The Secretary
Senate Community Affairs References Committee
Parliament House
Canberra

Dear Secretary and Committee,

Inquiry into workplace exposure to toxic dust

In my submission to the Inquiry, I wish to focus the Committee's attention on the **Alumina industry** where workers and surrounding communities have been exposed to numerous toxic elements contained in:

- bauxite dust
- caustic chemicals used to process bauxite into alumina
- the alumina itself
- dusts produced from liquor burning and oxalate destruction
- the residue or red mud.

I have summarized the hazards under various headings below and would be pleased to provide further information at the public hearings if required.

This submission is part of my wider study of hazards in the alumina industry.

I believe the Committee should examine the health and cause of death of former alumina industry workers, particularly those employed in the early years from 1960 to the 1980s as these workers can be expected to have developed more occupational disease.

Yours sincerely,

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Toxic Dust Hazards in the Alumina Industry

Workers are exposed to heavy airborne dust loads from bauxite loading and grinding, caustic dusts inside washers, thickeners and sand traps, dusts from calciners, the liquor burner, oxalate kiln, alumina and hydrate dusts in the bagging department and residue (red mud) when driving to the red mud lakes.

Over 50% of red mud particles are less than 10.0 μm [Glenister 1990].

Alumina producers accumulate red mud waste and are not permitted to dump it in the sea, one reason being that it leads to agglutination of fish gill tissues [Dethelsen, Sigmond]. It must be kept wet at all times and producers have emergency crews with tankers if sprinkler systems fail.

Worksafe time-weighted average limits are 10 mg/m^3 for inhalable and 5 mg/m^3 for respirable dusts. The NEPM for PM10 is 0.05 mg/m^3 for a one day average and the NHMRC interim indoor air quality goal is 0.09 mg/m^3.

Alcoa Kwinana reported 410,000 kg of particulate matter below 10.0 μm emitted to air annually in 1998-9 [NPI Kwinana]. This is 1200 kg per day. It would require 120,000,000 m^3 of air to dilute it to 10 mg/m^3. This equates to a square kilometer to a height of 120 metres. Clearly on no wind days this level would be exceeded.

In 1997 it was **assumed in the absence of measurements** that dust emissions from the liquor burner at Kwinana would be 250 mg/m^3 [Galton-Fenzi].

The liquor burner at Kwinana emitted dust at the level of 39 mg/m^3 in December 2001.

Animal studies of dust load have been made.

Male rats were injected into their trachea with a suspension of dust of ore mine dead rock of high quartz content and mixed composition (particle size: 0-60 micrometers) [Tatrai].

Three and six months after treatment, foreign body granulation and after twelve months a non-fibrotic diffuse lung disease was seen in the animals.

After twelve months of exposure, non-specific, mature sinus histiocytosis and small focal epitheloid cell reaction were found to develop in the cervical, lung-hilar and retroperitoneal lymph nodes. Comparison of the histopathological changes with those seen in experimental silicosis induced by DQ12 quartz showed that the hazardous effect of industrial dusts is not identical with their fibrogenic effect.

At least one study has been made on particle deposition efficiency of artificial aerosols in the human maxillary sinus [Hyo N]. Deposition efficiencies in the human maxillary sinus were found to be 3%, for particles of 3-10 microns in diameter.

It has been found that 4-fold or greater risk increases of cancer of the nasal cavity and sinuses were associated with a history of chronic nasal diseases, including those occurring 10 or more years prior to cancer diagnosis [Zheng et al].

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 [Southwell y].

Measurements of dust during manual descaling found average levels of 2.9 with a geometric standard deviation of 3.2 mg/m^3

Other measurements comparing various sites at the Alcoa plant showed manual descaling with the high dust exposure of 7.9 (single measurement) and maximum levels of 1.5 for a residue operator, 5.6 for calciner operator/train loading and 8.1 reclaimer for operator. One sample measured 17.0 mg/m^3 for a calciner operator and this was rejected as excessive sample for further analysis.

The mass median aerodynamic diameter (MMAD, defined as the equivalent aerodynamic diameter such that 50% of the mass of the sample is associated with smaller particles) was found [Terry 1998] from 8 valid tests (from 11 measurements) to be 22.5 μm with a geometric standard deviation of 1.6 mg/m^3

Another measurement during manual descaling found the MMAD to be 18.8 μm.

Although much is made of small dust particles' health risks due to their potential to travel far into the lung, risks associated with larger particles should not be discounted.

Underestimation of Dust Loads

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.

All **Alcoa measurements listed here have not been corrected** on the basis of the 1998 findings by Terry *et al*.

