Health Risk Assessment of Dioxin Emissions from MM Kembla, Port Kembla, Illawarra, NSW

June 2013
Executive Summary

On 24 April 2013 the NSW Environment Protection Authority (EPA) made NSW Health aware of levels of dioxin in air emissions in excess of the environmental protection licence limits set for MM Kembla in Port Kembla, NSW.

Dioxins are a group of environmentally persistent chemicals that are not manufactured intentionally but are by-products of combustion. They are everywhere in our environment and people are routinely exposed to minute quantities of dioxins mainly through food. At certain levels dioxins may cause a range of health effects. Exposure to dioxins is generally decreasing in Australia due to better industrial controls.

Licence limits on air emissions of dioxins from MM Kembla are set to minimise the emission of air pollutants and achieve good environmental performance. It is important to understand that licence limits are not health-based limits and emissions above the licence limits do not necessarily indicate human health risks or issues.

Routine emissions testing at MM Kembla in February 2013 showed levels that were four times above the licence limits resulting in a cessation of horizontal billet caster operations. Testing two years previously had showed MM Kembla complied with the licence limits. Further testing was undertaken in April 2013 where a short term test firing of the horizontal billet caster showed levels that were 170 times above the licence limits.

Upon learning of the test results NSW Health undertook this health risk assessment to determine whether, using conservative assumptions, an estimated dioxin exposure from the MM Kembla factory emissions was likely to be above the recommended levels of intake of dioxins set by the Australian National Health and Medical Research Council, and whether there was any need for any specific health advice to prevent exposure.

Key Findings

- This health risk assessment confirms that dioxin emissions from the MM Kembla factory are unlikely to pose a short or long term health risk to the surrounding community.
- In undertaking the assessment NSW Health considered two scenarios. One assuming that the level taken during operation (0.41ng/m³) – four times the licence limit with a spike to 17ng/m³ for two weeks – was emitted continuously for the previous two years, and another assuming the single highest measured concentration of dioxin (17ng/m³) – 170 times the licence limit - was emitted continuously for the previous two years.
- NSW Health identified consumption of home grown produce including eggs, as the main pathway of any possible exposure.
- In keeping with good practice, residents are reminded to always wash any home grown produce before consumption.
- Given that dioxins are persistent within our environment NSW Health emphasise the importance of the company complying with the NSW Environment Protection Authority licence limits.
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0. Structure of Risk Assessment
This risk assessment follows the methods outlined in the enHealth ‘Environmental Health Risk Assessment; guidelines for assessing human health risks from environmental hazards’ (enHealth 2012a). The risk assessment model is outlined in Figure 1.

Figure 1. Environmental Health Risk Assessment Model

SOURCE: Environmental Health Risk Assessment: Guidelines for assessing human health risks from environmental hazards
1. Issue Identification

On 24 April 2013, the NSW Environment Protection Authority (EPA) informed NSW Health that levels of dioxin in excess of the licence limits had been measured on routine inspection of the horizontal billet caster at MM Kembla in Port Kembla, NSW.

MM Kembla is situated in Port Kembla, on the east coast of Australia around 100 km south of Sydney and specializes in making copper products and equipment. The plant is surrounded by a mixed density population. It is located within 500m of residential property and a school, and within kilometres of childcare centres (Figure 2). MM beach is situated on the coast adjacent the factory. It is an unpatrolled beach and less popular than other beaches in the area but is still visited by local residents for recreation.

Figure 2 shows an aerial view of the MM Kembla site and surrounding area, with schools and child care centres highlighted.

Figure 2. Aerial view of MM Kembla and surrounding area. Port Kembla, NSW

In 2010 MM Kembla commissioned a new billet caster to make flat copper sheets. The process involves melting down recycled copper in a billet caster, attached to a chimney system that evacuates all exhausts from the process. The chimney has a number of filters (bags) which are designed to capture particle pollutants including dioxins.

The caster was initially assessed by the EPA in 2010. The EPA operating licence for the caster specifies biennial testing and an emission concentration limit for dioxins at less than 0.1 ng/m³. This is world’s best practice under the Australian National Action Plan for Dioxins but not a health based measurement. During the commissioning period in 2010, dioxins were demonstrated to comply with EPA licensing.

The billet caster has now been operating for approximately two years.
On 11 February 2013 MM Kembla undertook routine testing in keeping with its operating licence conditions. The initial measurement taken on this day was 0.41 ng/m³ dioxins. Following this, the caster was shut down and the EPA closed its operation until it could be demonstrated to operate at or below licence limits. The caster was allowed to operate again only to collect two further samples on 9th and 10th April. These further samples returned dioxin levels many times higher than the initial readings at around 17 and 12 ng/m³ (Table 1).

Table 1. Test results: Horizontal Billet Caster at MM Kembla

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>100 percentile concentration limit</th>
<th>Sample date</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>11 February 2013</td>
</tr>
<tr>
<td>Dioxins and Furans (TEQ*) (ng/m³)</td>
<td>0.1</td>
<td>0.41 (Particulate)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3.2 (Gaseous)</td>
</tr>
</tbody>
</table>

*Toxic Equivalent

On Wednesday 24 April NSW Health was notified of the incident. After expert panel evaluation, NSW Health issued the following advice to local media approximately 4 hours after notification.

NSW Health has tonight been notified by the NSW Environment Protection Authority (EPA) of a breach of licence limits for dioxin by Port Kembla based company MM Kembla.

The plant is not currently operational and NSW Health has been advised that there are no ongoing dioxin emissions from the plant.

Early indications are that the air levels of dioxins emitted from the stack are low enough not to expect acute (ie immediate) health effects. The possibility of long term health effects appears unlikely but needs careful evaluation. As a precaution, NSW Health is investigating the matter further.

Dioxin occurs in small amounts in air, water and soil throughout the world. Nearly all people in developed countries have dioxins in their body from the food they eat. As always, NSW Health advises people growing their own fruit and vegetables to wash them thoroughly before consumption and use town water supplies where available for drinking and washing vegetables.

A NSW Health factsheet that provides further information on dioxin may be found at www.health.nsw.gov.au/environment/Pages/dioxins.aspx

This risk assessment investigates the potential for long term health effects from dioxin emissions from the MM Kembla factory.
2. Hazard Assessment

2.1 Hazard Identification

Dioxins

The term ‘dioxins’ is used to describe a group of environmentally persistent halogenated aromatic hydrocarbon chemicals that are not manufactured intentionally, but are by-products of combustion (NHMRC 2002).

Dioxins are formed naturally by volcanoes and forest fires. It is thought that bushfires contribute at least 20-30% of the total release of dioxin-like compounds to the Australian environment (AGDEH 2004). In the industrial setting, they are formed through processes such as waste incineration and the synthesis of certain chemicals (NHMRC 2002).

For the general Australian population, around 95% of exposure to dioxins occurs through the diet, with foods of animal origin such as meat, dairy and fish being the main sources (AGDoH 2005).

Dioxins are bio-persistent and bio-accumulative and are stored in the fat of animals and humans, with an average half life in humans of approximately 7 years (NHMRC 2002). This long half-life means that over decades, even a low rate of exposure leads to accumulation of dioxins in the body (NHMRC 2002). Estimates of the half-life of 2,3,7,8-TCDD on the soil surface range from 9 to 15 years, whereas the half-life in subsurface soil may range from 25 to 100 years (Paustenbach 1992).

Dioxins and dioxin-like compounds are generally found as complex mixtures in which the toxicity of individual chemicals may vary by orders of magnitude (NAF 2006). The overall toxicity of a mixture of dioxins is expressed as a Toxic Equivalent (TEQ) which is the sum total of the amounts of each chemical when they are expressed as an equivalent toxic amount of 2,3,7,8 TCDD, the most toxic of the group (NFA 2006).

The International Agency for Research on Cancer have nominated TCDD, the most toxic of the conveners, as a Class 1 carcinogen, meaning a “known human carcinogen” (WHO 2007); however dioxins are non-genotoxic carcinogens, meaning they are capable of producing cancer by some secondary mechanism not related to direct gene damage (Hayashi 1992).

Dioxins are readily absorbed following ingestion and are also likely to be absorbed following inhalation. Studies also suggest that dermal (through skin) absorption is possible (Wakefield 2008).

