

**Comments CRCCARE - Final Report- 11010506 (2009)**  
**Adsorbed Organic Species on Respirable Alumina Particles**

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**Summary**

The Report by D'Angelo *et al* (2009) was supplied to me for my preliminary assessment.

I am available to be commissioned to produce a more thorough literature review of the issues that arise if required.

This recently released report (D'Angelo *et al* 2009) repeats and extends earlier work (Li *et al* 2001 – cited in the report, Ross 1986 - not cited) that shows Alumina particles adsorb carcinogenic volatile organic compounds (VOCs) and promote reactions generating other carcinogens. The conversion might also be enhanced in the presence of sunlight. Many of these compounds are also known sensitizers i.e. they can prime the body producing an allergic reaction to tiny subsequent exposures, a complaint known as Chemical Hypersensitivity, sometimes called Multiple Chemical Sensitivity (MCS). As a general rule, sensitizers are carcinogens.

The report confirms that the adsorbed carcinogens are released from the particles when they encounter water, as found in the eyes, skin, nose and extended airways and fluids of the human body.

The conclusion to be reached from this report (D'Angelo *et al* 2009) is that all previous measurements of airborne carcinogens emitted into the community are void (**gross underestimates**) and that the real dose received by those affected workers and residents is very much higher than previously admitted by ALCOA and the WA Health Department. The smaller the particles are, the larger the surface area, the greater the dose delivered.

**Dust area and carcinogen load versus dust diameter**

This report (D'Angelo *et al* 2009) used a model with dust particles of 50 µm diameter for practical handling. In reality, the surface area of 1 µm dust (most easily transported from stacks and red mud waste) is approximately 1/50<sup>th</sup> of the size of the particles studied. Therefore the carcinogenic load delivered to humans by the smaller particles will be very much larger weight for weight.

Measurement of dusts can be difficult and it was shown in 1998 that measurements performed on Western Australian mine sites, including Alcoa sites, prior to that study were likely to have **underestimated dust loads by a factor of 2.4 to 3.4** [Terry 1998].

The authors emphasized that this would affect epidemiological investigations and compliance with statutory exposure standards – including radiation risk.

Uncontaminated Alumina is hazardous in its own right. In **1968** it was found that aluminium oxide showed evidence of atypical cellular proliferation and **squamous cell atypical**. The authors concluded that aluminium compounds induce a **specific proliferative response of the respiratory epithelium** [Kobayashi].

According to a NIOSH study in **1973** of an alumina plant, “Air sampling and medical investigations indicate that **alumina dust and sinter dust are toxic at the concentrations found**. Workers in the alumina bagging area noted occasional burning of the nose and had a history of skin irritation.”

Although so far not confirmed as a carcinogen in humans,  $\gamma$ -alumina, formed by heating alumina hydrate to 900 to 1000 °C, has been found to be highly fibrogenic when injected into rat lungs, with **carcinogenic potential**.

Exposure of rabbits to low level (0.56 mg/m<sup>3</sup>) alumina dust for 8 hours per day, 5 days per week, for 5 months caused Aluminium to continuously increase in serum and move to various organs.

Percentage increases in the following tissues:

Brain	247%
Lung	15800%
Kidney	165%
Heart	70%
Bone	122%

"brain aluminium concentration is regarded as disturbing in view of the known neurotoxicity of aluminium" [Rollin 1991].

A study of 10 horses living near an alumina plant indicated a cluster of granulomatous enteritis. Ulceration was evident on the buccal, oesophageal, gastric, small and large intestinal mucosa [Fogarty 2003].

Alumina stays in the lungs and acts as a reservoir of toxins, carcinogens and radioactivity. A study sponsored by the alumina industry found that a clearance half-time, due to dissolution of the retained, inhaled deposit, is in the order of 2000 days (5.5 years). “A consequence of the above is that, under conditions of continuous intake, aluminium will accumulate in the body.” [Priest Chapter 10 in Priest 1997].

