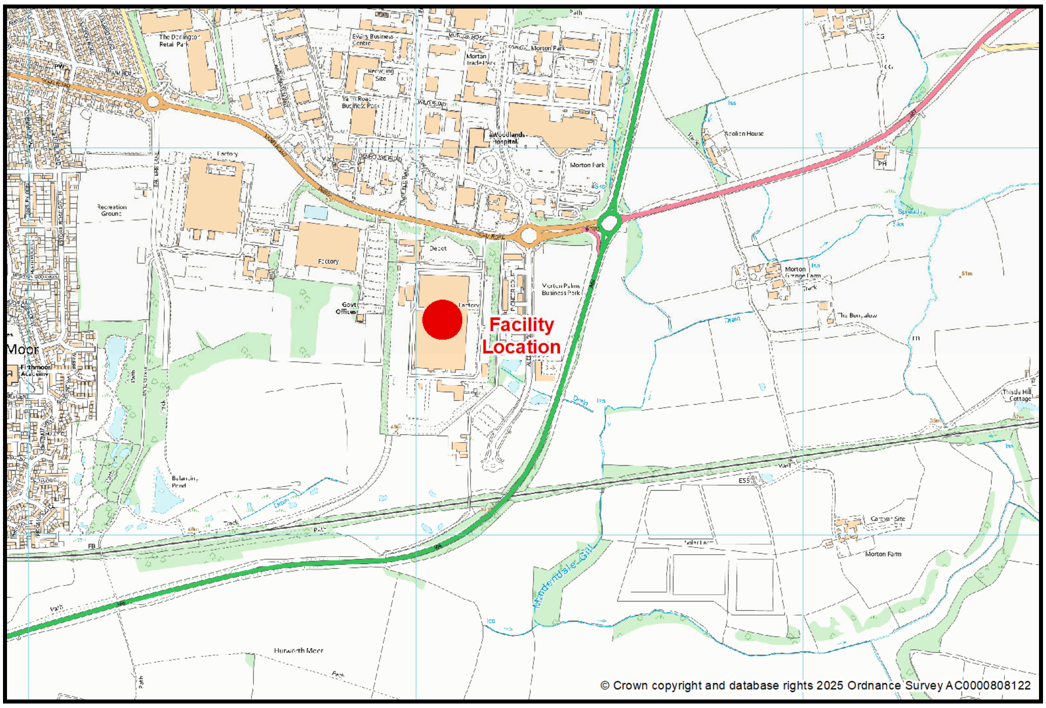
SOL ENVIRONMENT LTD

CLEVELAND BRIDGE FACILITY:

HUMAN HEALTH RISK ASSESSMENT



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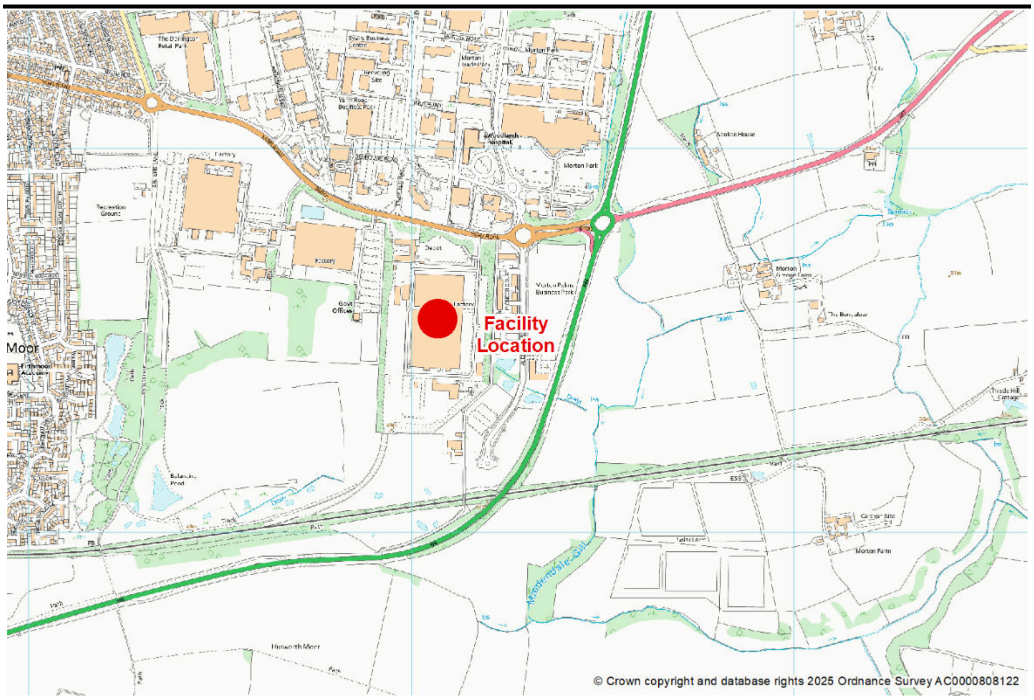
1 INTRODUCTION

1.1 PURPOSE OF THE ASSESSMENT

Gair Consulting Ltd has been commissioned by Sol Environment Limited on behalf of Endolys Limited to undertake an assessment to consider the effects on human exposure from a proposed plastic waste recycling facility on land to the southeast of Darlington, County Durham. The facility will utilise Advanced Thermal Treatment (pyrolysis) to manufacture pyrolysis oil (PyOil) from around 120,000 tonnes of waste plastic film per annum. This human health risk assessment (HHRA) supports the Environmental Permit application for the facility.

The proposed facility is located to the southeast of Darlington, County Durham. The location of the facility is presented in *Figure 1.1*. The area surrounding the site is a mix of light industrial use but land use to the east and south is rural. The nearest residential (hotel) receptor is located to the north of the site to the north of Yarm Road. The site is located within the administrative area of Darlington Borough Council (DBC). DBC does not currently have any Air Quality Management Areas (AQMAs) within their administrative area. The nearest AQMA to the proposed development is 28 km to the north in Durham.

FIGURE 1.1 LOCATION OF THE PROPOSED PLASTICS RECYCLING FACILITY



The assessment is provided to support the Environmental Permit application for the proposed facility. The Human Health Risk Assessment (HHRA) supplements the air quality assessment provided for the facility. The HHRA only considers emissions to air as in this case human exposure to any harmful pollutants discharged directly to the aquatic environment and from solid waste disposal is considered to be negligible.

An air quality assessment of emissions from the facility has been provided by Sol Environmental Ltd [1]. The air quality assessment provides a comparison of predicted concentrations of pollutants at off-site locations with background air quality and air quality standards and guidelines for the protection of human health. The air quality assessment assumes the theoretical position that the maximum permissible emission limit values (ELVs), stipulated for legal compliance for the facility, are emitted during all times of operation. This position is considered unlikely to be a realistic operating scenario because, in reality, the emissions will be lower.

Given the above theoretical operating scenario, the emissions from the facility would contain a number of substances that cannot be evaluated in terms of their effects on human health simply by reference to ambient air quality standards. Health effects could occur through exposure routes other than purely inhalation. As such, an assessment needs to be made of the overall human *exposure* to the substances by the local population and then the *risk* that this exposure causes.

1.2 BACKGROUND TO THE ASSESSMENT

As the assessment supports the Environmental Permit application for the facility, it has been prepared in accordance with our understanding of the requirements of the Environment Agency for waste incineration and waste co-incineration plants. The Environment Agency requirements are for a human health risk assessment of dioxin/furan emissions from the facility based on the US EPA HHRAP methodology in the absence of UK or EU methods.

Human exposure to dioxins and furans has been compared against the Committee of Toxicity (COT) Tolerable Daily Intake (TDI) of 2 pg/kg per day. An assessment of exposure to dioxin-like polychlorinated biphenyls (PCBs) has also been included.

It should be noted that the former Her Majesty's Inspectorate of Pollution (HMIP) method does not have the capability to consider dioxin-like PCBs and the US EPA HHRAP method is limited in this respect. The HHRAP method does not contain physical properties or exposure parameters for individual dioxin-like PCBs but does provide information for two dioxin-like PCB mixtures (Aroclor 1016 and Aroclor 1254). Therefore, for these two substances typical emissions for dioxin-like PCBs have been included in the Industrial Risk Assessment Program (IRAP) model and these have been assumed to comprise

1 Cleveland Bridge Facility: Air Quality Assessment, Sol Environment Report Reference SOL_25_P051_PYR_AQA (January 2026)

entirely of Aroclor 1016 or Aroclor 1254 depending on which substance gives rise to the highest exposure.

1.3 SCOPE OF THE ASSESSMENT

The emissions from the facility during the modelled operational scenario would contain a number of substances that cannot be evaluated in terms of their effects on human health simply by reference to ambient air quality standards. Health effects could occur through exposure routes other than purely inhalation. As such, an assessment needs to be made of the overall human *exposure* to the substances by the local population and then the *risk* that this exposure causes.

The assessment presented here considers the potential impact of substances released by the installation on the health of the local population at the point of maximum exposure. These substances are those that are 'persistent' in the environment and have several pathways from the point of release to the human receptor. Essentially, they can be described as dioxins/furans and dioxin-like polychlorinated biphenyls (PCBs) and are present in extremely small quantities and are typically measured in mass units of nanograms (ng = 10^{-9} g), picograms (pg = 10^{-12} g) and femtograms (fg = 10^{-15} g).

Unlike substances such as nitrogen dioxide, which have short term, acute effects on the respiratory system, dioxins/furans and dioxin-like PCBs have the potential to cause effects through long term, cumulative exposure. A lifetime is the conventional period over which such effects are evaluated. A lifetime is taken to be 70 years.