2.2 Dose-Response Assessment

Being non-genotoxic carcinogen a threshold dose may be established for dioxins. This means people may intake dioxins at certain levels without health effects. The Australian National Health and Medical Research Council has set a level of dioxin intake it has judged to be free of health effects. This level, known as a Tolerable Monthly Intake (TMI), equates to 70pg total toxic equivalent per kilogram body weight (TEQs/kg-b) (NHMRC 2002) and is the value used in this assessment. This level is based on animal studies that show that the most sensitive adverse effects (hormonal, reproductive and developmental effects) are observed at doses in the range of 10-50 nanogram per kilogram body weight (ng/kg-b) in animals (NHMRC 2002).
The most widely recognised effect following high dose exposure to dioxins in humans is chloracne, a rare acne-like skin condition which is usually temporary (NAF 2006).

In an industrialised society, background level of dioxins mean that the vast majority of the population have dioxins in their bodies mainly from ingestion of dioxin contaminated food.
3. Exposure Assessment
This section involves identifying the pathways whereby a resident may be exposed to dioxins from the MM Kembla chimney. Environmental monitoring and modelling is used to determine the levels of exposure on each exposure pathway.

3.1 Conceptual Site Model
A conceptual site model is a summary of conditions at a site that identifies the type and location of all potential sources of contamination and how and where people, plants or animals may be exposed to the contamination. It is used here to determine and display those pathways by which residents may be exposed to dioxins from the MM Kembla chimney. Figure 3 shows a conceptual site model for the MM Kembla emissions.
Figure 3. Conceptual site model for residents’ potential exposure to dioxins from the MM Kembla chimney

<table>
<thead>
<tr>
<th>Source</th>
<th>Release mechanism</th>
<th>Exposure Route</th>
<th>Mechanism</th>
<th>Parameters</th>
</tr>
</thead>
</table>
| Billet caster   | Emissions         | Dermal contact (through dust deposition on soil)         | General inhalation         | Scenario 1: Average ground level concentration 3.54E-06 ng/m³/year  
                              |                                  |                                           | Scenario 2: Average ground level concentration 5.73E-05 ng/m³/year  |
|                 |                   | Ingestion (entry into food chain through dust deposition on plants and plant/animal absorption from soil) | Inhalation of resuspended dust | Scenario 1: Average Deposition 1.51E+00 ng/m²/year  
                              |                                  |                                           | Scenario 2: Average Deposition 1.00E+02 ng/m²/year  |

**Basic assumptions**:

<table>
<thead>
<tr>
<th>Exposure receptors</th>
<th>Local residents (Adult, Child 3-16 years and Child 1-3 years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposure time</td>
<td>24 hours/day</td>
</tr>
<tr>
<td>Exposure frequency</td>
<td>365 days/year</td>
</tr>
<tr>
<td>Exposure duration</td>
<td>2 years</td>
</tr>
</tbody>
</table>

1 Note: A casual visitor to the beach was not considered in this risk assessment as their exposures and exposure time would be less than that of the resident and therefore so would their potential risk.
The horizontal billet caster (chimney) released emissions that local residents could potentially be exposed to via three exposure routes; inhalation, dermal contact and/or ingestion.

Dioxins in ambient air could be inhaled (general inhalation), and dioxin particles deposited on the soil could be resuspended upon agitation and again be available for inhalation (inhalation of resuspended dust).

Contact with the soil may expose residents to dioxins (dermal contact).

Residents could ingest dioxins through the ingestion of soil containing dust from the MM Kembla chimney, home grown vegetables grown in soil containing dust from the MM Kembla chimney and/or home grown eggs produced in the exposed area. All humans ingest a certain amount of soil daily (soil ingestion). Eating home grown vegetables could further expose residents to dioxins through dust/dirt on the surface of the food and/or dioxins incorporated into the growing vegetable through growth in contaminated soil. Finally, chickens kept locally could be exposed to dioxins through ingestion of dust and/or vegetable scraps and therefore produce eggs with higher dioxin levels.

3.2 Exposure Concentrations

Air dioxin concentration and annual deposition were determined through dispersion modeling undertaken by the EPA. Two scenarios were modeled:

1. Scenario 1 assumes that emissions from testing conducted in February 2013 (0.41 ng/m³ dioxin) occur continuously for 51 weeks of the year and maximum measured emissions (17.2 ng/m³ dioxin) occur for 1 week of the year.

2. Scenario 2 assumes maximum measured emissions (April 2013) (17.2 ng/m³ dioxin) occurring continuously for an entire year.

Both scenarios use the location with the highest modeled residential concentration of dioxins.

The modeling results are presented in Table 2.

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Assumptions</th>
<th>Annual Average GLC (ng/m³)</th>
<th>Annual Average Deposition ng/m²/year</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Variable emissions</td>
<td>3.54E-06</td>
<td>1.51E+00</td>
</tr>
<tr>
<td>2</td>
<td>Max concentration for 365 days</td>
<td>5.73E-05</td>
<td>1.00E+02</td>
</tr>
</tbody>
</table>

It should be noted that the modeling incorporates a set of assumptions and limitations, which are listed in Appendix A.

3.3 Exposure Calculations

The full methodology adopted for the calculation of chemical intake via inhalation, dermal contact and ingestion pathways is presented in Appendix B and Appendix C. Equations were derived from United States Environment Protection Agency (US EPA) guidelines and enHealth (2012a) methods where possible. Inhalation of resuspended dust was derived from work undertaken by the United States Nuclear Regulatory Commission (US NRC 2002).
Estimates of chemical intake via the inhalation, dermal contact and ingestion pathways are presented below:

3.3.1 Intake via inhalation

Two pathways and three calculations were considered for the intake of dioxin via the inhalation route; general inhalation, and inhalation of resuspended dust (two calculations). The equation for intake via general inhalation is presented below. For further information on calculation of intake via inhalation, see Appendix B.

Intake via General Inhalation


\[ CI = \frac{CA \times InhR \times EF \times ED \times FI}{BW \times AT} \]

Where:

- CI (mg/kg-day) = chemical intake;
- CA (mg/m³) = contaminant concentration in the air;
- InhR (m³/day) = inhalation rate;
- EF (days/year) = exposure frequency;
- ED (years) = exposure duration;
- FI (unitless) = fraction inhaled;
- BW (kg) = body weight; and
- AT (day) = averaging time (period over which exposure is averaged)

3.3.2 Intake via dermal contact

The calculation from the dermal route and its supporting equations were undertaken as outlined in enHealth (2012a). The dermal absorption of dioxin was considered via contact with contaminated soil. The equation for the calculation of this pathway is presented below.

Intake via Dermal absorption from soil

Derived from enHealth (2012a)

\[ CI = \frac{CA \times AH \times SA \times B \times EF \times ED \times CF}{BW \times AT} \]

Where:

- CI (mg/kg-day) = chemical intake;
- CA (mg/kg) = contaminant concentration in the soil;
3.3.3 Intake via ingestion

The ingestion of dioxin was considered for four separate pathways – ingestion of soil; ingestion of contaminated home grown root vegetables, ingestion of home grown eggs; and ingestion of contaminated home grown leafy vegetables. The equation for the calculation of ingestion of contaminated home grown root vegetables is presented below as an example. For further information on calculation of intake via ingestion and supporting equations, see Appendix B.

Intake via Ingestion of contaminated home grown vegetables

*Derived from US EPA (1989)*

\[
CI = \frac{CA \times IR \times FI \times B \times EF \times ED \times CF}{BW \times AT}
\]

Where:

- **CI (mg/kg-day)** = chemical intake;
- **CA (mg/kg)** = contaminant concentration in produce (mg/kg wet weight);
- **IR (mg/day)** = ingestion rate of root vegetables (mg/day wet weight);
- **FI (unitless)** = fraction ingestion which is home grown;
- **B (unitless)** = Bioavailability;
- **EF (days/year)** = exposure frequency;
- **ED (years)** = exposure duration;
- **CF (unitless)** = Conversion factor (mg to kg);
- **BW (kg)** = body weight; and
- **AT (day)** = averaging time (period over which exposure is averaged)
3.4 Background Exposure
The Australian National Dioxins Program aimed to estimate the extent to which Australians are exposed to dioxins, and determine the best actions Australia could take to reduce the population’s exposure. It involved surveys of dioxin levels in the environment and people throughout Australia and provided the basis for assessing the potential risks of dioxins to the environment and to human health (AGDoH 2005).

