.01 =	1.27E-09	0.1
OCDF	2.28E-04	x (	1.79E-12	1.79E-03	) x 0.0003 =	1.23E-10	0.0
PCB-77		x (			) x 0.0001 =		#VALUE!
PCB-81		x (			) x 0.0003 =		#VALUE!
PCB-126		x (			) x 0.1 =		#VALUE!
PCB-169		x (			) x 0.03 =		#VALUE!
PCB-105		x (			) x 0.00003 =		#VALUE!
PCB-114		x (			) x 0.00003 =		#VALUE!
PCB-118		x (			) x 0.00003 =		#VALUE!
PCB-123		x (			) x 0.00003 =		#VALUE!
PCB-156		x (			) x 0.00003 =		#VALUE!
PCB-157		x (			) x 0.00003 =		#VALUE!
PCB-167		x (			) x 0.00003 =		#VALUE!
PCB-189		x (			) x 0.00003 =		#VALUE!

Total WHO-TEQ ADE 1.65E-06 pg WHO-TEQ kg<sup>-1</sup> BW day<sup>-1</sup>  
 TDSI 1.00E+00 pg WHO-TEQ kg<sup>-1</sup> BW day<sup>-1</sup>  
 Hazard Index 0.00