



## Orica Independent Monitoring Committee

### Report for Botany Groundwater Community Liaison Committee on Task 26 for the CLC meeting on 15 June 2010

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At the March 2010 meeting of the CLC, I was requested to provide comment on the following issue:

**Task 26:** To review the 2008 Human Health and Environmental Risk Assessment (HHERA) for soil and groundwater mercury contamination associated with the former ChlorAlkali Plant.

This report is intended to supplement the information in the Human Health & Environmental Risk Assessment (HHERA) on the former Chloralkali Plant prepared by URS and dated 21 August 2008. My report expands on the toxicology of mercury in its various elemental forms, the basis for setting health-based exposure standards, and some brief comment on potential sources of mercury exposure, contrasting these with the exposure sources identified in the URS report.

#### **Toxicology of mercury**

The toxicity of mercury, both in terms of its toxic effects (target organs) and potency, depends to a large extent on its elemental form (oxidation state) – i.e whether the mercury is metallic, as inorganic salts, or complexed with an organic molecule (i.e. in organic form).

Elemental (metallic) mercury is toxic to the nervous system. There is a substantial body of evidence showing that long-term exposures in occupational settings (e.g. workers in plants producing chlorine products; dentists and their clinic staff) can cause subtle behavioural and sensory changes consistent with brain damage. It has also been known that excessive exposure to an organic form of mercury in the diet (methylmercury, mainly from fish) can also result in neurobehavioural impairments in adults and children. Based on this knowledge, women have been advised to limit their fish consumption patterns when pregnant, to avoid damaging the developing brains of their babies.

Another source of exposure to elemental mercury is from dental fillings using mercury amalgam. The possible health impacts from this dental source have been controversial, but reviews by competent Dental and Health authorities (e.g. the Australian National Health & Medical Research Council 1999)

have not implicated this source of mercury exposure as posing a sufficient health risk to require any remedial actions.

The toxicity of mercury has been extensively reviewed (ATSDR 1999; WHO 1991, 2003; Clarkson, Magos & Myers 2003; Clarkson & Magos 2006; Magos & Clarkson 2006). These reviews have summarised the key findings derived from experimental studies in animals, as well as the substantial body of directly relevant information on toxicity to humans. The human database is derived mainly from studies of occupational exposures (mainly elemental and other inorganic forms of mercury), outcomes associated with major environmental exposures (e.g. Minnamata disease), the potential health effects associated with mercury accumulation in seafood (mainly methylmercury) and, more recently, the controversial issues around whether release of mercury vapour from dental amalgam represents a significant health risk to dentists, their staff or their patients. Most of these studies have focussed on risks from chronic low-level exposures, but there are some informative studies of acute toxicity associated with accidents, intentional exposures (suicide attempts) and medical misadventures associated with inappropriate dosing with medicinal products.

There are substantial differences in toxicity associated with inorganic and organic forms of mercury. Dyspnoea (difficulty with breathing) is often the first observable symptom of an acute intoxication with mercury, along with chest pain, nausea and vomiting. Neurotoxicity (brain damage) and nephrotoxicity (kidney damage) are generally considered to be outcomes of mercury toxicity of most concern, although the most sensitive target organs really depend on the chemical speciation of mercury and the exposure route. Adverse health outcomes tend to be different when exposure has occurred to metallic elemental mercury, or to salts of mercury in mercurous ( $\text{Hg}^+$ ) or mercuric ( $\text{Hg}^{++}$ ) valence states, or to Hg bound in organic compounds (e.g. methyl and ethyl mercury; mercury-based antifungals and preservatives such as thiomersol or phenylmercuric acetate). These different chemical forms of mercury can have quite variable systemic absorption (bioavailability) according to the exposure pathway (oral, inhalational, dermal or parenteral), as well as different patterns of tissue distribution and accumulation, and rate of clearance from the body by various elimination pathways.

