Kronospan Low Carbon CHP Facility

Appendix 6E

Dioxin Pathway Intake Assessment

Prepared for: Kronospan

December 2025

DNS5-4-026

1.0 INTRODUCTION

1.1.1 As the fuel combusted in the CHP Facility will be sourced from waste wood, the emission limit values (ELVs) will be set in the EP based on those outlined in Chapter IV and Annex VI of the Industrial Emissions Directive (IED) (2010/75/EU) for waste incineration and co-incineration plants and the and the Waste Incineration BREF[1]. This includes limits on emissions of dioxins and furans (collectively referred to as "dioxins" for the purpose of this assessment). The existing EP also includes ELVs for dioxins for the K8 biomass plant. The ELVs for the K8 biomass plant and those proposed for the CHP Facility are different as the K8 biomass plant is classified as an "existing plant" whilst the CHP Facility would be a "new plant".

1.1.2 The advice from health specialists such as the UK Health Security Agency (formerly the Health Protection Agency, "HPA") is that the damage to health from emissions from incineration and co-incineration plants is likely to be very small, and probably not detectable. Nevertheless, the specific effects on human health of the CHP Facility have been considered. This includes a review of published literature on the health effects of energy recovery facilities, and a quantitative assessment of the effect of the CHP Facility in combination with the existing K8 biomass plant.

1.1.3 For most substances released from the CHP Facility, the most significant effects on human health will arise by inhalation. However, for dioxins and dioxin-like polychlorinated biphenyls (PCBs) which accumulate in the environment, inhalation is only one of the potential exposure routes.

[1] *Best Available Techniques (BAT) Reference Document for Waste Incineration (2019)*

1.1.4 For dioxins and dioxin-like PCBs the health assessment criteria are expressed as the total intake from ingestion and inhalation. Therefore, this assessment considers exposure routes other than just inhalation.

2.0 LITERATURE REVIEW

2.1.1 The HPA, whose role was taken over by Public Health England (PHE) and more recently by the UK Health Security Agency, published a note RCE-13 "The Impact on Health of Emissions to Air from Municipal Waste Incinerators", in 2009[2] . The summary states:

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

2.1.2 PHE commissioned further research in 2012, while continuing to state that the conclusions of RCE-13 remain applicable. These studies were commissioned from the Small Area Health Statistics Unit, which is based at Imperial College London and Kings College London. The methodology and results of the studies have been published in a series of papers in scientific journals. The three most recent papers, known as Ghosh et al (2018)[3], Freni-Sterrantino et al (2019)[4] and Parkes et al (2019)[5], are the most relevant.

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

2.1.4 Ghosh et al (2018) concluded that:

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

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[2] https://www.gov.uk/government/publications/municipal-waste-incinerators-emissions-impact-on-health
[3] Ghosh RE, Freni Sterrantino A, Douglas P, Parkes B, Fecht D, de Hoogh K, Fuller G, Gulliver J, Font A, Smith RB, Blangiardo M, Elliott P, Toledano MB, Hansell AL. (2018) Fetal growth, stillbirth, infant mortality and other birth outcomes near UK municipal waste incinerators; retrospective population based cohort and case-control study. Environment International.
[4] Freni-Sterrantino, A; Ghosh, RE; Fecht, D; Toledano, MB; Elliott, P; Hansell, AL; Blangiardo, M. (2019) Bayesian spatial modelling for quasi-experimental designs: An interrupted time series study of the opening of
[5] Parkes B, Hansell A.L., Ghosh R.E, Douglas P., Fecht D., Wellesley D., Kurinczuk J.J., Rankin J., de Hoogh K., Fuller G.W, Elliot P., and Toledano M.B. "Risk of congenital anomalies near municipal waste incinerators in England and Scotland: Retrospective population-based cohort study". Environment International (Parkes et al).

2.1.5 Freni-Sterrantino et al (2019) concluded that:

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

2.1.6 The objective of Parkes et al (2019) was as follows:

"To conduct a national investigation into the risk of congenital anomalies in babies born to mothers living within 10 km of an MWI associated with: i) modelled concentrations of PM_{10} as a proxy for MWI emissions more generally and; ii) proximity of residential postcode to nearest MWI, in areas in England and Scotland that are covered by a congenital anomaly register."

2.1.7 Under objective (i), which related congenital anomalies to modelled concentrations and so would be considered the more representative approach, the study found no association with congenital abnormalities. Under objective (ii), there was a small excess risk, but the paper's authors note that this may be due to residual confounding.

2.1.8 The Imperial College website includes Frequently Asked Questions on this study. One of these is "Does the study show that MWIs are causing increased congenital anomalies in populations living nearby?" The answer is as follows.

"No. The study does not say that the small excess risks associated with congenital heart disease and genital anomalies in proximity to MWIs are caused by those MWIs, as these results may be explained by residual confounding factors i.e., other influences which it was not possible to take into account in the study. This possible explanation is supported further by the fact that the study found no increased risk in congenital anomalies due to exposure to emissions from incinerators."

2.1.9 A further study by Parsons et al (2024)[6] aimed to measure levels of dioxins and PCBs in mothers' milk, with the measurements being taken between 2013 and 2015. This concluded that there was no detectable change in dioxin and PCB levels for individuals residing closer to their nearest MWI. However, when the modelled PM_{10} from the MWIs was considered (as a spatial proxy for other pollutants), a doubling of mean MWI modelled PM_{10} contribution resulted in small (<10%) increases in dioxin and furan levels in breast milk. The study concludes that although diet is the main source of exposure, MWI emissions may make a small contribution to the body burden of dioxins and PCBs. The paper does not attempt to make any link between this small contribution from MWIs and effects on human health.

2.1.10 These four recent papers consider facilities in the UK, operating under the same regulatory regime which would apply to the CHP Facility and operating to the standards of the IED. The papers found no conclusive evidence of an association of waste incineration facilities with the health outcomes considered. Given that the CHP Facility will operate to tighter standards, as it is subject to the reduced ELVs from the Waste Incineration BREF, the conclusions are directly relevant and support PHE's position statement that "*any potential damage to the health of those living close-by is likely to be very small, if detectable*".

2.1.11 Therefore, it can be concluded that the effect of emissions from the CHP Facility of pollutants that accumulate in the environment would not be significant. Nonetheless, a quantitative assessment of the effect of emissions from the CHP Facility in combination with the existing K8 biomass plant has been undertaken and is presented in the following sections.

[6] Parsons, R., Douglas, P., Ashworth, D., Hansell, A., Sepai, O., Chadeau-Hyam, M., Toledano, M., & , (2024). Polychlorinated dibenzo-dioxin/furan and polychlorinated biphenyl concentrations in the human milk of individuals living near municipal waste incinerators in the UK: Findings from the Breast milk, Environment, Early-life, and Development (BEED) human biomonitoring study. Environmental Research, 120588

3.0 ISSUE IDENTIFICATION

3.1 Issue

3.1.1 The key issue for consideration is the release of substances to atmosphere from the CHP Facility which have the potential to harm human health. Details of the dispersion modelling can be found in the **Appendix 6C**.

