SENATE COMMUNITY AFFAIRS COMMITTEE
INQUIRIES INTO WORKPLACE EXPOSURE TO TOXIC DUST

PERSONAL DETAILS

Name: Gavin William Kele

Employer's Details: S.W.Kele & Co. Pty. Ltd.

EXECUTIVE SUMMARY

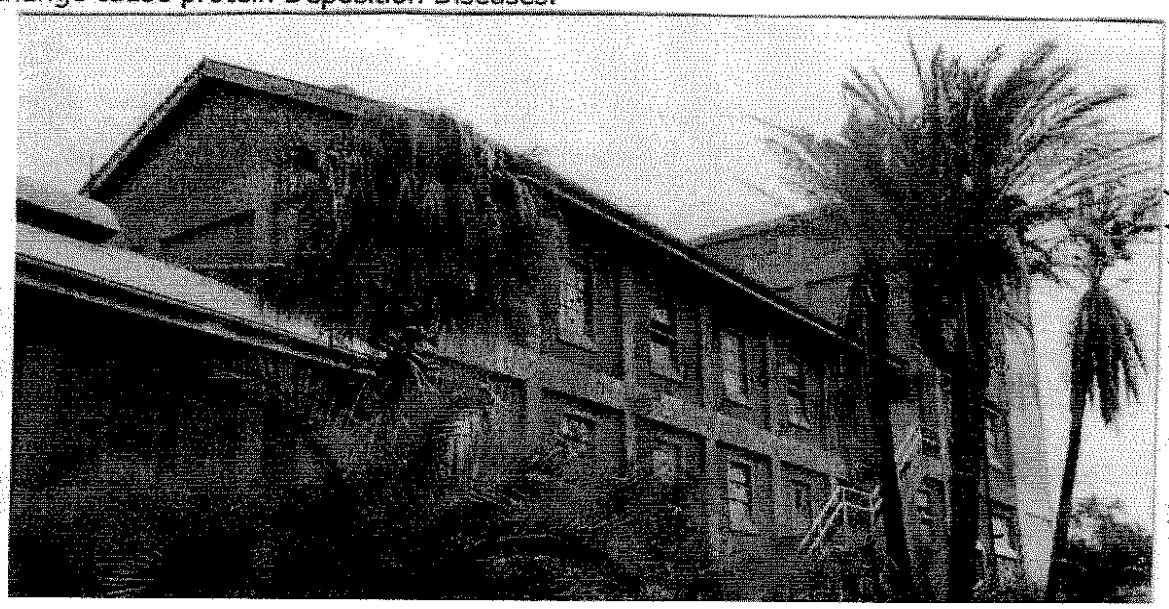
Our problem started when we purchased the lease of the Mt Charlton Water Works from the Rockhampton City Council. We owned the surrounding freehold land and our intentions were to establish a tourist venue highlighting the rural sector past and present.

The first step in setting up this venture was to shape and landscape the site. As this was to take many years we used the Water Works buildings as a base and storage area. As this project progressed, workers on the site experienced many health problems especially in the hot and dry period from September to Christmas each year.

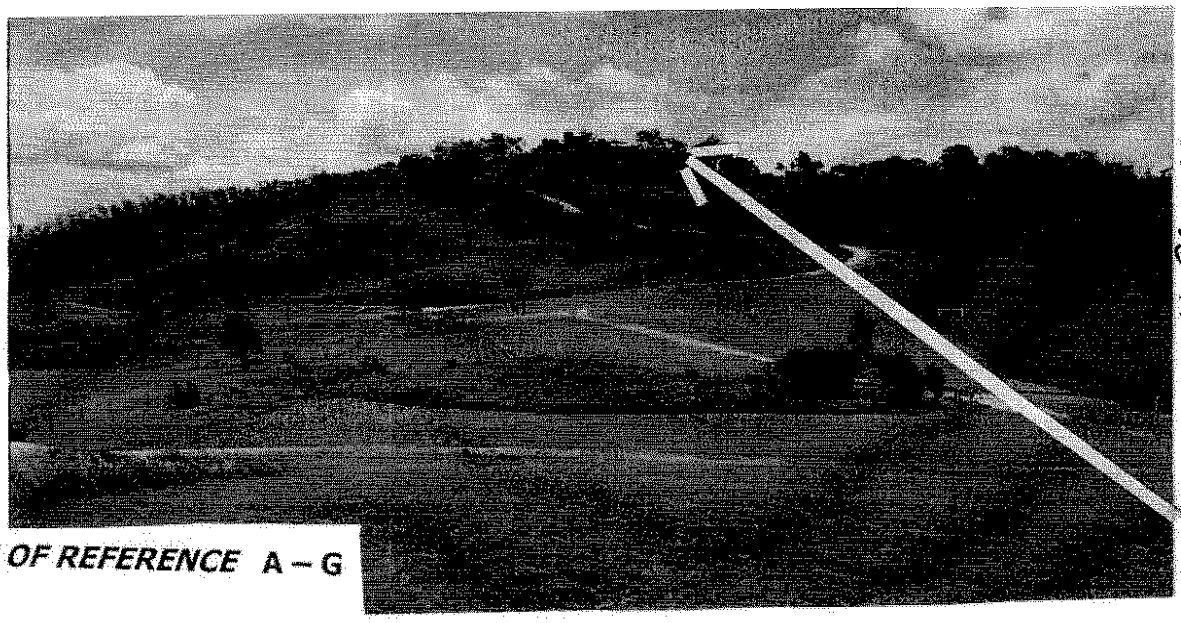
Some of these conditions included skin, asthma, lung and heart problems. When these workers were away from the site their health improved but deteriorated when back at this site. Our first clue as to what might be causing the health problems came when we discovered a white dust that accumulated on the plastic covering machinery was acidic with a ph between 3 & 4. We found this dust was predominantly oxidised alum originating from a council owned silo on a separate lease.

Our submission is based on the inherent properties of this dust substantiated by reference to M.S.D.S reports and other published material. To this date the Queensland and Commonwealth Health Department considers this material chemically inert.

Material Safety Data Sheet Reports on light metals are based on mortality rather than morbidity, this is strange as "Chronic Effects" headline the danger of cumulative effects. A large proportion of "Chronic Health Effects" relate to Protein based diseases, which effect the heart, liver, spleen, kidney, brain and nervous system, pancreas, bone, lungs, skin and diabetes. Proteins that fold themselves into defective shapes rather than proteins that have undergone harmful chemical change cause protein Deposition Diseases.



Mt. Charlton Waterworks Building:



Position of Mt. Charlton Waterworks Building:

TERMS OF REFERENCE A – G

A

Health impact of workplace toxic dust cannot be assessed until Oxidised Light Metal Dust is recognised as toxic. At present no one's prepared to admit this problem because it would involve massive compensation. By keeping the casual agent as some thing mystified, no one's to blame. As with asbestos dust, the only member of this family of dusts to be recognised as toxic, the morbidity period can be 20 to 40 years. Also with asbestos there is significant subject variability. Uptakes are a combination of gene type and environment exposure. Statical information can be used to track oxidised dust concentrations. Affected areas have significantly increased rates of cancer and protein deposition disease incidents.

B

M.S.D.S reports mention almost all of the dust characteristics but authorities refuse to recognise the oxidised dusts as toxic. The most significant characters of oxidised light metal dusts are:

1. Hydrophilic – craves moisture
2. Electrostatic – attracted to and accumulates articles with reverse polarity
3. Chelating – claw like gripping action

Attached letters to and from Queensland Health are typical of questions asked and answers received.

C

Employers are not aware of the fact that oxidised light metal dusts are toxic. M.S.D.S reports are written to read that the casual agent is something mystic. When you become aware of the characteristics of these dusts and their potential effects, careful reading of M.S.D.S reports can show concealed facts.

D

Doctors haven't been informed about the oxidised dust problem, although its health effects are seen everyday.

E

As the dusts are not recognised as toxic, no records are available on the extent of problems. Each medical condition is being treated individually the common influence is yet to be recognised.

F

Compensation is out of the question until oxidised light metal dusts are recognised as toxic. The legal precedents set with asbestos should be applied to oxidised light metal dust related medical conditions which include heart, liver, spleen, kidney, brain and nervous system, pancreas, bone, lung, skin, diabetes and well as lung cancer, melanoma and mesothelioma.

20th March 2003

Mr Paul Florian
Director Environmental Health
Central Public Health Unit Network
Rockhampton Queensland

Dear Paul,

It would be greatly appreciated if your department could answer some questions that we have in relation to the safety of certain types of aluminium dust.

The dust that we are concerned about is oxidised acidic aluminium dust. In particular, dust of this type that is of a very small size, that is, less than a micron in diameter.

This type of dust is commonly produced from the breakdown of alum (aluminium sulphate) commonly used in the water treatment industry. Similar dust is produced from the refinery processes of many alumina products and the corrosion of aluminium materials

Can you provide us with specific information in regards to how this strongly electrostatic dust accumulates on certain types of materials, such as plastics? This type of dust is also strongly hydrophilic and attracted to the moisture of the human body.

What human health concerns are there associated with this dust when it comes into contact with the moist tissues of the human body? Is the risk heightened for sensitive tissues such as sweat glands, eyes, esophageus, nerve, aural, lungs, and digestive?

Many of the Material Safety Data Sheets (MSDS) and other associated literature comment on the possibilities of malformed proteins and the various resulting diseases from exposure to certain levels of aluminium. How dangerous is this dust in relation to the risk of protein malformation.

What are the dangers of the cumulative effects of this aluminium dust? Most MSDS's focus on mortality rates, quite often ignoring the morbidity potential of a chemical. What is the morbidity potential of this dust?

What monitoring techniques are currently being used to monitor this dust in Queensland? What amounts of this dust is produced by Queensland industries and how far does it spread? At what level of this type of aluminium dust would the Queensland State Government consider it to be a health hazard?

Your help in answering these questions would be greatly appreciated.

Yours Sincerely,

Gavin Kele

Gavin Kele

2 MAY 2003

Hon. Wendy Edmond MP
Member for Mount Coot-tha

Queensland Government

cc 21/04/03

Minister for
Health

Minister
Assisting the Premier on
Women's Policy

MI110229
4300-0181-001

The Honourable R Schwarten MP
Minister for Public Works and
Minister for Housing
Member for Rockhampton
PO Box 100
ROCKHAMPTON Q 4700

Dear Minister

Thank you for your letter dated 24 March 2003, on behalf of Mr Gavin Kele, regarding aluminium dust.

I have been advised that Mr Paul Florian, Director Environmental Health Services, Central Public Health Unit–Rockhampton, has previously had contact with Mr Kele regarding aluminium in the environment and its impacts on health.

I have referred a copy of the correspondence from Mr Kele and a copy of this letter to the Minister for the Environment and the Minister for Industrial Relations. A response from these Ministers is more appropriate for issues relating to behaviour of aluminium dust, monitoring techniques and emissions of aluminium dust from industry in Queensland.

Queensland Health does not routinely undertake toxicological research and is therefore reliant on national and international reviews on the health effects of chemical substances. Reviews by the Agency for Toxic Substances and Disease Registry (ATSDR) and the National Environmental Health Forum indicate that aluminium has not been demonstrated to pose a health risk to healthy non-occupationally exposed humans. However, the mechanism by which aluminium impacts on neurogenerative disease is not well understood.

