Aberdeen Recycling and Energy

Energy from Waste Plant, East Tullos

Human Health Risk Assessment of Daily Intake of PCDD/Fs due to Process Emissions

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Amec Foster Wheeler Environment
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1. Introduction

Amec Foster Wheeler Environment and Infrastructure UK Limited ('Amec Foster Wheeler') has been instructed by Aberdeen City Council Waste and Recycling Service to prepare various assessments to support the planning application for an Energy from Waste (EfW) facility at East Tullos Industrial Estate, Aberdeen.

The proposed development would provide an EfW facility within the East Tullos Industrial Estate which would convert residual municipal waste into heat and power. The EfW would accept residual municipal waste from Aberdeen City Council, Aberdeenshire Council and Moray Council following all efforts to recycle.

The proposed capacity of the EfW facility is approximately 150,000 tpa and the preferred process is moving grate technology. The proposed development would comprise buildings that would include waste reception, waste bunker, boiler, turbine hall and stack, together with ancillary buildings and infrastructure, including air cooled condensers, offices and weighbridge. Flue gas treatment (FGT), including selective non-catalytic reduction (SNCR), lime and carbon injection upstream of a bag filtration plant, will be used to abate emissions from the stack.

This report presents an assessment which aims to evaluate the potential risk to human health due to daily intake of polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzonfurans (PCDFs) associated with process emissions to air from the main EfW stack.

The risk assessment uses a combination of detailed dispersion modelling and the methodologies proposed by Her Majesty's Inspectorate of Pollution (HMIP, 1996) and the United States Environmental Protection Agency (US EPA, 2005) to quantify the daily intake of PCDDs and PCDFs through inhalation and dietary ingestion for a Hypothetically Maximum Exposed Individual (HMEI). The resulting modelled daily intake is then compared to guidelines published by the Food Standards Agency's (FSA) Committee on Toxicity (COT) on the Tolerable Daily Intake (TDI) for these compounds in order to evaluate the risk to human health.

The information used to undertake the human health risk assessment includes:

- Design, process and emissions data;
- Baseline air quality data from surveys undertaken by Government bodies, Local Authorities and third parties;
- Ordnance Survey (OS) maps of the local area;
- Meteorological data supplied by Atmospheric Dispersion Modelling Ltd from Met Office and/or World Meteorological Organisation (WMO) affiliated weather stations; and
- Data and algorithms contained within reports by the HMIP, FSA, US EPA and the World Health Organisation (WHO).

The structure of the remainder of the report is set out as follows:

Table 1.1 Report Structure

Section	Aims and Objectives
Section 2	Details the background to PCDDs and PCDFs and emissions from UK waste facilities
Section 3	Describes the methodology used to undertake the human health risk assessment
Section 4	Details the results of the modelled background and incremental intake of PCDDs and PCDFs due to emissions from the facility
Section 5	Concludes the assessment

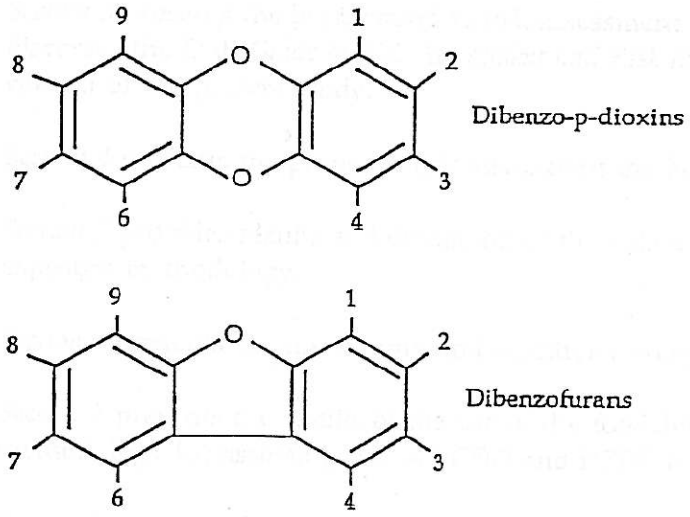
2. Background to PCDDs and PCDFs and UK Emissions to Air

2.1 Structure and Composition

PCDDs and PCDFs (hereafter, referred to, collectively, as PCDD/Fs for simplicity), are a family of compounds containing two benzene rings, connected by oxygen atoms, and one or more chlorine atoms. In the case of PCDDs, the benzene rings are connected by two oxygen atoms whereas, in PCDFs, the benzene rings are connected by a carbon bond and an oxygen atom. PCCD/Fs are also sometimes grouped together and simply termed “dioxins”.

Figure 2.1 depicts the basic structure of PCDD/Fs.