## Dust from Red Mud Areas

Dust emissions from Residue Areas have been estimated with annual emission of 268 tonnes dust plume 1 km wide, 10 metres high to levels exceeding 100 micrograms per cubic metre (estimated average 26). [Alcoa].

Over 50% of red mud particles are less than 10.0  $\mu\text{m}$  [Glenister 1990]. Alcoa Kwinana reported 410,000 kg of particulate matter below 10.0  $\mu\text{m}$  emitted to air annually in 1998-9 [NPI Kwinana]. This is 1200 kg per day. In a Mines Department study of the relationship between respiratory disease and particle size inhaled by workers [Hewson and Terry 1996], there is an interesting section on alumina and bauxite dusts that refers to lung overload. Hewson and Terry state that **dusts previously considered nuisance dusts could induce cancer** and that current exposure standards for respirable dusts could be an order of magnitude too large [Pritchard 1989]. Analysis of particulates from the Kwinana oxalate kiln found 15% below 2.5  $\mu\text{m}$ , and 43% below 10  $\mu\text{m}$  [Stackair 1997].

In 2003 Alcoa lost its self-policing rights for dust after it was disclosed that an employee had **falsified dust level recordings** on a database and Alcoa was fined for breaching dust emissions from its mud lakes [Gault, Southwell].

The very small particles will carry the carcinogens further into the body and deliver concentrated doses of carcinogens to the most sensitive tissues.

Epistaxis , or more commonly known as blood nose, is a common occurrence among Alcoa workers and can be induced by caustic dusts [Power 2002] and certain aldehydes (see below).

Inhalation of dust is the major source of radiation dose, not discussed further here.

The cancer and death rates in workers and the community can thus be expected to be much higher than calculated from the concentration of carcinogens previously measured in filtered. This provides further evidence that resources are required so that all people ever exposed to the air and dust pollution emitted by Alumina refineries should be tracked over their entire lives to determine age at death and cause of death.

## The Carcinogens Mentioned

The authors cover only a few of the known carcinogens emitted by ALCOA refineries. The report (D'Angelo *et al* 2009) is limited due to funding and time constraints, as well as the hazardous nature of the necessary analytical standard samples required. For example it is understandable that the researchers did not study acrolein (2-propenal), known to be

emitted by the refineries (Oxalate furnace and precipitation area), light sensitive and an extremely dangerous material to transport and use in the laboratory. Likewise they did not study Methacrolein (2-methyl-2-propenal), 2-methylpropanal, 2-methylpropenal, 4-methylhexanal or 2,4-Hexadienal which are also emitted by the refineries.

Below I provide some relevant information on the following selected compounds mentioned in the report (D'Angelo *et al* 2009).

Acetaldehyde, Acetone, Diacetone alcohol and Mesityl Oxide, Acetophenone, Benzaldehyde, Benzene, 2-Butanone = Methyl Ethyl Ketones (MEK), Crotonaldehyde = 2-butenal, Propionaldehyde, 2-methyl-2-pentanal, 2-methyl-2-pentenal, Trans-2-hexenal, 2-hydroxybutanal, 3-hydroxybutanal

### **Acetaldehyde**

Egle [1970] determined acetaldehyde retention in eight volunteers after mouth and nose inhalation of concentrations of 100 to 800 mg/m<sup>3</sup> for 1 to 4 minutes from a recording respirometer. Total retention varied between 45 and 70% independently of whether inhalation was through the nose or the mouth.

Acetaldehyde has been shown to dissolve into the saliva and to be a **local carcinogen** in the **human upper digestive tract** [Salaspuro 2006]. DNA adducts have been studied in **human buccal epithelial cells** exposed to acetaldehyde *in vitro* [Vaca 1998].

Bittersohl [1974] reported a **fivefold** higher than expected incidence of cancer among 200 German factory workers exposed to 1 to 7 mg/m<sup>3</sup> acetaldehyde as well as to other aldehydes. The workers had **squamous-cell cancers of the mouth** (N = 2).