Please find attached information that describes aluminium and its potential impacts on health. However, if further assistance is required, Mr Kele may wish to contact Mr Florian on telephone (07) 4920 6895.

Thank you for bringing this matter to my attention and I trust this information is of assistance.

Yours sincerely

Wendy Edmond MP
Minister for Health and
Minister Assisting the Premier on Women's Policy

28 APR 2003

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ATTACHMENT

Aluminium occurs naturally in soil, water and air. The most common pathway of exposure to aluminium for humans is ingestion of food, water and medicines. Exposure to aluminium dust is generally considered to be very low. Aluminium dust occurs naturally, but can also be generated by industrial processes. Aluminium particles released from combustion processes are usually attached to very small particles, while aluminium contained in wind-borne soil is generally found in larger particles.

While it is well established that exposure to particles can lead to eye and respiratory irritation, non-occupational exposures to aluminium dust are not associated with significant adverse health risks. Respiratory effects have been observed in some workers exposed to aluminium dust; however, exposures in the workplace are generally exposed for periods of 8 to 12 hours per day and are orders of magnitude higher than those reported to occur in urban and industrial areas.

Little is known about the potential impacts of aluminium dust on moist tissues of the body. Aluminium compounds are widely used in antiperspirants without harmful effects to the skin or other organs. However, some individuals develop skin rashes in response to the use of some types of aluminium-containing antiperspirants.

Experimental studies of animals and humans indicate that once aluminium enters the body only a very small percentage (0.1-1%) is absorbed and less that 5% of the amount absorbed is retained in the body. The amount of absorption is dependent on the form of the aluminium compound and the presence of dietary constituents. The kidney is the major route of excretion and occupational studies indicate that after short-term exposures to aluminium, it is excreted from the body relatively quickly (half of the original aluminium absorbed is excreted in the first 8 hours after exposure). Occupational studies also suggest that the longer the exposure to aluminium, the longer the body's retention time. The low absorption and relatively rapid excretion via urine indicates that the potential for accumulation of aluminium in the body as a result of non-occupational exposures to aluminium dust is very low.

More information on aluminium toxicity can be found in Queensland Health's Guidance Note on Aluminium available at: www.health.qld.gov.au or at the US Department Of Health and Human Services toxicological profile database at: www.atsdr.cdc.gov.

G

Particle size with all dusts is most important. Any dust particles less that 1-micron can penetrate the body's defences. Modern research indicates toxic dust particle size more relevant than the number of particles per cubic meter of air.

FREEFORM LETTERS

Our submission is not based on any individual's health problems, but on what maybe causing the dramatic increase in protein deposition diseases and some cancers. Statistics show the increase started in the early 1900's. This table compares the known characteristics for Aluminium, magnesium and asbestos. I will endeavour to explain how we found each characteristic and give references.

Dust Characteristics	Aluminium Oxide Dust with Compounds. Anthropogenic (man made)	Magnesium Oxide Dust with compounds Anthropogenic (man made)	5 asbestos types of Natural Material MgSi0 Common to all types of asbestos.
(1) Hydrophilic (Takes up CO2 & H2O from air) **Translucent** (Invisible to eye when saturated)	yes yes	yes yes	Magnesium is found in Brucite layers in asbestos.
(2) Electrostatic (Attracted to anything with reverse polarity)	yes	yes	yes
(3) Chelating (claw-like or pincer like) (not a chemical reaction)	Amyloid Protein Deposits	Bonding	Fibrous - particles attach to cause plaque deposits
(4) Distribution	2.5 micron & less Infinity Dust similar to smoke	2.5 micron & less Infinity Dust similar to smoke	10m due to reactivity
(5) Solubility	Partly Hydrophilic	Partly Hydrophilic	Partly
(6) Amyloid Proteins Plaque formation	MSDS on Aluminium report plaque formation enhanced by aluminium	Production areas for Aluminium Magnesium compounds have CJD, (Prions) Incidents	Plaque with mesothelioma and Asbestos.
(7) .05 micron & less most lethal.	MSDS on Aluminium report .5-micron dust penetrates lung and brain.	yes	Fibres less that 5-micron most dangerous.
(8) Compounds with other metals.	yes	yes	5 main types all have magnesium, silicate & oxygen common.
(9) Morbidity Period 20 to 40 years.	yes	yes	yes
MSDS mortality rather than mobility cumulative effects.	No long-term research recorded. Danger of cumulative effects.	No long-term research recorded.	MSDS- Cancer forming 20 – 40 year incubation.
(11) Health spikes in production areas (statistics)	yes	yes	yes
(12) Common links in Protein Deposition Diseases.	Amyloid protein plaque deposits.	Amyloid Protein deposits which include Prion replication.	Amyloid Protein plaque deposits.
(13) Cancer Forming	Aluminium workers 30% to 50% higher than average with mesothelioma and melanoma.	No information available.	Asbestos Mesothelioma Courts attribute 19% of the health problems to James Hardie. How much of the other 81% should be attributed to Aluminium and Magnesium
(14) Time Frame for increase in incidents of protein deposition diseases.	Production of Aluminium increase started early 1900's. PDD increase in line	Production on increase.	Production stopped, incidents yet to peak.

In late 1993 we decided to abandon the project at Mt Charlton for a number of reasons, health concerns being one. At first, all we knew was we had an acidic white dust, that we later found to be oxidised alum, coming predominately from a silo on an adjacent lease. The doors and windows at the top of this silo were open to the weather and every time it rained the alum in this silo was wet. The continual wetting and drying out of this alum accelerated the oxidising. Because of the height of the silo the dust rained down like confetti on the surrounding area. The extreme concentration of accumulated dust around the silo was 200 to 500 times above the environmental criteria. The unique circumstances at Mt Charlton exaggerated the dust problem, but eventually helped lead us to the answers.

Oxidised bagged Alum on top floor of silo:

Points 1-2-3:

1 Hydrophilic
2 Electrostatic
3 Chelating

The first significant point found about the dust characters was that the dust is hydrophilic, i.e. craves moisture in periods of low humidity. It migrates to find sources of moisture. This explained our problem in the hot and dry weather leading up to the wet season.

The slow process of investigating this dust has had many highs and lows. An early event was when we questioned the safety of the site with the landlord i.e. Queensland Government Natural Resources Department and it was mutually decided to commission the Queensland Department of Environment to investigate the site. After a long delay, a draft report was submitted by the Environment Department to the Natural Resources Department who took this draft to the Rockhamtpon City Council but refused us access, when the final report was provided we had to use freedom of information action to get a copy of the report.

Page 3 of this report shows the analysis of the dust in the silo. MSDS on Alum dust currently has TLV TWA 1mg/m3, the dust in the silo for aluminium and sulphated component was 479 & 515 mg/m3.

Two samples of the alum from the top of the silo were taken for laboratory analyses to test for major elements and for the presence of contaminants. The results of analyses are in Table 1.

Table 1. Results of laboratory analyses.

Component	SAMPLE		Assessment criteria	
(mg/kg, ppm)	Bagged material	Floor and silo	Environmental	Health
Aluminium	95,000	99,000	--	--
Sulphate	420,000	380,000	2,000	--
Arsenic	2.1	2.6	100	20
Cadmium	< 2	< 2	20	--
Copper	13	11	100	60
Lead	< 20	< 20	300	300
Nickel	< 10	< 10	100	60
Zinc	350	180	500	200
pH	3.0	3.3	6 to 8*	--

* pH criteria are expressed as the normal environmental background range.

In comparing the analysis results with the health and environmental assessment criteria it should be remembered that the criteria are intended for assessment of the risk to health or the environment from contaminated soil. In this case the material is the chemical alum (predominantly aluminium sulphate), and not a soil.

However, for the purposes of assessing the potential for site contamination the criteria can be used to assess the health and environmental implications of a large quantity of this material being released into the soil. The analyses also provide the basis for determining the options available for reuse or disposal of this material.

Several results of analyses are of concern:

- elevated sulphates (about 200 times the environmental criterion)
- elevated zinc (above the health criterion)
- very acid pH (well outside the environmental background, pH 7 is neutral)

No environmental or health criteria for aluminium, or health criteria for sulphate or pH are available for use in the context of this report.

The implications of these results are that the alum present in the silo and associated areas:

- should not be allowed to be dispersed or disposed of on the site
- should not be allowed to enter stormwater, waterways of groundwater
- will require special personal protective precautions during handling and removal.

While the alum in the silo is reasonably effectively contained at present, the residual should eventually be removed to prevent accidental or vandal release to the environment. The potential for dispersal is not limited to the movement of solids, alum is highly soluble and could be transported by water. In the interim the structural integrity of the silo should be maintained.

PRECAUTIONS FOR USE ...

INGREDIENT DATA

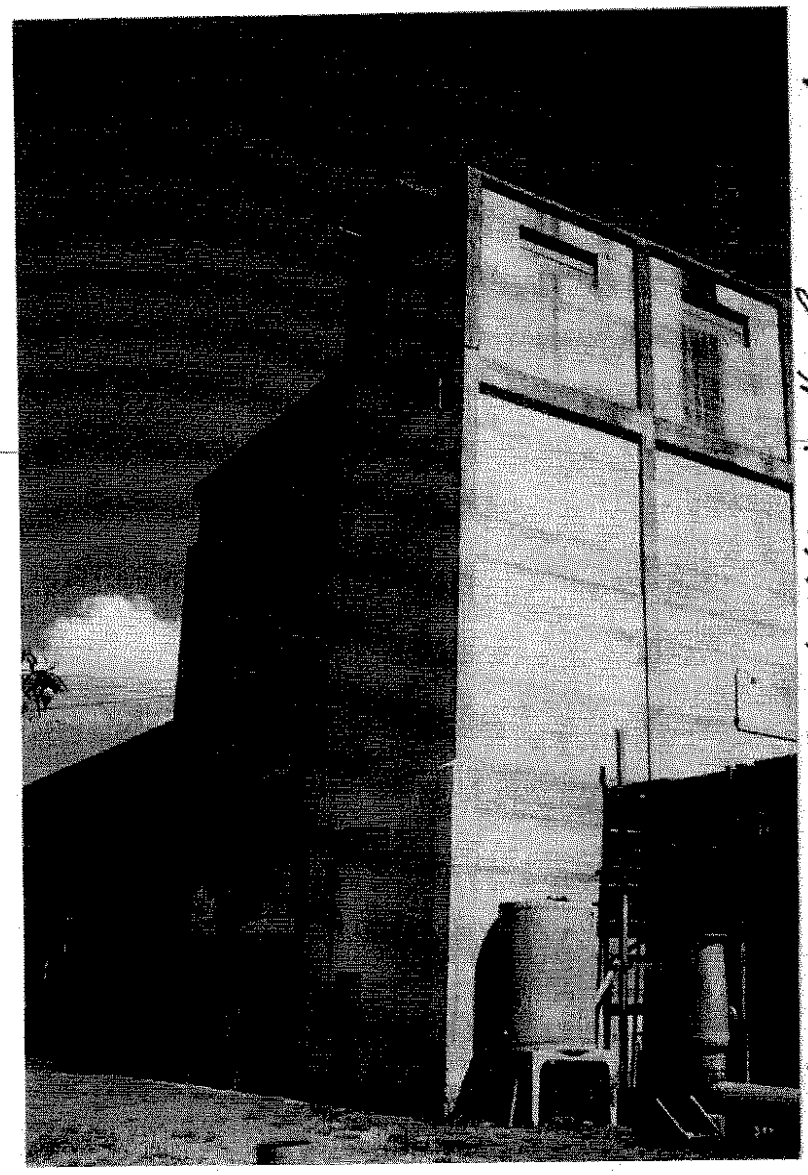
ALUMINIUM SULFATE:
TLV TWA: 2 mg/m³ soluble salts [ACGIH] ← 1-1-05 Reduced to 1 mg/m³
aluminium soluble salts, as Al (A.Wt: 26.98)
ES TWA: 2 mg/m³
TLV TWA: 2 mg/m³
The TLV is based on the exposures to aluminium chloride and the amount of hydrolysed acid and the corresponding acid TLV to provide the same degree of freedom from irritation. Workers chronically exposed to aluminium dusts and fumes have developed severe pulmonary reactions including fibrosis, emphysema and pneumothorax. A much rarer encephalopathy has also been described.