Figure 2.1 Basic Structure of PCDD/Fs



Source: HIMP (1996)

The convention used to identify different PCDD/Fs is based on the position on the benzene rings where chlorine atoms can be substituted (e.g., 2,3,7,8-tetrachlorodibenzo-*p*-dioxin has chlorine atoms located at positions 2, 3, 7 and 8 in **Figure 2.1**). There are 75 PCDDs and 135 PCDFs, each differing in the number and location of chlorine atoms on the benzene rings. Each individual PCDD/F is known as a congener. Congeners with the same number of chlorine atoms are termed homologues and are often abbreviated for convenience e.g., monochloro DDs are abbreviated to MDDs, whilst the fully chlorinated octachloro congeners are abbreviated to OCDD and OCDF, respectively.

Table 2.1 details the various PCDD/F homologues and congeners.

Table 2.1 PCDD/F Homologues and Congeners

Homologue	Number of Congeners	
	PCDDs	PCDFs
Monochloro (M)	2	4
Dichloro (D)	10	16
Trichloro (Tr)	14	28
Tetrachloro (T)	22	38
Pentachloro (Pe)	14	28
Hexachloro (Hx)	10	16
Heptachloro (Hp)	2	4
Octachloro (O)	1	1
TOTAL	**75**	**135**

2.2 Toxic Equivalents

The toxic response to individual PCDD/F congeners is similar but in different degrees of magnitude; the most toxic PCDD/F is 2,3,7,8-TCDD. Rather than assessing the toxic effects of each individual congener, it is more common to assess the combined effect of a mixture of PCDD/Fs, since these are rarely found in isolation in the environment.

In order to assess the toxicity of a mixture of PCDD/Fs, a Toxic Equivalent Factor (TEF) can be assigned to the individual congeners. As the most toxic of the PCDD/F congeners, 2,3,7,8-TCDD is assigned a TEF value of 1.0. The remaining PCDD/F congeners are then assigned lower TEFs, relative to that of 2,3,7,8-TCDD. When the TEF is applied to the congener-specific concentration, the resulting product is known as a Toxic Equivalent Quotient (TEQ), with units identical to that in which the concentrations of the individual congeners are expressed. The toxicity of the mixture is obtained by summing the individual TEQs.

There have been a number of toxicity rating schemes developed to prescribe TEFs to PCDD/Fs. In general, the scheme that has been internationally adopted is that of the NATO Committee on the Challenges of Modern Society (NATO/CCMS, 1988), under which the TEFs are known as international TEFs (I-TEFs). An alternative scheme adopted in the UK, and various other countries, is that proposed by the WHO, under which the TEFs are known as WHO-TEFs. There have been various updates to the WHO scheme, the most recent occurring in 2005 (Van den Berg *et al*., 2006). The Industrial Emissions Directive (2010/75/EU) states the I-TEF in Annex VI, Part 2.

The TEFs under these schemes for various PCDD/F congeners are detailed in **Table 2.2**.

Table 2.2 I-TEFs and WHO-TEFs

PCDD/F Congener	Toxicity Rating Scheme	
	I-TEF	WHO-TEF
	PCCDDs	
2,3,7,8-TCDD	1.0	1.0
1,2,3,7,8-PeCDD	0.5	1.0
1,2,3,4,7,8-HxCDD	0.1	0.1

PCDD/F Congener	Toxicity Rating Scheme	
	I-TEF	WHO-TEF
1,2,3,6,7,8-HxCDD	0.1	0.1
1,2,3,7,8,9-HxCDD	0.1	0.1
1,2,3,4,6,7,8-HpCDD	0.01	0.01
OCDD	0.001	0.0003
	PCDFs	
2,3,7,8-TCDF	0.1	0.1
1,2,3,7,8-PCDF	0.5	0.03
2,3,4,7,8-PCDF	0.05	0.3
1,2,3,4,7,8-HxCDF	0.1	0.1
1,2,3,6,7,8-HxCDF	0.1	0.1
1,2,3,7,8,9-HxCDF	0.1	0.1
2,3,4,6,7,8-HxCDF	0.1	0.1
1,2,3,4,6,7,8-HpCDF	0.01	0.01
1,2,3,4,7,8,9-HpCDF	0.01	0.01
OCDF	0.001	0.0003

It should be noted that only those PCDD/F congeners with chlorine atoms in the 2,3,7 and 8 positions are of concern for human health impacts. All non 2,3,7,8 positional congeners are assigned TEF values of 0.