3.1.2 The CHP Facility will be required to meet the ELVs which will be set in the EP as outlined in the Waste Incineration BREF. Limits will be set for pollutants known to be produced during the combustion of waste wood which have the potential to impact upon the local environment either on human health or ecological receptors. In addition, the existing EP the requirements of the Waste Incineration BREF have been implemented on the K8 biomass plant which also includes an ELV for dioxins and as such has been included in this assessment. Dioxins and dioxin-like PCBs can accumulate in the environment, which means that inhalation is only one of the potential exposure routes. The health assessment criterion is expressed as the total intake from ingestion and inhalation. Pathway modelling considering the intake from inhalation and ingestion has been carried out using the software "Industrial Risk Assessment Program-Human Health" (IRAP-h View - Version 5.1, "IRAP"). In addition, a review of published literature on the health effects of energy recovery facilities has been undertaken.

3.2 Chemicals of Potential Concern (COPC)

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

i) PCDD/Fs (individual congeners), i.e., dioxins; and
ii) Dioxin-like PCBs.

3.2.2 This risk assessment investigates the potential for long term health effect of these COPCs through other routes than just inhalation.

4.0 ASSESSMENT CRITERIA

4.1.1 IRAP calculates the total exposure through each of the different pathways so that a dose from inhalation and ingestion can be calculated for each receptor. By default, these doses are then used to calculate a cancer risk, using the United States Environment Protection Agency's (USEPA)'s approach. However, this assessment applies the approach set out in the Environment Agency's document "Human Health Toxicological Assessment of Contaminants in Soil", ref SC050021 (2009).

4.1.2 For the COPCs considered, which have a threshold level for toxicity, a Tolerable Daily Intake (TDI) is defined. This is "*an estimate of the amount of a contaminant, expressed on a bodyweight basis, which can be ingested daily over a lifetime without appreciable health risk.*" A Mean Daily Intake (MDI) is also defined, which is the typical intake from background sources (including dietary intake) across the UK. In order to assess the impact of the CHP Facility, the predicted intake of a substance due to emissions from the CHP Facility and the K8 biomass plant (the other source of dioxins from the Kronospan site) is added to the MDI and compared with the TDI.

4.1.3 The following table outlines the MDIs (the typical intake from existing background sources) and TDIs for dioxins and dioxin-like PCBs. These figures are defined in the "Contaminants in soil: updated collation of toxicology data and intake values for humans: dioxins, furans and dioxin-like PCBs"(Environment Agency 2009).

Table 4.1 – Assessment Criteria for Intake of Dioxins

Item	Units	70 kg adult	20 kg child
Tolerable Daily Intake (TDI)	pg WHO-TEQ/kg bw/day	2.0	
Mean Daily Intake (MDI)	pg WHO-TEQ/kg bw/day	0.7	1.8
	% of TDI	35.00%	90.00%
Source: *Contaminants in soil: updated collation of toxicology data and intake values for humans: dioxins, furans and dioxin-like PCBs, Environment Agency 2009*[7]*.*			

4.1.4 To allow comparison with the TDI for dioxins, intake values for each dioxin are multiplied by a factor known as the WHO-TEF. A full list of the WHO-TEF values for each dioxin is provided in **Table 7.3**.

[7] *This document has been archived by the EA. The page detailing the TDI and MDI has been appended as Annex* ***Error! Reference source not found.****.*

4.1.5 The TDI has been set at a level which can be ingested daily over a lifetime without appreciable health risk. Therefore, if the total exposure is less than the TDI, it can be concluded that the impact is not significant.

5.0 CONCEPTUAL SITE MODEL

5.1 Conceptual Site Model

5.1.1 IRAP, created by Lakes Environmental, is based on the USEPA Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities[8]. This Protocol is a development of the approach defined by Her Majesty's Inspectorate on Pollution (HMIP) in the UK in 1996[9], taking account of further research since that date. The exposure pathways included in the IRAP model are shown in **Table 5.1**.

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

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

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

Table 5.2 – Pathways Considered

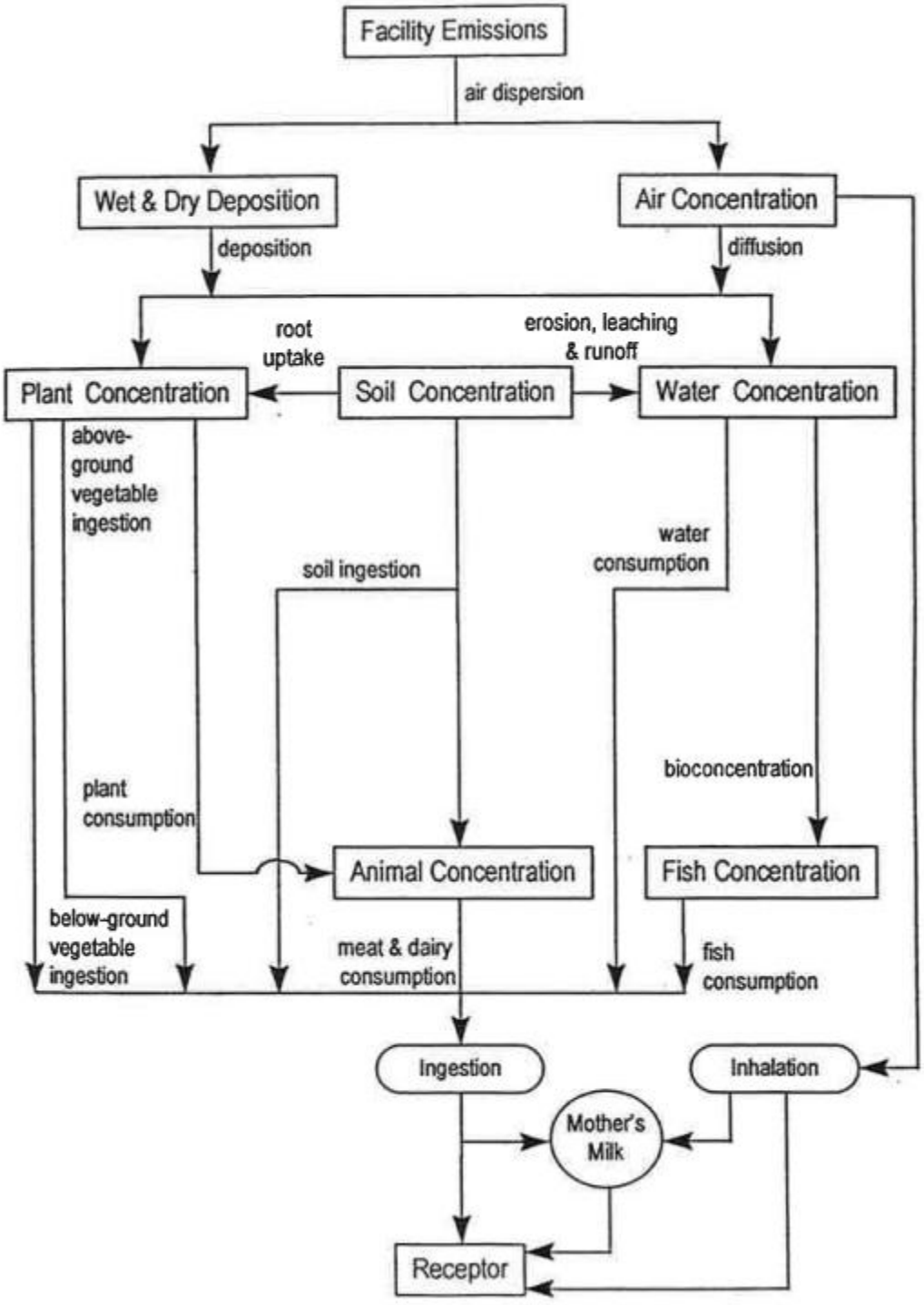
Pathway	Residential	Allotment	Agricultural
Direct inhalation	Yes	Yes	Yes
Ingestion of soil	Yes	Yes	Yes
Ingestion of home-grown produce	Yes	Yes	Yes
Ingestion of drinking water	Yes	Yes	Yes
Ingestion of eggs from home-grown chickens	-	Yes	Yes
Ingestion of home-grown poultry	-	Yes	Yes
Ingestion of home-grown beef	-	-	Yes
Ingestion of home-grown pork	-	Yes	Yes
Ingestion of home-grown milk	-	-	Yes
Ingestion of breast milk (infants only)	Yes	Yes	Yes