ENGINEERING CONTROLS

Use in a well-ventilated area
None required when handling small quantities.
OTHERWISE:
Local exhaust ventilation is required where solids are handled as powders or crystals; even when particulates are relatively large, a certain proportion will be powdered by mutual friction.
Exhaust ventilation should be designed to prevent accumulation and recirculation of particulates in the workplace.
If in spite of local exhaust an adverse concentration of the substance in air could occur, respiratory protection should be considered. Such protection might consist of:
(a): particle dust respirators, if necessary, combined with an absorption cartridge;
(b): filter respirators with absorption cartridge or canister of the right type;
(c): fresh-air hoods or masks
Build-up of electrostatic charge on the dust particle, may be prevented by bonding and grounding.
Powder handling equipment such as dust collectors, dryers and mills may require additional protection measures such as explosion venting.

CHRONIC HEALTH EFFECTS
Danger of cumulative effects.

Not considered to cause discomfort through normal use.
Principal routes of exposure are by accidental skin and eye contact and inhalation of generated dusts.
Long term exposure to high dust concentrations may cause changes in lung function (i.e. pneumoconiosis) caused by particles less than 0.5 micron penetrating and remaining in the lung. A prime symptom is breathlessness. Lung shadows show on X-ray.



Silo is the unpainted building in the foreground. Damaged timber doors and windows were open for the duration of our occupancy.

The explanation of how this dust accumulated on black plastic sheeting we had covering machinery came soon after. (When reading the MSDS one day, I noticed in the advice to fireman, a caution about build up of electrostatic charge.) The dust was attracted to the static electrical charge in the plastic sheeting on the machinery stored, both inside and outside the buildings, by mild electrical attractions.

When we moved the plastic on machinery the accumulated hydrophilic dust moved to body moisture both external and internal. The Inchem report mentions electrically charged particles on numerous occasions i.e. cation – positively charged particles, anion – negatively charged particles. Some of these particles remain permanently charged.

The next part of our investigation had us puzzled for quite some time. The black plastic covering the machines at Mt Charlton retained some of the white dust on the outside. In time the outside turned white while the underside remained black. A layer of white dust was permanently fixed and could not be removed. In time we found this characteristic to be well known and called chelating. A chemistry book dating back to 1872 details how alum is used to dye Denim cloth and leather, this process is still used to this day. Most MSDS and Inchem reports mention chelating aluminium as a characteristic of aluminium dust.

1055. Alum has a peculiar sour-sweet and astringent taste. It acts like a strong acid on litmus-paper, and evolves hydrogen when zinc is added to its solution. It is used to a considerable extent in medicine. It is largely employed in the preparation of leather, and still more abundantly in dyeing, and in the preparation of pigments. Its application to the two latter purposes depends upon its attraction for organic colouring matters, and its affinity for textile fibres. The former property may be observed by adding to a solution of colouring matter, such as madder, cochineal, or litmus, first alum, and then solution of ammonia, when a precipitate of aluminic hydrate will be produced, and will carry down the colouring matter combined with it. Such compounds are called lakes.

1056. The attraction of alumina for the fibre of cloth may be shewn by filtering aluminic acetate, or a solution of common alum which has been partially neutralised by sodic carbonate, through linen or cotton, when part of the alumina will be

(3)

abstracted by these tissues, and retained. Colours otherwise fugitive are thus fixed, the cloth to be dyed being first impregnated with alumina, by soaking it in either of the solutions above mentioned, and leaving it to dry in a warm place. When dry, it is boiled in a solution of the required colouring matter, when the alumina attaches itself to the fibre on the one hand, and the dye-stuff on the other, so that a coloured compound is produced, insoluble in water, and not destroyed by soap, &c. Patterns are produced by printing upon the calico with a solution of aluminic acetate, thickened with gum. It is then immersed in the dye-vat, and afterwards thoroughly washed in many changes of hot water. The colouring matter is washed away, except in those parts where the aluminic salt, or 'mordant,' as it is called, has been applied.

Inchem 7.3.1

The severity of developmental aluminium toxicity by the oral route is highly dependent on the form of aluminium and the presence of organic chelators that influence bioavailability.

Chelating agent – compound that can react with a metal ion and form a stable compound.

Harmful if swallowed. May cause irritation. Avoid breathing vapors, or dusts. Use with adequate ventilation. Avoid contact with eyes, skin, and clothes. Wash thoroughly after handling. Keep container closed.

Section 4 First Aid Measures

Harmful if swallowed. May cause irritation. Avoid breathing vapors, or dusts. Use with adequate ventilation. Avoid contact with eyes, skin, and clothes. Wash thoroughly after handling. Keep container closed.

FIRST AID: SKIN: Wash exposed area with soap and water. If irritation persists, seek medical attention.

EYES: Wash eyes with plenty of water for at least 15 minutes, lifting lids occasionally. Seek Medical Aid. INHALATION: Remove to fresh air. If not breathing, give artificial respiration. If breathing is difficult, give oxygen.

INGESTION: If swallowed, induce vomiting immediately after giving two glasses of water. Never give anything by mouth to an unconscious person.

Section 5 Fire Fighting Measures

Fire Extinguisher Type:	Any means suitable for extinguishing surrounding fire
Fire/Explosion Hazards:	None specified by manufacturer.
Fire Fighting Procedure:	Wear self-contained breathing apparatus and protective clothing to prevent contact with skin and clothing.

Boiling Point:	3600 Deg C	Volume:	0
Vapor Pressure:	Information not available	Evaporation Rate:	0
Vapor Density:	Information not available	Evaporation Standard:	
Solubility in Water:	Very soluble	Auto ignition Temperature:	Not applicable
Appearance and Odor:	None specified	Lower Flamm. Limit in Air:	Not applicable
Flash Point:	Not applicable	Upper Flamm. Limit in Air:	Not applicable

Section 10 Stability and Reactivity Information

Stability: Stable Conditions to Avoid: Takes up CO2 and H 2O from air.

Materials to Avoid: Interhalogens, phosphorus pentachloride.

Hazardous Decomposition Products: Not known to occur.

Hazardous Polymerization: Will Not Occur.

Condition to Avoid: None known.

Section 11 Additional Information

If comes in contact with skin wash with soap and water. If comes in contact with eyes wash with water for atleast 15 minutes and seek medical advice if necessary. If inhaled remove to fresh air and support breathing. If ingested call al physician immediately. Target organs : None. Persons with pr-existing disorders may be more susceptible.

DOT Classification: Not Regulated

SKIN

If product comes in contact with the skin:
1: Wash affected areas thoroughly with water (and soap if available).
2: Seek medical attention in event of irritation.

INHALED

1: If dust is inhaled, remove to fresh air.
2: Encourage patient to blow nose to ensure clear breathing passages.
3: Rinse mouth with water. Consider drinking water to remove dust from throat.
4: If irritation or discomfort persists seek medical attention.

ADVICE TO DOCTOR

1. Manifestation of aluminium toxicity include hypercalcaemia, anaemia, Vitamin D refractory osteodystrophy and a progressive encephalopathy (mixed dysarthria-apraxia of speech, asterixis, tremulousness, myoclonus, dementia, focal seizures).
   Bone pain, pathological fractures and proximal mycpathy can occur. Symptoms usually develop insidiously over months to years (in chronic renal failure patients) unless dietary aluminium loads are excessive.
3. Serum aluminium levels above 60ug/ml indicate increased absorption. Potential toxicity occurs above 100 ug/ml and clinical symptoms are present when levels exceed 200 ug/ml.
4. Deferoxamine has been used to treat dialysis encephalopathy and osteomalacia. CaNa2EDTA is less effective in chelating aluminium

(Ellenhorn and Barceloux: Medical Toxicology)

Copper, magnesium, aluminium, antimony, iron, manganese, nickel, zinc (and their compounds) in welding, brazing, galvanising or smelting operations all give rise to thermally produced particulates of smaller dimension than may be produced if the metals are divided mechanically. Where insufficient ventilation or respiratory protection is available these particulates may produce "metal fume fever" in workers from an acute or long term exposure.

1. Onset occurs in 4-6 hours generally on the evening following exposure. Tolerance develops in workers but may be lost over the weekend. (Monday Morning Fever)
2. Pulmonary function tests may indicate reduced lung volumes, small airway obstruction and decreased carbon monoxide diffusing capacity but these abnormalities resolve after several months.
3. Although mildly elevated urinary levels of heavy metal may occur they

Points 4 & 7: Distribution and Particle Size

Health Effects of Particle Matter

(a) Particle size: Less that 1 micron can penetrate body organs.

(b) Intensity of exposure: When humidity is low, oxidized light metal dust craves moisture and migrates to find moisture. Electrostatic moving dust accumulates on articles with reverse polarity. Accumulated dust can reach extreme concentrations over long periods of time, if not disturbed. Normal dust monitoring doesn't show these concentrations. How safe are air-conditioned buildings with low humidity and the abundance of light metals in plant, ducting and building products.

(c) Interaction with human tissue: Oxidised light metal dust are mostly chemically inert, but, as with Asbestos the chelating (claw like) action attaches to tissue and proteins causing a chain reaction leading to numerous medical conditions.

(d) Susceptibility: Research shows that fair skinned people are more susceptible, but in time everyone is susceptible with high concentrations of oxidised dust. Inchem report shows that people with Downes Syndrome absorb aluminium 5 times higher that rate than controls. Alzheimer type neuropathy is almost universal in Downes Syndrome subject 25-35 years of age.

(e) Distribution of Dust:

Particles Size	Travel Distance
50-micron	Short travel distance
10 micron	can remain in air for several days
Less than 2.5 micron	May remain in air indefinitely as with smoke

Point 5: Solubility

Pure aluminium with Ph less than 5 are higher than 8 soluble. As a metal to counteract this tendency, aluminium is compounded (alloid) with other metals. One of the most dangerous of these is zinc. all of the roofing iron is now coated with zinc alume, i.e. Aluminium Zinc compound. This material oxidises at a very high rate.

Alum (Aluminium sulphate) is used on town water treatment works, dying of clothes, shoes and for soil treatment. About 1500 tonnes of alum are added to Rockhampton's water supply each year to settle the mud in river water. The filters remove most of the dissolved aluminium but not all.