2.3 Sources, Occurrence and Levels of PCDD/Fs in the Environment

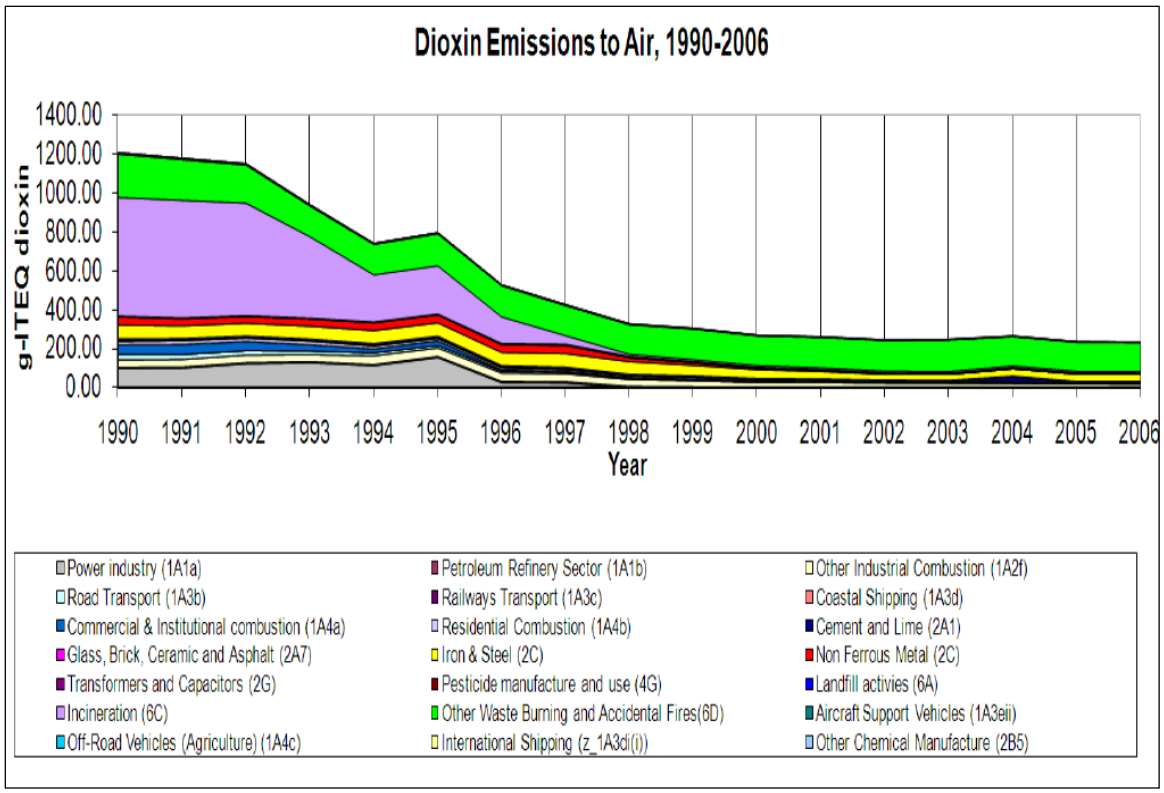
Quantities of PCDD/Fs in environmental media (water, air soil, vegetation etc.) are expressed in terms of picogrammes (i.e., 1/1 000 000 000 000 or 10^{-12} of a gramme) or femtogrammes (i.e., 1/1 000 000 000 000 000 or 10^{-15} of a gramme).

UK emissions to air of PCDD/Fs have declined by approximately 80% over the period 1990-2008 (NAEI, 2010). By far and large, the greatest reduction has been in the waste incineration sector, brought about by the introduction of a compulsory Emission Limit Value (ELV) of 0.1 ng I-TEQ m^{-3} for PCDD/Fs emissions from waste incineration plant. In 1990, emissions of PCDD/Fs from waste incineration accounted for approximately 51% of total UK emissions. By 2006, this figure had reduced more than ten-fold to 4.6% (Defra, 2010a).

The largest contributor to total UK emissions of PCDD/Fs is now uncontrolled domestic waste burning, bonfires and other accidental fires, accounting for more than 62% of total UK emissions in 2006 (Defra, 2010a).

Figure 2.2 presents estimates of the UK's annual emissions to air of PCDD/Fs by sector during the period 1990-2006.

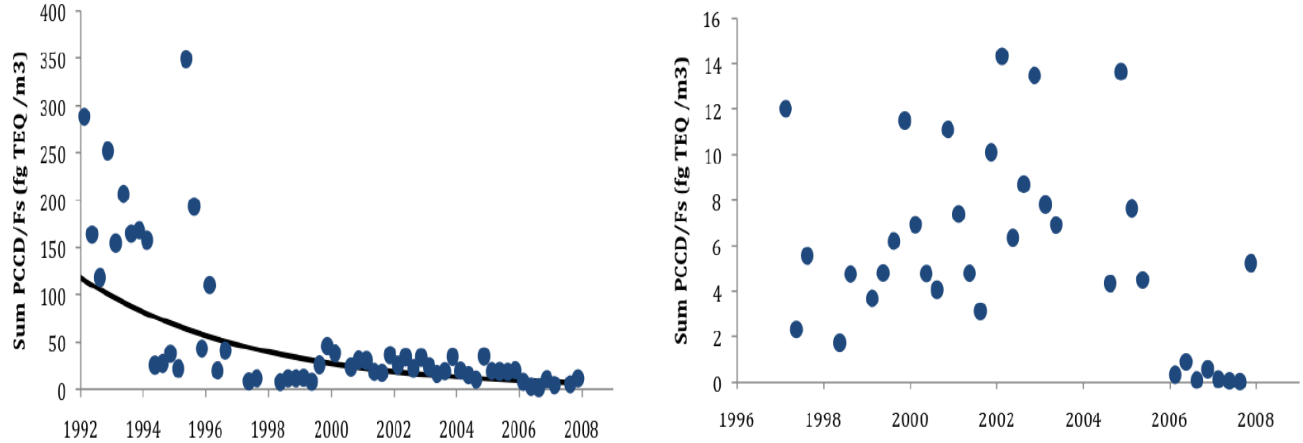
Figure 2.2 UK Emissions to Air of PCDD/Fs by Sector