The program found that the upper bound mean total dioxin intake for adults in Australia was 15.6 pg WHO-TEQ / kg bodyweight/month. For young children (approx. 1-4 years of age), the total intake was 37.5 pg WHO-TEQ / kg bodyweight/month. These estimates are used in this assessment as background exposure levels and represent 22% and 54% of the TMI of 70 pg WHO-TEQ / kg bodyweight/day respectively. It should be noted that as upper bound intake estimates, they are likely to be conservative overestimates and the average intake by these two age groups is likely to be lower (AGDoH 2005).
4. Risk Characterisation

Risk Characterisation involves bringing together the information from the hazard assessment and exposure assessment and describes the risks to individuals and populations in terms of nature, extent and severity of potential adverse health effects. Additionally, this section evaluates the overall quality of the assessment, and the degree of confidence in the estimates of risk and conclusions drawn.

4.1 Risk Calculations

As dioxins are non genotoxic carcinogens, a threshold approach using a Hazard Index (HI) was used to determine potential risk to human health.

Table 3 highlights these hazard indices by scenario.

**Table 3. Hazard Indices by age and scenario**

<table>
<thead>
<tr>
<th></th>
<th>Adult</th>
<th>Child (3-16)</th>
<th>Child (1-3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scenario 1</td>
<td>0.2</td>
<td>0.6</td>
<td>0.6</td>
</tr>
<tr>
<td>Scenario 2</td>
<td>1.5</td>
<td>1.6</td>
<td>2.5</td>
</tr>
</tbody>
</table>

Table 4 (scenario 1) and Table 5 (scenario 2) show the contributions from each route of exposure to total risk, the HI and percentage of Total Daily Intake (TDI) from the potential MM Kembla exposure.

For all groups, ingestion of dioxins from home grown eggs and above ground vegetables were the main contributors to exposure, comprising around 40% and 60% of the total exposure from this incident respectively.

Under Scenario 1, the HI for adults is 0.2, and for children 3-16 years and 1-3 years the HI is 0.6. These hazard indices are well below 1, indicating that no adverse human health effects are expected to occur.

Under Scenario 2, the HI is 1.5 for adults, 1.6 for children 3-16 years and 2.5 for children 1-3 years. A hazard quotient greater than 1 implies that the estimated exposure to the contaminant exceeds the reference value. Given that this level is less than 10 it is argued that consideration of assumptions, uncertainties and refinements to the risk assessment is a valid course of action. It is noted that the HI for a child of 1-3 years would be reduced to 1 if it is assumed a quarter of all leafy vegetables eaten are thoroughly washed and home grown, and that the child consumes the mean amount of eggs and eggs products for their age group as specified in enHealth (2012b), keeping all other conservative assumptions the same.
Table 4. Scenario 1 (maximum receptor / running concentration) risk calculation by route of exposure

<table>
<thead>
<tr>
<th>Route of Exposure</th>
<th>Adult</th>
<th>% Contribution of exposure</th>
<th>Child (3-16)</th>
<th>% Contribution of exposure</th>
<th>Child (1-3)</th>
<th>% Contribution of exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inhalation of Vapours</td>
<td>1.01E-03</td>
<td>2.2</td>
<td>1.06E-03</td>
<td>2.8</td>
<td>3.58E-03</td>
<td>5.0</td>
</tr>
<tr>
<td>Inhalation from Resuspended Particles (Indoor)</td>
<td>1.80E-04</td>
<td>0.4</td>
<td>1.89E-04</td>
<td>0.5</td>
<td>4.21E-04</td>
<td>0.6</td>
</tr>
<tr>
<td>Inhalation from Resuspended Particles (Outdoor)</td>
<td>3.79E-09</td>
<td>0.0</td>
<td>3.98E-09</td>
<td>0.0</td>
<td>1.27E-08</td>
<td>0.0</td>
</tr>
<tr>
<td>Ingestion of soil</td>
<td>7.50E-05</td>
<td>0.2</td>
<td>1.05E-04</td>
<td>0.3</td>
<td>7.00E-04</td>
<td>1.0</td>
</tr>
<tr>
<td>Ingestion from Home Grown Root Vegetables</td>
<td>1.13E-05</td>
<td>0.0</td>
<td>1.29E-05</td>
<td>0.0</td>
<td>2.49E-05</td>
<td>0.0</td>
</tr>
<tr>
<td>Ingestion from Home Grown Eggs</td>
<td>1.49E-02</td>
<td>32.5</td>
<td>1.75E-02</td>
<td>46.6</td>
<td>2.30E-02</td>
<td>32.0</td>
</tr>
<tr>
<td>Ingestion of above ground vegetables</td>
<td>2.96E-02</td>
<td>64.7</td>
<td>1.87E-02</td>
<td>49.8</td>
<td>4.40E-02</td>
<td>61.4</td>
</tr>
<tr>
<td>Total Intake (pg/kg-d)</td>
<td>4.58E-02</td>
<td></td>
<td>3.76E-02</td>
<td></td>
<td>7.18E-02</td>
<td></td>
</tr>
<tr>
<td>Background (pg/kd-d)</td>
<td>5.20E-01</td>
<td></td>
<td>1.25E+00</td>
<td></td>
<td>1.25E+00</td>
<td></td>
</tr>
<tr>
<td>TDI (pg/kg-d)</td>
<td>2.30E+00</td>
<td></td>
<td>2.30E+00</td>
<td></td>
<td>2.30E+00</td>
<td></td>
</tr>
<tr>
<td>Hazard Index</td>
<td>0.2</td>
<td></td>
<td>0.6</td>
<td></td>
<td>0.6</td>
<td></td>
</tr>
<tr>
<td>% of TDI from exposure</td>
<td>2.0</td>
<td></td>
<td>1.6</td>
<td></td>
<td>3.1</td>
<td></td>
</tr>
</tbody>
</table>
Table 5. Scenario 2 (maximum receptor / maximum concentration) risk calculation by route of exposure

<table>
<thead>
<tr>
<th>Route of Exposure</th>
<th>Adult (pg/kg·d)</th>
<th>Child (3-16)</th>
<th>Child (1-3)</th>
<th>% Contribution of exposure</th>
<th>% Contribution of exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inhalation of Vapours</td>
<td>1.64E-02</td>
<td>1.72E-02</td>
<td>3.82E-02</td>
<td>0.5</td>
<td>0.7</td>
</tr>
<tr>
<td>Inhalation from Resuspended Particles (Indoor)</td>
<td>1.19E-02</td>
<td>1.25E-02</td>
<td>2.78E-02</td>
<td>0.4</td>
<td>0.5</td>
</tr>
<tr>
<td>Inhalation from Resuspended Particles (Outdoor)</td>
<td>2.51E-07</td>
<td>2.63E-07</td>
<td>8.39E-07</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Ingestion of soil</td>
<td>4.96E-03</td>
<td>6.95E-03</td>
<td>4.63E-02</td>
<td>0.3</td>
<td>0.3</td>
</tr>
<tr>
<td>Ingestion from Home Grown Root Vegetables</td>
<td>7.48E-04</td>
<td>8.56E-04</td>
<td>1.65E-03</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Ingestion from Home Grown Eggs</td>
<td>9.86E-01</td>
<td>1.16E+00</td>
<td>1.52E+00</td>
<td>33.1</td>
<td>33.4</td>
</tr>
<tr>
<td>Ingestion of above ground vegetables</td>
<td>1.96E+00</td>
<td>1.24E+00</td>
<td>2.91E+00</td>
<td>65.8</td>
<td>64.1</td>
</tr>
<tr>
<td>Total Intake (pg/kg·d)</td>
<td>2.98E+00</td>
<td>2.43E+00</td>
<td>4.55E+00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Background (pg/kd·d)</td>
<td>5.20E-01</td>
<td>1.25E+00</td>
<td>1.25E+00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TDI (pg/kg·d)</td>
<td>2.30E+00</td>
<td>2.30E+00</td>
<td>2.30E+00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hazard Index</td>
<td>1.5</td>
<td>1.6</td>
<td>2.5*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of TDI from exposure</td>
<td>130</td>
<td>106</td>
<td>198</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*This HI can be reduced to 1 by refining the assumptions associated with the model such that:

- the assumed egg consumption is the mean consumption for this age group (6.7 g egg/day as estimated in enHealth 2012b)
- % of all produce eaten is home grown (87.5% of all people enHealth 2012b)
- all dirt and dust is thoroughly washed off any home-grown produce before consumption
5. Conclusions

After independent review by a clinical toxicologist and considering the uncertainties outlined in ‘Uncertainties’ section 6, it is concluded that the dioxin emissions from the MM Kembla factory are unlikely to pose a short or long term health risk to the surrounding community.