[8] USEPA (2005) Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities.
[9] HMIP (1996) Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes.

5.1.4 Some households may keep chickens and consume eggs and potentially the birds. The impact on these households is considered to be between the impact at an agricultural receptor and a standard resident receptor as such a modified 'allotment' type receptors has also been considered which has the same parameters as the 'agricultural' type receptor but excludes the pathways from home-grown beef and home-grown milk products. The approach used considers each of the receptor types at the point of maximum impact as a complete worst case.

5.1.5 As shown in **Image 5.1**, the pathway from the ingestion of mother's milk in infants is considered within the assessment. The IRAP model calculates the amount of dioxins entering the mother's milk and being passed on to the infants. IRAP does not include data on individual PCBs, but it does include data for take-up and accumulation rates within the food chain for two groups of PCBs, known as Aroclor 1254 and Aroclor 1016. IRAP does not include these when determining the intake via mother's milk. Therefore, a safety factor of 1.5 has been applied to the dioxin and dioxin-like PCBs emission rate when considering the impact of the intake via mother's milk. The impacts are then compared against the TDI.

Image 5.1 – Conceptual Site Model – Exposure Pathways



5.2 Pathways Excluded from Assessment

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

Dermal Absorption

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

5.2.3 The USEPA[10] provide an example from the risk assessment conducted for the Waste Technologies, Inc. hazardous thermal treatment in East Liverpool, Ohio. This indicated that for an adult subsistence farmer in an area with high exposures, the risk resulting from soil ingestion and dermal contact was 50-fold less than the risk from any other pathway and 300-fold less than the total estimated risk.

5.2.4 The HMIP document[11] provides a screening calculation using conservative assumptions, which states that the intake via dermal absorption is 30 times lower than the intake via inhalation, which is itself a minor contributor to the total risk.

5.2.5 As such the pathway from dermal absorption is deemed to be an insignificant risk and has been excluded from this assessment.

Groundwater

5.2.6 Exposure via groundwater can only occur if the groundwater is contaminated and consumed untreated by an individual.

5.2.7 The USEPA[12] have concluded that the build-up of dioxins in the aquifer over realistic travel times relevant to human exposure was predicted to be so small as to be essentially zero.

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[10] USEPA (2005) Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities.
[11] HMIP (1996) Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes.
[12] USEPA (2005) Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities.

5.2.8 As such the pathway from groundwater is deemed to be an insignificant risk and has been excluded from this assessment.

Surface Water

5.2.9 A possible pathway is via deposition of emissions directly onto surface water - i.e., local drinking water supplies or rainwater storage tanks.

5.2.10 Surface water generally goes through several treatment steps and as such any contaminants would be removed from the water before consumption. Run off to rainwater tanks may not go through the same treatment. However, rainwater tanks have a very small surface area and as such the potential for deposition and build-up of COPCs is limited. As such, the pathway from contaminated surface water is deemed to be an insignificant risk and has been excluded from this assessment.

Fish Consumption

5.2.11 The consumption of locally caught fish has been excluded from the assessment. Whilst fish makes up a proportion of the UK diet, it is not likely that this would be sourced wide-scale from close proximity to the Kronospan Facility.

5.2.12 A review of the local waterbodies has been undertaken to determine if there are any game fishing lakes in the local area[13]. The closest game fishing lake is Chirk Fisheries approximately 1.6 km south-west of the Kronospan Facility. Although this is within the area potentially affected by emissions from the Facility, the likelihood of persons sourcing a large proportion of their diet from a game fishery is very low. Game fishing may also take place along rivers in the local area. However, the accumulation of pollutants in river systems is not of significant concern, as any pollutants will be washed downstream rather than accumulating. Therefore, the fish consumption pathway has been excluded from this assessment.

[13] *https://anglingtrust.net/map/*

6.0 SENSITIVE RECEPTORS

6.1.1 This assessment considers the possible effects on human health at key receptors, where humans are likely to be exposed to the greatest impact from the Kronospan Facility, and at the point of maximum impact of annual mean emissions.

6.1.2 For the purposes of this assessment, receptor locations have been categorised as 'residential', ‘allotment’ or 'agricultural'. Residential receptors represent a known place of residence that is occupied within the study area. Agricultural receptors represent a farm holding or area land of horticultural interest, and ‘allotment’ has been used for those which may grow and consume home grown produce, as a worst-case this includes schools.

6.1.3 The specific receptors identified in **Table 6.1** have been considered in this assessment. An additional receptor has been included at the point of maximum impact of emissions, this point is on the land to the immediate east of the Kronospan Facility and has been included to demonstrate the theoretical maximum impact. Reference should be made to **Figure 1** at the end of this Appendix which shows the location of these receptors with respect to the Kronospan Facility.