Source: Defra (2010a)

In the UK, Defra's Toxic Organic Micropollutants (TOMPS) survey is the principal source of data on the measured concentrations of PCDD/Fs at six locations (two urban sites, three rural sites and one semi-rural site). PCDD/F concentration data from the TOMPS survey has shown corresponding decreases in ambient concentrations of PCDD/Fs in urban locations since the early 1990s but no clear trend in concentrations at the rural sites has been observed (**Figure 2.3**). However, the rural values are much lower than for the urban locations.

Figure 2.3 Variation in UK Ambient PCDD/F Concentrations at an Urban and Rural Location



Quarterly averaged PCCD/F concentrations in London (urban location; left image) and High Muffles (rural location; right image) as measured by the TOMPS survey. Source: Defra (2010b)

2.4 Human Exposure to PCDD/Fs and Tolerable Daily Intakes

PCDD/Fs are lipophilic, have very low solubility in water and are persistent and likely to bioaccumulate in the environment. They are, therefore, more likely to accumulate in fatty foods such as milk and milk products, and in certain meats and fish, than in other foodstuffs (Food Standards Agency (FSA), 2000). In the UK, cereals, fats and oils are major components of our diet and, therefore, these foods contribute a significant proportion of total PCDD/F intake (FSA, 2002). Although less likely to be present in vegetables, PCDD/Fs may be present in soil adhered to vegetables if not thoroughly washed or peeled.

Long-term exposure to high levels of PCDD/Fs has been shown to cause a wide range of effects in animal studies, including cancer and damage to the immune and reproductive system, although it appears that humans may be less susceptible to these effects (FSA, 2010).

Over 90% of human background exposure to PCDD/Fs is estimated to arise from the diet (WHO, 1998), with the remainder from sources such as inhalation, drinking water and skin absorption. Of these, only inhalation is likely to be measurable and it has been estimated (Defra/Environment Agency, 2003) that, based upon available measurements of ambient air concentrations of these compounds in the UK, the daily intake for adults by inhalation would be about 2 picogrammes per day ($2x10^{-12}$ g d^{-1}), equivalent to 0.03 pg kg^{-1} d^{-1} for an “average” 70 kg adult.

In 2001 the FSA’s Committee on Toxicity (COT) issued a statement that recommended revised Tolerable Daily Intake (TDI) values for PCDD/Fs (FSA, 2001). The revised TDI is 2 picogrammes as WHO-TEQ per kilogramme of body weight per day (2 pg WHO-TEQ kg^{-1} d^{-1}). It was considered that this TDI would be adequate to protect against toxicity from PCDD/Fs, particularly since this incorporates a ten-fold safety factor. The Committee also noted that, at the time, the intake estimates for the UK population were 1.8 pg kg^{-1} d^{-1} for the average consumer and 3.1 pg kg^{-1} d^{-1} for the 97.5 percentile consumer and that dietary intake was decreasing.

The Government, through the FSA, undertakes regular monitoring of foodstuffs for a range of contaminants, including PCDD/Fs, and a comparison of measurements undertaken in 1997 and 2001 (FSA, 2003) identified the following:

- Estimated total dietary intakes by all age groups fell by around 50% between 1997 and 2001;
- The estimated average intakes by adults from the UK diet have fallen from 1.8 pg kg^{-1} d^{-1} in 1997 to 0.9 pg kg^{-1} d^{-1} in 2001 and were, therefore, well within the TDI. The percentage of adults estimated to exceed the TDI fell from 35% in 1997 to 1.1% in 2001;
- Average intakes by schoolchildren have fallen from between 1.6 and 4.0 pg kg^{-1} d^{-1} in 1997 to between 0.7 and 1.8 pg kg^{-1} d^{-1} in 2001, with the younger children being at the upper end of these ranges. The percentage of schoolchildren estimated to exceed the TDI fell from 62% in 1997 to 10% in 2001;
- Average intakes by toddlers have also fallen over the period 1997 to 2001 and are estimated to be close to or slightly above the new UK safety guideline. The percentage of toddlers estimated to exceed the TDI from the whole diet fell from 97% in 1997 to 37% in 2001; and
- The concentrations of PCDD/Fs found in samples of foodstuffs were all within the relevant EU regulatory limits.