The study showed that the previously used 7-hole (7H) and closed face (CF) samplers should be replaced with the Institute of Occupational Medicine sampler.

Alcoa made 43 measurements of "total" dust in K56 Digestion Building 17 from January 1990 to December 1998, averaging 7 to a maximum of 119 mg/m^3 over the period with the occupational exposure limit of 10 mg/m^3 exceeded on 6 sampling occasions.

Alcoa made 8 measurements of "respirable" dust in K53 descalers from September 1979 to November 1993, showing a maximum of 31.1 and a mean of 6.3 mg/m^3 over the period. The OEL was exceeded in 3 samples.

Alcoa made 4 measurements of "respirable" dust in K53 descale from January 1996 to December 2000, showing a decline from 1 to 0.2 mg/m^3 over the period.

Alcoa made 23 measurements of respirable dust in K56 Digestion Building 15 from January 1990 to December 1998, averaging 0.59 to a maximum of 1.58 mg/m^3 over the period .

Alcoa made 28 measurements of respirable dust in K56 Digestion Building 16 from January 1990 to December 1998, averaging 0.47 to a maximum of 3.1 mg/m^3 over the period .

Alcoa made 27 measurements of respirable dust in K56 Digestion Building 17 from January 1990 to December 1998, averaging 0.38 to a maximum of 1.2 mg/m^3 over the period .

Alumina a possible carcinogen

One of the most prominent dusts at refineries is of course alumina (aluminium oxide).

Alumina is classified by the International Agency for Research on Cancer (IARC) in Group 3, i.e. cannot be classified as to carcinogenity to humans.

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

Lung Overload associated with Alumina and Bauxite

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.

Particulates often designated PNOC (particulates not otherwise classified) by the ACGIH TLV committee might in fact be more hazardous than assumed.

In rats at lung dust loadings of 1 mg g^{-1} lung tissue, particle clearance is delayed, however at 10 mg g^{-1} particle pulmonary clearance virtually ceased, leading to the term **dust overloading**. It was found that macrophages containing about 150 particles become immobilized and may release enzymes and other substances causing fibrosis [Morrow 1988, Vincent 1989 Muhle 1990, Mastomatteo 1993].

It was found volume, not mass is critical to immobilization.

Hewson and Terry state that dusts previous considered nuisance dusts could induce cancer and that current exposure standards for respirable dusts could be an order of magnitude too large [Pritchard 1989].

The ACGIH proposed a respirable dust standard of 3 mg m^{-3} for PNOC compared with the Western Australian standard of 5 mg m^{-3}

Arsenic

Arsenic was detected in Kwinana refinery ambient air in March 2002.

Sampling was done at typical worker locations including "in the liquor burner plume".

Arsenic was detected in:-

Building 30	in heater steam plume
Building 48	Granivor overflow vent odour
Guardhouse	Outside in liquor burner plume
Building 44	Baro Flash tank PRV vent steam
Building 25	#6 Rodmill discharge top door
Building 51	Tank top walkway liquor burner plume

The maximum arsenic level detected was 0.15 $\mu g/m^3$. It was confirmed that the liquor burner plume descends to ground level within the plant.

A study on the occupational etiology of sinonasal cancer in three cases, which had arisen among glass workers, included an adenocarcinoma, a melanoma, and a squamocellular carcinoma. Evidence was found for cancer induction by arsenic dust [Battista 1996].

Increased incidence of bladder cancer has been found at a sulfuric acid factory with airborne arsenic levels of 11 micrograms/m^3 [Englander 1988].

Arsenic inhalation is known to be associated with skin cancer [Hewson Terry 1996].

Arsenic is present in mud thickener launder scale (26.5 ppm) and floor scale 12 ppm.

The USEPA has set a target of zero for arsenic in drinking water.

Beryllium

The Committee will no doubt receive many submissions on this element following the recent publicity concerning beryllium disease in Navy personnel arising from descaling of ship surfaces.

Beryllium is present in red mud thickener launder scale (0.2 ppm) and floor scale (0.1 ppm).

Beryllium is present in red mud waste at 0.7 ppm.

The US National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, considers workers as exposed if the airborne concentration of beryllium is 100 ng/m^3 [Henneberger].

This concentration will be present if red must dust is present at 143 mg/m^3

Beryllium is known to induce debilitating and often fatal disease at low chronic exposures and significant lung function improvement may be seen following cessation of beryllium exposure [Cullen 1987, 2004].

A useful test was devised for chronic beryllium disease involving the proliferative response of bronchoalveolar lymphocytes to beryllium [Cullen 1989]

Screening for chronic beryllium disease will be vital for alumina and aluminium workers [Cullen 2005].