Residents can reduce their exposure to dioxins and other environmental toxins by thoroughly washing all vegetables to remove any dirt and dust before consumption. It is noted that the simple action of thoroughly washing any home grown vegetables, as is normal good practice, would significantly reduce any potential risk.

Given that dioxins are persistent within our environment it is important that the company comply with NSW Environment Protection Agency licence limits.
6. Uncertainties / Assumptions

Uncertainty in this Health Risk Assessment may come from the emissions inventory, dispersion model and/or the exposure assumptions.

5.1 Emissions Inventory

There is no concrete evidence to determine which scenario (1 or 2) is correct, however expert opinion from the NSW EPA has indicated that scenario 1 is more likely than scenario 2. Given this information Scenario 2 may be considered as a sensitivity analysis, or a likely upper bound of exposure.

5.2 Dispersion modeling assumptions

Limitations and assumptions for the dispersion model are outlined in Appendix 1. Of particular note is the 40% operational time of the caster. In scenario 1 and 2 it is assumed that the caster is operational for 100% of the time leading to a likely overestimation of actual exposure.

5.3 Exposure assumptions

Assumptions under both scenario 1 and 2 are conservative, and assume that a person:

- Is located at the place with the highest predicted concentration of dioxins 24 hours per day, 365 day per year, for 2 years.
- Half their vegetable consumption is homegrown, which is not washed before eaten.
- If an adult, consumes approximately 3 home grown eggs per day, 365 days per year for 2 years
- If a child (3-16 years), consumes approximately 2 home grown eggs per day, 365 days per year for 2 years
- If a child (1-3 years), consumes approximately 1 home grown egg per day, 365 days per year for 2 years.
- The chickens that produce the eggs eat soil and vegetation from the highest predicted dust deposition location.
- All home grown produce (and produce eaten by the chickens) is also located in this area.
- This location is also where the person will spend 4 hours per day 365 days per year, for 2 years undertaking activities vigorous enough to resuspend the soil that will then be breathed in. The soil will also coat their arms, legs, hands and feet.
- The person lives in a house located at the highest predicted concentration that has not been dusted for 1 year, spending 20 hours a day indoor and creating enough of a disturbance to resuspend this dust which is then breathed in.
Appendix A. Dispersion Model Assumptions and Limitations – (provided by NSW EPA)

1. Receptor locations (that is locations where the public may be) were chosen to reflect likely worst case residential locations.

2. The manufacturing process at MM Kembla is a batch process but modeling assumed there are continuous operations all hours in the year i.e. modelling assumes the castor operated continually for the whole two years, whereas MM Metal Kembla report that the caster only operated intermittently, for about 40% of the year. This has likely caused an over estimation of exposure concentration. With the information available there is no more realistic way to assign when emissions occur through the year. As a crude estimate, one could divide the predictions by the proportion of total yearly hours the plant normally operates.

3. The modeling approach is based on MM consultant’s configuration from 2011. The configuration should not be considered best practice and is suitable for screening level predictions only.

4. The simulation is based on meteorology in the region from 2003.

5. The model does not account for potentially significant influences of terrain and coastal influences

6. Modeling and test results do not account for variation in plant operating load.

7. Deposition modeling was based on total particulate matter (PM) emission rates and proportioned based on a dioxin to PM ratios in the stack tests.

8. When estimating particle deposition, particle characteristics have been loosely based on US-EPA AP-42 general particle size distributions for secondary metal production. With the information available, there is no way to quantify accuracy and applicability of this data to MM processes.

9. The MM Kembla site particulate matter (PM) control – baghouse – appears to have failed but the extent of the failure was not known to the EPA at the time of modeling.
Appendix B. Calculation of Chemical Intake - Methodology

B1.1 General Methodology

The following presents the general methodology adopted for the calculation of chemical intake via inhalation, ingestion and dermal pathways. Equations were derived from United States Environmental Protection Agency (US EPA) and enHealth (2012) methods where possible. Inhalation of resuspended dust was derived from work undertaken by the United States Nuclear Regulatory Commission (US NRC 2002).

B1.2 Intake via Inhalation

Two pathways and three calculations were considered for the intake of dioxin via the inhalation route. General inhalation; and inhalation of resuspended dust (two calculations).

The calculation from the inhalation routes were undertaken as outlined in US EPA (1989) and enHealth (2012). While it is acknowledged that the US EPA updated these guidelines for inhalation in 2009 (US EPA RAGS F), as the Tolerable Monthly Intake (TMI) used in this risk assessment was in terms of (pg/kg-day) the 1989 equation was used. It is noted that this is more conservative for young children than using the US EPA 2009 RAGS F calculation which may be converted to (pg/kg-day) by using standard adult factors for inhalation and body weight. The equations used (1-3) are presented below.

B1.2.1 Intake via General Inhalation

Derived from US EPA (1989 RAGS A)

\[
CI = \frac{CA \times InhR \times EF \times ED \times FI}{BW \times AT}
\]  

Where:

- CI (mg/kg-day) = chemical intake;
- CA (mg/m³) = contaminant concentration in the air;
- InhR (m³/day) = inhalation rate;
- EF (days/year) = exposure frequency;
- ED (years) = exposure duration;
- FI (unitless) = fraction inhaled;
- BW (kg) = body weight; and
- AT (day) = averaging time (period over which exposure is averaged)
B1.2.2 Intake via Inhalation of indoor resuspended dust

Derived from US EPA (1989 RAGS A)

\[
CI = \frac{CA \times \text{InhR} \times ET \times EF \times ED \times FI \times IO \times RF}{BW \times AT}
\]  

Equation 2

Where:

- CI (ug/kg-day) = chemical intake;
- CA (ug/m²) = contaminant concentration (deposited) on surface;
- InhR (m³/d) = inhalation rate;
- ET (hour/hour) = exposure time (unitless since using an inhalation rate per day);
- EF (days/year) = exposure frequency;
- ED (years) = exposure duration;
- FI (unitless) = fraction inhaled;
- IO (unitless) = Indoor to outdoor concentration;
- RF (m⁻¹) = Resuspension factor; and
- BW (kg) = body weight; and
- AT (day) = averaging time (period over which exposure is averaged)

B1.2.3 Intake via Inhalation of outdoor resuspended dust

Derived from US EPA (1989 RAGS A)

\[
CI = \frac{CA \times \text{InhR} \times ET \times EF \times ED \times FI \times \left(\frac{1}{PEF}\right)}{BW \times AT}
\]  

Equation 3

Where:

- CI (mg/kg-day) = chemical intake;
- CA (mg/kg) = contaminant concentration deposited in soil;
- InhR (m³/hour) = inhalation rate;
- ET (hour/hour) = exposure time (unitless since using an inhalation rate per day);
EF (days/year) = exposure frequency;
ED (years) = exposure duration;
FI (unitless) = fraction inhaled;
PEF (m³/kg) = Particle emission factor; and
BW (kg) = body weight; and
AT (day) = averaging time (period over which exposure is averaged)
Appendix B  
Calculation of Chemical Intake

B1.3 Intake via Ingestion

The calculation from the ingestion routes and their supporting equations were undertaken as outlined in US EPA (1989), US EPA (2003), US EPA (2005) and enHealth (2012). The ingestion of dioxin was considered for four separate pathways – ingestion of soil; ingestion of contaminated home grown root vegetables, ingestion of home grown eggs; and ingestion of contaminated home grown leafy vegetables. The equations (4-7) for the calculation of these pathways are presented below.