Table 6.3 – Sensitive Receptors

ID	Receptor Name	Location		Type of Receptor
		X (m)	Y (m)	
MAX	Point of maximum impact	328960	338530	Agricultural / Residential
R1	Brynkinallt Home Farm	329717	338038	Agricultural
R2	Highfield Farm	329767	338684	Agricultural
R3	Chirk Green Farm	329513	338537	Agricultural
R4	Ley Farm	329867	339214	Agricultural
R5	Lodge Farm	329227	339568	Agricultural
R6	Afon-Bradley Farm	328687	339451	Agricultural
R7	New Hall Farm	327543	338884	Agricultural
R8	Pontfaen Farm	328380	336965	Agricultural
R9	Chirk Infant School	329166	338427	Allotment
R10	Ysgol y Waun Primary School	329338	338327	Allotment
R11	Chirk Green Road Allotments	329778	338571	Allotment
R12	Holyhead Road	329023	338452	Residential
R13	20 Ewart Street	329074	338766	Residential
R14	4 Crogen	328979	339008	Residential
R15	102 Crogen	329194	339065	Residential
R16	2 Maes Yr Ysgol	329130	338229	Residential

7.0 IRAP MODEL ASSUMPTIONS AND INPUTS

7.1.1 The following section details the user defined assumptions used within the IRAP model and provides justifications where appropriate.

7.2 Concentrations in Soil

7.2.1 The concentration of each chemical in the soil is calculated from the deposition results of the air quality modelling for vapour phase and particle phase deposition. The critical variables in calculating the accumulation of pollutants in the soil are as follows:

i) the lifetime of the CHP Facility and K8 biomass plant is taken as 30 years; and
ii) the soil mixing depth is taken as 2 cm in general and 20 cm for produce.

7.2.2 The split between the solid and vapour phase for the substance considered depends on the specific physical properties of each chemical.

7.2.3 In order to assess the amount of substance which is lost from the soil each year through volatilisation, leaching and surface run-off, a soil loss constant is calculated. The rates for leaching and surface runoff are taken as constant, while the rate for volatilisation is calculated from the physical properties of each substance.

7.3 Concentrations in Plants

7.3.1 The concentrations in plants are determined by considering direct deposition and air-to-plant transfer for above ground produce, and root uptake for above ground and below ground produce.

7.3.2 The calculation takes account of the different types of plant. For example, uptake of substances through the roots will differ for below ground and above ground vegetables, and deposition onto plants will be more significant for above ground vegetables.

7.4 Concentrations in Animals

7.4.1 The concentrations in animals are calculated from the concentrations in plants, assumed consumption rates and bio-concentration factors. These vary for different animals and different substances, since the transfer of chemicals between the plants consumed and animal tissue varies.

7.4.2 It is also assumed that 100% of the plant materials eaten by animals is grown on soil contaminated by emission sources. This is likely to be a highly pessimistic assumption for UK farming practice.

7.5 Concentrations in Humans

Intake via Inhalation

7.5.1 This is calculated from inhalation rates of typical adults and children and atmospheric concentrations. The inhalation rates used for adults and children are:

i) adults - 20 m^3/day; and
ii) children - 7.2 m^3/day.

7.5.2 These are as specified within the Environment Agency's document "Human Health Toxicological Assessment of Contaminants in Soil". The calculation also takes account of time spent outside, since most people spend most of their time indoors.

Intake via Soil Ingestion

7.5.3 This calculation allows for the ingestion of soil and takes account of different exposure frequencies. It allows for ingestion of soil attached to unwashed vegetables, unintended ingestion when farming or gardening and, for children, ingestion of soil when playing.

Ingestion of Food

7.5.4 The calculation of exposure due to ingestion of food draws on the calculations of concentrations in animals and plants and takes account of different ingestion rates for the various food groups by different age groups.

7.5.5 For most people, locally-produced food is only a fraction of their diet and so exposure factors are applied to allow for this.

Breast Milk Ingestion

7.5.6 For infants, the primary route of exposure is through breast milk. The calculation draws on the exposure calculation for adults and then allows for the transfer of chemicals in breast milk to an infant who is exclusively breast-fed.

7.5.7 The only pathway considered for dioxins for a breast feeding infant is through breast milk. The modelled scenario consists of the accumulation of pollutants in the food chain up to an adult receptor, the accumulation of pollutants in breast milk and finally the consumption of breast milk by an infant.

7.5.8 The assumptions used are:

i)	Exposure duration of infant to breast milk	1 year
ii)	Proportion of ingested dioxin that is stored in fat	0.9
iii)	Proportion of mother's weight that is stored in fat	0.3
iv)	Fraction of fat in breast milk	0.04
v)	Fraction of ingested contaminant that is absorbed	0.9
vi)	Half-life of dioxins in adults	2,555 days
vii)	Ingestion rate of breast milk	0.688 kg/day
viii)	Safety factor on total dioxin intake to account for PCBs	1.5

7.6 Estimation of COPC Concentration in Media

7.6.1 The IRAP-h model uses a database of physical and chemical parameters to calculate the COPC concentrations through each of the different pathways identified. The base physical and chemical parameters have been used in this assessment.

7.6.2 Meteorological data has been obtained for the period 2020 to 2024 from the Shawbury weather station, as used within the dispersion modelling and fully described in **Appendix 6C**. This provides the annual average precipitation which can be used to calculate the general IRAP-h input parameters, as presented in **Table 7.1**.

Table 7.4 – Site-Specific Assumptions

Input Variable	Assumption	Value (cm/year)
Annual average evapotranspiration	70% of annual average precipitation	55.22
Annual average irrigation	0% of annual average precipitation	0.00
Annual average precipitation	100% of annual average precipitation	78.88
Annual average runoff	10% of annual average precipitation	7.89

7.6.3 The average wind speed was taken as 3.99 m/s, calculated from the average of the five years of weather data from Shawbury.

7.6.4 A number of assumptions have been made with regard to the deposition of the different phases. These are summarised in the following table.

Table 7.2 – Deposition Assumptions

Deposition Phase	Dry Deposition Velocities (m/s)	Radio Dry Deposition to Wet Deposition	
		Dry Deposition	Wet Deposition
Vaour	0.005	1.0	2.0
Particle	0.010	1.0	2.0
Bound particle	0.010	1.0	2.0

7.6.5 These deposition assumptions have been applied to the annual mean concentrations predicted using the dispersion modelling, to generate the inputs needed for the IRAP modelling. For details of the dispersion modelling methodology please refer to the **Appendix 6C**.