For example, metallic mercury is very poorly absorbed when given orally, but up to 80% is absorbed via inhalation of mercury vapours. Mercuric ( $\text{Hg}^{++}$ ) salts such as mercuric chloride are quite corrosive, and acute exposure to high doses is often marked by extensive local tissue damage (e.g. the gut wall if taken orally). Acrodynia (painful extremities) was a common symptom in Pink Disease associated with archaic therapeutic administration of mercurous chloride. Both inorganic and organic forms of mercury have been reported to cause contact dermatitis through a sensitisation mechanism. Irrespective of the exposure route and mercury speciation, it is likely that the ultimately toxic form of mercury in tissues arises from *in situ* oxidation to  $\text{Hg}^{++}$  and the main form of clearance from the body is via urinary excretion of inorganic  $\text{Hg}^{++}$ .

Studies on potential Hg-related neurotoxicity have focussed on more objectively measurable effects, such as tremor and finger-to-nose co-ordination, both reasonably sensitive indicators of cerebellar dysfunction. Other studies have addressed some of the more subtle neurotoxic outcomes, such as effects on motor performance and neuropsychological effects such as emotional lability, memory loss, and attention deficits. One study of a cohort of 58 mercury-exposed workers in a chloralkali plant and 35 unexposed controls used sensitive and reproducible measurements of eye-hand coordination and rapid alternating rotation of the forearms to assess neuromotor function (Wastensson et al 2008). The

same sensitive techniques have measured significant deficits associated with MeHg and manganese exposures, as well as other neuromotor disease states.

The importance of the Wastensson study is that, while it demonstrated age- and smoking-related effects on neuromotor functions, it failed to demonstrate any deficits associated with low level elemental mercury exposures in an exposed worker cohort - a median urinary mercury excretion of 5.9  $\mu\text{g/g}$  creatinine (range 1.3-25) compared with controls 0.7  $\mu\text{g/g}$  creatinine (range 0.2-4.1), likely to be representative of mercury exposure in the general community. Using the conversion factor derived by Roels *et al* (1987), this equates to an average air mercury concentration of 4.8  $\mu\text{g/m}^3$  for exposures in the worker cohort. The apparently safe level of exposure to airborne mercury in this study is several orders of magnitude above the estimated concentrations of mercury vapour in off-site areas associated with the former Botany Chloralkali plant in the URS HHERA (0.0000018  $\mu\text{g/m}^3$  indoors and 0.00000041  $\mu\text{g/m}^3$  outdoors).

### **Health-based exposure standards**

Unlike many other hazardous chemicals in the environment, where controlled studies in toxicity testing systems based on animals usually provide the basic dose-response data for derivation of health-based standards, most of the occupational and public health standards for mercury are based on human studies of either occupational exposures, or epidemiological assessments of mercury intakes in the food chain.

#### Inhalational standards for metallic mercury

The health-based exposure standards used in the URS HHERA to benchmark against potential exposure estimates have been based on human data. It is noted that URS has used the more conservative WHO airborne Tolerable Concentration 0.2  $\mu\text{g/m}^3$  for assessing chronic exposures. However, the most widely used benchmark for exposure to elemental (metallic) mercury in air is the 0.3  $\mu\text{g/m}^3$  US EPA (IRIS) Reference Concentration for Inhalation Exposure (RfC), last revised in 1995. The RfC is defined as:

“an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalational exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime”.

The RfC is derived from a range of occupational studies with humans in which hand tremor, memory disturbances, and autonomic nervous system dysfunction were the critical effects evaluated with respect to elemental mercury exposure. The human studies were complemented to some extent by experimental observations in animals. Most of the key studies on which the RfC is based were conducted in the 1980-90s.

It is important to note that most of key studies used to establish the RfC failed to demonstrate a No Observable Adverse Effect Level (NOAEL) for mercury exposure. The majority of the studies demonstrated an 8-hr TWA exposure Lowest Observable Adverse Effect Level (LOAEL) of 0.025 mg  $\text{Hg/m}^3$  (equivalent to 25  $\mu\text{g/m}^3$ ). This is the same value currently accepted as the Australian National Exposure Standard for Hg in the workplace. The exposure estimates which underpin the LOAEL are reasonably robust because the workplace air monitoring data are complemented in some cases by

biomonitoring data, where urinary Hg excretion could be correlated with airborne Hg concentrations using conversion factors determined by Roels *et al* (1987).