The exposure scenarios used here represent highly unrealistic situations in which all exposure assumptions are chosen to represent a worst case and should be treated as an extreme view of the risks to health. While individual high-end exposure estimates may represent actual exposure possibilities (albeit at very low frequency), the possibility of all high-end exposure assumptions accumulating in one individual is, for practical purposes, never realised. Therefore, intakes presented here should be regarded as an extreme upper theoretical representation of exposure that would be over and above that which would actually be experienced by the real population in the locality.

1.4 APPROACH TO THE ASSESSMENT

The risk assessment process is based on the application of the US EPA Human Health Risk Assessment Protocol (HHRAP) [2]. This protocol has been assembled into a commercially available model, Industrial Risk Assessment Program (IRAP, Version 5.1.4) and marketed by Lakes Environmental of Ontario.

2 US EPA Office of Solid Waste (September 2005) Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities

The approach seeks to quantify the *hazard* faced by the receptor, the *exposure* of the receptor to the substances identified as being a potential hazard and then to assess the *risk* of the exposure, as follows.

- *Quantification of the exposure*: an exposure evaluation determines the dose and intake of key indicator chemicals for an exposed person. The dose is defined as the amount of a substance contacting body boundaries (in the case of inhalation, the lungs) and intake is the amount of the substance absorbed into the body. The evaluation is based upon worst-case, conservative scenarios, with respect to the following:

    - location of the exposed individual and duration of exposure;
    - exposure rate;
    - emission rate from the source.

- *Risk characterisation*: following the above steps, the risk is characterised by examining the toxicity of the chemicals to which the individual has been exposed, and evaluating the significance of the calculated dose by a comparison of intakes with the tolerable daily intake (TDI) for dioxins/furans and dioxin-like PCBs.

2 METHODOLOGY FOR ESTIMATING EXPOSURE TO EMISSIONS

2.1 INTRODUCTION

An exposure assessment for the purposes of characterising the health impact of the facility emissions requires the following steps:

(1) Measurement or estimation of emissions from the source.

(2) Modelling the fate and transport of the emitted substances through the atmosphere and through soil, water and biota following deposition onto land. Concentrations of the emitted chemicals in the environmental media are estimated at the point of exposure, which may be through inhalation or ingestion.

(3) Calculation of the uptake of the emitted chemicals into humans coming into contact with the affected media and the subsequent distribution in the body.

With regard to Step (3), the exposure assessment considers the uptake of polychlorinated dibenzo-para-dioxins and polychlorinated dibenzofurans (PCDD/Fs, often abbreviated to 'dioxins/furans') and dioxin-like PCBs by various categories of human receptors.

2.2 POTENTIAL EXPOSURE PATHWAYS

There are two primary exposure 'routes' where humans may come into contact with chemicals that may be of concern:

- direct, via inhalation; or
- indirect, via ingestion of water, soil, vegetation and animals and animal products that become contaminated through the food chain.

There are four other potential exposure pathways of concern following the introduction of substances into the atmosphere:

- ingestion of drinking water;
- dermal (skin) contact with soil;
- incidental ingestion of soil; and
- dermal (skin) contact with water.

2.3 EXPOSURE PATHWAYS CONSIDERED IN THE ASSESSMENT

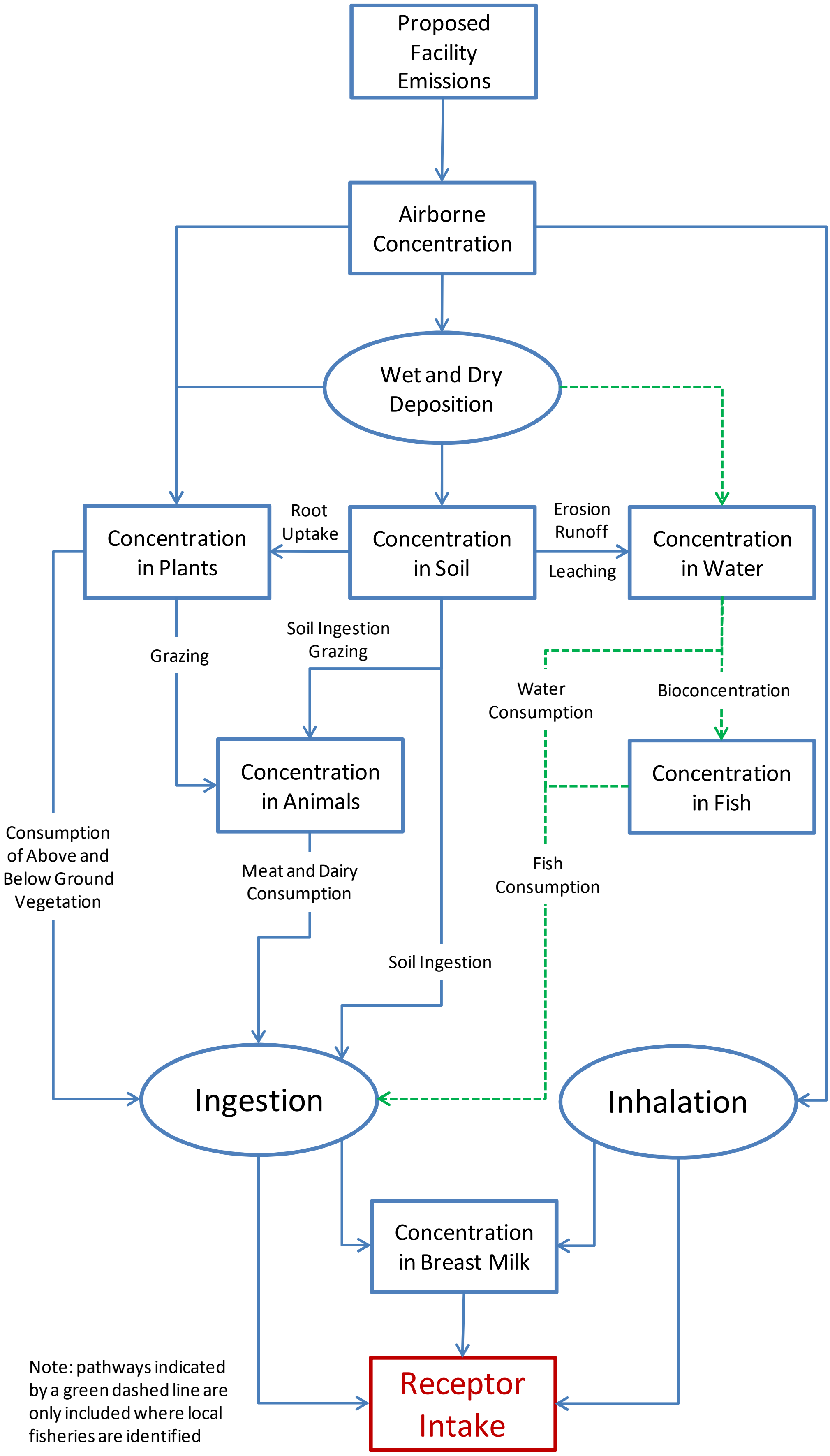
The possible exposure pathways included in the IRAP model are presented in *Figure 2.1*. Dermal contact with soil is an insignificant exposure pathway on the basis of the infrequent and sporadic nature of the events and the very low dermal absorption factors for this exposure route, coupled with the low plausible total dose that may be experienced (when considered over the lifetime of an individual). Health risk assessments of similar emissions (Pasternach (1989) *The Risk Assessment of Environmental and Human Health Hazards*, John Wiley, New York) have concluded that dermal absorption of soil is at least one order of magnitude less efficient than lung absorption.

Similar arguments are relevant with respect to the elimination of aquatic pathways from consideration; swimming, fishing and other recreational activities are also sporadic and unlikely to lead to significant exposures or uptake of any contamination into the human body via dermal contact with water.

Exposure via drinking water requires contamination of surface drinking water sources local to the point of consumption. The likelihood of contamination reaching a level of concern in the local water sources and ground water supplies is extremely low, particularly where there is no large-scale storage (e.g. reservoirs) or catchment areas for local water supplies. However, the US EPA's HHRAP does include the ingestion of drinking water from surface water sources as a potential exposure pathway where water bodies and water sheds have been defined within the exposure scenario. The ingestion of groundwater as a source of local drinking water is not considered by the HHRAP as it is considered to be an insignificant exposure pathway for emissions derived from combustion processes.

The ingestion of drinking water from surface water sources is only considered a potential exposure pathway where there is a local surface water body which provides local drinking water. However, it is our experience that drinking water from a reservoir located close to this type of facility makes a very small contribution to the total exposure. Therefore, exposure via drinking water is generally only considered where there is the potential for exposure via the ingestion of fish and the presence of edible fish farms (e.g. trout or salmon farms). There are no edible fish farms identified within 3 km of the facility. The nearest fishing venue (Fighting Reservoir) is located to the east of the facility at a distance of around 2 km. However, these are stocked with coarse fish (carp, roach and perch). Therefore, it is a recreational fishing venue where fish are not normally taken for human consumption.

FIGURE 2.1 EXPOSURE PATHWAYS FOR RECEPTORS



On the basis of the assessment of the potential significance of the exposure pathways, the key exposure pathways which are relevant to the assessment and, hence, subject to examination in detail are as follows:

- inhalation;
- ingestion of food; and
- ingestion of soil.