A1.3.1 Intake via Ingestion of soil

Derived from US EPA (1989 RAGS A)

\[
CI = \frac{CA \times IR \times FI \times B \times CF \times EF \times ED}{BW \times AT}
\]  
Equation 4

Where:

- CI (mg/kg-day) = chemical intake;
- CA (mg/kg) = contaminant concentration in the soil;
- IR (mg/day) = ingestion rate;
- FI (unitless) = fraction ingested;
- B (unitless) = Bioavailability;
- CF (unitless) = Conversion factor (mg to kg);
- EF (days/year) = exposure frequency;
- ED (years) = exposure duration;
- BW (kg) = body weight; and
- AT (day) = averaging time (period over which exposure is averaged)
B1.3.2 Intake via Ingestion of contaminated home grown vegetables

Derived from US EPA (1989 RAGS A)

\[
CI = \frac{CA \times IR \times FI \times B \times EF \times ED \times CF}{BW \times AT}
\]

Equation 5 and 7

Where:

CI (mg/kg-day) = chemical intake;
CA (mg/kg) = contaminant concentration in produce (mg/kg wet weight);
IR (mg/day) = ingestion rate of root vegetables (mg/day wet weight);
FI (unitless) = fraction ingestion which is home grown;
B (unitless) = Bioavailability;
EF (days/year) = exposure frequency;
ED (years) = exposure duration;
CF (unitless) = Conversion factor (mg to kg);
BW (kg) = body weight; and
AT (day) = averaging time (period over which exposure is averaged)
B1.3.3 Intake via Ingestion from home grown chicken eggs

Derived from US EPA (1989 RAGS A)

\[ CI = \frac{CA \times IR \times LF \times FI \times B \times EF \times ED}{BW \times AT} \]  

Where:

- \( CI \) (pg/kg-day) = chemical intake;
- \( CA \) (pg/g lipid) = contaminant concentration in produce (pg/g lipid);
- \( IR \) (g/day) = ingestion rate of eggs (g/day);
- \( LF \) (g lipid /g) = Fraction of egg that is lipid
- \( FI \) (unitless) = fraction ingestion which is home grown;
- \( B \) (unitless) = Bioavailability;
- \( EF \) (days/year) = exposure frequency;
- \( ED \) (years) = exposure duration;
- \( BW \) (kg) = body weight; and
- \( AT \) (day) = averaging time (period over which exposure is averaged)
B1.4 Intake via Dermal Absorption

The calculation from the dermal route and its supporting equations were undertaken as outlined in enHealth (2012a). The dermal absorption of dioxin was considered via contact with contaminated soil. The equation (8) for the calculation of this pathway is presented below.

A1.4.1 Intake via Dermal absorption from soil

*Derived from enHealth (2012a)*

\[
CI = \frac{CA \times AH \times SA \times B \times EF \times ED \times CF}{BW \times AT}
\]  

*Equation 8*

Where:

- \(CI\) (mg/kg-day) = chemical intake;
- \(CA\) (mg/kg) = contaminant concentration in the soil;
- \(AH\) (mg/cm²-d) = soil adherence;
- \(SA\) (cm²) = surface area of skin exposed;
- \(B\) (unitless) = Bioavailability;
- \(CF\) (unitless) = conversion factor (mg to kg);
- \(EF\) (days/year) = exposure frequency;
- \(ED\) (years) = exposure duration;
- \(BW\) (kg) = body weight; and
- \(AT\) (day) = averaging time (period over which exposure is averaged)
B1.4 Supporting equations

In order to determine the concentration of dioxin in soil, root vegetables and leafy vegetables the following equations were used.

B1.4.1 Cumulative Soil Concentration from Dust Deposition

*Derived from US EPA (2005) Equation 5.1*

\[
C_s = \frac{D_s \times \left(1 - e^{-ks \times td}\right)}{Z_s \times BD \times ks}
\]

Where:

- \(C_s\) (mg/kg) = Average soil concentration over exposure period;
- \(D_s\) (mg/m² −year) = Deposition rate;
- \(k_s\) (1/y) = 0.03 [2,3,7,8 TCDD soil loss constant (taken from US EPA HHRPA Compendium database assuming only biodegradation)];
- \(t_d\) (y) = time period over which deposition occurs;
- \(Z_s\) (m) = Soil mixing zone depth 0.02 for resuspension, ingestion, chickens, 0.2 for root vegetables (US EPA 2005);
- \(BD\) (kg/m³) = 1400kg/m³ Soil Bulk Density (Average taken from APPENDIX 2. Australian Soil Resources Information System);
B1.4.2 Concentration in root vegetables

Derived from US EPA (2005) Equation 5.20B

\[ Pr = \frac{Cs \times RCF \times VG}{Kds} \] \hspace{1cm} \text{Equation 10}

Where:

- \( Pr \) (mg/kg) = Concentration in produce due to root uptake;
- \( Cs \) (mg/kg) = Average soil concentration over exposure period;
- \( RCF \) (ug-g/ug-ml) = Root concentration factor \( 4 \times 10^4 \) [2,3,7,8 TCDD (taken from US EPA HHRPA Compendium database)];
- \( VG \) (unitless) = Empirical correction for below ground produce \( 0.01 \) (US EPA 2005 log Kow > 4);
- \( Kds \) (l/kg) = Soil / water partition coefficient \( 2.92 \times 10^5 \) [2,3,7,8 TCDD (taken from US EPA HHRPA Compendium database)];

B1.4.2 Concentration in above ground vegetables

Above ground vegetables may absorb dioxin from three pathways – dust deposition, vapour absorption and root uptake. As dioxin concentration was modelled considering total concentrations of emissions were particles, vapour absorption was not considered. Thus concentration in above ground vegetation is the combination of the following two equations.


\[ Pd = \frac{Dr \times Rp \times (1 - e^{-kp \times tp})}{Yp \times kp} \] \hspace{1cm} \text{Equation 11}

Where:

- \( Pd \) (mg/kg) = Concentration in produce due to direct deposition;
- \( Dr \) (mg/m2-y) = Deposition rate;
- \( Rp \) (unitless) = Interception fraction of the edible portion of plant \( 0.39 \);
Appendix B
Calculation of Chemical Intake

kp (1/y) = Plant surface loss coefficient \(18\) (US EPA 2005);

tp (y) = Length of plant exposure to deposition per harvest of the eatable portion \(0.164\) (US EPA 2005);

Yp (kgDW/m2) = Yield or standing crop biomass of edible portion of the plant Soil / water partition coefficient \(2.24\) (US EPA 2005);

Derived from US EPA (2005) Equation 5.20A – Above ground root uptake

\[ P_d = CS \times BR \] \hspace{1cm} Equation 12

Where:

Pd (mg/kg) = Concentration in produce due to direct deposition;
Cs (mg/kg) = Average soil concentration over exposure period;
Br (unitless) = Plant – soil bioconcentration factor \(1.03\) [2,3,7,8 TCDD (taken from US EPA HHRPA Compendium database)];

B1.4.2 Concentration in chicken eggs


\[ C_{egg} = (BCF \times DFs \times Cs \times CF) + (BCF \times DFg \times Bg \times Pd \times CF) \] \hspace{1cm} Equation 13

Where:

Cegg (pg/g egg fat) = Concentration in produce due to direct deposition;
BCF (kg/kg egg fat) = Bioconcentration factor \(7.8\) (US EPA 2003 for 2,3,7,8 TCDD);
DFs (unitless) = Fraction of chicken diet soil \(0.1\) (US EPA 2003);
Cs (mg/kg) = Average soil concentration over exposure period;
DFg (unitless) = Fraction of chicken diet incidental vegetation \(0.05\) (US EPA 2003);
Appendix B
Calculation of Chemical Intake

\[
\begin{align*}
B_g (\text{unitless}) &= \text{Bioavailability on vegetation vehicle relative to soil vehicle (0.65)} \\
&\quad \text{(US EPA 2003)}; \\
P_d (\text{mg/kg}) &= \text{Concentration in above ground produce}; \\
C_F (\text{mg/kg – pg/g}) &= 1 \times 10^6; 
\end{align*}
\]
Appendix C. Exposure Factors

C1.1 General Methodology

Exposure factors were sourced on a hierarchy of evidence. If actual exposure measurements or modelled estimated were available they were used. Where no exposure measures or estimates were available default Australian exposure factors were used. If no default Australian factors could be found, United States exposure factors were sourced.

Note: -

Two exposure scenarios have been used based on advice from NSW EPA.

a) Scenario 1 assumes that emissions from testing conducted in February 2013 (0.41 ng/m3 dioxin) occur continuously for 51 weeks of the year and maximum measured emissions occur for 1 week of the year (CA₁).

b) Scenario 2 assumes maximum measured emissions (April 2013) (17.2 ng/m3 dioxin) occur continuously for an entire year (CA₂).

Receptor locations were chosen to reflect likely worst case residential locations.