Question: What are the harmful effects generated by adding dissolved aluminium to the town water supply?

Inchem 8.1.3.5 states a positive relationship between aluminium in drinking water and Alzheimer's disease.

Question: Why are we still using aluminium in water treatment?

Inchem 6.1.2.1 stated that even aluminium compounds that are insoluble in water are bioavailable when introduced in to the respiratory system 'Insoluble' particles may be slowly dissolved and thus enter blood circulation. Owing to chemical properties of aluminium, the absorption of aluminium metal or its compounds by the respiratory systems depends on the aluminium species inhaled and the biological environment in the tissue compartment where they are deposited.

Point 6: Amyloid Protein Deposits

M.S.D.S – Chronic Health Effects

disability if excessive concentrations of particulate are inhaled.

CHRONIC HEALTH EFFECTS

Not considered to cause discomfort through normal use. Principal routes of exposure are by accidental skin and eye contact and inhalation of generated dusts. Long term exposure to high dust concentrations may cause changes in lung function (i.e. pneumoconiosis) caused by particles less than 0.5 micron penetrating and remaining in the lung. A prime symptom is breathlessness. Lung shadows show on X-ray. Chronic exposure to aluminium oxides of particle size 1.2 microns did not produce significant systemic or respiratory system effects in workers. When hydrated aluminas were injected intratracheally, produced dense and numerous nodules of advanced fibrosis in rats, a reticulin network with occasional collagen fibres in mice and guinea pigs, and only a slight reticulin network in rabbits. Shaver's disease, a rapidly progressive and often fatal interstitial fibrosis of the lungs, is associated with a process involving the fusion of bauxite (aluminium oxide) with iron, coke and silica at 2000 deg. C. Occupational exposure may produce asthma, chronic obstructive lung disease and pulmonary fibrosis. Long-term overexposure may produce dyspnea, cough, pneumothorax, variable sputum production and nodular interstitial fibrosis; death has been reported. Chronic interstitial pneumonia with severe cavitations in the right upper lung and small cavities in the remaining lung tissue, have been observed in gross pathology. Shaver's Disease may result from occupational exposure to fumes or dusts; this may produce respiratory distress and fibros... with large blebs. Animal studies produce no indication that aluminium or its compounds are carcinogenic. Controversy exists over whether aluminium is the cause of degenerative brain disease (Alzheimer's disease or AD). Several epidemiological studies show a possible correlation between the incidence of AD and high levels of aluminium in drinking water. A study in Toronto, for example, found a 2.6 times increased risk in people residing for at least 10 years in communities where drinking water contained more than 0.15 mg/l aluminium compared with communities where the aluminium level was lower than 0.1 mg/l. A neurochemical model has been suggested linking aluminium exposure to brain disease. Aluminium concentrates in brain regions, notably the hippocampus, cerebral cortex and amygdala where it preferentially binds to large pyramid-shaped cells - it does not bind to a substantial degree to the smaller interneurons. Aluminium displaces magnesium in key metabolic reactions in brain cells and also interferes with calcium metabolism and inhibits phosphoinositide metabolism. Phosphoinositide normally controls calcium ion levels at critical concentrations. Under the microscope the brain of AD sufferers show thickened fibrils (neurofibrillary tangles - NFT) and plaques consisting of amyloid protein deposited in the matrix between brain cells. Tangles result from alteration of "tau" a brain cytoskeletal protein. AD tau is distinguished from normal tau because it is hyperphosphorylated. Aluminium hyperphosphorylates tau in vitro. When AD tau is injected into rat brain NFT-like aggregates form but soon degrade. Aluminium stabilises these aggregates rendering them resistant to protease degradation. Plaque formation is also enhanced by aluminium, which induces the accumulation of amyloid precursor protein in the thread-like extensions of nerve cells (axons and dendrites). In addition aluminium has been shown to depress the activity of most neuro- transmitters similarly depressed in AD (acetylcholine, norepinephrine, glutamate and GABA). Aluminium enters the brain in measurable quantities, even when trace levels are contained in a glass of tap water. Other sources of bioavailable aluminium include baking powder, antacids and aluminium products used for general food preparation and storage (over 12 months, aluminium levels in soft drink packed in aluminium cans rose from 0.05 to 0.9 mg/l). [Walton, J and Bryson-Taylor, D. - Chemistry in Australia, August 1995].

Point 8: Compounds

Oxidised dust originating from light metals is toxic on its own but, more toxic when compounded with other metals and chemicals. Compounds can be anthropic (man made) and also formed by electrostatic, chelating dusts compounding in the atmosphere by accumulation.

Aluminium and Magnesium are the two light metals in most common use today. Sometimes they are compounded together, as in motorbike and aeroplane castings, but can be compounded with any number of other metals. They are compounded to increase strength, durability and hardness etc. Incidents of CJD (the human equivalent to Mad Cow Disease) are higher than normal in area adjacent to aluminium magnesium alloy production.

When light metals oxidise and migrate to find moisture they can attract to anything with reverse polarity as well as chelating with other oxidised metal dust.

Fine dust compounds are released when sandblasting.

Some of the materials released are:

(a) Paints of all types

(b) Protective coatings including galvanize (zinc)

(c) Metals from materials being blasted

(d) Silica sand and other blasting materials, which include ground furnace, shag which contains any number of metals.

Abrasive grinding and cutting discs also have high aluminium content, which is released to atmospheric dust when grinding and cutting. In almost every case where silicosis is a health problem it's in conjunction with one of the light metals i.e. asbestos, magnesium & silica, coal silicate & aluminium.

Another dust very prevalent in parts of Australia where coal is mined, carted and stores is oxidised coal dust. Coal dust slowly oxidises when exposed to air. Benzene type compound are released as the coal dust oxidises.

Inchem 1.8 mentions Pulmonary Fibrosis in workers exposed to aluminium particles coated with oil.

Question – What is the result when oxidised light metal oxidises and coal dust and coal fired boiler emissions mix in the environment? In Central Queensland large volumes of aluminium, magnesium and coal dust are released into the environment. We have some of the highest P.D.D and cancer rates in Australia. Is there a connection or are we as the government implies all too fat? What research has been done on particulate matter coming from coal fired power stations and boilers with high concentrations of aluminium in their emissions.

Attached sheets show emissions for 12 months from 01/07/2001 – 30/06/2002 from Gladstone QAL chimneystack the particulate matter less that 10 microns was 1800 tonnes i.e. approximately 5 tonnes per day. As aluminium is considered inert NO breakdown of the aluminium content has to be published for both chimney and refinery emissions.

SPONTANEOUS COMBUSTION, THE CURSE OF COAL MINERS, AND A HEALTH AND ENVIRONMENTAL HAZARD TO ALL

Dr. Mike Clarke, CPEng, FIEAust., MAusIMM, RPEQ, M.E.T.T.S. Pty Ltd, Brisbane, www.metts.com.au

Spon Com (aka Spontaneous Combustion) is a curse of those who mine coal, handle coal, use coal and who are responsible for reclaimed coal mining areas. Spon Com has been the cause of mine fires, a contributor to mine explosions, a management and safety headache to coal handlers and users, and an on-going festering sore to those who have responsibility for managing, mine spoil heaps, reject and coal tailings impoundments and reclaimed mining areas. Spon Com has been responsible for at least one war, the Spanish-American War, which was initially blamed on an act of sabotage on the USS Maine in Havana Harbour, but which is now believed to have been caused the ignition of munitions by smouldering bunkered coal [1].

Spon Com of coal and colliery wastes has environmental and health effects and consequences. The uncontrolled partial oxidation of coal produces toxic emissions that contain the following; carbon monoxide, aromatic hydrocarbons (benzene type compounds), sulphur oxides, nitrous oxide, ammonia and cyanide. The environmental emission hazards include the greenhouse gases, carbon dioxide, methane and nitrous oxide, as well as the greenhouse gas contributors, ammonia and carbon monoxide. The emissions have the propensity to poison man and nature, and furthermore be an on-going threat, where the Spon Com cannot be prevented or fully controlled.

Dr Mike Clarke

What is Spon Com?

For Moses it is was burning foliage at the foot of Mt. Horeb. For farmers and compost makers it is haystacks and dung piles bursting into flame. It is the self ignition of combustible material, or the self heating of combustible material, and/or the assisted self heating of combustible material. For the coal industry it is, the heating of coal at 'the mining face', the smouldering of coal in storage, the loss of energy values from coal going to market, the restriction of markets for certain coals, and an environmental, health and sometimes life threatening hazard.

The mechanism of spontaneous combustion is the build up of heat in a combustible mass, until the temperature is reached where self ignition occurs. In other words, combustible material, eg coal, in contact with oxygen (or oxygen sources) reaches it activation energy/temperature where the oxidation will proceed to actual combustion. But how can a rock reach a temperature where oxidation becomes a run-away reaction?

Spon Com mechanisms

These are poorly understood [2]. Coal does slowly oxidise once exposed to air, as evidenced by the changing surface chemistry of coal undergoing flotation or flocculation in coal washeries. Freshly mined coal will show markedly different performance in coal washing processes that rely on surface chemistry, than coal that has been stored for considerable time. Further in testing coal particles for their surface potential, the 'oxidation' of surfaces will be indicated by significant changes in measured surface potential [3].

This slow oxidation of coal surfaces does produce heat, however the heat liberated is small per unit mass per unit time, and there is opportunity for this heat to disperse in most situations before self heating can occur. Other possibilities for temperature increases are the oxidation of mineral sulphides and sulphur compounds contained in coal with or without the assistance of microbes in heating. Research undertaken by Dr. Joseph Shonhardt at the University of New South Wales in the 1980's however discounts these factors as being the prime initiators of self heating [4].

Public Contact Email:	fawkesr@qal.com.au
Web Address:	www.qal.com.au
Number of Employees:	1050
Main Activities:	Refining Weipa bauxite into alumina.
Primary ANZSIC Industry Class:	Alumina Production
Subsidiary ANZSIC Industry Class(s):	
ANZSIC Industry Group:	Basic Non-Ferrous Metal Manufacturing

Emissions for 01-Jul-2001 to 30-Jun-2002 to All Destinations

All emissions have been rounded to two significant figures. Note that totals may differ from the sum of the individual amounts because of this rounding. Substance emissions are ranked on a scale of 1-100: 1=lowest; 100=highest. Rankings are shown as: ○=0-25; ◔=26-50; ◕=51-75; ●=76-100. Actual rankings are shown in brackets [ ] - see further explanation of ranking below.

