

## **Technical review of analytical results for dioxins in soil & sludge.**

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### **1. Introduction**

Norfolk Island Administration (NIA 2008a) have requested Toxikos to urgently consider a set of dioxin analytical results from samples collected at various locations of the Island.

Toxikos was provided with soil and sludge sampling results in the following communication.

- NIA (2008a) Personal correspondence by email dated 12<sup>th</sup> December “Quotation on interpretation of soil and sludge dioxin samples”, containing an analytical report ALS (2008a).

Toxikos has not been involved with the data collection and have not assessed its veracity. A summary of the sampling methods and sampling locations was provided by personal communication (NIA 2008b) and the information is summarised in Section 4 of this report.

As described in more detail in Section 2, the concentrations of dioxins are expressed as picogram (pg) (expressed as TEQ) per gram (g) or nanogram per kilogram (kg) of soil, or per litre (L) of sludge.

1 pg is equal to 1/1,000,000,000,000 ( $1 \times 10^{-12}$ ) of a gram (or one million millionth of a gram).

Because a kilogram is 1,000 grams, 1 ng is one million millionth of a kilogram (or 1/1,000,000,000,000 or  $1 \times 10^{-12}$  kg).

## 2. Brief introduction to dioxins and their toxicity

Polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) are collectively called 'dioxins'. There are 75 kinds of PCDDs, and 135 PCDFs.. The different types of dioxins are called congeners.

Dioxins have no known industrial use but occur as unwanted by-products of some industrial and combustion processes such as metal smelting and burning wastes and fuel. Dioxins are ubiquitous in the Australian environment (e.g. Gaus et al. 2001), and it has been estimated that bushfires contribute at least 20 – 30% of the total release of dioxin-like compounds to the Australian environment. Thus humans have been exposed to low levels of dioxins, and human metabolism has coped with dioxins, for thousands of years (OCS 2004). More than 95% of dioxin intake by the general public is via the diet, with the majority of this due to fatty foods derived from animals and fish/shell fish (Liem et al. 2000, Llobet et al. 2003, WHO 2000).

Humans are invariably exposed to a complex mixture of many dioxins and furans, but the degree of toxicity of different dioxins varies from compound to compound. Not all congeners produce toxicity, only a subset. Tetrachlorinated dibenzo-p-dioxin with chlorine atoms attached in the 2, 3, 7 and 8 positions (2,3,7,8-TCDD usually simplified to TCDD) is known to possess the highest toxic potency and toxic effects of this congener have been the most studied.

Because dioxin congeners that cause toxicity appear do so via a common mode of biological action (binding to a specific receptor inside cells<sup>1</sup>) it is possible to rank the toxicity of various dioxins, furans relative to the toxicity of TCDD. Thus the 'toxic' dioxins are assigned a 'Toxicity Equivalency Factor' (TEF) relative to TCDD according to their ability to bind to the receptor and elicit the activated receptor mediated biochemical and toxic responses. The TEFs developed by the World Health Organisation – WHO (van den Berg et al. 1998) are widely accepted as being

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<sup>1</sup> A biological receptor is a protein either on or within a cell that has a specific three dimensional structure allowing a substance to tightly bind to it; an oft referred analogy is a lock and key where the receptor is the lock. When the receptor is thus occupied it changes shape and is able to then initiate a number of biological processes. A substance that activates the receptor is called an agonist but one that binds to it but does not cause the required conformational changes needed for activation is called an antagonist. The receptor for dioxins is the aryl hydrocarbon receptor, abbreviated to *Ah* receptor. Activation of the *Ah* receptor causes activation of tyrosine kinase (an enzyme pivotal to other enzyme networks and receptor signalling) and also increases the transcription of a variety of genes and regulation of other gene networks. The strength of receptor activation and the ensuing responses is dependent upon the concentration of dioxins in the tissue (i.e. the extent of exposure); this determines the number of receptors activated. Activation of the *Ah* receptor in animals by relatively large doses of dioxin can result in endocrine and paracrine disturbances and alterations in cell functions including growth and differentiation. Different dioxin congeners have different ability to activate the *Ah* receptor. Thus in a dioxin mixture a weak agonist bound to the receptor may block the action of a stronger congener.

the most appropriate for human risk assessment and have been adopted by Australian authorities (OCS 2004).

The toxic potency of a dioxin mixture is estimated by multiplying the mass concentration of each individual congener by its respective TEF. The sum of the products provides the TCDD toxic equivalence (TEQ) for the mixture. Thus TEF values for individual congeners in combination with their chemical concentration can be used to calculate the total TCDD toxic equivalent concentration (TEQs) contributed by all dioxin-like congeners using the following equation (assuming dose additivity).

$$TEQ = \sum (PCDD_i \times TEF_{WHO\ i}) + \sum (PCDF_i \times TEF_{WHO\ i})$$

.....**Equation 1**

The mass concentration of individual congeners in soil is commonly below the analytical limit of reporting – LOR (also referred to as a detection limit). However even though a congener is below the LOR it does not necessarily mean that the concentration is zero. Thus for congeners below the LOR it is common practice (Environment Australia Dioxins Program – eg Mueller 2004, US Environmental Protection Agency US EPA 2004) by regulatory authorities to use a value of half the LOR to represent the concentration for these congeners.