Therefore, the exposures arising from ingestion are assessed with reference to the following:

- milk from home-reared cows;
- eggs from home-reared chickens;
- home-reared beef;
- home-reared pork;
- home-reared chicken;
- home-grown vegetable and fruit produce;
- breastmilk; and
- soil (incidental).

The inclusion of all food groups in the assessment conservatively assumes that both arable and pasture land are present in the vicinity of the predicted maximum annual average ground level concentration. This is, in reality, a highly unlikely scenario, but it has been included as a means of building a high degree of conservatism into the assessment and, hence, reducing the risk of exposures being underestimated. However, it should be noted that not all exposure scenarios will result in the ingestion of home-reared meat and animal products and these food products are only considered by the HHRAP for farmers and the families of farmers.

Similarly, the ingestion of fish is only considered where there is a local water body that is used for fishing and where the diet of the fisher (and family) may be regularly supplemented by fish caught from these local water sources. As discussed previously, there are no edible fish farms identified within 3 km of the facility. Therefore, the ingestion of locally caught edible fish from an inland closed water source has not been considered as consumption rates are likely to be very small.

2.4 BASELINE CONDITIONS

Dioxins and furans are ubiquitous in the environment and are present in air, soil and dietary products.

The latest assessment of dietary exposure to PCCD/Fs was documented in 2003 based on the 2001 Total Diet Study (TDS) [3]. This estimated that the average intake for adults decreased from 1.8 pg TEQ kg^{-1} d^{-1} (1997) to 0.9 pg TEQ kg^{-1} d^{-1} in 2001. For younger children, the average exposure decreased from 4.0 pg TEQ kg^{-1} d^{-1} to 1.8 pg TEQ kg^{-1} d^{-1}. These reductions were likely due to the significant reduction in emissions during the 1990s from waste incineration facilities.

The 2001 TDS is over twenty years old and there have been further reductions in emission since this study was published. This is evidenced by PCCD/F emissions data obtained from the UK National Atmospheric Emissions Inventory which indicates that total PCDD/F emissions in the UK decreased from 523 g TEQ a^{-1} in 1997 to 335 g TEQ a^{-1} in 2001 and further to 181 g TEQ a^{-1} in 2019.

An updated TDS was undertaken in 2012 [4] but this study did not consider dietary exposure to PCDD/Fs. The report provides the concentration of PCDD/Fs and dioxin-like PCBs in a range of food products. Using dietary intake data from the National Diet and Nutrition Survey [5] (NDNS) an estimate of the dietary exposure to PCDD/Fs has been calculated as follows.

- For each food group the ng TEQ kg^{-1} fat basis has been obtained from the 2001 and 2012 TDS for adults and children (4 to 10 years).
- The fat intake (%) for each receptor type (adults and children) has been obtained from the NDNS. Data for Years 5 to 6 were used corresponding with 2012. Data were normalised to 100%.
- The average daily fat intake was calculated based on a total fat intake of 67.8 g d^{-1} for an adult and 54.4 g d^{-1} for a child.
- The intake was calculated by multiplying the PCDD/F concentrations in food (ng TEQ kg^{-1}) by the intake g d^{-1} and then converting to units of pg TEQ kg^{-1} d^{-1}.

The results of this analysis are presented in *Annex C*. The analysis was also applied to the 2001 TDS to provide a comparison with published intakes. A summary of the results is presented in *Table 2.1*.

3 Dioxins and dioxin-like PCBs in the UK Diet: 2001 Total Diet Study Samples, Food Survey Information Sheet 38/03 (July 2003)

4 Organic Environmental Contaminants in the 2012 Total Diet Study Samples, Report to the Food Standards Agency, The Food and Environment Research Agency (December 2012)

5 https://www.gov.uk/government/statistics/ndns-results-from-years-7-and-8-combined

TABLE 2.1 COMPARISON OF PUBLISHED AND ESTIMATED INTAKES OF PCDD/FS AND DIOXIN-LIKE PCBS FOR 2001 AND 2012

Scenario	Adult (pg TEQ kg^{-1} d^{-1})	Child (pg TEQ kg^{-1} d^{-1})
2001 TDS Published	0.9	1.8
2001 Estimated Intake	0.68	1.70
2012 Estimated Intake	0.47	1.11
2012 Estimate normalised to 2001	**0.62**	**1.17**

The 2001 estimates are slightly lower than the published estimates, particularly for the adult. Therefore, the 2012 estimates have been normalised based on the difference between the published and estimated 2001 data. This results in 2012 daily intakes of 0.62 and 1.17 pg TEQ kg^{-1} d^{-1} for the adult and child, respectively.

2.5 EMISSIONS AND DISPERSION MODELLING INPUT DATA

2.5.1 Compounds of Potential Concern (COPCs)

The substances which have been considered in the assessment are referred to as the Compounds of Potential Concern (COPCs) and include the seventeen PCDD/F congeners that are known to be toxic (refer *Section 2.5.3*). In addition, the IRAP model includes two dioxin-like PCBs (Aroclor 1016 and Aroclor 1254). These comprise a mixture of congeners with one to four chlorine atoms for Aroclor 1016 with a chlorine content of 41% by mass (average of three chlorine atoms). Similarly, Aroclor 1254 has between four and seven chlorine atoms and a chlorine content of 54% by mass (average of five chlorine atoms).

2.5.2 Emission Parameters

There are two types of emission which have the potential to give rise to PCDD/F emissions and include the two pyrolysis heating units (A1a and A1b) and the two excess syngas boilers (A2a and A2b). Emission parameters (per unit) for the two pyrolysis heating units are as follows:

- stack height of 25 m above ground level;
- internal stack diameter of 0.8 m;
- emission temperature of 50°C.
- emission velocity of 10.2 m s^{-1}; and
- normalised flow rate of 4.34 Nm^3 s^{-1} (273K, dry and 11% O_2).

Emission parameters (per unit) for the two excess syngas boilers are as follows:

- stack height of 25 m above ground level;
- internal stack diameter of 0.81 m;
- emission temperature of 180°C.

- emission velocity of 10.2 m s^{-1}; and
- normalised flow rate of 3.55 Nm^3 s^{-1} (273K, dry and 11% O_2).

2.5.3 Emission Concentrations for the COPCs

The general term dioxins denotes a family of compounds, with each compound composed of two benzene rings interconnected with two oxygen atoms. There are 75 individual dioxins, with each distinguished by the position of chlorine or other halogen atoms positioned on the benzene rings. Furans are similar in structure to dioxins, but have a carbon bond instead of one of the two oxygen atoms connecting the two benzene rings. There are 135 individual furan compounds. Each individual furan or dioxin compound is referred to as a congener and each has a different toxicity and physical properties with regard to its atmospheric behaviour. It is important, therefore, that the exposure methodology determines the fate and transport of PCDD/Fs on a congener specific basis. It does this by accounting for the varying volatility of the congeners and their different toxicities. Consequently, information regarding the PCDD/F annual mean ground level concentrations on a congener specific basis is required.

For the purposes of the exposure assessment, the congener profile for the proposed facility is presented in *Table 2.2*, which is a standard profile for municipal waste incinerators derived by Her Majesty's Inspectorate of Pollution (HMIP), one of the predecessors of the Environment Agency. The international toxic equivalency factors are given and used to derive the toxic equivalent emission (I-TEQ). It is assumed that PCDD/F emissions are 0.06 ng I-TEQ Nm^{-3} (reference conditions 273K, dry and 11% O_2).

Information on dioxin-like PCB emissions has been obtained from the Best Available Techniques (BAT) Reference Document for Waste Incineration (BREF) [6]. Figure 8.118 of the BREF provides a comparison of measured concentrations of PCDD/F and dioxin-like PCBs from the same sample for a number of different types of waste incinerators. The highest dioxin-like PCB emission concentration was 0.06 ng I-TEQ Nm^3 for a UK municipal solid waste incinerator. The corresponding PCDD/F concentration was approximately 0.045 ng I-TEQ Nm^3. Therefore, as a worst-case, an emission concentration of 0.06×10^{-6} mg I-TEQ m^{-3} has been assumed for dioxin-like PCBs.

For the dioxin-like PCBs, a toxic equivalent factor (TEF) of 0.1 has been used to provide an actual emission concentration (i.e. 0.6×10^{-6} mg Nm^{-3}). The same equivalence factor has been used to convert the total actual dose back to the total toxic equivalent dose.