Substance	Ranking [1 - 100]	Total (kg)	Air	Land	W
Acetone	○ [Low - 9]	13,000	13,000		
Arsenic & compounds	○ [Low - 3]	1,000	53	270	
Beryllium & compounds	○ [Low - 1]	1.8	1.8		
Boron & compounds	○ [Low - 1]	1,000	57	940	
Cadmium & compounds	○ [Low - 1]	0.91	0.39	0.52	
Carbon monoxide	○ [Low - 1]	2,500,000	2,500,000		
Chromium (III) compounds	○ [Low - 1]	150	110	40	
Chromium (VI) compounds	○ [Low - 1]	2.5	2.5		
Cobalt & compounds	○ [Low - 1]	26	23	1.6	

Copper & compounds	[Low - 1]	60	42	5.3	
Ethanol	[Low - 1]	110	110		
Fluoride compounds	[Medium - 47]	260,000	79,000	25,000	1
Hydrochloric acid	[Low - 1]	6,800	6,800		
Lead & compounds	[Low - 1]	36	26	10	
Magnesium oxide fume	[Low - 0]	0.00	0.00		
Manganese & compounds	[Low - 1]	750	750		
Mercury & compounds	[Low - 20]	190	190	0.050	
Nickel & compounds	[Low - 1]	38	30	2.4	
Oxides of Nitrogen	[Low - 23]	11,000,000	11,000,000		
Particulate Matter 10.0 um	[Low - 7]	1,800,000	1,800,000	= approx 5 Tonnes per day	
Polychlorinated dioxins and furans	[Low - 1]	0.000090	0.000090		
Polycyclic aromatic hydrocarbons	[Low - 2]	410	410		
Sulfur dioxide	[Low - 3]	6,200,000	6,200,000		
Sulfuric acid	[Low	72,000	72,000		

Points 9 & 10: Morbidity Period 20 to 40 years

Inchem 1.6.2 highest levels of aluminium may be found in lungs, where it may be present as inhaled insoluble particles. Aluminium is distributed in most organs within the body with accumulation occurring mainly in bone at high dose levels. Aluminium passes blood-brain barrier and may also distributed to the fetus.

ALUMINIUM OXIDE

Section 4 - HAZARDS IDENTIFICATION ...

POTENTIAL HEALTH EFFECTS

ACUTE HEALTH EFFECTS

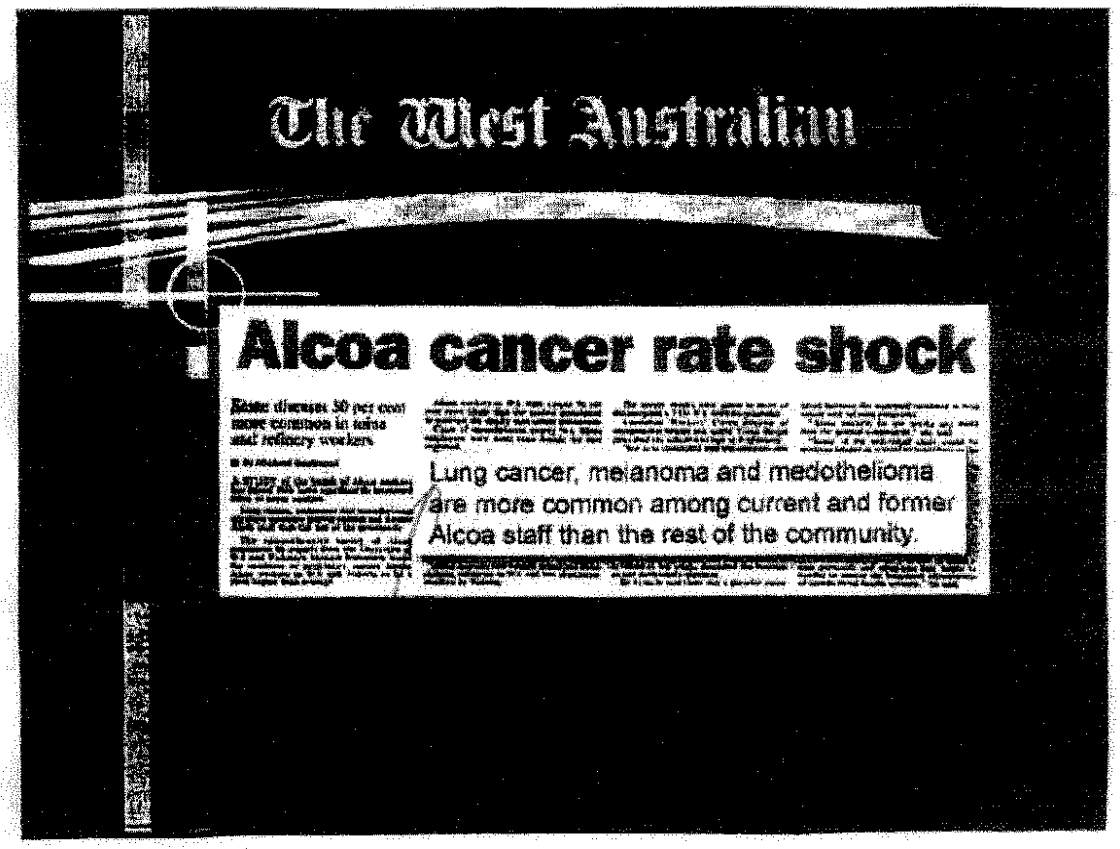
SWALLOWED

Although ingestion is not thought to produce harmful effects (as classified under EC Directives), the material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.

Point 11

M.S.D.S, Inchem and published articles, show that 30 to 50% higher than average P.D.D and cancer rates in vicinity of major production areas for light metals. Central Queensland with aluminium, magnesium and coal production has some of the highest rates in Australia.

Points 12 & 13: Common link in Protein Deposition Diseases


The West Australian

Alcoa cancer rate shock

Lung cancer, melanoma and medothelioma are more common among current and former Alcoa staff than the rest of the community.

M.S.D.S report states plaque formation is also enhanced by aluminium, which induces the accumulations of amyloid precursor protein in thread like extension of nerve cells.

Most of the current health concerns with asbestos are cancers in one form or another. Has the P.D.D incidents with asbestos workers been assessed if not why?

Aluminium workers in Western Australia report 30 to 50% higher than average cancer rates. In Central Queensland cancer rates for most cancers are above the Australian average.

The West Australian

Alcoa cancer rate shock

By Michael Southwell

A STUDY of the health of Alcoa workers has found they have significantly increased rates of some cancers.

Lung cancer, melanoma and mesothelioma are more common among current and former Alcoa staff than the rest of the community.

The comprehensive survey of Alcoa employees by experts from the University of WA and Victoria's Monash University found the incidence of respiratory cancers among Alcoa workers in WA and Victoria to be a third higher than average.

Alcoa workers in WA were almost 50 per cent more likely than the general population to contract the deadly skin cancer melanoma.

Cases of mesothelioma among WA Alcoa employees were more than double the rate expected.

The study said the results were statistically significant.

The West Australian revealed late last year that Alcoa was aware as early as 1990 that a liquor burner at its Kwinana refinery was emitting big volumes of harmful volatile organic compounds, including known carcinogens such as benzene.

A liquor burner was installed at the Wagerup refinery in 1996.

The Kwinana liquor burner was last month shut to allow installation of pollution control equipment.

Alcoa operates two bauxite mines and three alumina refineries in WA and two aluminium smelters in Victoria.

The survey results were given to most of the company's 3700 WA workers yesterday.

Australian Workers' Union director of occupational health and safety Yosse Berger described the cancer findings as frightening.

"We have demanded that the company pay for cancer screening of every worker and we want more work done to find out what is causing this," he said.

The findings come from the interim report of a cancer and mortality study of past and present Alcoa workers up to 1996. More up-to-date results are not expected until next year.

03/07/2002

Proteins, diseases linked

NEW research suggests illnesses as diverse as Alzheimer's, Creutzfeldt-Jakob disease and adult-onset diabetes are caused by proteins that fold themselves into defective shapes, rather than proteins that have undergone harmful chemical changes.

As they develop, these aberrant protein forms can clump together and wreak molecular havoc on healthy cells, according to two studies published in the journal *Nature*.

The effect may be common to any protein in the body, the researchers reported. "There is lots of evidence that any protein can form these structures in principle," said Christopher Dobson of the University of Cambridge in England.

Thomas Sudhof, of the Howard Hughes Medical Institute, said the findings demonstrated the particular stage at which the toxic effects of the misfolded proteins could be gauged.

Proteins are the agents of change and chemical messengers in cells, and they carry out the instructions encoded in genes. The specific roles they play are dictated by the three-dimensional shape they take on when folded. On occasion, that folding can go awry. Accumulations of these rogue proteins are common in what researchers categorise as protein deposition diseases.

In Creutzfeldt-Jakob disease — a degenerative brain disorder that resembles mad cow disease in its effects — a type of misfolded protein known as a prion may replicate itself and spread to other cells to cause damage.

In diabetes, protein misfolding may be responsible for blood-vessel damage.

HEART STOPPER

Why you're more likely to die from a heart attack here than almost anywhere else in the nation

Rocky man's fatal illness still mystery

By NATASHA HOLLAND

RELATIVES of Rockhampton man Paul Lawrie are mystified about the illness that killed him within months.

It was speculated that Mr Lawrie may have contracted variant Creutzfeldt-Jakob disease (the human strain of mad cow disease).

Mr Lawrie died on December 30 in Royal Brisbane Hospital after going into a coma.

His mother-in-law, Joan Rynne, of Dalby, said the family still did not know what the terminal illness was.

The well-known sporting and business man became ill four months ago. His health rapidly deteriorated.

Another crushing blow to Mr Lawrie and wife Karen was the theft of personal items including a wedding ring from their rented Rickart Street home.

A thief smashed windows to gain access.

Neighbours notified police of the incident.

As well as the wedding ring, children's money boxes also were taken.

Mrs Rynne said the family was appealing for the ring to be returned.

"So far nothing has turned up," she said.

Mr Lawrie is survived by wife Karen and children Stacey, Hayden and Kayla.

A funeral will be held at Mt Gravatt on January 7.

Point 14 Time frame for increases in Protein Deposition Diseases

The slow but sure increase in P.D Diseases started about the same time that aluminium became more available i.e. in the early 1900's. The increase in P.D.D and aluminium production follow a close statistical path, allowing for the fact that P.D.D can take 20 to 40 years to become evident.

One point to take into consideration is the massive increase in production and general use of aluminium and other light metals over the last 20 years. Combine this with statistical projection for increases in P.D.D in the next 30 years.

Present	Future	Time Line
A.D – 160,000	600,000	30 years
Diabetes 1,000,000	2,000,000	5 years
Heart disease	1 in 4 will have heart problems	25 years

Others would question how this theory can account for the outbreak the kuru strain of TSE that exclusively erupted in an isolated tribe in e Fore region of the New Guinea Highlands. The conventional gma blames this outbreak upon the Fore tribe's traditional practice of nnibalism. Whilst cannibalism may have played a role in the bioac- mulation of manganese—particularly if the pituitary tissues were gested in these cannibalistic binges —the fact that virtually every be across New Guinea had adhered to a cannibalistic lifestyle, yet mained free of kuru, needs to be addressed by those who promote is theory. And furthermore, considering that cannibalism had been ditionally practised for centuries across New Guinea, why did kuru il to erupt until a few years after World War II?

My investigations suggest that the cause of kuru stems from the me template of eco-factors: the Fore tribe's self-sufficient lifestyle on pper-deficient soils, coupled with their scavenging of manganese- uminium sheet metal from the fuselages of several Japanese bomber t which had crashed in their area of the Highlands during World I. The Fore folk moulded the salvaged metal to make tools, ng pans and bowls, and these consequently contaminated their ods. They also accidentally exploded some of the bombs on board e crashed aircraft. These infamous explosions—well remembered by e surviving Fore folk—infrasonically irradiated their local vironment.