Acetaldehyde is reasonably anticipated to be a human carcinogen based on **sufficient evidence of carcinogenicity** in experimental animals [IARC 2005].

Acetaldehyde is listed as a carcinogen by the State of California under Proposition 65, California's Safe Drinking Water and Toxic Enforcement Act of 1986. Acetaldehyde associated with alcohol consumption is regarded as 'carcinogenic to humans' (IARC Group 1), with **sufficient evidence** available for the oesophagus, **head and neck** as sites of carcinogenicity. It forms DNA adducts in **human buccal** cells.

Acetaldehyde induces squamous cell carcinomas and adenocarcinomas in rats and **laryngeal** cancer in hamsters [IARC Acetaldehyde].

ACGIH recommends a ceiling of 25 ppm (45 mg/cubic metre). The Western Australian Government described a level of 210 ppb as "acceptable" ground level concentration [K481]. **This is 42 times the OEHHA REL.**

The Liquor burner at Wagerup emitted 4.0 mg/m<sup>3</sup> (**444 times the OEHHA REL**) of acetaldehyde which translates to 120 gm/hr assuming 30,000 m<sup>3</sup> per hour flow. Note this is almost one tenth the emission from the Kwinana burner.

Alcoa has shown that acetaldehyde is 224 mg/m<sup>3</sup> (**24,000 times the OEHHA REL**) in **green liquor tank vent emissions**, together with a host of compounds including acetone at 2000 mg/m<sup>3</sup>.

Acetaldehyde is also present in **causticisation vent emissions** at 16 mg/m<sup>3</sup> (**4000 times the OEHHA REL**) and the Wagerup Mill vent showed 14 mg/m<sup>3</sup>.

Acetaldehyde is a sensitizer or adjuvant, increasing the incidence of respiratory tract tumors in hamsters exposed to benzo[a]pyrene (also a proven oral carcinogen in human tissue).

According to Alcoa [2002] acetaldehyde has an extremely low odour threshold of 0.00002 mg/m<sup>3</sup>. Respondents can find the smell at this level pleasant, but irritating at higher concentrations.

It self-condenses to paraldehyde and metaldehyde (snail poison).

**NIOSH recommends that occupational exposure to acetaldehyde be limited to the lowest feasible concentration** [NIOSH NTP]. Acetaldehyde is likely to come under closer scrutiny after reclassification of formaldehyde. Further work on characterizing Alcoa worker and community exposure to acetaldehyde is clearly required.

### **Acetone, Diacetone alcohol and Mesityl Oxide**

The study (D'Angelo *et al* 2009) demonstrated that Acetone is transformed to Diacetone alcohol (4-hydroxy-4-methyl-2-pentanone – a known eye and skin irritant) and Mesityl Oxide (4-methyl-3-penten-2-one – a severe skin irritant and lachrymator) on the alumina particles. Further review on cancer potential from these compounds would be useful.

### **Benzaldehyde**

Emitted in large quantities from Oxalate destruction. Further review on cancer potential from this compound would be useful.

### **Benzene**

As early as the 1920s, scientists knew benzene caused cancer. However, it was not until some twenty years later that officials instituted 100ppm as the “permissible” exposure level, which was lowered to 10ppm in 1978, and 1 ppm 1990. As a renowned benzene researcher (Infante 1988) noted, the evolution of what was considered a permissible level of benzene was not driven by dramatic improvements in scientific knowledge regarding the mechanisms by which benzene caused cancer, but rather was the result of **a continued**

**struggle for health** by unions, workers, physicians, and scientists **against powerful economic interests. The debate regarding a permissible exposure level for benzene exposure continues with mounting evidence that there is no safe threshold for this carcinogen.**"

Benzene was measured by Alcoa at Kwinana in the range of 35 to 50 mg/cubic metre (**over 800 times the REL**), giving emission rate above the USEPA reportable limit. Actual flow of LB feed was closer to 50,000 cubic metres per hour at 74 degrees C.