2.5 Summary of PCDDs and PCDFs in UK

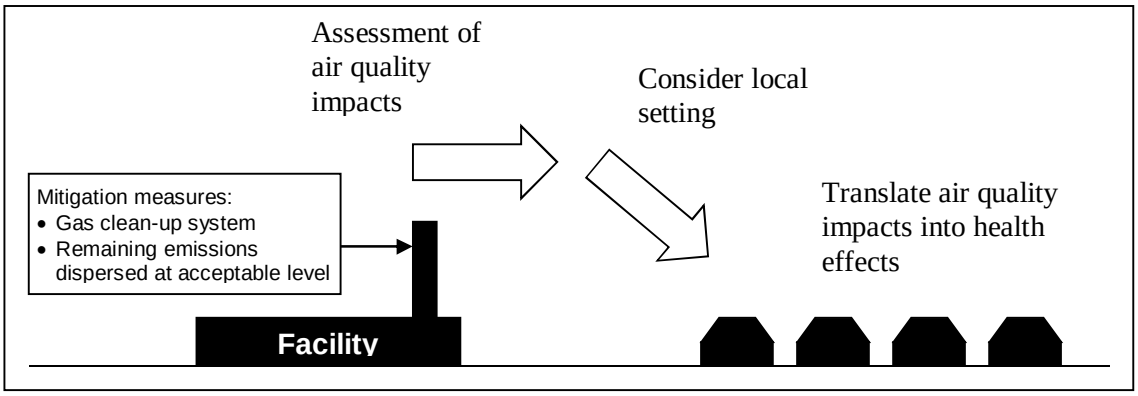
In summary, UK emissions to air of PCDD/Fs has decreased almost five-fold over the period 1990 to 2008, primarily as a result of significant improvements in combustion and flue gas treatment techniques in the waste incineration sector. The ambient concentration of PCDD/Fs in populated areas has shown a corresponding decrease over that period, whilst dietary intake of PCDD/Fs decreased by an average of 50% between 1997 and 2001.

3. Risk Assessment Methodology

3.1 General Approach

The general approach to assessing the risk posed to human health due to PCDD/F emissions from the thermal treatment plant follows the methodologies contained within the HMIP's 'Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes' (HMIP, 1996) and the US EPA's 'Human Health Risk Assessment Protocol (HHRAP) for Hazardous Waste Combustion Facilities' (US EPA, 2005). This process is represented in **Figure 3.1**.

Figure 3.1 Schematic Representation of Analysis Undertaken

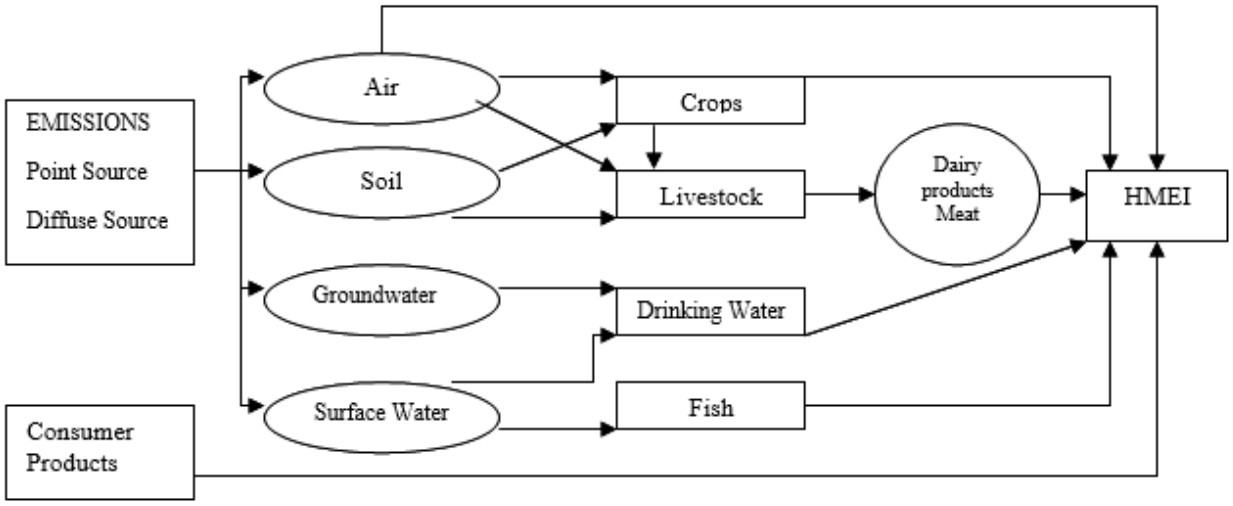


Following the advice in these documents, this assessment considers PCDD/F intake for a Hypothetically Maximally Exposed Individual (HMEI). The concept of a HMEI assumes that this individual:

- Lives for his/her entire life at the location of maximum predicted ground level PCDD/F concentration;
- Breathes the air at this maximum concentration for his/ her entire life;
- Drinks water exposed to this maximum concentration/ deposition of PCDD/F for his/her entire life;
- Eats crops grown in the soil at this location of maximum concentration/ deposition of PCDD/F for his/her entire life;
- Eats meat and produce from animals grazing on vegetation grown in the soil at this location of maximum concentration/ deposition of PCDD/F for his/her entire life; and
- Eats fish that have grown in a closed water body (such as a reservoir) exposed to this maximum concentration/deposition of PCDD/F for his/her entire life.

Thus, exposure of the HMEI to PCDD/F concentrations in environmental media resulting from emissions from the generic thermal treatment plant is as high as theoretically possible, to the extent of being an extreme worst-case assessment. The exposure routes for a HMEI are provided in **Figure 3.2**.

Figure 3.2 Schematic Representation of Pollutant Exposure Pathways for HMEI



The assessment uses the basic algorithms in HMIP (1996) to quantify the daily intake of PCDD/Fs from predicted ground level concentrations obtained from an advanced dispersion model. However, the HHRAP contains revised congener specific physical and chemical properties and biotransfer factors based on more recent research post-1996. Consequently, the latest parameter values advocated by the HHRAP have been used in preference to the values contained within the original HMIP methodology.

3.2 Hazard Identification

The HMIP methodology considers eight potential direct and indirect human exposure pathways of PCDD/F emissions from waste incineration processes:

- Inhalation of air;
- Ingestion of soil;
- Ingestion of surface water;
- Ingestion of vegetable produce and fruits;
- Ingestion of animal meats and other animal food products;
- Ingestion of milk and milk products;
- Ingestion of fish reared in a closed water body such as a reservoir; and
- Ingestion of mother's breast milk.

The following pathways were considered by HMIP but were later screened out due to the low intake predicted:

- Dermal absorption of PCDD/Fs from contact with soil and water;
- Impacts of PCDD/Fs on groundwater resources;
- Impact of PCDD/Fs on users of rivers and estuaries following their release in scrubber water discharges; and
- Off-site disposal of contaminated fly-ash.

As previously discussed, the assessment considers the PCDD/F intake for a HMEI. In this report, the assessment is limited to an adult HMEI, since HMIP (1996) concluded that this category of individual had the highest daily PCDD/F intake when compared to a child HMEI.

With regard to breast-fed infants, Defra/Environment Agency (2003) report:

> *"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 markedly less dramatic. Peak infant body burdens are only around twice those of an adult, a consequence of the infant's rapidly expanding body weight and lipid volume, as well as a possibly faster elimination rate."*

3.3 Dispersion Modelling

The methodology used to predict maximum ground level concentrations of PCDD/Fs from the facility is consistent with that detailed in the *Technical Methodology Report of Point Source Emissions to Air* (Amec Foster Wheeler, 2016).

As monitored emissions data is not yet available for the facility, the assessment adopts a worst-case approach by assuming PCDD/F emissions from the facility are at the respective emission limit value (ELV) contained within Annex VI of the Industrial Emissions Directive. The speciation of individual PCDD/F congeners follows the typical emission profile contained within HMIP (1996).

Table 3.1 summarises the model input data used in the assessment.

Table 3.1 Model Input Data

Parameter	Value
Stack Height (m)	80
Stack Diameter (m)	1.85
Efflux Temperature (°C)	130
Efflux Velocity (m s^{-1})	15
PCDD/F Emission Rate (ng I-TEQ s^{-1})	2.84
PCDD/F Congener Speciation (%w/w)	
2,3,7,8-TCDD	3.1%
1,2,3,7,8-PeCDD	24.4%
1,2,3,4,7,8-HxCDD	2.9%
1,2,3,6,7,8-HxCDD	2.6%
1,2,3,7,8,9-HxCDD	2.0%
1,2,3,4,6,7,8-HpCDD	1.7%
OCDD	0.1%
2,3,7,8-TCDF	2.8%
1,2,3,7,8-PeCDF	0.8%
2,3,4,7,8-PeCDF	16.0%
1,2,3,4,7,8-HxCDF	21.7%
1,2,3,6,7,8-HxCDF	8.0%
1,2,3,7,8,9-HxCDF	0.4%

2,3,4,6,7,8-HxCDF	8.7%
1,2,3,4,6,7,8-HpCDF	4.4%
1,2,3,4,7,8,9-HpCDF	0.4%
OCDF	0.1%

Meteorology

The selection of meteorological data used to drive the dispersion model is discussed in detail in the *Technical Methodology Report of Point Source Emissions to Air* (Amec Foster Wheeler, 2016). Mean wind speed and annual rainfall data from the year of meteorological data producing the highest annual mean ground level concentration has been used to specify the site specific parameters required by the HMIP methodology. These are detailed in **Table 3.2** below.