This story goes on. At the mouth of e Fuji River valley in Japan is a man- anese-aluminium alloy factory that anufactured these metal aircraft panels om the late 1930s and still makes Mn- l alloy products today. The man- anese-enriched chimney emissions dis- ersed downwind, permeating the entire ength of the valley. Intriguingly, a luster of CJD has blighted the residents f the Fuji River basin for 50 years. ote that it can take up to 20 years for he toxic effects of metal/chemical xposure to manifest.

The role of prion protein genetics is also surely compatible with the environmental facets as part of the overall multifactorial actiology of TSEs. For it is well established that prion protein genetics plays a major role in dictating which individuals are most susceptible to TSEs—where susceptibility hinges upon the expression of a defective prion protein that can only bind two or three atoms of copper instead of the usual five. But the sole focus of TSE susceptibility studies to date has almost exclusively concentrated upon

New drug test adds to Alzheimer's hope

The facts about dementia

- Alzheimer's is the greatest form of dementia.
- More than 160,000 Australians have dementia.
- One in 15 Australians over 84 years of age have dementia.
- By age 85, one in four Australians have dementia.
- About 6500 Australians under 65 have dementia.
- By 2041 some 500,000 Australians will have dementia if a treatment is not found, rising to 580,000 by 2050.
- The cost of dementia in Australia is expected to increase by 82 per cent in the next decade.
- In 2002, the estimated cost of dementia in Australia was about 1 per cent of GDP.
- By 2050 this is expected to increase to about 3 per cent of GDP.

A NEW drug testing system developed by Austr lian and US scientists could lead to a treatment f Alzheimer's disease within the next decade.

One of the world's leading dementia researc ers, Professor Ashley Bush, said the system shou cut decades from the race to develop drugs th prevent Alzheimer's.

And it could also help develop treatments f other age-related diseases with chemical simil ities to Alzheimer's.

"It's a breakthrough because it's the first tir ever that we've been able to consistently stop t pathology of the disease in animals by maki predictions from the test tube," he said.

"Our team is now investigating whether t system can be applied to Parkinson's disease a cataracts."

The process enabled thousands of drugs to systematically tested against other chemicals positive reactions with the protein amyloid, loved to cause Alzheimer's.

He said this reduced the first stage of identi ing drugs by decades.

The process has already led to clinical trials the drug Clioquinol, with early results showin stops Alzheimer's disease progressing.

"Clioquinol itself is still in clinical trials an all goes well that drug might be available as ea as within three to five years," he said.

Professor Bush said the next generation drugs developed by the new technology could available within five to 10 years.

Alzheimer's disease affects 160,000 Ausralia with that number expected to increase to 500, by 2041 if effective treatments are not found.

Professor Bush said it was now believed eve one developed Alzheimer's, but not everyone li long enough to show symptoms.

Alzheimer's Australia executive director L ette Moore said Australia was already in midst of a dementia epidemic.

CONCLUSION

Initially all our investigations were with aluminium oxide, but over the last 12 months we have been looking at the "Big Picture" for both oxidised light metal dust contributors and possible health effects.

Examined light metal oxidised dust, including asbestos, has similar characteristics that lead to the formation of plaque (amyloid protein) deposits. At present asbestos is being held solely responsible for plaque deposits causing lung cancer, melanoma, and mesothelioma. Considering that all the examined oxidised light metal dusts have similar characteristics how many asbestos related health problems have a source other than asbestos. Inchem 8.2.1.1 (the authors stated that aluminium oxide was the most likely cause of the development of interstitial fibrosis in workers and that asbestos could be ruled out). One of many such statements in the Inchem report.

Circumstances in our case show that the Queensland Government and The Rockhamtpon City Council knew of the problems with oxidised alum dust. The most disturbing element in this whole matter is that both Government and Council administrators are still risking workers and other lives every day. The moment they alter work practices that have been in practice for decades they admit knowledge, and therefore do nothing and avoid compensation.

With all the health problems linked to amyloid protein, plaque deposits, isn't it about time we acknowledge the common influence? The general public needs to be informed. It's only then that actions will be implemented to fix this problem. The media have skirted around the edges with health stories but media management will not address the big picture because of possible legal actions.

If we acted today to fix this problem, related health cases will still be showing up in the middle of this century.

8.2.1.1 Restrictive pulmonary disease

Historically, pulmonary fibrosis has been associated with various jobs within the aluminium industry. Shaver's disease (described in the 1940s) was a form of silicosis associated with the production of corundum abrasives (Shaver & Riddell, 1947). Another historically important occupational exposure associated with pulmonary fibrosis was experienced by "pyro powder" workers, who were exposed to very fine stamped aluminium powder (generally < 1 µm), including that used in the manufacture of explosives and fireworks (Doese, 1938; Meyer & Kasper, 1942; Mitchell et al., 1961; Jordan, 1961; McLaughlin et al., 1962; Gross et al., 1970). In that process, oils and solvents were used to coat particles to prevent naturally occurring oxidation, and nearly all cases of fibrosis were reported in workers exposed to mineral-oil-coated particles. That process is no longer used (Dinman, 1988a) and only one case has been reported since 1960 (McLaughlin et al., 1962). This syndrome indicates the potential pulmonary effect of non-oxidized aluminium metal, but such exposures do not occur in nature.

In a report of nine cases of workers exposed to aluminium oxide (mean duration of exposure 25 years), abnormal chest roentgenograms were described, as well as pathological lung functions in three of the cases (Jederlinic et al., 1990). Biopsies were taken from these three patients and analysed by electron microscopy and microprobe analysis. Interstitial fibrosis was the main histological finding. Metals occurred in amounts several orders of magnitude above background levels and the majority was aluminium oxide. The authors stated that aluminium oxide was the most likely cause for the development of interstitial fibrosis in these workers and that asbestos could be ruled out. Exposure to a "mixed dust", including free silica, also seemed to be a possible explanation.

With the exception of this exposure, pathological findings associated with aluminium exposure listed in Table 23 refers to mixed exposures, and cannot be solely attributed to aluminium. Other exposures, such as to silica or other metals, must be considered.

8.2.1.2 Obstructive pulmonary disease

a) *Asthma*

A potentially persistent form of occupational asthma related to primary aluminium smelting (pot room asthma) has been reported over the past 35 years; reversible symptoms, airflow limitation and increased bronchial responsiveness have been described (O'Donnell et al., 1989). The likely causes are irritant airborne particulate and fumes contributed by cryolite (sodium aluminium fluoride), gaseous hydrogen fluoride and other agents that may be adsorbed onto aluminium. A close relationship in aluminium potroom workers between levels of exposure to fluoride, which may be one of a number of

Table 23. Clinical and pathological pulmonary findings in aluminium-expo

Exposure	Clinical effects	Pathological changes
Aluminium powder grinder for 6 years	cough, DOE, abnormal X-ray, restrictive PFTs	pulmonary alveolar prot
Polisher for	cough, DOE	bronchogenic cancer; d

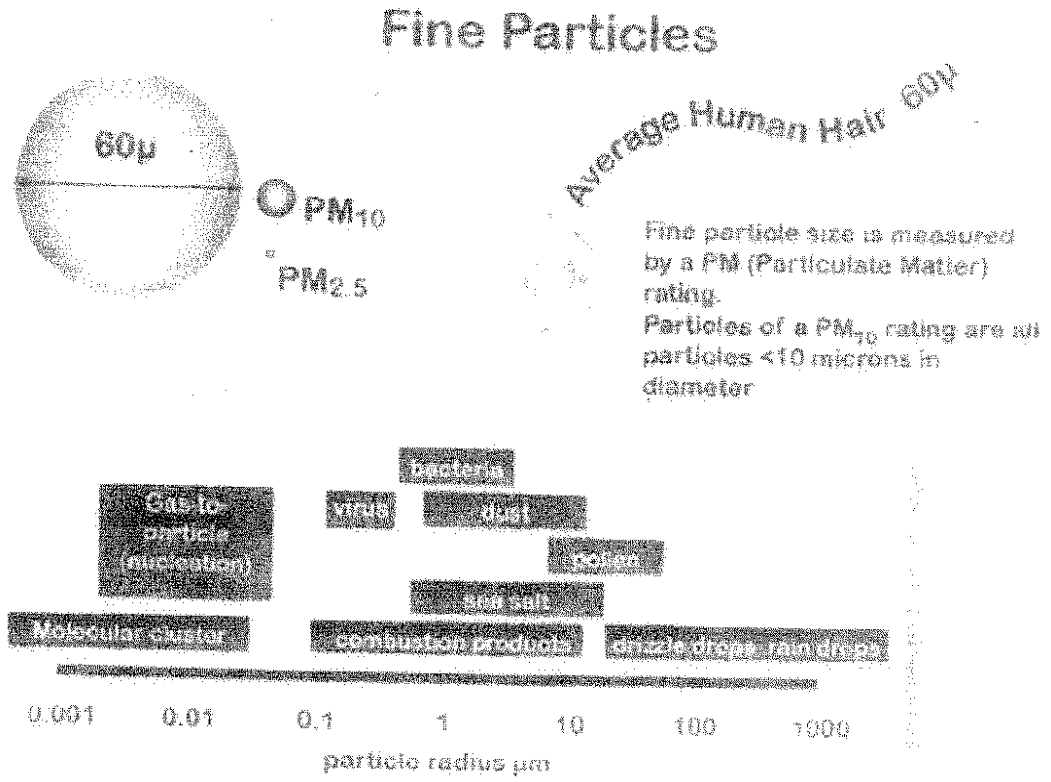
may want to expand their facilities and regulators who can use the model to assess development applications

Airborne particulate matter

Environmental effects of particulate matter
Health effects of particulate matter
Measurement of particulate matter
Dustfall deposit gauge
Aerosols
TSP
PM_{10}
$PM_{2.5}$

Particulate matter is the term used to describe particles that are suspended in the air. Particles may be solid or liquid and are one of the most obvious forms of pollution as they are visible in the hazes that cover a city or region.

Size is the main determinant of the behaviour of an atmospheric particle. The size is usually expressed in terms of the 'aerodynamic diameter' which refers to unit density of spherical particles with the same aerodynamic properties, such as the falling speed.



Larger particles (greater than 50µm) usually only remain in the air for a few minutes and settle near the source. A µm is one millionth of a metre, or 0.000001m). Smaller particles (less than 10µm, known as PM_{10}) can remain in the air for several days, and can be spread by winds over wide areas or long distances from the original source.

Fine particles (between 0.1-2.5µm) may remain in the atmosphere indefinitely. Fine particles are capable of scattering light, causing a reduction in visibility.

Particles are generally removed from the atmosphere by rain or when they come into contact with surfaces

Some particles may have other pollutants attached to them, which may react with those surfaces.

Windblown dusts, pollens from plants and sea salts are natural sources of particles in the atmosphere. Bushfires, agricultural and forest hazard-reduction burning release smoke particles into the air.

Combustion processes using coal and other fossil fuels, such as power generation, industrial operations and motor vehicle fuels, emit most of the particulate matter in urban areas. Other noticeable sources of particles include agricultural burning practices (e.g. burning of sugar cane prior to harvesting) and emissions from domestic solid fuel heaters and woodstoves.