C1.2 Inhalation exposure factors and equations

C1.2.1 Exposure factors for General Inhalation with equation used

<table>
<thead>
<tr>
<th>Source</th>
<th>Infant (1-3 years)</th>
<th>Child (3 – 16 years)</th>
<th>Adult</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW (kg)</td>
<td>15</td>
<td>50</td>
<td>70</td>
<td>enHealth (2012 a&amp;b)</td>
</tr>
<tr>
<td>InhR (m³/d)</td>
<td>10</td>
<td>15</td>
<td>20</td>
<td>enHealth (2012 a&amp;b)</td>
</tr>
<tr>
<td>AT (d)</td>
<td>730</td>
<td>730</td>
<td>730</td>
<td>USEPA (1989)</td>
</tr>
<tr>
<td>FI (unitless)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Assumed 100%</td>
</tr>
<tr>
<td>EF (d/y)</td>
<td>365</td>
<td>365</td>
<td>365</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>ED (y)</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>CA₂ (µg/m³)</td>
<td>5.73x10⁻⁸</td>
<td>5.73x10⁻⁸</td>
<td>5.73x10⁻⁸</td>
<td>NSW EPA (2013)</td>
</tr>
<tr>
<td>CA₁ (µg/m³)</td>
<td>3.54x10⁻⁹</td>
<td>3.54x10⁻⁹</td>
<td>3.54x10⁻⁹</td>
<td>NSW EPA (2013)</td>
</tr>
</tbody>
</table>

Derived from US EPA (1989 RAGS A)

\[ CI = \frac{CA \times InhR \times EF \times ED \times FI}{BW \times AT} \] ....Equation 1
### C1.2.2 Exposure factors for Inhalation of resuspended indoor dust with equation used

<table>
<thead>
<tr>
<th></th>
<th>Infant (1-3 years)</th>
<th>Child (3 – 16 years)</th>
<th>Adult</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW (kg)</td>
<td>15</td>
<td>50</td>
<td>70</td>
<td>enHealth (2012 a&amp;b)</td>
</tr>
<tr>
<td>InhR (m³/d)</td>
<td>10</td>
<td>15</td>
<td>20</td>
<td>enHealth (2012 a&amp;b)</td>
</tr>
<tr>
<td>AT (d)</td>
<td>730</td>
<td>730</td>
<td>730</td>
<td>USEPA (1989)</td>
</tr>
<tr>
<td>IO (unitless)</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>Assumed half the outdoor concentration moves indoors</td>
</tr>
<tr>
<td>FI (unitless)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Assumed total dust absorbed</td>
</tr>
<tr>
<td>ET (h/h)</td>
<td>0.83</td>
<td>0.83</td>
<td>0.83</td>
<td>Assume 20 hour per day indoors and undertaking activities vigorous enough to resuspend particles.</td>
</tr>
<tr>
<td>EF (d/y)</td>
<td>365</td>
<td>365</td>
<td>365</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>ED (y)</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>CA₂ (µg/m²)</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>NSW EPA (2013) Assumed full year deposition with no degeneration</td>
</tr>
<tr>
<td>CA₁ (µg/m²)</td>
<td>0.0015</td>
<td>0.0015</td>
<td>0.0015</td>
<td>NSW EPA (2013) Assumed full year deposition with no degeneration</td>
</tr>
<tr>
<td>RF (m⁻³)</td>
<td>1 x 10⁻⁶</td>
<td>1 x 10⁻⁶</td>
<td>1 x 10⁻⁶</td>
<td>US NRC (2002)</td>
</tr>
</tbody>
</table>

Derived from US EPA (1989 RAGS A)

\[
CI = \frac{CA \times InhR \times ET \times EF \times ED \times FI \times IO \times RF}{BW \times AT}
\]  

Equation 2
C1.2.3 Exposure factors for Inhalation of resuspended outdoor dust with equation used

<table>
<thead>
<tr>
<th></th>
<th>Infant (1-3 years)</th>
<th>Child (3 – 16 years)</th>
<th>Adult</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW (kg)</td>
<td>15</td>
<td>50</td>
<td>70</td>
<td>enHealth (2012 a&amp;b)</td>
</tr>
<tr>
<td>InhR (m³/d)</td>
<td>10</td>
<td>15</td>
<td>20</td>
<td>enHealth (2012 a&amp;b)</td>
</tr>
<tr>
<td>AT (d)</td>
<td>730</td>
<td>730</td>
<td>730</td>
<td>USEPA (1989)</td>
</tr>
<tr>
<td>FI (unitless)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Assumed total dust absorbed</td>
</tr>
<tr>
<td>ET (h/h)</td>
<td>0.16</td>
<td>0.16</td>
<td>0.16</td>
<td>Assume 4 hour per day outdoors and undertaking activities vigorous enough to resuspend particles.</td>
</tr>
<tr>
<td>EF (d/y)</td>
<td>365</td>
<td>365</td>
<td>365</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>ED (y)</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>CA₂ (mg/kg)</td>
<td>6.9x10⁻⁶</td>
<td>6.9x10⁻⁶</td>
<td>6.9x10⁻⁶</td>
<td>Equation 9</td>
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<tr>
<td>CA₁ (mg/kg)</td>
<td>1.1x10⁻⁷</td>
<td>1.1x10⁻⁷</td>
<td>1.1x10⁻⁷</td>
<td>Equation 9</td>
</tr>
<tr>
<td>PEF (m³/kg)</td>
<td>1.32x10⁹</td>
<td>1.32x10⁹</td>
<td>1.32x10⁹</td>
<td>US EPA (1996)</td>
</tr>
</tbody>
</table>

Derived from US EPA (1989 RAGS A)

\[
CI = \frac{CA \times InhR \times ET \times EF \times ED \times FI \times \left( \frac{1}{PEF} \right)}{BW \times AT} \tag{Equation 3}
\]
Appendix C
Exposure Factors

C1.3 Ingestion exposure factors and equations

C1.3.1 Exposure factors for Ingestion of soil with equation used

<table>
<thead>
<tr>
<th></th>
<th>Infant (1-3 years)</th>
<th>Child (3 – 16 years)</th>
<th>Adult</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW (kg)</td>
<td>15</td>
<td>50</td>
<td>70</td>
<td>enHealth (2012 a&amp;b)</td>
</tr>
<tr>
<td>IR (mg/d)</td>
<td>100</td>
<td>50</td>
<td>50</td>
<td>enHealth (2012a)</td>
</tr>
<tr>
<td>B (unitless)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Assumed 100%</td>
</tr>
<tr>
<td>CF (unitless)</td>
<td>1x10^{-6}</td>
<td>1x10^{-6}</td>
<td>1x10^{-6}</td>
<td>Conversion factor (mg to kg)</td>
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<td>FI (unitless)</td>
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<td>1</td>
<td>1</td>
<td>Assumed total intake from backyard</td>
</tr>
<tr>
<td>AT (d)</td>
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<td>730</td>
<td>730</td>
<td>USEPA (1989)</td>
</tr>
<tr>
<td>EF (d/y)</td>
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<td>365</td>
<td>365</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>ED (y)</td>
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<td>2</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>CA₂ (mg/kg)</td>
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<td>6.9x10^{-6}</td>
<td>6.9x10^{-6}</td>
<td>Equation 9</td>
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<tr>
<td>CA₁(mg/kg)</td>
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<td>1.1x10^{-7}</td>
<td>1.1x10^{-7}</td>
<td>Equation 9</td>
</tr>
</tbody>
</table>

Derived from US EPA (1989 RAGS A)

\[
CI = \frac{CA \times IR \times FI \times B \times CF \times EF \times ED}{BW \times AT} \quad \text{Equation 4}
\]
## Appendix C
### Exposure Factors

#### C1.3.2 Exposure factors for Ingestion of home grown root vegetables with equation used

<table>
<thead>
<tr>
<th>BW (kg)</th>
<th>Infant (1-3 years)</th>
<th>Child (3 – 16 years)</th>
<th>Adult</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15</td>
<td>50</td>
<td>70</td>
<td>enHealth (2012 a&amp;b)</td>
</tr>
<tr>
<td>IR (mg/d)</td>
<td>52000</td>
<td>90000</td>
<td>110000</td>
<td>enHealth (2012b) – average intake of potatoes, carrots and other root vegetables</td>
</tr>
<tr>
<td>FI (unitless)</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>enHealth (2012b)</td>
</tr>
<tr>
<td>B (unitless)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Assumed 100%</td>
</tr>
<tr>
<td>AT (d)</td>
<td>730</td>
<td>730</td>
<td>730</td>
<td>USEPA (1989)</td>
</tr>
<tr>
<td>CF (unitless)</td>
<td>1x10^{-6}</td>
<td>1x10^{-6}</td>
<td>1x10^{-6}</td>
<td>Conversion factor (mg to kg)</td>
</tr>
<tr>
<td>EF (d/y)</td>
<td>365</td>
<td>365</td>
<td>365</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>ED (y)</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>CA₂ (mg/kg)</td>
<td>9.5x10^{-10}</td>
<td>9.5x10^{-10}</td>
<td>9.5x10^{-10}</td>
<td>Equation 10</td>
</tr>
<tr>
<td>CA₁ (mg/kg)</td>
<td>1.4x10^{-11}</td>
<td>1.4x10^{-11}</td>
<td>1.4x10^{-11}</td>
<td>Equation 10</td>
</tr>
</tbody>
</table>