Beryllium compounds are irritating to the respiratory system and can cause skin sensitization.

Diesel particulates

Work inside alumina refinery tanks operating diesel equipment is comparable to underground mining where the respirable fraction of dust is about 50% of the inspirable aerosol [Terry 1998].

The following table is a summary of literature on cancer induced by diesel particulates.

Cancer	Odds Ratio or relative risk	Year	Reference
laryngeal cancer	OR 5 - 5.4	1994	Goldberg
lung	70 –year unit risk 2.1-5.5 per 10,000 workers per µg/m3	2001	Dawson Alcoa 8.35
lung	RR 1.3 95% CI 1.3-1.4	2001	Bofetta Alcoa 8.34
lung	1.7 95% CI 0.58-6.0 after 20 years exposure	1999	Saverin Alcoa 8.27
lung	RR 1.63 95% CI 1.14 – 2.33	2000	Gustavsson Alcoa 8.31
lung	RR 2.4 95% CI 1.3 – 4.5		Gustavsson Alcoa 8.6
lung	RR 1.44 95% CI 1.01-2.05 Adjusted for smoking	2000	Larkin Alcoa 8.32
lung	2.31 95% CI 1.44-3.70 heavy equipment operators	1999	Bruske-Hohlfeld Alcoa 8.25
lung	OR 1.43 95% CI 1.23-1.67 Adjusted for asbestos and smoking	2000	Bruske-Hohlfeld Alcoa 8.29
lung	RR 1.47 95% CI 1.29-1.67		Lipsett Alcoa 8.26
lung	OR 1.21 95% CI 0.78-2.02	1990	Alcoa 8.5
Sinonasal	SIR 1.16 95% CI 0.98 – 1.36	2001	Bofetta Alcoa 8.34

Thorium

Alumina refinery workers are exposed to dust containing up to 400 ppm Thorium. This is more than 100 times the thorium concentration of basalt and 40 times the average for continental upper crust.

Thorium is an α-particle emitter. A series of unstable daughter decay products are formed, including radium and radon.

Thorium was found in the 1960s to cause maxillary sinus cancer [Goren]. An x-ray contrast medium called Thorotrast was previously injected and shown to remain in the sinus for years [Galati].

Squamous cell Carcinoma of the parotid gland was found in 2 cases following sialography with Thorotrast 28 and 45 years previously [Nielsen 1979]. Both cases were histologically established as carcinoma and the presence of Thorotrast in the tumours was confirmed by autohistoradiography.

It was suggested that the tumours may have developed from metaplastic ductal epithelium after many years of exposure to the alpha radiation from Thorotrast deposits in the gland.

Estimation of risk from radiation exposure at Kwinana

Risk from inhalation of radioactive material at Kwinana requires knowledge of breathing rate, assumed to average 1.2 m^3 /hr, 2000 hours per year, average radon and inhaled dust concentrations and composition of the dusts.

Unfortunately the amount of data is limited.

Estimates of geometric mean dose of 5.0 – 7.8 milliSieverts (mSv) have been made for underground bauxite mines in Europe [Bernhard 1984 cited in Hewson 1991].

This level of exposure would result in up to 200 excess cancers per million person years based on average energy dose to the whole body [Selinger 1989], however these figures do not allow for specific tissue susceptibility and damage power of particular isotopes.

When considering isotopes that decay in series, the energy contributions of all daughters (or progeny) have to be added.

Estimates of excess deaths from inhalation of radionuclides at Kwinana

Tables of mortality from cancers induced by inhaled radionuclides are available [Eckerman 1998].

Considering the data for Thorium-232 and its progeny only, a figure of approximately 6 deaths per million workers is expected for either acute one-time or chronic inhalation of **one Becquerel**. Note this **excludes** contributions of Radon- 220, Polonium-212, Polonium 216 and Thallium-208.

The red mud dust at Kwinana contains 400 ppm Thorium. Using this figure we can calculate that Thorium alone contributes at least 1.5 Becquerel per gram activity to the dust. This is in agreement with limited published data of up to 26 Becquerel per gram total activity for bauxite residue [O'Connor].

If we conservatively assume 3 Becquerel per gram total activity for bauxite residue at Kwinana and a dust level of 10 mg/m^3 a worker exposed for 1920 hours per year and breathing 1.2 m^3 / hour would inhale 23 g/year or 69 Becquerel.

The cancer mortality rate is therefore 414 per million.

If the worker is breathing this amount for 20 years, the cancer mortality rate is therefore 8280 per million. As noted above the actual inhalation of radionuclides will vary for each worker.

To this figure must be added the excess deaths expected from external radiation at the plant.

