

Dr. Tidu van der Merwe.

B.Sc.(Hons)(Genet), B.Med. Sci., MB.Ch.B., DOH.
Occupational Medical Practitioner.

Klibbe Rd 69,
Valhalla,
Pretoria, 0185.

PO Box 21014,
Valhalla, 0137
E mail: tidu@telkomsa.net

Tel.: 012 6601023
Fax: 0880 12 660 1023
Cell: +27 83 262 0096

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Baseline Community Health Assessment Report

Prepared for:
Pretoria Portland Cement Company Ltd

By

Dr. Tidu van der Merwe.

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1. Introduction.

The concept of burning Hazardous Waste (HW) in Cement Kilns has been in operation for many years in several countries in the world. In the US alone some 110+ cement kilns are currently burning HW for its inherent energy content. In the European Union, many countries had accepted the idea and are using the process. Similarly, in Australia the process has been accepted. Much controversy is generated with valid arguments for and against co-processing of hazardous chemical substances in cement kilns. With regard to the possible health impacts of the processes, publications emanating from the Cement Industry tend to present a picture of no hazard. The activist groups on the other hand paint a picture of significant hazard to people's health. In this report we will focus on the health impact only, and make a recommendation as to the desirability in terms of possible health outcomes of co-processing. The inescapable interaction and interdependency of the multitude of environmental systems, of which humans form part, will necessitate observations with regard to impacts other than emissions only.

The reaction of populations to low level exposures is extremely complex and poorly understood. It would be folly to predict specific outcomes and therefore we would endeavor to present a balanced, qualitative rather than quantitative conclusion. Epidemiological studies fall beyond the scope of this risk assessment and the assessment will be made on the basis of available information.

2. Scope.

- 2.1 The study will evaluate the possible current impact of air emissions is on local communities living close to the PPC plants

3. Terms of Reference;

The terms of reference for this study are as follows:

- 3.1 On the basis of available information, make qualitative recommendation regarding the health impacts and consequences of PPC cement kilns operations.

4. Limitations.

Some significant limitations exist to this study. These may be summarized as follows:

- 4.1 Information from rural hospitals can not be readily extrapolated to metropolitan centers.
- 4.2 The quality of the public health information: information regarding the prevailing current morbidity profile of the population is sketchy and limited. The data is often grouped with other data in terms of diagnosis and often of low specificity with regards to aetiology (cause).
- 4.3 In many cases where the ICD 10 (International Classification of Diseases, Version10)(World Health Organisation Publication) is used, the final classification has limited accuracy as the categorization of a specific disease is often ambiguous and difficult, for instance 'C97 – Malignant neoplasms of independent (primary) multiple sites' can easily be confused with 'D00 – D09 In situ neoplasms'. For this reason data capturing can also be a problem in that it occurs in an office environment rather than in the clinical setting.
- 4.4 We have encountered problems with obtaining information from the public sector service providers for the determination of the current situation. The

- reasons were unavailability of information in some cases. In other cases where information was available, it was of such a nature that it did not indicate the size of the reference population, therefore incidence and prevalence figures could not be calculated. The figures were merely that of numbers of patients seen, with no indication as to the size and demographics of the serviced population.
- 4.5 The availability of data from other governmental sources also presented a problem in that it was grouped in a format not suited for determination of specific aetiology (cause).
 - 4.6 The influence of HIV on the morbidity profile of the relevant populations might skew data significantly, obscuring minor increases in eg. respiratory diseases. The diseases that occur 'normally' in people, occur with increased frequency in the HIV positive person. Only later in the course of disease will some specific disease like Pneumocystis Carinii (pneumonia) occur. In the absence of HIV, an increase in chronic bronchitis may become noticeable. However, an increase is expected in the increasing HIV positive prevalence setting.
 - 4.7 Occupational diseases are seldom recognized as such in the clinical setting, both private and public health care. Where it may be recognized, reporting of such is poor. This is supported by the low figures reported to the Compensation Commissioner.
 - 4.8 Where occupationally related diseases are treated in the referral hospitals, the data is not captured as such, but rather under diagnosis. Retrieval and collation of such data would be difficult and incomplete at best. In most cases, the diagnosis will be in the format of 'pneumonitis' rather than 'Arsenic pneumonitis', thereby making retrieval of relevant data improbable.
 - 4.9 Due to the complexity of the pathological processes, as well as the non-specificity of obtainable data, the study will endeavor to provide qualitative information.
 - 4.10 No data on the prevalence or incidence of occupationally related cancers (except for asbestosis-related diseases) in the general population is available. None of the sources in Occupational Medicine provide information as to population incidences as all information relates to incidents and captured populations (groups of exposed workers)
 - 4.11 Many other exposures will go undetected as the clinical approach tends to address symptoms rather than aetiology, for example the psychological symptoms of Manganese or Mercury poisoning will be treated symptomatically and the true diagnosis may never be revealed.
 - 4.12 No studies exploring the current impact of cement kilns in South Africa were found.