To convert this estimate to one relevant to continuous non-occupational exposures, the 25 µg/m<sup>3</sup> figure is adjusted to 9 µg/m<sup>3</sup> using estimates of daily respiratory volumes and adjusting from a working week of 5 days to a continuous exposure period of 7 days/week. The LOAEL is further adjusted (divided by 30x Uncertainty Factor) to derive the RfC of 3 x 10<sup>-4</sup> mg/m<sup>3</sup> (equivalent to 0.3 µg/m<sup>3</sup>). In this case, the 30x UF incorporates a 10 fold factor for protection of sensitive human subpopulations together with the forced use of a LOAEL rather than NOAEL, as well as an additional 3 fold factor for use of a database which lacked studies on potential developmental and reproductive studies.

While the earlier studies used in deriving the RfC were based on workers exposed in chloralkali plants, and demonstrated LOAELS within a fairly tight range of measured TWAs, some of the later studies examined workers in different occupational settings. For example, Ngim *et al* (1992) assessed neurobehavioural performance in dentists, where the airborne Hg vapour concentrations were measured by personal sampling badges and confirmed with blood samples. The LOAEL assessed in this study was 0.023 mg/m<sup>3</sup>, which converted to a continuous exposure Human Equivalent Concentration (HEC) LOAEL of 6 µg/m<sup>3</sup>. Liang *et al* (1993) measured Hg exposure levels (TWA 33 µg/m<sup>3</sup>, urinary Hg excretion average 25 µg/L) in a fluorescent lamp factory and demonstrated some decrements in selected neurobehavioural tests.

In summary, the inhalational RfC is based on a robust set of occupational studies, and includes conservative adjustments designed to be protective of more sensitive humans over quite long periods of exposure.

#### Other health-based mercury standards

The WHO Provisional Tolerable Weekly Intake (PTWI) level of 5 µg/kg body wt (equivalent to a daily intake of 0.71 µg/kg body wt) is for **total** mercury intake (inorganic and organic). This PTWI is strongly influenced by the potential intake of methylmercury in fish and other dietary sources. Ingestion of elemental mercury is a negligible contributor to the derivation of the oral PTWI because of its very low solubility in the gut. The WHO PTWI is based on assessment of epidemiological data on neurological and developmental deficits in human populations exposed to relatively high fish intakes or from well-studied environmental disasters, such as the health effects on Japanese populations arising from industrial pollution of waterways in the Minnamatta region. Where methylmercury is identified as the predominant source of mercury exposure, an alternative PTWI of 1.6 µg/kg body wt (equivalent to a daily intake of 0.23 µg/kg body wt) may be used to ensure protection of the developing foetus of pregnant women who may be exposed. The Provisional Tolerable Weekly Intake (PTWI) for mercury in fish (mainly in the form of MeHg) was revised down to 1.6 µg/kg bw/week by the WHO Joint Expert Committee on Food Additives & Contaminants (JECFA) in 2003. Marien & Patrick (2001) surveyed populations who were relatively heavy consumers of a fish diet, and found that many such groups could exceed a Tolerable Daily Intake of total mercury in the range 0.035 – 0.08 µg/kg bw/day.

The fact that background dietary methylmercury intake can be such a strong contributor to total mercury exposure, means that the adjustments for background exposure in the URS HHERA have been conservatively set at 15% for adults and 80% for children.

## Exposure sources

There are various estimates of daily intake of mercury available in the scientific literature. One such source is the 2003 WHO review that considered health impacts of inorganic mercury (metallic and salts).

### Estimated average daily intake in $\mu\text{g}$ of inorganic Hg

(source: table 1 from the WHO 2003 CICAD, based on the 1999 ATSDR updated toxicological profile of Hg).