Silica

Silica makes up about 21% of red mud waste. The committee will no doubt receive much data on this hazard in other submissions.

Silica is usually associated with lung cancer [Bruske-Hohlfeld, IARC Silica].

Nasal cancer risk in foundries show odds ratios up to 5.9 and this could be at least partially due to airborne silica [Comba 1992].

Alcoa made 4 measurements of silica in K53 descale from January 1996 to December 2000. Concentrations ranged from 0.0005 to 0.04 mg/m^3

Alcoa made 8 measurements of silica in K56 Building 17 digestion from January 1996 to December 1998. Concentrations ranged from 0.002 to 0.09 mg/m^3

Sodium hydroxide (caustic soda) and Bayer liquor

Workers at alumina plants are required to use recycled caustic waters to hose down items of equipment as directed. During this hosing, caustic mists and, in dry air, dusts are generated.

Prolonged exposure to high concentrations of caustic soda can cause ulceration of nasal passages and severe eye and skin injury.

Irreversible obstructive lung disease following chronic occupational exposure as well as after a one-time high-level exposure to sodium hydroxide is known [Hansen and Isager 1991, Rubin *et al* 1992].

In 1969 the carcinogenic potential of sodium hydroxide was known.

Damage to the esophagus by sodium hydroxide can induce cancer scarring after swallowing [Lansing 1969]. Malignancy may develop even years after the removal of such tumors in the remaining part of the gullet. Total oesophagectomy was suggested instead of bypass [Csikos].

The patients with scar cancer comprised 7.2% of the overall oesophageal carcinoma cases. The interval between the caustic burn and the diagnosis of scar carcinoma was found to be 46.1 years. It was 50.9 years in those patients who drank lye before the age of 12, but 14 years less when it happened in adulthood.

Sodium and potassium hydroxide induce skin cancer with a long latency period in mice [Ingram].

Caustic substances cause tissue destruction through liquefaction or coagulation reactions and the intensity of destruction depends on the type, concentration, time of contact and amount of the substance ingested [ACGIH].

Rats that inhaled unmeasured concentrations of sodium hydroxide aerosols for 30 minutes per day suffered pulmonary damage after 2.5 months. [Mamede]

Workers at Kwinana report numerous rat deaths.

In humans, burning/redness of the nose, throat and eyes is found on exposure to caustic mists with NaOH concentrations between 0.005 and 0.7 mg/m^3.

Alcoa made 11 measurements of NaOH mists in K53 Descalers from September 1979 to November 1993, averaging 0.26 to a maximum of 0.45 mg/m^3 over the period.

Alcoa made 57 measurements of NaOH mists in K58 Precipitation Building 45 from November 1993 to May 2002, to a maximum of 2 mg/m^3 over the period.

Alcoa made 40 measurements of NaOH mists in K58 Precipitation Building 46 from November 1993 to May 2002, averaging 0.2 to a maximum of 2 mg/m^3 over the period.

Alcoa made 20 measurements of NaOH mists in K58 Precipitation Building 60 from November 1993 to May 2002, averaging 0.2 to a maximum of 15.4 mg/m^3 over the period. The occupational exposure limit was exceeded by a factor of nearly 8 times on one sampling occasion.

On August 9, 2002, Alcoa issued a "World Wide Health Protocol - Mandatory Requirement" for all personnel exposed to dust, welding fume or caustic fumes to fill in a questionnaire annually.

Hill Brothers Chemical Company, a major supplier of sodium hydroxide warns of cancer risk in its Material Safety Data Sheet [Hill Brothers].

Bayer Liquor, a mixture of sodium aluminate, hydroxide, sulfate and chloride has its own Alcoa MSDS. It says "Vapors and mists can cause severe irritation of respiratory tract and lung damage". Alcoa recommends use of a personal respirator if the caustic mist exceeds 2 mg /m^3.

In 2003, environmental sampling for operation and maintenance workers found "unacceptable" caustic mist concentrations in calcination at KW 59 (building 50) and "significant" in **K58** (buildings 45 and 46) and K28.

On the basis of these measurements, the Occupational Hygienist recommended personnel must wear a minimum of Class P2 disposable respirator, precipitation tanks on caustic wash cycle must be barricaded with Class P2 respirators provided for people entering the area.

Caustic soda washing of precipitators was increased in 2000 to economize on contractor descaling costs.

Sodium oxalate

This compound is a waste product, sent to kiln for thermal destruction or stored at red mud lakes. Single exposure causes severe redness and swelling of mucous membranes [Merck].

A very rare disease, antrolithiasis, involving deposition of calcium oxalate nodules, is most commonly found in maxillary sinus [Nass Duce 2003, Mori 2000].