Thus in this report the dioxin concentration is expressed as ng TEQ/kg (equivalent to pg/g) where TEQ is the WHO TEQ at half detection LOR (WHO-TEQ<sub>0.5LOR</sub>).

### **3. Methodology for evaluation of results**

#### **a. Soil**

In judging the likelihood of adverse health effects of chemicals in soil it is usually sufficient to simply compare the maximum measured soil concentration of a chemical with an Australian health investigation level (HIL) established for standard land use scenarios. The HIL's and land use exposure scenarios are designed to be conservative. If the soil concentration is less than the HIL then no further assessment is required. However, exceedance of a HIL does not necessarily equate to an unacceptable or imminent health risk to individuals who may come in contact with the soil, rather it flags the need for site specific assessment. Therefore HIL's provide a trigger for when risk is negligible or for further evaluation. Where an Australian HIL does not exist for a contaminant, an equivalent overseas soil guideline may be used or a HIL can be derived from toxicological information for the contaminant of concern and relevant guidance documents (enHealth 2001b).

An Australia HIL is not available for dioxins hence overseas soil guidelines are used as criteria for assessing the acceptability of dioxins in soil.

In addition the reported dioxin soil levels are placed in context by comparison to the range of dioxin soil concentrations commonly found in uncontaminated soil around Australia.

The US Department of Health Agency for Toxic Substances and Disease Registry have derived a soil screening value based on a minimal risk level (equivalent to a tolerable daily intake) of 1 pg/kg/day TCDD<sup>2</sup>, which is approximately two orders of magnitude below any health effect levels demonstrated either experimentally or in epidemiologic studies for both cancer and noncancer health end points (ATSDR 1997). The conservative nature of both the soil screening guideline and the minimal risk level reflects adjustments made for recognized areas of uncertainty, perhaps spanning two to three orders of magnitude. As such, these values are well below levels of exposures associated with demonstrated health effects and are therefore considered to be protective of human health.

#### **b. Sludge**

Sludge is defined as the wet sediment at the bottom of a dam or at the bottom of a water tank. The sludge is described (NIA 2008b) as a mixture of sediment and water. The analytical results are expressed as pg/L indicating the analysis was done on a water sample. The analysis was

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<sup>2</sup> The Australian tolerable daily intake is 2.3 pg TEQ/kg/day (OCS 2004).

conducted on a mixed sample of sediment and water rather than the dry solids (sediment) and possibly pore water (i.e. water that residues in the pore spaces between sediment).

There are no health based guideline values for sludge. In order to comment on the measured dioxin levels in sludge a qualitative assessment was carried out to consider the measured concentrations, possible exposure routes and the likelihood for adverse health impacts.

#### 4. Summary of data collected and analytical results

Two types of samples were collected and analysed (NIA 2008a &b):

- Three composite soil samples made up of soil collected via a sample plug to a depth of 10 cm. The samples were collected randomly from three properties near the burning grounds at Kingstone Reserve in the south west corner of Norfolk Island.
- Chimney samples; these are soil samples at the opposite (east) side of the island (ie controls sites)
- Samples of sludge (i.e. water + sediment) samples were collected from the bottom of two rainwater tanks south east SE of burning pit (samples designated "MENGHETTI", "KING") and the bottom of a dam and a rainwater tank from properties NE of the burning pit (samples designated "DAM", "Adams"). The sludge results are reported in units of pg/L and it is unclear what the analytical results represent water + sediment concentration of dioxins or pore water within the sediment sample collected.

A summary of the sampling locations, samples collected and the analytical results are presented in Table 1.

Results are expressed in this report as dioxin toxicity equivalents - WHO TEQ (refer Section 2 below), the units are presented in nanograms per kilogram or nanograms per litre.

The highest TEQ concentration for the four sample locations in soil expressed as WHO-TEQ<sub>0.5LOR</sub> was 11.72 pg TEQ/g equivalent to 11.72 ng TEQ/kg (ALS 2008a).

**Table 1. Summary of sample locations, samples and analytical results**

<b>Environmental media &amp; Sample name</b>	<b>Description of sample location <sup>a</sup></b>	<b>Analytical result ng/kg or ng/L <sup>b</sup></b>
Soil – Menghetti	Rural property used for cattle grazing.	5.2 (5.18)
Soil – Watson	Residential yard.	8.6 (8.60)
Soil - King	Residential yard.	6.1 (6.06)
2 Chimney samples	Rural property used for cattle grazing.	11.7 (11.72)
Sludge - Dam	Located in a creek,	8.3 ng/L sludge
Sludge - Menghetti Rain water tank	Residential rainwater tank, sludge at bottom of tank. Water used for domestic water supply.	3.9 ng/L sludge
Sludge - King Rain water tank	Residential rainwater tank, sludge at bottom of tank. Water used for domestic water supply	0.56 ng/L sludge
Sludge - Adams	Residential rainwater tank, sludge at bottom of tank. Water used for domestic water supply	3.8 ng/L sludge

<sup>a</sup>. Personal communication NIA (2008).