6 Best Available Techniques (BAT) Reference Document for Waste Incineration, JRC Science for Policy Report (December 2019)

TABLE 2.2 PCDD/F CONGENER PROFILE FOR THE PROPOSED FACILITY

Congener	Annual Mean Emission Concentration (ng Nm^{-3}) (a)	I-TEF toxic equivalent factors)	Annual Mean Emission Concentration (b) (ng I-TEQ Nm^{-3})
2,3,7,8-TCDD	0.0031	1.0	0.0019
1,2,3,7,8-PeCDD	0.025	0.5	0.0074
1,2,3,4,7,8-HxCDD	0.029	0.1	0.0017
1,2,3,7,8,9-HxCDD	0.021	0.1	0.0013
1,2,3,6,7,8-HxCDD	0.026	0.1	0.0016
1,2,3,4,6,7,8-HpCDD	0.17	0.01	0.0010
OCDD	0.40	0.001	0.00024
2,3,7,8-TCDF	0.028	0.1	0.0017
2,3,4,7,8-PeCDF	0.054	0.5	0.016
1,2,3,7,8-PeCDF	0.028	0.05	0.00084
1,2,3,4,7,8-HxCDF	0.22	0.1	0.013
1,2,3,7,8,9-HxCDF	0.0040	0.1	0.00024
1,2,3,6,7,8-HxCDF	0.081	0.1	0.0049
2,3,4,6,7,8-HxCDF	0.087	0.1	0.0052
1,2,3,4,6,7,8-HpCDF	0.44	0.01	0.0026
1,2,3,4,7,8,9-HpCDF	0.040	0.01	0.00024
OCDF	0.40	0.001	0.00024
Total (ng I-TEQ Nm^{-3})			**0.06**

(a) Congener profile from Table 7.2a DOE (1996) Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes Contract No. HMIP/CPR2/41/1/181

(b) Reference conditions of 273K, 1 atmosphere, dry and 11% O_2 and normalised to 0.06 ng I-TEQ Nm^{-3}

The emission rates for each substance as input to the IRAP model are provided in *Table 2.3.*

TABLE 2.3 PCDD/F EMISSION RATES USED IN THE IRAP MODEL

Congener	Emission Concentration (mg Nm^{-3})	Emission Rate (A1a and A1b) (g s^{-1})	Emission Rate (A2a and A2b) (g s^{-1})
2,3,7,8-TCDD	0.0019×10^{-6}	8.4×10^{-12}	6.6×10^{-12}
1,2,3,7,8-PeCDD	0.015×10^{-6}	6.7×10^{-11}	5.2×10^{-11}
1,2,3,4,7,8-HxCDD	0.017×10^{-6}	7.9×10^{-11}	6.2×10^{-11}
1,2,3,7,8,9-HxCDD	0.013×10^{-6}	5.7×10^{-11}	4.5×10^{-11}
1,2,3,6,7,8-HxCDD	0.016×10^{-6}	7.1×10^{-11}	5.5×10^{-11}
1,2,3,4,6,7,8-HpCDD	0.10×10^{-6}	4.6×10^{-10}	3.6×10^{-10}
OCDD	0.24×10^{-6}	1.1×10^{-9}	8.5×10^{-10}
2,3,7,8-TCDF	0.017×10^{-6}	7.6×10^{-11}	6.0×10^{-11}
2,3,4,7,8-PeCDF	0.032×10^{-6}	1.5×10^{-10}	1.1×10^{-10}
1,2,3,7,8-PeCDF	0.017×10^{-6}	7.6×10^{-11}	6.0×10^{-11}
1,2,3,4,7,8-HxCDF	0.13×10^{-6}	5.9×10^{-10}	4.6×10^{-10}
1,2,3,7,8,9-HxCDF	0.0024×10^{-6}	1.1×10^{-11}	8.5×10^{-12}
1,2,3,6,7,8-HxCDF	0.049×10^{-6}	2.2×10^{-10}	1.7×10^{-10}
2,3,4,6,7,8-HxCDF	0.052×10^{-6}	2.4×10^{-10}	1.9×10^{-10}
1,2,3,4,6,7,8-HpCDF	0.26×10^{-6}	1.2×10^{-9}	9.4×10^{-10}
1,2,3,4,7,8,9-HpCDF	0.024×10^{-6}	1.1×10^{-10}	8.5×10^{-11}
OCDF	0.24×10^{-6}	1.1×10^{-9}	8.5×10^{-10}
Aroclor 1016/1254	0.60×10^{-6}	2.7×10^{-9}	2.1×10^{-9}

2.6 DISPERSION MODELLING ASSUMPTIONS

The air quality assessment supporting the planning application for the facility has relied upon the use of ADMS to estimate ground level concentrations of pollutants. The HHRA model has been designed to accept output files from the US EPA ISC or AERMOD dispersion models, reflecting its North American origins and its need to follow the US EPA risk assessment protocol. The use of ADMS is consistent with the air quality assessment undertaken for the facility and the emissions data and model set up are identical to that carried out for the air quality assessment [1].

Therefore, to maintain consistency with the air quality assessment, it has been possible to use output from the ADMS model with IRAP using the following procedure:

- generation of ISC input files and output files for the study area;
- generation of ADMS output data using the approach outlined in the US EPA risk assessment protocol; and

- inserting the ADMS results into the ISC output files.

For the modelling, all emission properties, building heights, and other relevant factors were retained from the air quality assessment provided for the facility. As the health risk assessment requires information on the deposition of substances to surfaces as well as airborne concentrations of substances, the ADMS dispersion model has also been used to predict the following:

- the airborne concentration of vapour, particle and particle bound substances emitted;
- the wet deposition rate of particle and particle bound substances; and
- the dry deposition rate of vapour, particle and particle bound substances.

For dry deposition of particles and particle bound contaminants, a fixed deposition velocity of 0.01 m s^{-1} has been used. The facility will be equipped with particle filtration and the emitted particles are likely to be less than 1 -2 µm in diameter. For particles of this size, deposition velocities are likely to be of the order of 0.001 to 0.01 m s^{-1}. Therefore, as a worst-case, for the ADMS modelling a value of 0.01 m s^{-1} has been adopted. A gas dry deposition velocity of 0.005 m s^{-1} is used for the gas phase. For wet deposition, the following washout coefficients are used:

- Gas phase – washout coefficient A at 0.00016 and washout coefficient B of 0.64;
- Particle phase – washout coefficient A at 0.00028 and washout coefficient B of 0.64; and
- Particle bound phase – washout coefficient A at 0.00010 and washout coefficient B of 0.64.

2.7 DISPERSION MODELLING RESULTS

A summary of the key results from the ADMS dispersion model is presented in *Table 2.4*. These have been predicted using the 2022 Durham Tees Valley Airport meteorological data set. This year was selected, as out of the five years considered (2018 to 2022), it was the year that provided the highest predicted annual mean concentrations and deposition rates.

TABLE 2.4 MAXIMUM ANNUAL AVERAGE PARTICLE PHASE CONCENTRATIONS AND PARTICLE PHASE DEPOSITION RATES ESTIMATED BY ADMS

Pollutant	Max Annual Average Concentration (a)	Max Annual Average Deposition Rate (b)
	(fg m^{-3})	(ng m^{-2} $year^{-1}$)
2,3,7,8-TCDD	0.094	0.042
1,2,3,7,8-PeCDD	0.75	0.33
1,2,3,4,7,8-HxCDD	0.88	0.39
1,2,3,7,8,9-HxCDD	0.64	0.28
1,2,3,6,7,8-HxCDD	0.79	0.35
1,2,3,4,6,7,8-HpCDD	5.2	2.3
OCDD	12.2	5.4
2,3,7,8-TCDF	0.85	0.38
2,3,4,7,8-PeCDF	1.6	0.73
1,2,3,7,8-PeCDF	0.85	0.38
1,2,3,4,7,8-HxCDF	6.6	3.0
1,2,3,7,8,9-HxCDF	0.12	0.054
1,2,3,6,7,8-HxCDF	2.5	1.1
2,3,4,6,7,8-HxCDF	2.7	1.2
1,2,3,4,6,7,8-HpCDF	13.4	6.0
1,2,3,4,7,8,9-HpCDF	1.2	0.54
OCDF	12.2	5.4
Aroclor 1016/1254	30.5	13.6

(a) Where 1 fg m^{-3} is equal to 1×10^{-15} g m^{-3}

(b) Where 1 ng m^{-2} $year^{-1}$ is equal to 1×10^{-9} g m^{-2} $year^{-1}$

3 INPUT PARAMETERS FOR THE IRAP MODEL

3.1 INTRODUCTION

Exposure of an individual to a chemical may occur either by inhalation or ingestion (including food, water and soil). Of interest is the total dose of the chemical received by the individual through the combination of possible routes, and the IRAP model has been developed to estimate the dose received by the human body, often referred to as the external dose.

Exposure to COPCs is a function of the estimated concentration of the substance in the environmental media with which individuals may come into contact (i.e. exposure point concentrations) and the duration of contact. The concentration at the point of contact is itself a function of the transfer through air, soil, water, plants and animals that form part of the overall pathway. Exposure equations have been developed which combine exposure factors (e.g. exposure duration, frequency and medium intake rate) and exposure point concentrations. The dose equations therefore facilitate estimation of the received dose and account for the properties of the route of exposure, i.e. ingestion and inhalation.

For those substances that bio-accumulate, i.e. become more concentrated higher up the food chain, especially in body fats, the exposure to contaminated meat products and milk is of particular significance.

The IRAP model user has the ability to adjust some of the key exposure factors. An example is the diet of the receptor and the proportion of which is local produce, which may be contaminated. Obviously, if a nearby resident eats no food grown locally, then that person's diet cannot be contaminated by the emissions from the source, in this case the facility. It is conventional to investigate two types of receptor, a farmer and a resident. It is assumed that a farmer eats proportionately more locally grown food than a resident. Where the potential exists for the consumption of locally caught fish a fisher receptor may also be considered.

The receptor types can also be divided into adults and children. Children are important receptors because they tend to ingest soil and dusts directly and have lower body weights, so that the effect of the same dose is greater in the child than in the adult.

The IRAP model is designed to accept output files of airborne concentrations and deposition rates. From these, it proceeds to calculate the concentrations of the pollutants of concern in the environmental media, foodstuffs and the human receptor. The dose experienced by the human receptor can be compared to the tolerable daily intake (TDI) provided by the Committee on Toxicity for dioxins and dioxin like PCBs of 2 pg kg^{-1} d^{-1}.

The model requires a wide range of input parameters to be defined, these include:

- physical and chemical properties of the COPCs;
- site information, including site specific data; and
- receptor information – for each receptor type (e.g. adult or child, resident or farmer or fisher).