Derived from US EPA (1989 RAGS A)

\[
CI = \frac{CA \times IR \times FI \times B \times EF \times ED \times CF}{BW \times AT} 
\]  

Equation 5
### C1.3.3 Exposure factors for Ingestion of above ground home grown fruit and vegetables with equation used

<table>
<thead>
<tr>
<th></th>
<th>Infant (1-3 years)</th>
<th>Child (3-16 years)</th>
<th>Adult</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW (kg)</td>
<td>15</td>
<td>50</td>
<td>70</td>
<td>enHealth (2012 a&amp;b)</td>
</tr>
<tr>
<td>IR (mg/d)</td>
<td>53500</td>
<td>75700</td>
<td>168000</td>
<td>enHealth (2102b) Average intake from berry fruit, stone fruit, cabbage type vegetables, leaf and stalk, peas and beans, tomatoes and other fruiting vegetables</td>
</tr>
<tr>
<td>FI (unitless)</td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>enHealth (2012b)</td>
</tr>
<tr>
<td>B (unitless)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Assumed 100%</td>
</tr>
<tr>
<td>AT (d)</td>
<td>730</td>
<td>730</td>
<td>730</td>
<td>USEPA (1989)</td>
</tr>
<tr>
<td>CF (unitless)</td>
<td>1x10^{-6}</td>
<td>1x10^{-6}</td>
<td>1x10^{-6}</td>
<td>Conversion factor (mg to kg)</td>
</tr>
<tr>
<td>EF (d/y)</td>
<td>365</td>
<td>365</td>
<td>365</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>ED (y)</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>CA₂ (mg/kg)</td>
<td>1.6x10^{-6}</td>
<td>1.6x10^{-6}</td>
<td>1.6x10^{-6}</td>
<td>Equation 11 &amp; 12</td>
</tr>
<tr>
<td>CA₁ (mg/kg)</td>
<td>2.5x10^{-8}</td>
<td>2.5x10^{-8}</td>
<td>2.5x10^{-8}</td>
<td>Equation 11 &amp; 12</td>
</tr>
</tbody>
</table>

*Derived from US EPA (1989 RAGS A)*

\[
CI = \frac{CA \times IR \times FI \times B \times EF \times ED \times CF}{BW \times AT}
\]  
\[\text{Equation 7}\]
Appendix C
Exposure Factors

C1.3.4 Exposure factors for Ingestion of home grown eggs with equation used

<table>
<thead>
<tr>
<th></th>
<th>Infant (1-3 years)</th>
<th>Child (3 – 16 years)</th>
<th>Adult</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>BW (kg)</td>
<td>15</td>
<td>50</td>
<td>70</td>
<td>enHealth (2012 a&amp;b)</td>
</tr>
<tr>
<td>IR (g/d)</td>
<td>48.9</td>
<td>124</td>
<td>148</td>
<td>FSANZ (2012) Mean (for 1-3 years) and 97.5% (for 3-16 years) of 2-16 age group respondents. 97.5% for adults</td>
</tr>
<tr>
<td>FI (unitless)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Assume all eggs eaten are home grown</td>
</tr>
<tr>
<td>B (unitless)</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Assumed 100%</td>
</tr>
<tr>
<td>LF (g-lipid/g)</td>
<td>0.08</td>
<td>0.08</td>
<td>0.08</td>
<td>USEPA (2003)</td>
</tr>
<tr>
<td>AT (d)</td>
<td>730</td>
<td>730</td>
<td>730</td>
<td>USEPA (1989)</td>
</tr>
<tr>
<td>EF (d/y)</td>
<td>365</td>
<td>365</td>
<td>365</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>ED (y)</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td>CA₂ (pg/g-lipid)</td>
<td>5.38</td>
<td>5.38</td>
<td>5.38</td>
<td>Equation 13</td>
</tr>
<tr>
<td>CA₁(pg/g-lipid)</td>
<td>0.088</td>
<td>0.088</td>
<td>0.088</td>
<td>Equation 13</td>
</tr>
</tbody>
</table>

Derived from US EPA (1989 RAGS A)

\[
CI = \frac{CA \times IR \times LF \times FI \times B \times EF \times ED}{BW \times AT}
\]  

.................................................................Equation 6
Appendix C
Exposure Factors

C1.4 Dermal exposure factors and equation

C1.4.1 Exposure factors for dermal absorption of contact with soil with equation used

<table>
<thead>
<tr>
<th></th>
<th>Infant (1-3 years)</th>
<th>Child (3 – 16 years)</th>
<th>Adult</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BW (kg)</strong></td>
<td>15</td>
<td>50</td>
<td>70</td>
<td>enHealth (2012 a&amp;b)</td>
</tr>
<tr>
<td><strong>AH (mg/cm²-d)</strong></td>
<td>0.5</td>
<td>0.5</td>
<td>0.5</td>
<td>enHealth (2012b)</td>
</tr>
<tr>
<td><strong>SA (cm²)</strong></td>
<td>3000</td>
<td>7800</td>
<td>15400</td>
<td>enHealth (2012b) 95% surface area of arms, legs, hands and feet</td>
</tr>
<tr>
<td><strong>CF (unitless)</strong></td>
<td>1x10⁻⁶</td>
<td>1x10⁻⁶</td>
<td>1x10⁻⁶</td>
<td>Conversion factor (mg to kg)</td>
</tr>
<tr>
<td><strong>B (unitless)</strong></td>
<td>0.03</td>
<td>0.03</td>
<td>0.03</td>
<td>enHealth (2012b)</td>
</tr>
<tr>
<td><strong>AT (d)</strong></td>
<td>730</td>
<td>730</td>
<td>730</td>
<td>USEPA (1989)</td>
</tr>
<tr>
<td><strong>EF (d/y)</strong></td>
<td>365</td>
<td>365</td>
<td>365</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td><strong>ED (y)</strong></td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>Assumed constant exposure</td>
</tr>
<tr>
<td><strong>CA₂ (mg/kg)</strong></td>
<td>6.9x10⁻⁶</td>
<td>6.9x10⁻⁶</td>
<td>6.9x10⁻⁶</td>
<td>Equation 9</td>
</tr>
<tr>
<td><strong>CA₁(mg/kg)</strong></td>
<td>1.1x10⁻⁷</td>
<td>1.1x10⁻⁷</td>
<td>1.1x10⁻⁷</td>
<td>Equation 9</td>
</tr>
</tbody>
</table>

Derived from enHealth (2012)

\[
CI = \frac{CA \times AH \times SA \times B \times EF \times ED \times ED}{BW \times AT} \tag{Equation 8}
\]
Appendix D. Risk Calculation

D1.1 General Methodology

Health risk was calculated as outlined in the methods presented in Australian (enHealth 2012a) guidelines. Dioxin is considered a threshold chemical and as such risk is calculated using a Hazard Index approach. Under this approach dioxin intake from all relevant exposure pathways (inhalation, inhalation of outdoor dust, inhalation of indoor dust, ingestion of soil, ingestion of root vegetables, ingestion of above ground vegetables and fruit, ingestion of home grown eggs and dermal absorption) are added together along with the background intake of dioxin. This resultant amount is then compared to the tolerable intake as defined by the National Health and Medical Research Council.

The Hazard Index takes the following format.

\[
HI = \left( \frac{C_{II} + C_{Iii} + C_{Iio} + C_{Is} + C_{Ir} + C_{Iav} + C_{Ie} + C_{Id}}{TDI} + Background \right)
\]

Where:

\begin{align*}
C_{II} & = \text{chemical intake from inhalation;} \\
C_{Iii} & = \text{chemical intake from inhalation of indoor dust;} \\
C_{Iio} & = \text{chemical intake from inhalation of outdoor dust;} \\
C_{Is} & = \text{chemical intake from ingestion of soil;} \\
C_{Ir} & = \text{chemical intake from ingestion of root vegetables;} \\
C_{Iav} & = \text{chemical intake from ingestion of above ground vegetables and fruit;} \\
C_{Ie} & = \text{chemical intake from ingestion of eggs;} \\
C_{Id} & = \text{chemical intake from dermal absorption;} \\
TDI & = \text{Tolerable Daily Intake;}
\end{align*}

It is generally considered that a Hazard Index less than 1 represents an acceptable intake. A Hazard Index greater than 1 but less than 10 represents a possible exceedence of the tolerable intake and further consideration should be given to refining / assumptions made in the risk assessment.
D1.2 Risk Results

Hazard Indices were calculated for Scenario 1 and Scenario 2 in three separate age groups (young child, children and adults). Table D 1 displays these results. Table D2 & Table D3 show the breakdown of these results.