7.7 Modelled emissions

7.7.1 For the purpose of this assessment it is assumed that the CHP Facility will operate at the ELV for dioxins as set out in the WI BREF for a new for its entire operational life, and the K8 biomass plant at the ELVs set in the existing EP which implement the requirements of the WI BREF. In reality, each item of plant will be shut down for periods of maintenance and will typically operate below the ELVs. The monitoring from the K8 biomass plant has shown that the average monitored concentration between 2020 and 2024 was 0.05 ng-ITEQ/Nm^3 which is well below the ELV set in the existing EP.

7.7.2 The emissions from the K8 biomass plant used within the drying process and it is proposed that the CHP Facility would operate in the same way. As such it has been assumed that the release rate of dioxins from each of these sources vents to atmosphere via the associated dryers. This is detailed in **Appendix 6C**.

7.7.3 The following table presents the emissions rates of each COPC modelled and the associated emission concentrations which have been used to derive the emission rate.

Table 7.3 – COPC Emissions Modelled

COPC	Split of Congeners for a Release of 1 ng I-TEQ/Nm$^{3(1)}$	K8 Biomass Plant – via MDF 1 Dryer		CHP Facility – via MDF 2 Dryer	
		Emission Conc. (ng/Nm^3)$^{(2)}$	Emission Rate (ng/s)$^{(3)}$	Emission Conc. (ng/Nm^3)$^{(2)}$	Emission Rate (ng/s)$^{(3)}$
Sum I-TEQ dioxins	-	0.09	-	0.06	-
2,3,7,8-TCDD	0.031	0.0028	0.026	0.0019	0.085
1,2,3,7,8-PeCDD	0.245	0.0220	0.205	0.0147	0.675
1,2,3,4,7,8-HxCDD	0.287	0.0258	0.240	0.0172	0.790
1,2,3,6,7,8-HxCDD	0.258	0.0232	0.216	0.0155	0.710
1,2,3,7,8,9-HxCDD	0.205	0.0184	0.172	0.0123	0.564
1,2,3,4,6,7,8-HpCDD	1.704	0.1533	1.426	0.1022	4.691
OCDD	4.042	0.3637	3.382	0.2424	11.128
2,3,7,8-TCDF	0.277	0.0249	0.232	0.0166	0.763
1,2,3,7,8-PCDF	0.277	0.0249	0.232	0.0166	0.763
2,3,4,7,8-PCDF	0.535	0.0481	0.448	0.0321	1.473
1,2,3,4,7,8-HxCDF	2.179	0.1960	1.823	0.1307	5.999
1,2,3,6,7,8-HxCDF	0.807	0.0726	0.675	0.0484	2.222
1,2,3,7,8,9-HxCDF	0.042	0.0038	0.035	0.0025	0.116
2,3,4,6,7,8-HxCDF	0.871	0.0784	0.729	0.0522	2.398
1,2,3,4,6,7,8-HpCDF	4.395	0.3954	3.677	0.2636	12.100
1,2,3,4,7,8,9-HpCDF	0.429	0.0386	0.359	0.0257	1.181
OCDF	3.566	0.3208	2.984	0.2139	9.817
Total dioxins	20.150	1.8129	16.8599	1.2086	55.474
Dioxin-like PCBs$^{(4)}$	-	0.0138	0.128	0.0138	0.633

Notes:
(1) Split of the congeners taken from Table 7.2a from the HMIP document expressed at dry gas, 11% oxygen, 273.15K.
(2) All emissions are expressed at reference conditions of dry gas, 6% oxygen, 273.15K.
(3) Emission release rate calculated by multiplying the normalised volumetric flow rate by the emission concentration.
(4) Refer to note 2 below this table

7.7.4 A number of points should be noted for the two groups of COPCs:

1. Dioxins

7.7.5 The split of the different dioxins and furans is based on split of congeners for a release of 1 ng I-TEQ/Nm³ as presented in in **Table 7.2** and **Table 7.3**. This data is taken from Table 7.2a from the HMIP document "Risk Assessment of Dioxin Releases from Municipal Waste Incineration Processes".

7.7.6 To determine the emission rates, this split of the different dioxins factored to the ELV has been multiplied by normalised volumetric flow rate to determine the release rate of each congener.

2. Dioxin-like PCBs

7.7.7 There are a total of 209 PCBs, which act in a similar manner to dioxins, are generally found in complex mixtures and also have TEFs.

7.7.8 The UK Environment Agency has advised that 44 measurements of dioxin like PCBs have been taken at 24 MWIs between 2008 and 2010. The following data summarises the measurements, all at 11% reference oxygen content:

i) Maximum = 9.2×10^{-3} ng[TEQ]/m³
ii) Mean = 2.6×10^{-3} ng[TEQ]/m³
iii) Minimum = 5.6×10^{-5} ng[TEQ]/m³

7.7.9 For the purpose of this assessment, the maximum monitored PCB concentration has been used which has been converted to an emission rate using the volumetric flow. This has accounted for the difference in reference conditions.

7.7.10 The IRAP software, and the HHRAP database which underpins it, does not include any data on individual PCBs, but it does include data for take-up and accumulation rates within the food chain for two groups of PCBs, known as Aroclor 1254 and Aroclor 1016. Each Aroclor is based on a fixed composition of PCBs. Since we are not aware of any data on the specification of PCBs within incinerator or co-incinerator emissions, as a worst-case assumption it has been assumed that PCB emissions consist entirely of each of the two Aroclor compositions and the maximum impact of either composition has been presented.

7.7.11 As shown in **Table 4.1**, the MDI and TDI for dioxins and dioxin-like PCBs is given in pg WHO-TEQ/kg bw/day. However, the split of congeners shown in **Table 7.2** which are used to calculate the release rate of each dioxin are based on the I-TEFs listed in Annex VI Part II of the IED. To determine the total intake TEQ for comparison with the TDI, the output of the IRAP model has been multiplied by the relevant WHO-TEFs. Two sets of WHO-TEFs have been considered; those values published in 2005, and those published in 2022. The Environment Agency has not published any guidance relating to the publication of the 2022 WHO-TEFs. Correspondence with the Environment Agency has confirmed that the 2005 WHO-TEFs can continue being used for the main assessment, and a sensitivity analysis be undertaken using the 2022 WHO-TEFs. The I-TEFs and WHO-TEFs are shown in **Table 7.4**.