The HHRAP default values, which are incorporated into the IRAP model, have been used for the majority of these input values. These data are provided in the following sections.

3.2 INPUT PARAMETERS FOR THE COPCS

The IRAP model contains a database of physical and chemical parameters for each of the 206 COPCs. This database is based on default values provided by the HHRAP and all default values have been used for this assessment.

These parameters are used to determine how each of the COPCs behave in the environment and their presence and accumulation in various food products (meat, fish, animal products, vegetation, soil and water). For 2,3,7,8-TCDD (the most toxic of the PCDD/Fs), the default parameters are provided in *Table 3.1.*

TABLE 3.1 IRAP INPUT PARAMETERS FOR 2, 3, 7, 8-TCDD

Parameter Description	Symbol	Units	2,3,7,8-TCDD
Chemical abstract service number	CAS No.	-	1746-01-6
Molecular weight	MW	g $mole^{-1}$	322.0
Melting point of chemical	T_m	K	578.7
Vapour pressure	V_p	atm	1.97×10^{-12}
Aqueous solubility	S	mg L^{-1}	1.93×10^{-5}
Henry's Law constant	H	atm-m^3 mol^{-1}	3.29×10^{-5}
Diffusivity of COPC in air	D_a	cm^2 s^{-1}	0.104
Diffusivity of COPC in water	Dw	cm^2 s^{-1}	5.6×10^{-6}
Octanol-water partition coefficient	K_ow	-	6,309,573
Organic carbon-water partition coefficient	K_oc	mL g^{-1}	3,890,451
Soil-water partition coefficient	Kd_s	mL g^{-1}	38,904
Suspended sediments/surface water partition coefficient	Kd_sw	L kg^{-1}	291,784
Bed sediment/sediment pore water partition coefficient	Kd_bs	mL g^{-1}	155,618
COPC loss constant due to biotic and abiotic degradation	K_sg	a^{-1}	0.03
Fraction of COPC air concentration in vapour phase	f_v		0.664
Root concentration factor	RCF	mL g^{-1}	39,999

TABLE 3.1 IRAP INPUT PARAMETERS FOR 2, 3, 7, 8-TCDD

Parameter Description	Symbol	Units	2,3,7,8-TCDD
Plant-soil bioconcentration factor for below ground produce	br_root_veg	-	1.03
Plant-soil bioconcentration factor for leafy vegetables	br_leafy_veg	-	0.00455
Plant-soil bioconcentration factor for forage	br_forage	-	0.00455
COPC air-to-plant biotransfer factor for leafy vegetables	bv_leafy_veg	-	65,500
COPC air-to-plant biotransfer factor for forage	bv_forage	-	65,500
COPC biotransfer factor for milk	ba_milk	day kg^{-1}	0.0055
COPC biotransfer factor for beef	ba_beef	day kg^{-1}	0.026
COPC biotransfer factor for pork	ba_pork	day kg^{-1}	0.032
Bioconcentration factor for COPC in eggs	Bcf_egg	-	0.060
Bioconcentration factor for COPC in chicken	Bcf_chicken	-	3.32
Fish bioconcentration factor	BCF_fish	L kg^{-1}	34,400
Fish bioaccumulation factor	BAF_fish	L kg^{-1}	0
Biota-sediment accumulation factor	BSAF_fish	-	0.09
Plant-soil bioconcentration factor for grain	br_grain	-	0.00455
Plant-soil bioconcentration factor for eggs	br_egg	-	0.011
COPC biotransfer factor for chicken	ba_chicken	day kg^{-1}	0.019

3.3 SITE AND SITE SPECIFIC PARAMETERS

The IRAP health risk assessment model requires information relating to the location and its surroundings. The parameters required include the following.

- The fraction of animal feed (grain, silage and forage) grown on contaminated soils and quantity of animal feed and soil consumed by the various animal species considered.
- The interception fraction for above ground vegetation, forage and silage and length of vegetation exposure to deposition. The yield/standing crop biomass is also required.
- Input data for assessing the risks associated with exposure to breast milk, including:
    - body weight of infant;
    - exposure duration;
    - proportion of ingested COPC stored in fat;
    - proportion of mother's weight that is fat;

    - fraction of fat in breast milk;
    - fraction of ingested contaminant that is absorbed; and
    - half-life of dioxins in adults and ingestion rate of breast milk.
- Other physical parameters (e.g. soil dry bulk density, density of air, soil mixing zone depth).

For all of these parameters the IRAP/EPA HHRAP default values have been used and these are presented in *Annex A*.

Other site specific parameters are also required which are not provided by the IRAP model and include rainfall and average wind speeds. Rainfall and wind speed parameters were specified for the facility as follows:

- Annual average evapotranspiration rate of 43.4 cm a^{-1} (assumed to be 70% of total precipitation);
- Annual average precipitation of 62.0 cm a^{-1} (based on the average for the five year data set for the 2018 to 2022 meteorological data);
- Annual average irrigation of 0 cm a^{-1} since manual irrigation of crops in the UK is not generally required due to natural irrigation;
- Annual average runoff of 6.2 cm a^{-1} (assumed to be 10% of total precipitation);
- An annual average wind velocity of 4.5 m s^{-1} (average for the five years); and
- A time period over which deposition occurs of 30 years (the HHRAP default value).

3.4 RECEPTOR INFORMATION

Within the IRAP model there are three receptor types; Resident, Farmer and Fisher. Information relating to each receptor type (adult and/or child) is required by the model where these receptor types are used. The information required includes the following:

- Food (meat, dairy products, fish and vegetables), water and soil consumption rates for each receptor type. However, only Fishers are assumed to consume fish and only Farmers are assumed to consume locally reared animals and animal products.
- Fraction of contaminated food, water and soil which is consumed by each receptor type.
- Input data for the inhalation exposure including: inhalation exposure duration, inhalation exposure frequency, inhalation exposure time; and inhalation rate.

- Input data for the ingestion exposure including: exposure duration, exposure frequency, exposure time; and body weight of receptor.

For the purposes of this assessment the default IRAP/HHRAP parameters have been used mainly to define the characteristics of the receptors. The default input data are presented in *Annex B*. The only variation to this is the assumed body weight of a child receptor. The IRAP/HHRAP default value is 15 kg whereas in the UK a value of 20 kg is typically used. Therefore, a value of 20 kg has been used.

4 EXPOSURE ASSESSMENT

4.1 SELECTION OF RECEPTORS

In addition to defining specific locations for assessment, IRAP can be used to determine the location of the maximum impact over an area based on the results of the dispersion model. For each defined land-use area, IRAP selects the locations which represent the maximum predicted concentrations or deposition rates for the area selected. The locations of these various maxima are often co-located resulting in the selection of one to nine receptor locations per defined area. This approach is adopted by IRAP since the maximum receptor impact may occur at any one of the maximum concentration or deposition locations identified.

The nearest residential areas are located to the southeast of Darlington. Therefore, three areas of Darlington where residential exposure may occur have been defined (Firth Moor, Eastbourne and Heathfield).

The immediate area around the facility site is dominated by industrial activities. However, there are agricultural areas in close proximity to the east, north and south of the industrial site. It is assumed these areas are suitable for both arable and pasture use.

For each type of receptor up to nine locations are selected based on the maximum predicted airborne concentration, maximum predicted wet deposition rate and maximum dry deposition rate for the gas phase, particle phase and particle bound phase. For the assessment, five Farmer receptors and five Residential receptors have been assessed. It is considered that the likelihood of locally caught fish being consumed is low and fisher receptors have not been included in the assessment. For all of the receptor types, adult and child receptors have been considered. The locations of the Resident and Farmer receptors are described in *Table 4.1* and presented in *Figure 4.1*.

At other locations not specifically considered in the assessment, the predicted hazards and risks will be lower than predicted for the discrete receptors considered.