Source medium	Elemental Hg vapour	Other inorganic Hg
air	0.04 – 0.2	0
Food - fish	0	0.6*
Food – non fish	0	3.6
Drinking water	0	0.05
Dental amalgam	1.2 – 27	0
Total	1.2 – 27	4.3

\* This figure is inorganic Hg; the intake of organic Hg in the form of methylmercury is likely to be much higher.

It is clear from this table that background exposure from teeth filled with dental amalgam represents the largest potential source compared to environmental sources based on industrial and natural source emissions. The potential for adverse health effects to be associated with Hg vapours arising from amalgams used in restorative dentistry has been a controversial issue. It is not my intention to revisit this debate, but the matter was considered in some depth in a review by the NHMRC in 1999 (NHMRC 1999).

Partly because of the relatively high bioavailability by the inhalational route (80% for elemental Hg) compared to the oral route (<0.01%) airborne levels of Hg assume a relatively high significance for total environmental exposure. Ambient concentrations of  $0.01 - 0.02 \mu\text{g}/\text{m}^3$  have been commonly reported in U.S. air, but concentrations near point industrial sources such as mines, refineries and agricultural fungicide uses have been reported to be as high as  $10 - 15 \mu\text{g}/\text{m}^3$ . Air dispersion from crematoria is another significant localised source of airborne mercury emissions, derived from the mercury present in amalgam-filled teeth of human remains. Fitting mercury trapping devices to stacks is normally done to reduce this source of environmental emission.

In contrast, the URS HHERA modelled airborne elemental mercury in buildings constructed on the Chloralkali plant site to be of the order of  $10 \mu\text{g}/\text{m}^3$  (worst case) and  $3 \mu\text{g}/\text{m}^3$  in outdoor air. These estimates relate to air levels in the immediate vicinity of the site, and other modelling data suggest these would reduce to a maximum ground level concentration of  $0.5 \mu\text{g}/\text{m}^3$  around Block G of the site.

## Comment on URS HHERA

The approach taken in the URS HHERA report is consistent with current practice. It uses conventional modelling approaches to estimate likely exposures to all forms of mercury and all likely exposure routes. The predominant inhalational exposure pathways were identified as elemental mercury from impacted soils and dusts, with some potential exposure associated with extraction and industrial use of contaminated groundwater (noting such use would be subject to licensing agreements). Ingestion and skin contact of contaminated soil by on-site workers was the other potentially significant exposure pathway, most likely to be associated with inorganic and organic forms of mercury.

The risk assessments for mercury under a range of exposure scenarios are expressed in terms of Hazard Index (HI), where the combined exposures are compared with health-based standards, after adjustment for background intake from dietary sources and other sources not specific to the Botany Industrial Park. The effect of adjusting for background is that it permits a much smaller intake from the site-specific sources in the risk assessment.

**In most cases, the reported HIs for assessing site-related mercury exposure in the surrounding community are very low, and the risk has been correctly assessed as negligible.** Only a couple of scenarios have an assessed HI that approaches unity (0.44 for total mercury ingestion by a young child during irrigation activities; 0.1 to 0.5 total risk for all adult and child exposure scenarios (driven mainly by the risk estimate associated with inorganic and organic mercury in groundwater).

In contrast, the estimated HIs for chronic on-site worker exposures exceed 1 for some exposure routes. The total risk HI 35 is driven mainly by on-site inhalation of mercury vapours from impacted soils.

It must be remembered that even a HI estimate which exceeds 1 does not necessarily imply adverse health effects will occur. The health-based standards from which the HI is calculated incorporate conservative safety margins. However, a calculated HI >1 implies that risks should be managed and attenuated where possible. It is noted that the relatively high HI estimates for long-term worker exposure (240 days/year over 30 years) are based on the conservative assumption that exposure would be sustained at current levels and that it would not be reduced by subsequent remediation work.

While noting that the current off-site risk estimates identified in the URS HHERA report are low, the report does address the issue of potential migration of mercury (mainly inorganic) into down-plume groundwater. The URS HHERA report derives some estimates of risk-based soil concentrations (RBCs) and off-site groundwater concentration targets that could guide risk management of the site. These RBCs are conservative and well detailed, including a fair description of the uncertainties that underlie their derivation.



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