Table 3.2 Mean Wind Speed and Annual Rainfall

Parameter	Value
Mean wind speed (m s^{-1})	4.5
Annual rainfall (m y^{-1})	0.82

Deposition

The algorithms within the HMIP methodology have been used to quantify the mass of PCDD/Fs deposited to land based on the deposition velocity (HMIP default value of 0.2 cm s^{-1} used for all congeners), the fraction of individual PCDD/F congeners in the vapour and particulate phases (HHRAP parameters used) and the annual rainfall data detailed in **Table 3.2.**

Exposure Functions

Table 3.3 summarises the exposure functions for the adult HMEI scenario considered in this assessment. These figures replicate those recommended in the HMIP methodology. Data on the fraction of produce grown/ reared and consumed locally for a HMEI, and typical dietary daily intakes for an adult HMEI, can be found in HMIP (1996).

Table 3.3 Exposure Functions for Worst-Case Estimation of Adult HMEI

Parameter	Value
Inhalation rate (m3 h-1)	0.7
Body weight (kg)	70
Exposure frequency (days y-1)	365
Exposure duration (y)	75
Lifetime (days)	27,375
Averaging time (days)	27,375

3.4 Assessing the Risk

The methodology detailed above is used to estimate the incremental PCDD/F intake due to emissions from the EfW facility for an adult HMEI. The incremental intake is then added to the estimated background intake and the combined value assessed against the PCDD/F TDI of 2 pg WHO-TEQ kg^{-1} d^{-1}. With regard to the TDI, the FSA states (FSA, 2010):

> *"The TDI is set using a precautionary approach including a safety margin. Exceeding the TDI erodes the safety margin but does not necessarily result in a risk to health."*

Thus, the TDI can be viewed as a risk threshold, below which, there is a negligible risk of adverse health effects occurring and, above which, there is an increased risk of human health effects occurring. Consequently, two categories/ descriptors are used in this assessment to describe the potential risk to human health due to the total modelled PCDD/F intake (i.e., background intake plus incremental intake):

- Risk where the total intake is less than the TDI = ***Low or effectively zero; and***
- Risk where the total intake is greater than the TDI = ***Increased risk of adverse human health effects.***

Ambient concentrations of individual PCDD/F congeners have been taken from the TOMPS survey.

It is considered that the Manchester monitoring location in the TOMPS survey most closely represents the area surrounding the proposed development. Data contained within the most recently published TOMPS network annual report (2010 survey period) has been used. **Table 3.4** summarises this data.

Table 3.4 Background PCDD/F Concentrations used to Derive Background Intake

Congener	Concentration (fg WHO-TEQm^3)
2,3,7,8-TCDF	2.15
1,2,3,7,8-PeCDF	1.18
2,3,4,7,8-PeCDF	1.76
1,2,3,4,7,8-HxCDF	1.83
1,2,3,6,7,8-HxCDF	1.40
2,3,4,6,7,8-HxCDF	1.60
1,2,3,7,8,9-HxCDF	0.63
1,2,3,4,6,7,8-HpCDF	3.91
1,2,3,4,7,8,9-HpCDF	0.62
OCDF	3.27
2,3,7,8-TCDD	4.40
1,2,3,7,8-PeCDD	4.21
1,2,3,4,7,8-HxCDD	0.42
1,2,3,6,7,8-HxCDD	0.58
1,2,3,4,7,8-HxCDD	0.62
1,2,3,4,6,7,8-HpCDD	4.11
OCDD	8.93

4. Results of the Risk Assessment

This section details the results of the background and incremental PCDD/F intake for an adult HMEI using the methodology detailed in Section 3. When putting the modelled results into context, it is useful to reiterate the basic assumptions defining the HMEI scenario assessed:

- Emissions from the stack occur at ELV concentrations 24 hours a day, 365 days a year, for each year over the lifetime of the individual;
- This individual lives for his/her entire life at the location of maximum predicted ground level PCDD/F concentration, breathes the air at this maximum concentration for his/her entire life, eats crops grown in the soil at this location of maximum concentration/ deposition of PCDD/F for his/ her entire life and eats meat and produce from animals grazing on vegetation grown in the soil at this location of maximum concentration/ deposition of PCDD/F for his/ her entire life; and
- Worst-case meteorological data used.

Table 4.1 presents the results of the risk assessment.