Table 7.4 – Toxic Equivalency Factors for Dioxins and Furans

Congener	IED I-TEF Multiplier[(1)]	2005 WHO-TEF Multiplier[(1)]	2022 WHO-TEF Multiplier[(2)]
Dioxins			
2,3,7,8-TCDD	1	1	1
1,2,3,7,8-PeCDD	0.5	1	0.4
1,2,3,4,7,8-HxCDD	0.1	0.1	0.09
1,2,3,6,7,8-HxCDD	0.1	0.1	0.07
1,2,3,7,8,9-HxCDD	0.1	0.1	0.05
1,2,3,4,6,7,8-HpCDD	0.01	0.01	0.05
OCDD	0.001	0.0003	0.001
Furans			
2,3,7,8-TCDF	0.1	0.1	0.07
1,2,3,7,8-PCDF	0.05	0.03	0.1
2,3,4,7,8-PCDF	0.5	0.3	0.01
1,2,3,4,7,8-HxCDF	0.1	0.1	0.3
1,2,3,6,7,8-HxCDF	0.1	0.1	0.09
1,2,3,7,8,9-HxCDF	0.1	0.1	0.2
2,3,4,6,7,8-HxCDF	0.1	0.1	0.1
1,2,3,4,6,7,8-HpCDF	0.01	0.01	0.02
1,2,3,4,7,8,9-HpCDF	0.01	0.01	0.1
OCDF	0.001	0.0003	0.002

Notes:
(1) Contaminants in soil: updated collation of toxicological data and intake values for humans, Dioxins, furans and dioxin-like PCBs (Science report: SC050021/TOX 12), Environment Agency, 2009.
(2) The 2022 world health organization re-evaluation of human and mammalian toxic equivalency factors for polychlorinated dioxins, dibenzofurans and biphenyls, DeVito et al, 2023.

7.7.12 Given the difference in the ELVs for the K8 biomass plant and CHP Facility the IRAP model has been run for each source individually and then the impacts added together to determine the total impact of the Kronospan Facility.

8.0 RESULTS

8.1 Assessment against TDI - Point of Maximum Impact

8.1.1 The following tables present the impact of emissions of dioxins and dioxin-like PCBs from the Kronospan Facility during normal operations at the point of maximum impact of emissions for the three receptor types. This assumes that the emissions from the CHP Facility are used in the MDF 1 drier and released to atmosphere via the MDF 1 cyclone, and emissions from the K8 biomass plant are used in the MDF 2 drier and released to atmosphere via the MDF 2 cyclones.

Table 8.1 – Impact Analysis – Dioxins and Dioxin-Like PCBs – Point of Maximum Impact

Receptor Type	MDI (% of TDI)	Process Contribution (% of TDI)		Overall (% of TDI)
		K8 Biomass Plant	CHP Facility	
Adult				
Agricultural	35.00%	0.70%	2.23%	37.94%
Allotment	35.00%	0.04%	0.13%	35.17%
Residential	35.00%	0.02%	0.05%	35.07%
Child				
Agricultural	90.00%	1.00%	3.17%	94.18%
Allotment	90.00%	0.08%	0.26%	90.35%
Residential	90.00%	0.06%	0.18%	90.24%

8.1.2 As explained in **Section 2.0**, the worst-case receptor – the 'agricultural' receptor type - assumes the direct inhalation, and ingestion from soil, drinking water, and home-grown eggs and meat, beef, pork, and milk. This assumes that the person lives at the point of maximum impact and consumes home-grown produce (including milk) etc. Reference should be made to **Figure 1** for the location of the point in relation to the Kronospan Facility. This point is just outside the installation boundary and not at an area of public exposure. The more realistic receptor type for this point is the 'residential' type as it is close to the area of residential properties.

8.1.3 The TDI is an estimate of the amount of a contaminant, expressed on a bodyweight basis, which can be ingested daily over a lifetime without appreciable health risk. As shown in **Table 8.1**, at the point of maximum impact the overall impact (including the contribution from existing dietary intake) is less than the TDI for dioxins and dioxin-like PCBs. Therefore, there would not be an appreciable health risk based on the emission of these pollutants even if it is conservatively assumed that the worst-case receptor type is located at the point of maximum impact from emissions.

8.2 Breast milk exposure

8.2.1 The total accumulation of dioxins in an infant resulting from emissions from the Kronospan Facility, considering the breast milk pathway and based on an adult receptor at the point of maximum impact of emissions feeding an infant is set out in the following table.

Table 8.2 – Impact Analysis Breast Fed Infant – Point of Maximum Impact

Receptor Type	MDI (% of TDI)	Process Contribution (% of TDI)		Overall (% of TDI)
		K8 Biomass Plant	CHP Facility	
Breast milk fed infant				
Agricultural	-	6.05%	18.92%	24.97%
Allotment	-	0.35%	1.10%	1.45%
Residential	-	0.12%	0.37%	0.49%

8.2.2 There are no ingestion pathways besides breast milk ingestion for an infant receptor. As the process contribution is less than the TDI, it is considered that the emissions of dioxins from the Kronospan Facility will not increase the health risks from the accumulation of dioxins in infants significantly.

8.3 Maximum Impact at a Receptor

8.3.1 The following tables outline the impact of emissions at the most affected receptor (i.e., the receptor with the greatest combined impact from ingestion and inhalation of emissions from the Kronospan Facility) (R3 – Chirk Green Farm). This receptor has been classified as an agricultural receptor, which is conservative as it assumes that a significant proportion of the diet of the receptor is sourced from the receptor point assessed, including meat and milk products. In reality, people in the UK tend to source their diet from a wide geographical area.

Table 8.3 – Impact Analysis – Dioxins and Dioxin-Like PCBs – Point of Maximum Impact

Receptor	MDI (% of TDI)	Process Contribution K8 biomass plant (% of TDI)	Process Contribution CHP Facility (% of TDI)	Overall (% of TDI)
R3 – Agricultural Adult	35.00%	0.51%	1.33%	36.84%
R3 - Agricultural Child	90.00%	0.73%	1.89%	92.61%
R3 – Agricultural Breastfed Infant	-	4.40%	11.24%	15.64%

8.3.2 As shown, for the most impacted receptor the overall impact (including the contribution from existing dietary intake) is less than the TDI for dioxins and dioxin-like PCBs. Therefore, there would not be an appreciable health risk based on the emission of these pollutants.

8.3.3 Detailed results for all identified receptor locations are presented in the following tables. As shown, the predicted impact at all other receptor locations is well below the TDI.