FIGURE 4.1 LOCATION OF THE RESIDENT AND FARMER RECEPTORS

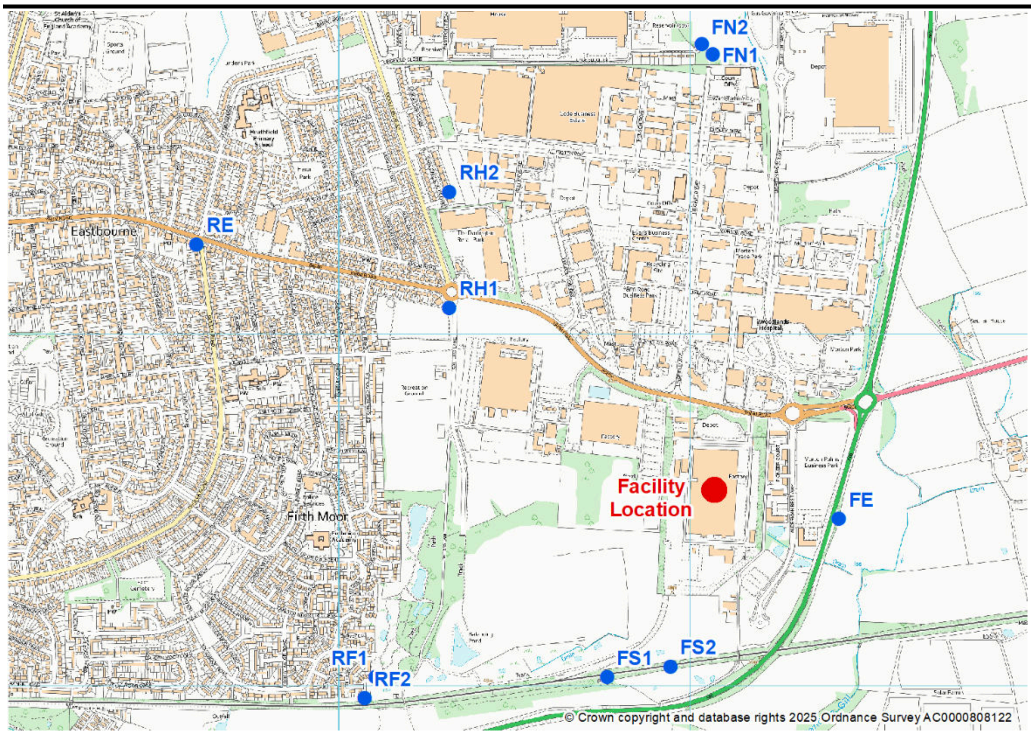


TABLE 4.1 DESCRIPTION OF RESIDENT AND FARMER RECEPTORS

Ref.	Name	Type	Easting	Northing
FE	Farmer East	Farmer	432425	513475
FN1	Farmer North 1	Farmer	432065	514795
FN2	Farmer North 2	Farmer	432035	514825
FS1	Farmer South 1	Farmer	431765	513025
FS2	Farmer South 2	Farmer	431945	513055
RE	Resident Eastbourne	Resident	430595	514255
RF1	Resident Firth Moor 1	Resident	431105	513025
RF2	Resident Firth Moor 2	Resident	431075	512965
RH1	Resident Heathfield 1	Resident	431315	514075
RH2	Resident Heathfield 2	Resident	431315	514405

4.2 ASSESSMENT OF INTAKE

4.2.1 Ingestion Dose

The ingestion intake is calculated as the Average Daily Dose (ADD) from all ingestion exposure routes (e.g. soil, above ground vegetables, meat and dairy products) where for example:

$$ADD_{Ing,\,TCDD} = \frac{I_{Ing,\,TCDD} \bullet ED \bullet EF}{AT \bullet 365}$$

Where: $ADD_{Ing,\ TCDD}$ = total ingestion dose for TCDD; ED is the exposure duration (dependent on the receptor type); EF is the exposure frequency (350 days per year); and AT is the averaging time, and for determining the TDI, is assumed to be equal to the ED. The total dose is the sum of the dose for each of the individual congeners.

4.2.2 Inhalation Dose

For inhalation, the ADD from inhalation exposure is calculated as follows:

$$ADD_{Inh,\,TCDD} = \frac{C_a \bullet IR \bullet ED \bullet EF}{AT \bullet 365}$$

Where: $ADD_{Inh,\ TCDD}$ is the total inhalation does for TCDD, C_a is the concentration of TCDD in air and IR is the daily inhalation rate. The total dose is the sum of the dose for each of the individual congeners.

4.3 EXPOSURE TO DIOXINS AND FURANS

4.3.1 Comparison of Dioxin/Furan Exposure with WHO and UK COT Guidance

Facility Contribution to Intake

The World Health Organization (WHO) recommends a tolerable daily intake for dioxins/furans of 1 to 4 pg I-TEQ $kg\text{-}BW^{-1}\ d^{-1}$ (picogrammes as the International Toxic Equivalent per kilogram bodyweight per day) [7]. The TDI represents the tolerable daily intake for lifetime exposure and short-term excursions above the TDI would have no consequence provided that the average intake over long periods is not exceeded. The average (lifetime) daily intake of dioxins/furans for the receptors considered is presented in *Table 4.2*. These are also compared to the Committee on Toxicity (COT) TDI for dioxins and dioxin-like PCBs of 2 pg I-TEQ $kg\text{-}BW^{-1}\ d^{-1}$.

7 Assessment of the Health Risk of Dioxins: Re-evaluation of the Tolerable Daily Intake (TD), WHO Consultation, May 25-29 1998, Geneva, Switzerland

TABLE 4.2 COMPARISON OF AVERAGE DAILY INTAKES WITH THE UK COT AND WHO'S TDI FOR DIOXINS/FURANS (pg I-TEQ kg-BW^{-1} d^{-1})

Receptor Name	Adult		Child	
	pg I-TEQ kg-BW^{-1} d^{-1}	%age of COT TDI	pg I-TEQ kg-BW^{-1} d^{-1}	%age of COT TDI
Farmer East	0.062	3.1%	0.091	4.6%
Farmer North 1	0.028	1.4%	0.041	2.1%
Farmer North 2	0.028	1.4%	0.040	2.0%
Farmer South 1	0.039	1.9%	0.057	2.9%
Farmer South 2	0.036	1.8%	0.053	2.6%
Resident Eastbourne	0.00025	<0.1%	0.00069	<0.1%
Resident Firth Moor 1	0.00054	<0.1%	0.0014	0.1%
Resident Firth Moor 2	0.00053	<0.1%	0.0014	0.1%
Resident Heathfield 1	0.00080	<0.1%	0.0022	0.1%
Resident Heathfield 2	0.00077	<0.1%	0.0021	0.1%
WHO TDI	*1 to 4 pg I-TEQ kg-BW^{-1} d^{-1}*			
Committee on Toxicity (COT) TDI	*2 pg I-TEQ kg-BW^{-1} d^{-1}*			

The maximum contribution from the facility to the COT TDI is 4.6% for the Farmer East child receptor and 3.1% for the Farmer East adult receptor. This assumes as a worst-case that these receptors produce their own home reared and home-grown food at the location of maximum impact for the area. Furthermore, this assumes that both arable land and pastureland are available at this location. Therefore, it is considered that the predicted impacts for this receptor represent a very worst-case.

For the residential receptors, the maximum contribution of the facility to the COT TDI is 0.1% for Resident Heathfield 1 child receptor and <0.1% for the Resident Heathfield 1 adult receptor. This assumes that these residents grow and consume all of their above ground vegetables.

Total Intake

The contribution of the facility to total intake is provided as follows:

- predicted incremental intake due to emissions from the facility;
- average daily background intake (i.e. that arising from other sources);
- the total intake (i.e. the sum of the predicted incremental intake and the background intake);
- a comparison of the total intake with the TDI for dioxin/furans.

A comparison of predicted intakes with the background intake and the TDI is presented in *Table 4.3*. Results are presented for Farmer East and Resident Heathfield 1 where highest farmer and resident exposures are predicted.

TABLE 4.3 COMPARISON OF TOTAL INTAKE WITH THE COT TDI

Receptor	Total Intake from the Facility (pg I-TEQ kg^{-1} d^{-1})	Total Intake Facility + Background (pg I-TEQ kg^{-1} d^{-1})	Facility as %age of TDI	Total Intake as %age of TDI
Farmer East Adult	0.062	0.68	3.1%	34.1%
Farmer East Child	0.091	1.26	4.6%	63.1%
Resident Heathfield 1 Adult	0.00080	0.62	<0.1%	31.0%
Resident Heathfield 1 Child	0.0022	1.17	0.1%	58.6%
COT TDI	*2*	*2*	-	-

For inhalation and oral intake of PCDD/Fs for adults, total intake is well below the TDI. Background exposure represents approximately 31% of total exposure. At worst, the facility contributes 3.1% to the TDI for adults and the total intake is 34.1% of the TDI for this receptor.

For inhalation and oral intake of PCDD/Fs for children, the background intake is relatively high at 59% of the TDI. At worst, the additional contribution from the facility for a child is 0.091 pg TEQ kg^{-1} d^{-1} (4.6% of the COT TDI). Combined with the background exposure for a child (1.17 pg TEQ kg^{-1} d^{-1}) the total intake would be well below the TDI (63.1%). Furthermore, it should be noted that the TDI for PCCD/Fs is set for the purposes of assessing lifetime exposure and these elevated background exposures for children are not representative of long-term exposure. Therefore, taking into account the extreme worst-case assumptions adopted for farmer receptors, it is concluded that the contribution of the facility to total intake would be not significant.

4.3.2 Infant Breast Milk Exposure to Dioxins and Furans

Another exposure pathway of interest is infant exposure to dioxins and furans via the ingestion of their mother's breast milk. This is because the potential for contamination of breast milk is particularly high for dioxin-like compounds such as these, as they are lipophilic (fat soluble) and hence likely to accumulate in breast milk. Further, the infant body weight is smaller and it could be argued that the effect is proportionately greater than in an adult.

This exposure is measured by the Average Daily Dose (ADD) on the basis of an averaging time of one year. In the US, a threshold value of 50 pg kg^{-1} d^{-1} of 2,3,7,8-TCDD TEQ is cited as being potentially harmful. The IRAP model calculates the ADD that would result from an adult receptor breast feeding an infant. It should be noted that the ADD from breast feeding calculated by IRAP does not consider dioxin-like PCBs. However, the dioxin-like PCB emission is a small fraction of the total emission and the inclusion of dioxin-like PCBs would not result in a significant increase in the ADD from breast feeding.