Table 4.1 Results of PCDD/F Human Health Risk Assessment

TDI (pg WHO-TEQ kg-1 d-1)	Background Intake (pg WHO-TEQ kg-1 d-1)	Incremental Intake (pg WHO-TEQ kg-1 d-1)	Total Intake (pg WHO-TEQ kg-1 d-1)	% Total Intake of TDI	Risk of Adverse Human Health Effects
2	0.41	0.01	0.42	21%	Low or effectively zero

Table 4.1 demonstrates that the modelled total intake of PCDD/Fs for an adult HMEI due to background intake, and incremental intake based on emissions from the EfW facility, is less than the TDI. The assessment indicates that the total intake for an adult HMEI is 21% of the TDI using these emission values, with emissions from the EfW facility contributing less than 1% of the TDI. With respect to predictions of impact for an infant HMEI, given the peak infant body burden of PCDD/Fs is typically double that of an adult (Defra/Environment Agency, 2003), the predicted values above would not result in an exceedence of the TDI for an infant HMEI.

As such, the risk of adverse human health effects due to PCDD/F emissions from the plant can be described as **low or effectively zero**.

5. Conclusion

This assessment has used a combination of detailed dispersion modelling and risk assessment methodology proposed by the HMIP and US EPA to ascertain the risk of adverse human health effects occurring due to emissions of PCDD/Fs from the proposed 150,000tpa EfW facility on the East Tullos Industrial Estate, Aberdeen.

The assessment considered total PCDD/F intake via inhalation and ingestion for an adult HMEI, incorporating 'worst-case' assumptions with regards to maximum exposure concentration, exposure frequency and dietary consumption.

Modelled total intake (background intake plus incremental intake due to site emissions) was calculated as being below the TDI, with the maximum total intake representing 21% of the TDI, and with the incremental intake due to site emissions contributing less than 1% of the TDI. With respect to intake for an infant HMEI, given the peak infant body burden of PCDD/Fs is typically double that of an adult, the predicted values above would not result in an exceedence of the TDI for an infant HMEI.

The conservative assumptions inherent in the theoretical basis of an HMEI, and worst-case assumptions adopted in the approach to the detailed dispersion modelling of PCDD/F emissions, are likely to further reduce the actual PCDD/F intake for individuals habiting or working in the proximity of the facility.

Consequently, this assessment concludes that the risk of adverse human health effects occurring due to PCDD/F emissions from EfW facility is ***low or effectively zero***.

6. References

Amec Foster Wheeler, 2014. 'Technical Methodology Report of Point Source Emissions to Air.'

CERC, 2003. 'The Met Input Module'. ADMS Technical Specification ADMS 3 P05/01N/03.

Defra/Environment Agency, 2003. 'Contaminants in Soil: Collation of Toxicological Data and Intake Values for Humans – Dioxins, Furans and Dioxin-Like PCBs.'

Defra, 2010a. 'Review and Update of the UK Source Inventories of Dioxins, Dioxin-Like Polychlorinated Biphenyls and Hexachlorobenzene for Emissions to Air, Water and Land. AEA Technology Report for the Department for Environment, Food and Rural Affairs, report reference AEAT/ENV/R/2767.

Defra, 2010b. 'Annual Report for 2008 on the UK Toxic Organic Micro-pollutants (TOMPS) Air Monitoring and Analysis Network.' Lancaster Environment Centre Report for the Department for Environment, Food and Rural Affairs.

FSA, 2000. 'Dioxins and PCBs in the UK Diet: 1997 Total Diet Study Samples.' Food Standards Agency, Food Surveillance Information Sheet No. 00/04.

FSA, 2001. 'Statement on the Tolerable Daily Intake for Dioxins and Dioxin-Like Polychlorinated Biphenyls'. Food Standards Agency Committee on Toxicity of Chemicals in Food.

FSA, 2002. 'Dioxins and PCBs in Fish Oil Dietary Supplements and Licensed Medicines.' Food Standards Agency Food Survey Information Sheet 26/02.

FSA, 2003. 'Dioxins and Dioxin-Like PCBs in the UK Diet: 2001 Total Diet Study Samples'. Food Standards Agency Report 38/3.

HMIP, 1996. 'Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes.' Her Majesty's Inspectorate of Pollution, report reference HMIP/CPR2/41/1/181.

NATO-CCMS, 1988. International Toxicity Equivalent Factor (I-TEF) Method of Risk Assessment for Complex Mixtures of Dioxins and Related Compounds.' North Atlantic Treaty Organisation, Committee on Challenges of Modern Society.

US EPA, 2005. 'Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities.' United States Environmental Protection Agency, report reference EPA530-Rx10-5-006.

Van den Berg, M., Birnbaum, S., Denison, M., De Vito, M., Farland, W., Feeley, M., Fiedler, H., Hakansson, H., Hanberg, A., Haws, L., Rose, M., Safe, S., Schrenk, D., Tohyama, C., Tritscher, A., Tuomitso., J., Tysklind, M., Walker, N. and Peterson., R., 2005. 'The 2005 World Health Organization Re-evaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds' *Toxicological Sciences*, **93**, 223-241.

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