Table 8.4 – Impact Analysis – Dioxins and Dioxin-Like PCBs – Impact at Receptors - Adult

Receptor	K8 Biomass Plant		CHP Facility		Total Kronospan Facility	
	Total Inhalation (fg WHO-TEQ kg^{-1} bw day^{-1})	Total Ingestion (fg WHO-TEQ kg^{-1} bw day^{-1})	Total Inhalation (fg WHO-TEQ kg^{-1} bw day^{-1})	Total Ingestion (fg WHO-TEQ kg^{-1} bw day^{-1})	Total Uptake (fg WHO-TEQ kg^{-1} bw day^{-1})	Comparison (% of TDI)
MDI (% of TDI)						35.00%
Point of maximum impact – agricultural	0.038	14.055	0.128	44.484	58.706	37.94%
Point of maximum impact – allotment	0.038	0.774	0.128	2.499	3.439	35.17%
Point of maximum impact – residential	0.038	0.288	0.128	0.917	1.371	35.07%
R1 - Brynkinallt Home Farm	0.010	3.759	0.024	8.423	12.216	35.61%
R2 - Highfield Farm	0.026	9.484	0.072	25.129	34.711	36.74%
R3 - Chirk Green Farm	0.028	10.219	0.076	26.435	36.758	36.84%
R4 - Ley Farm	0.017	6.323	0.051	17.696	24.087	36.20%
R5 - Lodge Farm	0.015	5.472	0.042	14.523	20.051	36.00%
R6 - Afon-Bradley Farm	0.016	5.809	0.044	15.284	21.153	36.06%
R7 - New Hall Farm	0.008	2.844	0.018	6.147	9.017	35.45%
R8 - Pontfaen Farm	0.007	2.491	0.014	5.017	7.530	35.38%
R9 - Chirk Infant School	0.033	0.674	0.096	1.877	2.681	35.13%
R10 - Ysgol y Waun Primary School	0.022	0.437	0.056	1.097	1.611	35.08%
R11 - Chirk Green Road Allotments	0.025	0.497	0.065	1.276	1.863	35.09%
R12 - Holyhead Road	0.037	0.279	0.116	0.833	1.266	35.06%
R13 - 20 Ewart Street	0.033	0.247	0.103	0.738	1.121	35.06%
R14 - 4 Crogen	0.025	0.185	0.074	0.530	0.813	35.04%
R15 - 102 Crogen	0.023	0.176	0.070	0.504	0.773	35.04%
R16 - 2 Maes Yr Ysgol	0.018	0.132	0.046	0.333	0.528	35.03%

Table 8.5 – Impact Analysis – Dioxins and Dioxin-Like PCBs – Impact at Receptors - Child

Receptor	K8 Biomass Plant		CHP Facility		Total Kronospan Facility	
	Total Inhalation (fg WHO-TEQ kg^{-1} bw day^{-1})	Total Ingestion (fg WHO-TEQ kg^{-1} bw day^{-1})	Total Inhalation (fg WHO-TEQ kg^{-1} bw day^{-1})	Total Ingestion (fg WHO-TEQ kg^{-1} bw day^{-1})	Total Uptake (fg WHO-TEQ kg^{-1} bw day^{-1})	Comparison (% of TDI)
MDI (% of TDI)						90.00%
Point of maximum impact – agricultural	0.048	20.022	0.161	63.291	83.523	94.18%
Point of maximum impact – allotment	0.048	1.593	0.161	5.117	6.919	90.35%
Point of maximum impact – residential	0.048	1.095	0.161	3.513	4.817	90.24%
R1 - Brynkinallt Home Farm	0.013	5.355	0.030	11.984	17.382	90.87%
R2 - Highfield Farm	0.033	13.510	0.091	35.754	49.388	92.47%
R3 - Chirk Green Farm	0.035	14.558	0.096	37.611	52.300	92.61%
R4 - Ley Farm	0.022	9.007	0.064	25.178	34.271	91.71%
R5 - Lodge Farm	0.019	7.795	0.053	20.663	28.529	91.43%
R6 - Afon-Bradley Farm	0.020	8.276	0.055	21.746	30.097	91.50%
R7 - New Hall Farm	0.010	4.051	0.022	8.746	12.830	90.64%
R8 - Pontfaen Farm	0.009	3.549	0.018	7.139	10.714	90.54%
R9 - Chirk Infant School	0.042	1.389	0.121	3.842	5.394	90.27%
R10 - Ysgol y Waun Primary School	0.027	0.899	0.071	2.245	3.242	90.16%
R11 - Chirk Green Road Allotments	0.031	1.023	0.082	2.612	3.748	90.19%
R12 - Holyhead Road	0.047	1.063	0.146	3.192	4.448	90.22%
R13 - 20 Ewart Street	0.041	0.940	0.129	2.827	3.939	90.20%
R14 - 4 Crogen	0.031	0.704	0.093	2.029	2.858	90.14%
R15 - 102 Crogen	0.030	0.669	0.088	1.930	2.717	90.14%
R16 - 2 Maes Yr Ysgol	0.022	0.501	0.058	1.276	1.857	90.09%

8.4 Uncertainty and Sensitivity Analysis

8.4.1 To account for uncertainty in the modelling the impact on human health was assessed for a receptor at the point of maximum impact.

8.4.2 To account for uncertainty in the dietary intake of a person, residential, allotment and agricultural receptors have been assessed. The agricultural receptor is assumed to consume a greater proportion of home grown produce, which has the potential to be contaminated by the COPCs released, than for a residential receptor. In addition, the agricultural receptor includes the pathway from consuming animals grazed on land contaminated by the emission source. This assumes that 100% of the plant materials eaten by the animals is grown on soil contaminated by emission sources. The agricultural receptor at the point of maximum impact is considered the upper maximum of the impact of the Kronospan Facility.

8.4.3 The IRAP software, and the HHRAP database which underpins it, does not include any data on individual PCBs, but it does include data for take-up and accumulation rates within the food chain for two groups of PCBs, known as Aroclor 1254 and Aroclor 1016. Each Aroclor is based on a fixed composition of PCBs. Since we are not aware of any data on the specification of PCBs within incinerator or co-incinerator emissions, as a worst-case assumption it has been assumed that PCB emissions consist entirely of each of the two Aroclor compositions and the maximum impact of either composition has been presented.

8.4.4 IRAP does not include these Aroclors (which are being used as a proxy for dioxin-like PCBs) when determining the intake via mother's milk. Therefore, a safety factor of 1.5 has been applied to the dioxin and dioxin-like PCBs emission rate when considering the impact of the intake via mother's milk.

8.5 Sensitivity Analysis - WHO-TEFs

8.5.1 As detailed in **Table 7.4** the output from the IRAP model for each congener has been multiplied by the appropriate WHO-TEF for comparison with the TDI. The WHO-TEFs were last updated in 2022, with the previous version being released in 2005. Correspondence with the Environment Agency has confirmed that the 2005 WHO-TEFs can continue being used for the main assessment, and a sensitivity analysis be undertaken using the 2022 WHO-TEFs.

8.5.2 The split of congeners from the HMIP document has been applied (refer to **Table 7.2**). A comparison of the results at the point of maximum impact and the maximum impacted receptor is presented in **Table 8.4**.