### **2-Butanone = Methyl Ethyl Ketone (MEK)**

Known to cause oral cancer (Burgaz 2002). Further review on cancer potential from this compound would be useful.

### **2-hydroxybutanal and 3-hydroxybutanal = acetaldo**

3-hydroxybutanal = acetaldo. Causes oral cancer. Covered in IARC 1985.

Further review on cancer potential from these compounds would be useful.

### **Crotonaldehyde = 2-butenal**

Crotonaldehyde - Butenal (2-Butenal) sources of release include:

Green Liquor (35A) Vent 76 mg/m<sup>3</sup>

Slurry Storage Tanks (25A) 48 mg/m<sup>3</sup>

This study (D'Angelo *et al* 2009) demonstrated that **alumina converts acetaldehyde to crotonaldehyde.**

Crotonaldehyde is considered as "**justifiably suspected of having carcinogenic potential**" in the German "MAK" list, and there is **no threshold limit value** at the work place [Eder 2001]. Crotonaldehyde is described as **geno toxic, mutagenic, and carcinogenic** [Chung 1986, Bittersohl 1974], and it forms exocyclic 1,N<sup>2</sup>-propanodeoxyguanosine adducts as the main DNA adducts [Wilson 1991, Hecht 2001].

Nath [1998] measured higher acrolein-dG and crotonaldehyde-dG levels in **gingival** tissue of smokers than in that of non-smokers.

A severe lachrymator. There is no "safe" level of crotonaldehyde.

### **Hexane**

Known to cause oral cancer (Burgaz 2002). Further review on cancer potential from this compound would be useful.

## **Propionaldehyde = Propanal, 2-methyl-2-pentanal and 2-methyl-2-pentenal**

Propionaldehyde is emitted from the Oxalate destruction. It is an irritant to the eyes, skin and respiratory system. This study demonstrated formation of the 2-methyl-2-pentanal and 2-methyl-2-pentenal on the alumina surface. Further review on cancer potential from these compounds would be useful.

## **Toluene**

Toluene is continually released in large quantities from the red mud waste areas as well as Liquor Burners.

Toluene produces **oral cancers** in test animals [Maltoni].

Toluene shows an important **geno toxic effect in cells of the human buccal mucosa** [González-Yebra 2009]. The incidence of nuclear abnormalities was significantly higher in the exposed group when compared to the control group. A positive relationship between the incidence of micronuclei and the toluene concentration in the environment was found.

The Liquor burner at Kwinana emitted 5.9 mg/m<sup>3</sup> of toluene.

Toluene was found in Wagerup Residue Areas at a concentration of 98 mg/m<sup>3</sup>

In a study of spray-painters exposed to toluene, isobutylacetate and dust histological examination showed in no case a normal nasal mucosa [Hellquist].

The authors suggested the possibility of an early pre-symptomatic detection of nasal mucosal disturbances and questioned whether the existing TLVs are adequate in preventing damage to the nasal mucosa and adherent clinical symptoms.

Toluene is decomposed yielding photochemical smog including other carcinogens.

## **Trans-2-hexenal**

2-hexenal is a bifunctional compound that forms 1,N<sup>2</sup>-propanodeoxyguanosine adducts and is **mutagenic** and **genotoxic** [Eder 1999].

In human studies [Dittberner 1997], using 7 non-smoking healthy volunteers, the number of micronuclei (MN) was determined in exfoliated **buccal mucosa cells** before and after rinsing the **mouth** with an aqueous 10 ppm solution of 2-trans-hexenal during 3 consecutive days. All individuals showed at least a doubling of the MN frequency during one of the next 4 days. An increase of the mean group MN frequency was observed on the fourth day, becoming significant between the sixth and the seventh day.

## **Xylenes**

Xylenes are known to cause **oral cancer** [CPDB, Maltoni]. The Liquor burner at Kwinana emitted 5.9 mg/m<sup>3</sup> of xylenes.

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