Environmental effects of particulate matter

Particulate air pollution can cause a wide range of damage to surfaces and materials. Merely by requiring more frequent cleaning, particulates can accelerate deterioration. If the particle is corrosive or has other pollutants, for example sulfur dioxide, attached to it then it may also react with or corrode the surface or material.

Health effects of particulate matter

Under normal conditions a human respiratory tract in good health is able to deal with inhaled particles without undue stress or long-term effects. In sensitive individuals, or when high levels of particles are present, particulate matter may contribute to increased rates of respiratory illnesses and symptoms.

Studies indicate that such adverse effects are dependent on a number of factors, including:

- particle size (whether particles can penetrate the lower airways),
- the intensity of the exposure,
- the chemical nature of the particles and their interaction with human tissue,
- the presence or absence of pre-existing conditions (especially diseases of the respiratory tract), and
- meteorological factors such as winds, humidity, a temperature inversion, rain or thunderstorms.

Air quality goal

Inhalable particles (those with diameter less than 10µm) are commonly understood to pose the greatest risk to human health. There have been extensive studies into the health effects of different levels of particles and pollution mixes. However, no studies have yet determined a threshold value for long-term or short-term exposure below which no adverse health effects are observed.

The national 24-hour exposure standard for PM_{10} in the Air NEPM is 50 µg/m^3. This is the same as the EPP(Air) goal for annual average PM_{10} concentrations. The EPA monitors PM_{10} in south-east Queensland, Gladstone, Rockhampton, Mackay and Townsville. The annual PM_{10} average in these areas for 2001 were below the EPP(Air) goal (see below). See what the current PM_{10} concentrations are at the EPA's monitoring stations throughout Queensland.

(front page)

UNITED NATIONS ENVIRONMENT PROGRAMME
INTERNATIONAL LABOUR ORGANISATION
WORLD HEALTH ORGANIZATION

INTERNATIONAL PROGRAMME ON CHEMICAL SAFETY

ENVIRONMENTAL HEALTH CRITERIA 194

Aluminium

Environmental Health Criteria 194

First draft prepared by Dr H. Habs, Dr B. Simon and Professor K.U. Thiedemann (Fraunhofer Institute, Hoanover, Germany) and Mr P. Howe (Institute of Terrestrial Ecology, Monks Wood, United Kingdom)

Published under the joint sponsorship of the United Nations Environment Programme, the International Labour Organisation, and the World Health Organization, and produced within the framework of the Inter-Organization Programme for the Sound Management of Chemicals.

World Health Organization
Geneva, 1997

ALUMINIUM OXIDE

IDENTIFICATION

STATEMENT OF HAZARDOUS NATURE

Not classified as hazardous according to Worksafe Australia criteria.

SUPPLIER

Company:
Tennant Limited (ABN: 32 080 478 717)
Aluminum Co.of America-Alcoa of Australia Ltd
Leco Australia Pty Ltd

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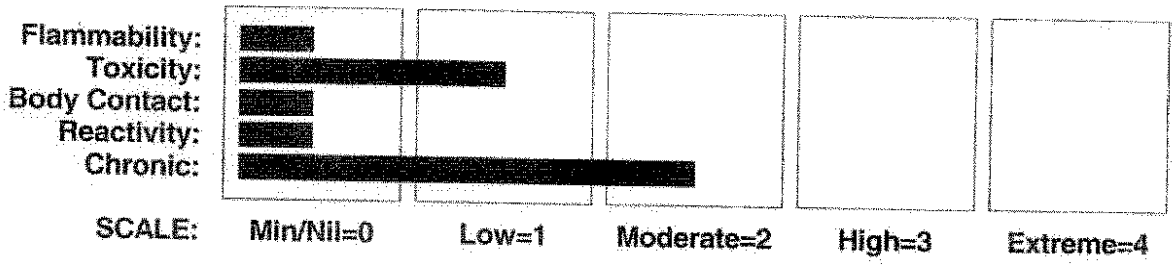
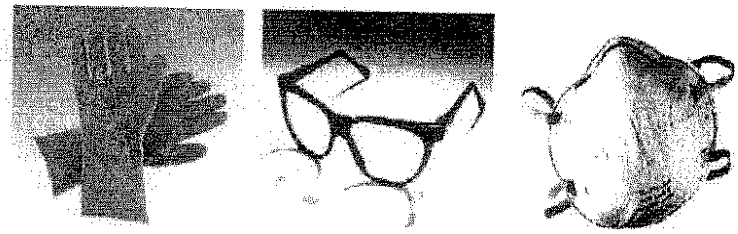
CHEMWATCH HAZARD RATINGS



PERSONAL PROTECTIVE EQUIPMENT FOR INDUSTRIAL/COMMERCIAL ENVIRONMENTS



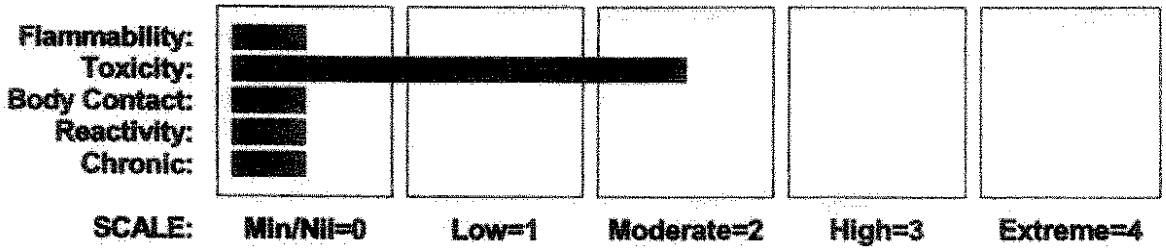
Product Name:	aluminium oxide
Other Names:	alumina Alcoa calcined aluminas - PCT Alcoa calcined aluminas - OF-2000 Alcoa calcined aluminas - A grades Polishing Powders, Aluminium Oxide

CAS RN No(s):	1344-28-1
UN Number:	None
Packing Group:	None
Dangerous Goods Class:	None
Subsidiary Risk:	None
Hazchem Code:	None
Poisons Schedule Number:	None

USE

Used as an adsorbent, desiccant. As a filler in paints & adhesives.
In the manufacture of alloys, ceramics, electrical insulators, resistors, dental cements, glass. As an abrasive and in metal polishes.
In the manufacture of artificial gems, in coatings for metals.
Gamma alumina - as a catalyst / catalyst support; used in chromatography.

Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION ...



Section 2 - HAZARDS IDENTIFICATION

STATEMENT OF HAZARDOUS NATURE

NON-HAZARDOUS SUBSTANCE. NON-DANGEROUS GOODS.

According to the Criteria of NOHSC, and the ADG Code.

POISONS SCHEDULE

None

RISK

Ingestion may produce health damage*.
* (limited evidence)

SAFETY

Do not breathe dust.
Take off immediately all contaminated clothing.
If you feel unwell contact Doctor or Poisons Information Centre. (Show the label if possible).

Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
magnesium oxide	1309-48-4.	> 96

Section 4 - FIRST AID MEASURES

SWALLOWED

- Immediately give a glass of water.
- First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor.

EYE

If this product comes in contact with the eyes:
- Immediately hold eyelids apart and flush the eye continuously with running water.
- Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
- Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes

Asbestos

Occupational Health & Safety Issues
Information Sheet No. 10

Description and Use

The term "Asbestos" is used to describe a group of naturally occurring fibrous silicate minerals that have a crystalline structure. There are two major groups of asbestos based upon the fibre type; these are:

- **Serpentine (wavy fibres):** The mineral Chrysotile commonly known as white asbestos
- **Amphibole (straight fibres):** The minerals Amosite (brown), Crocidolite (blue), Tremolite, Actinolite and Anthophyllite. The first two are the types commonly used for commercial purposes.

Asbestos has very good thermal resistance and corrosion resistance. Asbestos fibres are very strong and are capable of being spun and woven into fabric. Asbestos has been used as strengthening fibres in a large variety of materials such as cement, vinyl and cotton. It is electrically non-conductive.

Possible Health Effects

Asbestos fibres are made up of fine fibrils; thus each fibre can split into progressively finer fibres. Fine fibres are more likely to penetrate to the lower regions of the lungs (alveoli) and potentially cause disease. There is generally a latency period of 20 to 40 years between first exposure and onset of disease. Long-term over-exposure to airborne asbestos fibres can result in the following diseases:

- **Asbestosis:** is scarring of the lung tissues resulting from inhalation of large amounts of asbestos over a period of years.
- **Lung Cancer:** the risk of lung cancer is strongly related to the amount of fibre inhaled. (Note: the risk of lung cancer from exposure to both asbestos and cigarette smoke is much greater than the sum of the individual risks).
- **Mesothelioma:** cancer of the pleura (outer lung lining) or the peritoneum (lining of the abdominal cavity).

Worksafe Australia have set a standard of 0.1 fibres/ml for airborne asbestos to minimise the risk of developing one of the above health effects **for a person exposed to asbestos every day of their working life**. It is highly unlikely that anyone at Monash would be exposed to these sorts of levels of airborne asbestos nowadays. In the past staff working frequently with asbestos containing materials may have been.

There are two main types of asbestos containing products, these are:

- **Non-Friable:** Asbestos is bound within a matrix that does not allow airborne fibres to be readily generated (eg vinyl tiles, asbestos cement sheet). Asbestos fibres are not generally detected in the air (i.e. detection limit of 0.01 fibre/ml) near non-friable products unless the product is being physically damaged or abraded at the time of measurement.
- **Friable:** Products are easily damaged (eg pipe lagging) or their composition is such that airborne fibres can be generated readily (eg sprayed limpet). They do not generally give rise to detectable airborne asbestos levels unless they are physically disturbed for a significant proportion of the monitoring time. However airborne fibre are more readily produced from friable products.

Victorian Regulations

In 1992 Asbestos Regulations were introduced; they were replaced in 2003. The purpose of these regulations is to prevent asbestos-related disease in employees working in asbestos processes, and to protect the health of workers who may be exposed to airborne asbestos fibres due to asbestos products in their buildings or machinery. These regulations require the following:

- **Identify:** Employers and occupiers must identify whether asbestos is present.
- **Assess:** The risk of exposure from these products must be assessed.
- **Control:** Risk control measures must be implemented where necessary.

Asbestos at Monash University

A survey of the location and condition of friable forms of asbestos was performed at Clayton campus during 1984-85. Removal or encapsulation of high-risk items identified through this survey was done prior to 1990. Most asbestos-containing products at the Caulfield and Peninsula campuses were removed before merger with Monash. A survey of all types of asbestos-containing products, across all campuses, was commenced in 1995.

Labelling: Many items containing asbestos were labelled as part of the 1995 survey. Areas not generally accessed by the general public were labelled. Items that were not generally labelled were asbestos cement walls and ceilings, vinyl tiles and vermiculite ceilings. The warning on the labels of "serious inhalation health hazard" does not indicate that the current airborne fibre level constitutes a risk; it indicates that the product/ item should not be disturbed.

Remediation: A central budget exists for remediation and management of asbestos. Asbestos items are being sealed or removed to minimise the risk of staff, students and visitors being exposed to unacceptable levels of asbestos fibres. Where an immediate risk is thought to exist, air monitoring will be done to assess potential exposure. If this indicates high levels of air- borne asbestos then immediate remediation will occur.