Table 8.4 – Sensitivity of Results to Choice of WHO-TEFs

Receptor ID	Receptor Type	Total Intake as % of TDI (inclusive of MDI)		
		2005 WHO TEFs	2022 WHO TEFs	% Change in intake as % of TDI
Max	Adult - agricultural	37.94%	38.39%	0.46%
	Child - agricultural	94.18%	94.85%	0.67%
	Infant - agricultural	24.97%	19.33%	-5.64%
	Adult - allotment	35.17%	35.21%	0.04%
	Child – allotment	90.35%	90.45%	0.10%
	Infant - allotment	1.45%	1.19%	-0.26%
	Adult - residential	35.07%	35.09%	0.02%
	Child - residential	90.24%	90.32%	0.08%
	Infant - residential	0.49%	0.43%	-0.06%
R3	Adult - agricultural	36.84%	37.12%	0.29%
	Child - agricultural	92.61%	93.04%	0.42%
	Infant - agricultural	15.64%	12.11%	-3.53%

8.5.3 As shown, the 2022 WHO-TEFs result in a higher impact than using the 2005 WHO-TEFs for adult and child receptor types. Applying the 2022 WHO-TEFs, for an agricultural child receptor at the point of maximum impact the total impact including the MDI is 94.85% of the TDI. As this remains below the TDI, no significant effects are predicted based on the predicted impact using either the 2005 or 2022 WHO-TEFs.

9.0 CONCLUSIONS

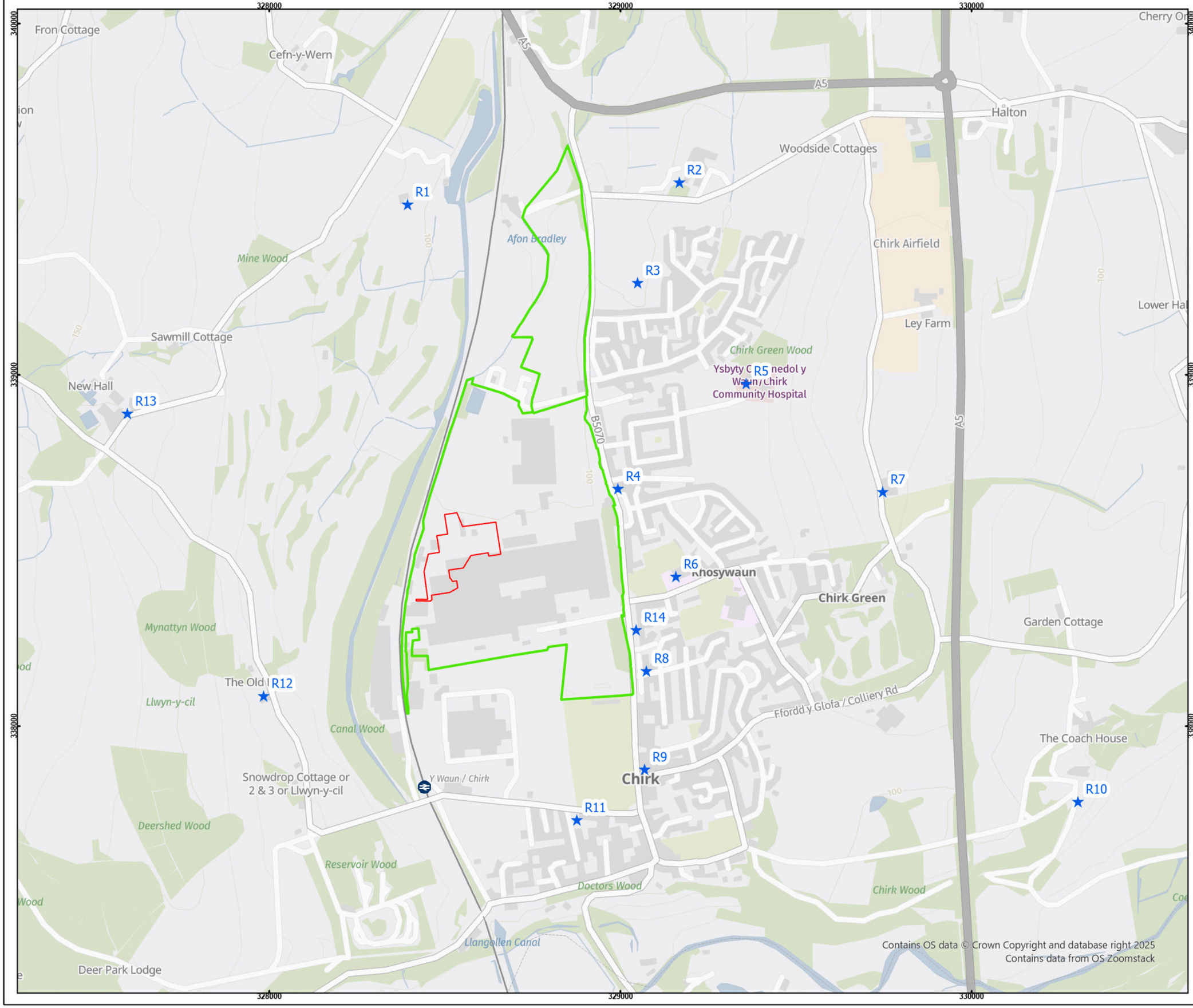
9.1.1 This Dioxin Pathway Intake Assessment has been undertaken based on the following conservative assumptions:

i) the K8 biomass boiler and the CHP Facility will operate continually at the ELV for dioxins, i.e., at the maximum concentrations which it is expected that the plant will operate at; and

ii) the hypothetical maximum impacted receptor (an agricultural receptor at the point of maximum impact) only ingests food and drink sourced from the area with the maximum contribution from the Kronospan Facility.

9.1.2 The results of the assessment show that, for the hypothetical maximum impacted receptor (an agricultural child receptor at the point of maximum impact of emissions from the Facility), the combined intake from the Kronospan Facility and the existing MDI intake of dioxins and dioxin-like PBCs via inhalation and ingestion is below the TDI. In addition, the ingestion of dioxins by an infant being breast fed by an agricultural receptor at the point of maximum impact of emissions from the Kronospan Facility is less than the TDI. The impact at identified receptor locations is lower. Therefore, there would not be an appreciable health risk based on the emission of dioxins and dioxin-like PCBs.

9.1.3 In conclusion, the impact of emissions of dioxins and dioxin-like PCBs from the Kronospan Facility following the proposed installation of the CHP Facility is predicted to be not significant.



Legend

- CHP Site Boundary
- Installation Boundary
- Human Sensitive Receptors

Client:	Axis
Site:	Kronospan - Chirk
Project:	CHP
Title:	Appendix 6E - Figure 1 - Human Sensitive Receptors
Drawn by: RSF	Date: 15/12/2025

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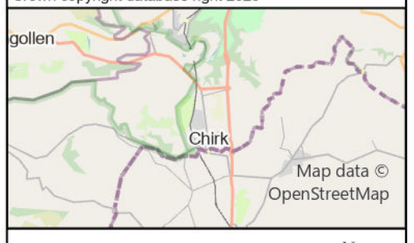

0 0.1 0.2 0.4 km

Scale: 1:10,000

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