A summary of the ADD for each of the infants of adult receptors considered for the assessment is presented in *Table 4.4.*

TABLE 4.4 ASSESSMENT OF THE AVERAGE DAILY DOSE FOR A BREAST-FED INFANT OF AN ADULT RECEPTOR

Receptor Name	Average Daily Dose from Breast Feeding (pg kg^{-1} d^{-1} of 2,3,7,8-TCDD)	Percentage of US EPA Criterion	Percentage of COT TDI
Farmer East	0.58	1.2%	29.2%
Farmer North 1	0.26	0.5%	13.1%
Farmer North 2	0.26	0.5%	12.8%
Farmer South 1	0.35	0.7%	17.6%
Farmer South 2	0.32	0.6%	16.1%
Resident Eastbourne	0.0011	<0.1%	0.1%
Resident Firth Moor 1	0.0023	<0.1%	0.1%
Resident Firth Moor 2	0.0023	<0.1%	0.1%
Resident Heathfield 1	0.0036	<0.1%	0.2%
Resident Heathfield 2	0.0034	<0.1%	0.2%
US EPA Criterion	*50*		
WHO criterion	*1 to 4*		
UK criterion (COT)	*2*		

The highest ADDs are calculated for the infants of farmer receptors and, in the absence of UK criterion for assessing exposure to dioxin-like compounds in breast milk, represent at worst 1.2% of the US EPA criterion of 50 pg kg^{-1} d^{-1} of 2,3,7,8-TCDD. The calculated ADDs for residential receptors are lower (less than 0.1% of the US EPA criterion) compared to the farmer receptors since the most significant exposure to dioxins/furans is via the food chain, particularly animals and animal products. The farmer receptors are assumed to consume contaminated meat and dairy products. However, the residential receptors are only assumed to consume vegetable products which are less significant with regard to exposure to dioxins/furans.

As a worst case, the ADD for the highest exposure for the infants of farmers (Farmer East) is 29.2% of the COT TDI. For these receptors it is assumed, as a worst-case, that all of the food consumed by their mothers is reared and grown locally at the location of maximum impact in the area. However, this represents an extreme worst-case. Furthermore, the duration of exposure is short and the average daily intake over the lifetime of the individual would be substantially less.

Taking into account the extreme worst-case basis for the assessment, it is concluded that infant exposure to breast milk would be not significant. Furthermore, the WHO recognises that breast-fed infants will be exposed to higher intakes for a short duration, but also that breast feeding itself provides associated benefits.

4.4 ABNORMAL EMISSIONS

The impact of abnormal emissions on long-term impacts has been assessed assuming that during abnormal conditions (which can occur for 60 hours per annum) the emission limit is exceeded by a factor of 100. It is considered that this is an extreme worst-case. On this basis, the dioxin and furan emission concentration averaged over one year would be 0.10 ng TEQ Nm^{-3} (8700 hours at 0.06 ng TEQ Nm^{-3} and 60 hours at 6 ng TEQ Nm^{-3}).

The impact of abnormal emissions on the long-term exposure to dioxins and furans is provided in *Table 4.5*.

TABLE 4.5 COMPARISON OF AVERAGE DAILY INTAKES WITH THE UK COT AND WHO'S TDI FOR DIOXINS/FURANS (pg I-TEQ kg-BW^{-1} d^{-1}) – ABNORMAL EMISSIONS

Receptor Name	Adult	Child
Farmer East	0.11	0.16
Farmer North 1	0.048	0.071
Farmer North 2	0.047	0.069
Farmer South 1	0.066	0.098
Farmer South 2	0.062	0.090
Resident Eastbourne	0.00043	0.0012
Resident Firth Moor 1	0.00092	0.0025
Resident Firth Moor 2	0.00091	0.0024
Resident Heathfield 1	0.0014	0.0037
Resident Heathfield 2	0.0013	0.0035
WHO TDI	*1 to 4 pg I-TEQ kg-BW^{-1} d^{-1}*	
Committee on Toxicity (COT) TDI	*2 pg I-TEQ kg-BW^{-1} d^{-1}*	

For these abnormal conditions, the maximum contribution of the facility to the COT TDI is 7.8% for the Farmer East child receptor and 0.2% for the Resident Heathfield 1 child receptor. Highest exposure is for a farmer receptor assumed to be exposed for a lifetime to the effects of the highest airborne concentrations and consuming mostly locally grown food at the location of maximum impact. This is an extremely conservative assumption. Therefore, taking into consideration the worst-case assumptions adopted for the assessment, it is concluded that the impact of abnormal emissions on long-term exposure to dioxins and furans would be not significant.

5 SUMMARY AND CONCLUSIONS

5.1 SUMMARY

The possible impacts on human health arising from dioxins and furans (PCDD/F) and dioxin-like PCBs emitted from the proposed facility have been assessed under the worst-case scenario, namely that of an individual exposed for a lifetime to the effects of the highest airborne concentrations and consuming mostly locally grown food. This equates to a hypothetical farmer consuming food grown on the farm, situated at the closest proximity to the facility. Where there are no active farming areas in close proximity, a residential receptor is considered where it is assumed that the resident consumes locally grown vegetables.

The assessment has identified and considered the most plausible pathways of exposure for the individuals considered (farmer and resident). Deposition and subsequent uptake of the compounds of potential concern (COPCs) into the food chain is likely to be the more numerically significant pathway over direct inhalation.

The maximum contribution of the facility to the COT TDI is 4.6% for the farmer receptors and 0.1% for the residential receptors. For the farmer this assumes as a worst-case that these receptors are located at the closest farming area to the facility and all of their food is reared and grown at this location and represents an extreme worst-case. Therefore, taking into account the worst-case assumptions, the impact of emissions on local sensitive receptors is considered to be not significant.

5.2 CONCLUSIONS

The risk assessment methodology used in this assessment has been structured so as to create worst case estimates of risk. A number of features in the methodology give rise to this degree of conservatism. It has been demonstrated that for the maximally exposed individual, exposure to dioxins, furans and dioxin-like PCBs is not significant.

ANNEX A

SITE PARAMETERS

Annex A: Site Parameters Defined for the Health Risk Assessment

Parameter	Parameter Value	IRAP Symbol	Units
Soil dry bulk density	1.5	bd	g cm^{-3}
Forage fraction grown on contam. soil eaten by CATTLE	1.0	beef_fi_forage	--
Grain fraction grown on contam. soil eaten by CATTLE	1.0	beef_fi_grain	--
Silage fraction grown on contam. eaten by CATTLE	1.0	beef_fi_silage	--
Qty of forage eaten by CATTLE each day	8.8	beef_qp_forage	kg DW d^{-1}
Qty of grain eaten by CATTLE each day	0.47	beef_qp_grain	kg DW d^{-1}
Qty of silage eaten by CATTLE each day	2.5	beef_qp_silage	kg DW d^{-1}
Grain fraction grown on contam. soil eaten by CHICKEN	1.0	chick_fi_grain	--
Qty of grain eaten by CHICKEN each day	0.2	chick_qp_grain	kg DW d^{-1}
Fish lipid content	0.07	f_lipid	--
Fraction of CHICKEN's diet that is soil	0.1	fd_chicken	--
Universal gas constant	8.205e-5	gas_r	atm-m^3 mol^{-1} K^{-1}
Plant surface loss coefficient	18	kp	a^{-1}
Fraction of mercury emissions NOT lost to the global cycle	0.48	merc_q_corr	--
Fraction of mercury speciated into methyl mercury in produce	0.22	mercmethyl_ag	--
Fraction of mercury speciated into methyl mercury in soil	0.02	mercmethyl_sc	--
Forage fraction grown contam. soil, eaten by MILK CATTLE	1.0	milk_fi_forage	--
Grain fraction grown contam. soil, eaten by MILK CATTLE	1.0	milk_fi_grain	--
Silage fraction grown contam. soil, eaten by MILK CATTLE	1.0	milk_fi_silage	--
Qty of forage eaten by MILK CATTLE each day	13.2	milk_qp_forage	kg DW d^{-1}
Qty of grain eaten by MILK CATTLE each day	3.0	milk_qp_grain	kg DW d^{-1}
Qty of silage eaten by MILK CATTLE each day	4.1	milk_qp_silage	kg DW d^{-1}
Averaging time	1	milkfat_at	a
Body weight of infant	9.4	milfat_bw_infant	kg
Exposure duration of infant to breast milk	1	milkfat_ed	a
Proportion of ingested dioxin that is stored in fat	0.9	milkfat_f1	--
Proportion of mothers weight that is fat	0.3	milkfat_f2	--
Fraction of fat in breast milk	0.04	milkfat_f3	--
Fraction of ingested contaminant that is absorbed	0.9	milkfat_f4	--
Half-life of dioxin in adults	2555	milkfat_h	d
Ingestion rate of breast milk	0.688	milkfat_ir_milk	kg d^{-1}
Viscosity of air corresponding to air temp.	1.81e-04	mu_a	g cm^{-1} s^{-1}
Fraction of grain grown on contam. soil eaten by PIGS	1.0	pork_fi_grain	--
Fraction of silage grown on contam. soil and eaten by PIGS	1.0	pork_fi_silage	--
Qty of grain eaten by PIGS each day	3.3	pork_qp_grain	kg DW d^{-1}
Qty of silage eaten by PIGS each day	1.4	pork_qp_silage	kg DW d^{-1}
Qty of soil eaten by CATTLE	0.5	qs_beef	kg d^{-1}
Qty of soil eaten by CHICKEN	0.022	qs_chick	kg d^{-1}
Qty of soil eaten by DAIRY CATTLE	0.4	qs_milk	kg d^{-1}
Qty of soil eaten by PIGS	0.37	qs_pork	kg d^{-1}
Density of air	1.2e-3	rho_a	g cm^{-3}
Solids particle density	2.7	rho_s	g cm^{-3}
Interception fraction - edible portion ABOVEGROUND	0.39	rp	--
Interception fraction - edible portion FORAGE	0.5	rp_forage	--
Interception fraction - edible portion SILAGE	0.46	rp_silage	--
Ambient air temperature	298	t	K
Temperature correction factor	1.026	theta	--
Soil volumetric water content	0.2	theta_s	mL cm^{-3}
Length of plant expos. to depos. - ABOVEGROUND	0.16	tp	a
Length of plant expos. to depos. - FORAGE	0.12	tp_forage	a
Length of plant expos. to depos. - SILAGE	0.16	tp_silage	a
Average annual wind speed	3.9	u	m s^{-1}
Dry deposition velocity	0.5	vdv	cm s^{-1}
Dry deposition velocity for mercury	2.9	vdv_hg	cm s^{-1}
Wind velocity	3.9	w	m s^{-1}
Yield/standing crop biomass - edible portion ABOVEGROUND	2.24	yp	kg DW m^{-2}
Yield/standing crop biomass - edible portion FORAGE	0.24	yp_forage	kg DW m^{-2}
Yield/standing crop biomass - edible portion SILAGE	0.8	yp_silage	kg DW m^{-2}
Soil mixing zone depth	2.0	z	cm