<sup>b</sup>. Results are from ALS (2008a) units of pg/g are equivalent to ng/kg. The WHO-TEQ at 0.5 LOR was used for the present assessment (refer section 1). Analytical results were rounded to one significant figure in this report.

## 5. Evaluation of soil results

At three locations around the south west corner of Norfolk Island (ie near the burning grounds at Headstone Reserve) and one location on the east side of the Island (control site), composite soil samples were collected at and below the surface (top 10 cm of soil). The dioxin soil concentrations in the South West (5.2 to 8.6 ng TEQ/kg) were marginally lower than those found on the east side of the Island (11.7 ng/kg).

Toxikos is of the opinion that dioxins detected in soil are unlikely to present a health risk to the general population for the following reasons:

- The maximum concentration in soil is within background ranges for dioxins in soil in Australia. The Australian Dioxins Program collected 116 samples from 86 locations across Australia. Dioxins were found in most soils sampled across Australia to be between 0.05 ng/kg and 23 ng/kg (Mueller et al. 2004).  
<http://www.environment.gov.au/settlements/publications/chemicals/dioxins/report-5/pubs/report-5a.pdf>
- The maximum soil concentration (11.7 ng TEQ/kg) is well below health based soil guideline value intended for the protection of public health (50 ng TEQ/kg ATSDR 1997).
- the analytical results show elevated levels of higher carbon length congeners in particular octa dioxin and furan). These results are consistent with the observations by Prange et al (2002) who suggest that natural formation is an important source for higher chlorinated PCDD/PCDF.in coastal areas of Australia (Prange et al 2002, Mueller et al. 2004)
- the dioxin concentrations at sampling sites near the burning grounds at Headstone reserve are not elevated compared to the sample taken from the other side of the Island (ie control sample).

## 6. Evaluation of sludge samples

Samples of sludge (i.e. water + sediment) samples were collected from the bottom of two rainwater tanks south east SE of burning pit (samples designated "MENGHETTI", "KING") and the bottom of a dam and a rainwater tank from properties NE of the burning pit (samples designated "DAM", "Adams"). The sludge results are reported in units of pg TEQ/L. It is assumed (needs to be confirmed) that the sludge analysis represents suspended sediment in pore water (ie water caught between sediment particles at the bottom of the tank).

The samples delivered to the laboratory were a mixture of sediment and water from the bottom of the tanks. The samples have been described by the laboratory as "mirky" samples indicating that the solid content was higher than that encountered in normal drinking water (Personal Communication ALS Laboratory Brisbane 17<sup>th</sup> December 2008). The samples were filtered (approx 55 micron glass filter disc) separating solids with water. The solids were not dried and weighed thus the proportion of solids in the sample and the dioxin sediment concentration cannot be calculated. The solids underwent solvent extraction as did the water sample and then the dioxins concentration in the solvent extract was measured.

The dioxin in dam/water tank sludge samples ranged between 0.56 and 8.3 ng TEQ/L. however it is difficult to interpret the meaning of these results. It is unlikely that the sludge dioxin concentrations are representative of the water concentrations in the tank. These results combine the; concentration of dioxins in sediment, the concentration of dioxins in suspended sediment in water and the concentration of dioxins in dissolved solids within the water.

Additional testing is required to separate the sample (sediment, pore water) and test the sediment and pore water collected in order to allow interpretation of the significance to public health.

## 7. References

Data provided by client:

ALS (2008a). Analytical results for dioxins and furans. Work Order EB0816355. Report issued 3<sup>rd</sup> December 2008. ALS Environmental Brisbane.

ALS (2008b). Quality Control Report. Work Order EB0816355 Report issued 3<sup>rd</sup> December 2008.

NIA (2008a). Personal correspondence by email dated 12<sup>th</sup> December "Quotation on interpretation of soil and sludge dioxin samples". Including attachments in both PDF form (ALS 2008a-b) and excel spreadsheet containing analytical results for dioxin/furans in soil.

NIA (2008b). Personal correspondence by email 16<sup>th</sup> and 17<sup>th</sup> December regarding sampling methods, and sample locations.

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US EPA (2004). Exposure and human health reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and related compounds. December 2003. NAS Review Draft National Center for Environmental Assessment Research and Development. U.S. Environmental Protection Agency, Washington, DC [www.epa.gov/ncea/dioxin](http://www.epa.gov/ncea/dioxin)

WHO (2000). Air Quality Guidelines for Europe 2nd Edition. WHO Regional Publications, European Series 91. World Health Organisation. Regional Office for Europe, Copenhagen. [http://www.euro.who.int/air/activities/20050223\\_4](http://www.euro.who.int/air/activities/20050223_4)