Recommendation

Where staff are concerned about the location of asbestos and the above health issues, these concerns should be raised with the relevant supervisor, safety officer or health and safety representative. Assistance in assessing the risk, and controlling exposure, if asbestos-containing items have to be disturbed can be obtained from Occupational Health, Safety and Environment. In addition, specific medical concerns can be discussed with the University's Consultant Occupational Physician.

For additional information, contact Occupational Health, Safety & Environment on:

Tel: 9905 1016
Fax: 9905 2580
Email: ohsehelpline@adm.monash.edu.au

April 2003

THE MINERAL SERPENTINE

- **Chemistry: (Mg,Fe)3Si2O5(OH)4, Magnesium Iron Silicate Hydroxide**
- **Class:** Silicates
- **Subclass:** phyllosilicates
- **Group: Kalolinite-Serpentine**
- **Uses: many industrial applications, including brake linings and fireproof fabrics and as an ornamental stone.**
- Specimens

Serpentine is a major rock forming mineral and is found as a constituent in many metamorphic and weather igneous rocks. It often colors many of these rocks to a green color and most rocks that have a green color probably have serpentine in some amount.

Serpentine is actually a general name applied to several members of a polymorphic group. These minerals have essentially the same chemistry but different structures. The following is a list of these minerals, their formulas and symmetry class:

- Antigorite; (Mg,Fe)3Si2O5(OH)4; monoclinic.
- Clinochrysotile; Mg3Si2O5(OH)4; monoclinic.
- Lizardite; Mg3Si2O5(OH)4; trigonal and hexagonal.
- Orthochrysotile; Mg3Si2O5(OH)4; orthorhombic.
- Parachrysotile; (Mg,Fe)3Si2O5(OH)4; orthorhombic.

Their differences are minor and almost indistinguishable in hand samples. However, the chrysotile minerals are more likely to form serpentine asbestos, while antigorite and lizardite form cryptocrystalline masses sometimes with a lamellar or micaceous character. Asbestos had been used for years as a fire retarding cloth and in brake linings. Its links to cancer however has led to the development of alternative materials for these purposes.

Serpentine's structure is composed of layers of silicate tetrahedrons linked into sheets. Between the silicate layers are layers of Mg(OH)2. These Mg(OH)2 layers are found in the mineral brucite and are called brucite layers. How the brucite layers stack with the silicate layers is the main reason for the multiple polymorphs. The stacking is not perfect and has the effect of bending the layers. In most serpentines, the silicate layers and brucite layers are more mixed and produced convoluted sheets. In the asbestos varieties the brucite layers and silicate layers bend into tubes that produce the fibers.

Serpentine can be an attractive green stone that takes a nice polish and is suitable for carving. It has been used as a substitute for jade and is sometimes difficult to distinguish from jade, a testament to the beauty of finer serpentine material.

Non-fiberous serpentine is not a cancer concern. Asbestos serpentines should be kept in closed clear containers, but makes an attractive specimen. Sometimes with a golden color as the name chrysotile in greek means golden fibers.

PHYSICAL CHARACTERISTICS:

- **Color** is olive green, yellow or golden, brown, or black.
- **Luster** is greasy, waxy or silky.
- **Transparency** crystals are translucent and masses are opaque.
- **Crystal System** is variable, see above.
- **Crystal Habits**: never in large individual crystals, usually compact masses or fibrous. Veins of viberous serpentine can be found inside of massive serpentine or other rocks.
- **Cleavage** the varieties of crysotile have none, in lizardite and antigorite it is good in one direction.
- **Fracture** is conchoidal in antigorite and lizardite and splintery in the crysotiles.
- **Hardness** is 3 - 4.5
- **Specific Gravity** is 2.2 - 2.6

- **Streak** white
- **Associated Minerals** include chromite, olivine, garnets, calcite, biotite and talc.
- **Other Characteristics:** serpentine in the rough has a silky feel to the touch and fibers are very flexible.
- **Notable Occurances** Val Antigorio, Italy; Russia; Rhodesia Switzerland; North Carolina, California, Rhode Island and Arizona, USA and Quebec, Canada.
- **Best Field Indicators** softness, color, silky feel and luster, asbestos if present and its flexibility.

Minerals | By_Name | By_Class | By_Groupings | Search | Silicates

THE MINERAL GIBBSITE

- **Chemical Formula: Al(OH)3, Aluminum Hydroxide**
- **Class:** Oxides and Hydroxides
- **Uses: A minor source of metallic aluminum.**
- Specimens

Gibbsite is an important ore of aluminum and is one of three minerals that make up the rock Bauxite. Bauxite is often thought of as a mineral but is really a rock composed of aluminum oxide and hydroxide minerals such as gibbsite, **boehmite**, AlO(OH) and **diaspore**, HAlO2, as well as clays, silt and iron oxides and hydroxides. Bauxite is a laterite, a rock formed from intense weathering environments such as found in richly forested, humid, tropical climates.

Gibbsite's structure is interesting and analogous to the basic structure of micas. The basic structure forms stacked sheets of linked octahedrons of aluminum hydroxide. The octahedrons are composed of aluminum ions with a +3 charge bonded to six octahedrally coordinated hydroxides with a -1 charge. Each of the hydroxides is bonded to only two aluminums because one third of the octahedrons are vacant a central aluminum. The result is a neutral sheet since +3/6 = +1/2 (+3 charge on the aluminums divided by six hydroxide bonds times the number of aluminums) and -1/2 = -1/2 (-1 charge on the hydroxides divided between only two aluminums); thus the charges cancel. The lack of a charge on the gibbsite sheets means that there is no charge to retain ions between the sheets and act as a "glue" to keep the sheets together. The sheets are only held together by weak residual bonds and this results in a very soft easily cleaved mineral.

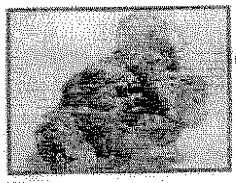
Gibbsite's structure is closely related to the structure of brucite, Mg(OH)2. However the lower charge in brucite's magnesium (+2) as opposed to gibbsite's aluminum (+3) does not require that one third of the octahedrons be vacant of a central ion in order to maintain a neutral sheet. The different symmetry of gibbsite and brucite is due to the different way that the layers are stacked.

It is the gibbsite layer that in a way forms the "floor plan" for the mineral corundum, Al2O3. The basic structure of corundum is identical to gibbsite except the hydroxides are replaced by oxygen. Since oxygen has a charge of -2 the layers are not neutral and require that they must be bonded to other aluminums above and below the initial layer producing the framework structure that is the structure of corundum.

Gibbsite is interesting for another reason because it is often found as a part of the structure of other minerals. The neutral aluminum hydroxide sheets are found sandwiched between silicate sheets in important clay groups: the Illite, Kaolinite and Montmorillonite/smectite groups. The individual aluminum hydroxide layers are identical to the individual layers of gibbsite and are referred to as the "gibbsite layers".

PHYSICAL CHARACTERISTICS:

- **Color** is white or colorless with shades of gray, blue and green.
- **Luster** is vitreous to dull; cleavage surfaces have a pearly luster.
- **Transparency** Crystals are translucent and rarely transparent.
- **Crystal System** is monoclinic; 2/m.
- **Crystal Habit** is usually massive but rare crystals are found in flattened tabular crystals. In many bauxite specimens gibbsite is found in a pisolitic habit. Also found as botryoidal encrustations, concretionary, stalactitic and foliated masses.
- **Cleavage** is perfect in one direction, basal.
- **Fracture** is uneven.
- **Hardness** is 2.5 - 3.5
- **Specific Gravity** is 2.4 (slightly below average)
- **Streak** is white.
- **Other Characteristics:** When breathed on, gibbsite gives off a noticeable clay smell.
- **Associated Minerals** are **boehmite, diaspore,** azurite, hydrozincite and aurichalcite.
- **Notable Occurrences** include Vogelsberg, Germany; Gant, Hungary; Les Baux, France; Lavrion, Greece; Guyana and Arkansas, USA.
- **Best Field Indicators** are crystal habits, hardness, smell and associations.



THE MINERAL BRUCITE

- **Chemical Formula: Mg(OH)2, Magnesium Hydroxide**
- **Class:** Oxides and Hydroxides
- **Group: Brucite**
- **Uses: A minor source of metallic magnesium, a source of magnesia and as a refractory additive.**
- Specimens

Brucite is a mineral that is not often used as a mineral specimen but does have some important industrial uses. It is a minor ore of magnesium metal and a source of magnesia. It is also used as an additive in certain refractories.

It is brucite's structure that is interesting. The basic structure forms stacked sheets of octahedrons of magnesium hydroxide. The octahedrons are composed of magnesium ions with a +2 charge bonded to six octahedrally coordinated hydroxides with a -1 charge. Each hydroxide is bonded to three magnesiums. The result is a neutral sheet since +2/6 = +1/3 (+2 charge on the magnesiums divided among six hydroxide bonds) and -1/3 = -1/3 (-1 charge on the hydroxides divided among three magnesiums); thus the charges cancel.

The lack of a charge on the brucite sheets means that there is no charge to retain ions between the sheets and act as a "glue" to keep the sheets together. The sheets are only held together by weak residual bonds and this results in a very soft easily cleaved mineral. Brucite is closely related to gibbsite, Al(OH)3. However the extra charge in gibbsite's aluminum (+3) as opposed to brucite's magnesium (+2) requires that one third of the octahedrons to be vacant of a central ion in order to maintain a neutral sheet.

Brucite is interesting for another reason because it is often found as a part of the structure of other minerals. How can this be? Well, the neutral magnesium hydroxide sheets are found sandwiched between silicate sheets in two important clay groups: the Chlorite and Montmorillonite/smectite groups. The individual magnesium hydroxide layers are identical to the individual layers of brucite and are referred to as the "brucite layers".

PHYSICAL CHARACTERISTICS:

- **Color** is white or colorless with shades of gray, blue and green.
- **Luster** is vitreous or waxy; cleavage surfaces have a pearly luster.
- **Transparency** Crystals are translucent and rarely transparent.
- **Crystal System** is trigonal; bar 3 2/m
- **Crystal Habit** is typically in flattened tabular crystals with rare rhombohedral terminations. Also found in lamellar and fibrous aggregates and as foliated masses. Brucite has been known to pseudomorph crystals of periclase.
- **Cleavage** is perfect in one direction, basal.
- **Fracture** is uneven.
- **Hardness** is 2 - 2.5
- **Specific Gravity** is 2.4 (slightly below average)
- **Streak** is white.
- **Other Characteristics:** cleavage flakes and fibers are flexible but not elastic.
- **Associated Minerals** are calcite, wollastonite, nepheline, talc, aragonite, serpentine, chromite, dolomite, magnesite, periclase and other magnesium minerals.
- **Notable Occurrences** include Unst, Shetland Islands, England; Aesbestos, Wakefield and Black Lake, Quebec, Canada; Aosta, Italy; Brewster, New York, Wood's Mine, Texas, Gabbs, Nevada, Crestmore, California and Berks Co., Pennsylvania, USA.
- **Best Field Indicators** are crystal habit, luster (especially on cleavage surfaces), lack of soapy or greasy feel and flexible but inelastic flakes and fibers.