ANNEX B

SCENARIO PARAMETERS

Annex B: Exposure Scenario Parameters

Parameter Description	Adult Resident	Child Resident	Adult Farmer	Child Farmer	Adult Fisher	Child Fisher	Units
Averaging time for carcinogens	70	70	70	70	70	70	a
Averaging time for noncarcinogens	30	6	40	6	30	6	a
Consumption rate of BEEF	0.0	0.0	0.00122	0.00075	0.0	0.0	kg kg^{-1} FW d^{-1}
Body weight	70	15	70	15	70	15	kg
Consumption rate of POULTRY	0.0	0.0	0.00066	0.00045	0.0	0.0	kg kg^{-1} FW d^{-1}
Consumption rate of ABOVEGROUND PRODUCE	0.00032	0.00077	0.00047	0.00113	0.00032	0.00077	kg kg^{-1} DW d^{-1}
Consumption rate of BELOWGROUND PRODUCE	0.00014	0.00023	0.00017	0.00028	0.00014	0.00023	kg kg^{-1} DW d^{-1}
Consumption rate of DRINKING WATER	1.4	0.67	1.4	0.67	1.4	0.67	L d^{-1}
Consumption rate of PROTECTED ABOVEGROUND PRODUCE	0.00061	0.0015	0.00064	0.00157	0.00061	0.0015	kg kg^{-1} DW d^{-1}
Consumption rate of SOIL	0.0001	0.0002	0.0001	0.0002	0.0001	0.0002	kg d^{-1}
Exposure duration	30	6	40	6	30	6	yr
Exposure frequency	350	350	350	350	350	350	d a^{-1}
Consumption rate of EGGS	0.0	0.0	0.00075	0.00054	0.0	0.0	kg kg^{-1} FW d^{-1}
Fraction of contaminated ABOVEGROUND PRODUCE	1.0	1.0	1.0	1.0	1.0	1.0	--
Fraction of contaminated DRINKING WATER	1.0	1.0	1.0	1.0	1.0	1.0	--
Fraction contaminated SOIL	1.0	1.0	1.0	1.0	1.0	1.0	--
Consumption rate of FISH	0.0	0.0	0.0	0.0	0.00125	0.00088	kg kg^{-1} FW d^{-1}
Fraction of contaminated FISH	1.0	1.0	1.0	1.0	1.0	1.0	--
Inhalation exposure duration	30	6	40	6	30	6	a
Inhalation exposure frequency	350	350	350	350	350	350	d a^{-1}
Inhalation exposure time	24	24	24	24	24	24	h d^{-1}
Fraction of contaminated BEEF	1	1	1	1	1	1	--
Fraction of contaminated POULTRY	1	1	1	1	1	1	--
Fraction of contaminated EGGS	1	1	1	1	1	1	--
Fraction of contaminated MILK	1	1	1	1	1	1	--
Fraction of contaminated PORK	1	1	1	1	1	1	--
Inhalation rate	0.83	0.30	0.83	0.30	0.83	0.30	m^3 h^{-1}
Consumption rate of MILK	0.0	0.0	0.01367	0.02268	0.0	0.0	kg kg^{-1} FW d^{-1}
Consumption rate of PORK	0.0	0.0	0.00055	0.00042	0.0	0.0	kg kg^{-1} FW d^{-1}
Time period at the beginning of combustion	0	0	0	0	0	0	a
Length of exposure duration	30	6	40	6	30	6	a

ANNEX C

ESTIMATION OF 2012 BACKGROUND PCDD/F INTAKES

Calculation of Dietary Intake of PCDD/Fs and Dioxin-like PCBs

Adult - 70 kg

Foodstuff	ng/kg WHO TEQ/kg fat basis 2001	ng/kg fat WHO TEQ upper 2012	NDNS Years 5-6 Total Fat Intake %	NDNS Years 5-6 Total Fat Intake % Normalised	Average Daily Fat Intake (g/d)	Intake pgTEQ/kgBW/d 2001 but 2012 Diet	Intake pgTEQ/kgBW/d 2012	Intake Normalised for 2001 Discrepancy 2012
Bread	0.35	0.277	4.2	4.6	3.1	0.0155	0.0123	0.016
Cereals	0.26	0.134	17.1	18.6	12.6	0.0469	0.0241	0.032
Carcass Meat	0.73	0.534	6.3	6.9	4.6	0.0485	0.0355	0.047
Offal	7.32	1.925	0.2	0.2	0.1	0.0154	0.0041	0.005
Meat Products	0.42	0.203	9.7	10.6	7.2	0.0429	0.0208	0.027
Poultry	0.71	0.148	6.1	6.6	4.5	0.0456	0.0095	0.013
Fish	4.63	3.499	4.6	5.0	3.4	0.2245	0.1696	0.224
fats & Oils	0.19	0.124	9.7	10.6	7.2	0.0194	0.0127	0.017
Eggs	0.44	0.463	4.3	4.7	3.2	0.0199	0.0210	0.028
Sugar	0.45	0.919	3.8	4.1	2.8	0.0180	0.0368	0.049
Green Vegetables	0.84	1.577	0.5	0.5	0.4	0.0044	0.0083	0.011
Potatoes	0.4	0.186	5.2	5.7	3.8	0.0219	0.0102	0.013
Other vegetables	0.37	0.965	1.65	1.8	1.2	0.0064	0.0168	0.022
Canned vegetables	0.45	0.392	1.65	1.8	1.2	0.0078	0.0068	0.009
Fresh Fruit	0.95	1.535	0.45	0.5	0.3	0.0045	0.0073	0.010
Fruit Products	1.26	1.778	0.45	0.5	0.3	0.0060	0.0084	0.011
Milk	0.9	0.421	5	5.4	3.7	0.0474	0.0222	0.029
Milk& Dairy Products	0.89	0.452	8.7	9.5	6.4	0.0816	0.0414	0.055
Nuts	0.2	0.045	2.3	2.5	1.7	0.0048	0.0011	0.001
			91.9	100	67.8	0.68	0.47	0.62

Child - 20 kg, 4 to 10 years

Foodstuff	ng/kg WHO TEQ/kg fat basis 2001	ng/kg fat WHO TEQ upper 2012	NDNS Years 5-6 Total Fat Intake %	NDNS Years 5-6 Total Fat Intake % Normalised	Average Daily Fat Intake (g/d)	Intake pgTEQ/kgBW/d 2001 but 2012 Diet	Intake pgTEQ/kgBW/d 2012	Intake Normalised for 2001 Discrepancy 2012
Bread	0.35	0.277	4.0	4.3	2.4	0.0413	0.0327	0.034
Cereals	0.26	0.134	21.0	22.8	12.4	0.1609	0.0829	0.088
Carcass Meat	0.73	0.534	3.6	3.9	2.1	0.0774	0.0567	0.060
Offal	7.32	1.925	0.1	0.1	0.1	0.0216	0.0057	0.006
Meat Products	0.42	0.203	9.5	10.3	5.6	0.1176	0.0568	0.060
Poultry	0.71	0.148	5.3	5.7	3.1	0.1109	0.0231	0.024
Fish	4.63	3.499	2.7	2.9	1.6	0.3684	0.2784	0.294
fats & Oils	0.19	0.124	8.9	9.6	5.2	0.0498	0.0325	0.034
Eggs	0.44	0.463	2.1	2.3	1.2	0.0272	0.0287	0.030
Sugar	0.45	0.919	4.9	5.3	2.9	0.0650	0.1327	0.140
Green Vegetables	0.84	1.577	0.4	0.4	0.2	0.0099	0.0186	0.020
Potatoes	0.4	0.186	5.8	6.3	3.4	0.0684	0.0318	0.034
Other vegetables	0.37	0.965	0.9	1.0	0.5	0.0098	0.0256	0.027
Canned vegetables	0.45	0.392	0.9	1.0	0.5	0.0119	0.0104	0.011
Fresh Fruit	0.95	1.535	0.3	0.3	0.2	0.0084	0.0136	0.014
Fruit Products	1.26	1.778	0.3	0.3	0.2	0.0111	0.0157	0.017
Milk	0.9	0.421	10.6	11.5	6.2	0.2811	0.1315	0.139
Milk& Dairy Products	0.89	0.452	9.8	10.6	5.8	0.2570	0.1305	0.138
Nuts	0.2	0.045	1.2	1.3	0.7	0.0071	0.0016	0.002
			92.3	100	54.4	1.70	1.11	1.17

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