The possibility that inhaled oxalate dusts at Kwinana result in precipitation of calcium oxalate particles in the sinus and other organs, especially the kidneys, should not be discounted. The carcinogenic potential of oxalate solids in the sinus should be investigated.

The sodium oxalate cake produced at Wagerup contains 0.9 ppm thorium, 3 ppm uranium, 84 ppm gallium, 20 ppm arsenic, 3.7 ppm mercury, 0.6 ppm nickel, 17 ppm selenium, 62 ppm vanadium.

When sent to the liquor burner or oxalate kiln, these elements can be expected in the particulate emissions.

Analysis of particulates from the Kwinana oxalate kiln found 15% below 2.5 μm, and 43% below 10 μm [Stackair 1997].

Strong acid mists including sulfuric acid

Alumina workers are exposed to strong acids in "acid cleaning" tasks e.g. when removing scale from cooling plates or similar equipment.

Strong acids are carcinogens of the upper and lower respiratory tract [Soskolne, NIOSH NTP].

Alcoa made 11 measurements over a period of 9 years of sulfuric acid mist in K56 Digestion Building 17 (labeled "draft only") with concentrations ranging from 0.001 to 13 mg/m^3

Thus on one occasion (date not recorded) a level 13 times the occupational exposure limit was found.

Alumina workers are also exposed to sulfuric acid as one of the constituents of diesel engine exhausts.

In 1995 the WA Mines Safety and Inspection Regulations specified a maximum sulphur content of 0.5% compared to the tougher USA regulations of 0.05% [Terry 1998].

A study concluded that Western Australian industry, were not using best practicable technology and suggested revision of Australian Standard 3640 and recommended immediate implementation of control measures to reduce diesel emissions [Terry 1998].

Vanadium compounds

A Western Australian study of minesite dusts identified respirable vanadium as a health concern [Terry 1998].

Red mud waste contains over 400 ppm vanadium.

Vanadate exposure of rats significantly decreased the pressor response to 1-noradrenaline, and increased the pressor response to sinus-carotid baroreceptor stimulation and the hypotensive responses to 1-isoprenaline and acetylcholine [Matera].

Vanadium pentoxide catalyzes formation of maleic anhydride from benzene in air.

In a Mines Department study of the relationship between respiratory disease and particle size inhaled by workers, vanadium dusts are said to produce **green tongue disease** and leave a metallic taste.

A number of workers complain of a metallic taste associated with alumina refinery emissions.

Summary table of airborne dusts in the alumina industry with acute and chronic health limits and examples of measured levels from one company's reports, where available (PINJ = Pinjarra refinery data, LB = Liquor Burner, OX = Oxalate residue).

CHEMICAL	ATSDR MRL		ALCOA KWINANA EMISSIONS In $\mu g/m^3$ unless otherwise marked
ARSENIC	Provisional Oral Acute Chr.	0.005 mg/ kg/ day 0.0003 mg/ kg/ day	48 ppm in mud 295 g/day PINJ 13.5 g/day LB
BERYLLIUM	Oral Chr.	0.002 mg/ kg/ day	0.7 ppm in mud
CADMIUM	Oral Chr.	0.0002 mg/ kg/ day	4.5 ppm in mud
CHROMIUM(VI), AEROSOL MISTS	Inh. Int.	0.000005 mg/ m^3	
CHROMIUM(VI), PARTICULATES	Inh. Int.	0.001 mg/ m^3	314 ppm in mud
COBALT	Inh. Chr. Oral Int.	0.0001 mg/ m 3 0.01 mg/ kg/ day	22 ppm in mud
FLUORIDE, SODIUM	Oral Chr.	0.05 mg/ kg/ day	1200 ppm in mud
MANGANESE	Inh. Chr.	0.00004 mg/ m 3	300 ppm in mud 2125 g/day PINJ
NICKEL COMPOUNDS	Inh. Int. Chr.	0.0002 mg/ m 3 0.00009 mg/ m 3	65 g/day PINJ 5 ppm in mud
PARTICULATES			200,000 LB 54,000 OX 271,296 g/day LB 528077 g/day PINJ
SILICA			21% in mud
SODIUM HYDROXIDE			
STRONG ACID MISTS INCLUDING SULFURIC ACID			
SELENIUM	Oral Chr.	0.005 mg/ kg/ day 3	120 LB 217 g/day PINJ 163 g/day LB 3 PPM in mud
SODIUM OXALATE			
VANADIUM	Inh. Acute	0.0002 mg/ m 3	

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Please note some are newspaper cuttings of unknown date

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