Table D 1. Hazard Indices by age and scenario

<table>
<thead>
<tr>
<th></th>
<th>Adult</th>
<th>Child (3 -16)</th>
<th>Child (1-3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scenario 1</td>
<td>0.2</td>
<td>0.6</td>
<td>0.6</td>
</tr>
<tr>
<td>Scenario 2</td>
<td>1.5</td>
<td>1.6</td>
<td>2.5</td>
</tr>
</tbody>
</table>
Table D 2 – Maximum receptor / running concentration (scenario 1) risk calculation by route of exposure

<table>
<thead>
<tr>
<th>Maximum receptor / Running concentration</th>
<th>Adult</th>
<th>% Contribution of exposure</th>
<th>Child (3-16)</th>
<th>% Contribution of exposure</th>
<th>Child (1-3)</th>
<th>% Contribution of exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>(pg/kg-d)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inhalation of Vapours</td>
<td>1.01E-03</td>
<td>2.2</td>
<td>1.06E-03</td>
<td>2.8</td>
<td>3.58E-03</td>
<td>5.0</td>
</tr>
<tr>
<td>Inhalation from Resuspended Particles (Indoor)</td>
<td>1.80E-04</td>
<td>0.4</td>
<td>1.89E-04</td>
<td>0.5</td>
<td>4.21E-04</td>
<td>0.6</td>
</tr>
<tr>
<td>Inhalation from Resuspended Particles (Outdoor)</td>
<td>3.79E-09</td>
<td>0.0</td>
<td>3.98E-09</td>
<td>0.0</td>
<td>1.27E-08</td>
<td>0.0</td>
</tr>
<tr>
<td>Ingestion of soil</td>
<td>7.50E-05</td>
<td>0.2</td>
<td>1.05E-04</td>
<td>0.3</td>
<td>7.00E-04</td>
<td>1.0</td>
</tr>
<tr>
<td>Ingestion from Home Grown Root Vegetables</td>
<td>1.13E-05</td>
<td>0.0</td>
<td>1.29E-05</td>
<td>0.0</td>
<td>2.49E-05</td>
<td>0.0</td>
</tr>
<tr>
<td>Ingestion from Home Grown Eggs</td>
<td>1.49E-02</td>
<td>32.3</td>
<td>1.75E-02</td>
<td>46.3</td>
<td>2.30E-02</td>
<td>31.9</td>
</tr>
<tr>
<td>Ingestion of above ground vegetables</td>
<td>2.96E-02</td>
<td>64.2</td>
<td>1.87E-02</td>
<td>49.5</td>
<td>4.40E-02</td>
<td>61.1</td>
</tr>
<tr>
<td>Dermal absorption of soil</td>
<td>3.47E-04</td>
<td>0.8</td>
<td>2.46E-04</td>
<td>0.6</td>
<td>3.15E-04</td>
<td>0.4</td>
</tr>
<tr>
<td>Total Intake (pg/kg-d)</td>
<td>4.62E-02</td>
<td></td>
<td>3.78E-02</td>
<td></td>
<td>7.21E-02</td>
<td></td>
</tr>
<tr>
<td>Background (pg/kd-d)</td>
<td>5.20E-01</td>
<td></td>
<td>1.25E+00</td>
<td></td>
<td>1.25E+00</td>
<td></td>
</tr>
<tr>
<td>TDI (pg/kg-d)</td>
<td>2.30E+00</td>
<td></td>
<td>2.30E+00</td>
<td></td>
<td>2.30E+00</td>
<td></td>
</tr>
<tr>
<td>Hazard Index</td>
<td>0.2</td>
<td></td>
<td>0.6</td>
<td></td>
<td>0.6</td>
<td></td>
</tr>
<tr>
<td>% of TDI from exposure</td>
<td>2.0</td>
<td></td>
<td>1.6</td>
<td></td>
<td>3.1</td>
<td></td>
</tr>
</tbody>
</table>
## Table D 3 – Maximum receptor / maximum concentration (scenario 2) risk calculation by route of exposure

<table>
<thead>
<tr>
<th>Maximum receptor / Maximum Concentration</th>
<th>Adult</th>
<th>% Contribution of exposure</th>
<th>Child (3-16)</th>
<th>% Contribution of exposure</th>
<th>Child (1-3)</th>
<th>% Contribution of exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>(pg/kg-d)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inhalation of Vapours</td>
<td>1.64E-02</td>
<td>0.5</td>
<td>1.72E-02</td>
<td>0.7</td>
<td>3.82E-02</td>
<td>0.8</td>
</tr>
<tr>
<td>Inhalation from Resuspended Particles (Indoor)</td>
<td>1.19E-02</td>
<td>0.4</td>
<td>1.25E-02</td>
<td>0.5</td>
<td>2.78E-02</td>
<td>0.6</td>
</tr>
<tr>
<td>Inhalation from Resuspended Particles (Outdoor)</td>
<td>2.51E-07</td>
<td>0.0</td>
<td>2.63E-07</td>
<td>0.0</td>
<td>8.39E-07</td>
<td>0.0</td>
</tr>
<tr>
<td>Ingestion of soil</td>
<td>4.96E-03</td>
<td>0.2</td>
<td>6.95E-03</td>
<td>0.3</td>
<td>4.63E-02</td>
<td>1.0</td>
</tr>
<tr>
<td>Ingestion from Home Grown Root Vegetables</td>
<td>7.48E-04</td>
<td>0.0</td>
<td>8.56E-04</td>
<td>0.0</td>
<td>1.65E-03</td>
<td>0.0</td>
</tr>
<tr>
<td>Ingestion from Home Grown Eggs</td>
<td>9.86E-01</td>
<td>32.8</td>
<td>1.16E+00</td>
<td>47.3</td>
<td>1.52E+00</td>
<td>33.3</td>
</tr>
<tr>
<td>Ingestion of above ground vegetables</td>
<td>1.96E+00</td>
<td>65.3</td>
<td>1.24E+00</td>
<td>50.5</td>
<td>2.91E+00</td>
<td>63.8</td>
</tr>
<tr>
<td>Dermal absorption of soil</td>
<td>2.29E-02</td>
<td>0.8</td>
<td>1.63E-02</td>
<td>0.7</td>
<td>2.08E-02</td>
<td>0.5</td>
</tr>
<tr>
<td>Total Intake (pg/kg-d)</td>
<td>3.00E+00</td>
<td></td>
<td>2.45E+00</td>
<td></td>
<td>4.57E+00</td>
<td></td>
</tr>
<tr>
<td>Background (pg/kd-d)</td>
<td>5.20E-01</td>
<td></td>
<td>1.25E+00</td>
<td></td>
<td>1.25E+00</td>
<td></td>
</tr>
<tr>
<td>TDI (pg/kg-d)</td>
<td>2.30E+00</td>
<td></td>
<td>2.30E+00</td>
<td></td>
<td>2.30E+00</td>
<td></td>
</tr>
<tr>
<td>Hazard Index</td>
<td>1.5</td>
<td>1.6</td>
<td>2.5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% of TDI from exposure</td>
<td>131</td>
<td>106</td>
<td>199</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
D1.2 Risk Conclusion

After independent review by a clinical toxicologist and considering the uncertainties, it is concluded that there is enough conservatism in the estimations to be confident that the dioxin emissions from the MM Kembla factory are unlikely to pose a short or long term health risk to the surrounding community.

Residents can reduce their exposure to dioxins and other environmental toxins by thoroughly washing all vegetables to remove any dirt and dust before consumption. It is noted that the simple action of thoroughly washing any home grown vegetables, as is normal good practice, would significantly reduce any potential risk.
Appendix E. References


FSANZ (2012) Personal communication between Adam Capon and Tracy Hambridge regarding upper 97.5% consumption patterns of eggs in the Australian population 29/11/12


Appendix E

References


Wakefield JC (2008) *Dioxins (2,3,7,8-Tetrachlorodibenzo-p-dioxin); Toxicological overview*, Health Protection Agency, United Kingdom.