5. List of substances investigated

- 5.1. Particulates.
 - 5.1.1 PM10
 - 5.1.2 Total Suspended Particulates (TSP)
- 5.2 Gases.
 - 5.2.1 SO_x
 - 5.2.2 NO_x
 - 5.2.3 CO
 - 5.2.4 HCL
 - 5.2.5 HF
 - 5.2.6 H₂SO₄
- 5.3 Metals

- 5.3.1 Arsenic(As)
- 5.3.2 Cadmium (Cd)
- 5.3.3 Chromium (Cr)
- 5.3.4 Cobalt (Co)
- 5.3.5 Copper (Cu)
- 5.3.6 Lead (Pb)
- 5.3.7 Manganese (Mn)
- 5.3.8 Mercury (Hg)
- 5.3.9 Nickel (Ni)
- 5.3.10 Thallium (Tl)
- 5.3.11 Tin (Sb)
- 5.3.12 Vanadium (V)
- 5.3.13 Zinc (Zn)

5.4 Hydrocarbons.

- 5.4.1 Dioxins
- 5.4.2 Furans

6. Tabulation of Known or Suspected Health Impacts.

Please note that the table in Appendix 1 list probable and possible exposure symptoms and signs related to various substances. (1 – 6). After exposure to a specific substance the person may or may not exhibit symptoms and signs, or the manifestation may be only months to years later. In a community setting, the symptoms can only be determined by very specific epidemiological studies – usually cohort studies spanning extended periods. In the event of clusters identified, the studies may retrospectively indicate increased incidences of certain diseases.

Table 1. Salient Points from Table Appendix 1¹

Substance name.	Health Impacts – salient points.	Notes.
SO_x as SO₂	Acute: pulmonary edema, death. Chronic: chronic bronchitis, eye effects.	Extremely common pollutant, Acid rain, Multiple sources of exposure throughout the country.
NO_x	Acute: Pulmonary irritation, edema, death Chronic: Transient lung opacities, dental complaints	Common in living environment, multiple sources of exposure, acid rain
CO	Acute: Insidious, unconsciousness, brain damage, death. Chronic: headache.	Lethal in acute exposures
HCL	Acute: Mucous membrane irritation, bronchospasm, laryngeal edema, pulmonary edema, ARDS (Adult Respiratory Distress Syndrome),	Common in living environment – pool acid. Acute exposures uncommon.
HF	Acute: Severe skin burns, penetrating. Pulmonary fibrosis, Osteofluorosis / Osteosclerosis, Cardiac arrhythmia, death.	Extremely toxic / hazardous in high doses, Rare in living environment
H₂SO₄	Acute: Mucosal irritation, bronchospasm, burns – 2 ^o , 3 ^o , charring. Chronic: Bronchospasm, asthma, Emphysema, pulmonary fibrosis, laryngeal cancer, Lung cancer	Strong acid, Common in living environment (Battery acid)
Arsenic(As), various forms. Arsine gas (AsH₃)	Acute: respiratory irritation ++, abdominal pain, hemorrhage, cardiogenic shock, nausea & vomiting, massive hemolysis, hemolytic anaemia, jaundice, acute oliguric renal failure, acute tubular necrosis, stupor, convulsions. coma, death. Chronic: GI symptoms, symmetric peripheral neuropathy, stocking-glove pattern, skin symptoms: Dermatitis, +/- depigmentation, Hyperpigmentation (scars, axillae, groin, nipples, neck), Mees' lines (Nails), desquamation palms & soles, cancer. Liver symptoms, cardiovascular symptoms, respiratory system: nasal septum perforation, lung carcinogen, immunosuppressant / immunotoxic , kidney failure	Uncommon in living environment, Associated with gold mining, previously commonly used in insecticides.
Cadmium (Cd)	Acute: Salivation, nausea, vomiting →	Toxic substance,

¹ Information for the country, obtained from the South African Medical Research Council (MRC), Burden of Disease Unit. (13)

Substance name.	Health Impacts – salient points.	Notes.
	<p>shock (after ingestion), chemical pneumonitis, pulmonary edema, death, metal fume fever, renal failure,</p> <p>Chronic: yellow rings around teeth, severe focal emphysema, osteomalacia, kidney damage, hypertension, lung carcinoma, prostate carcinoma.</p>	<p>common pollutant in living environment, used in colorants, batteries.</p>
<p>Chromium (Cr), as Cr III and Cr VI (Hexavalent)</p>	<p>Acute: Respiratory tract irritation, Allergic & irritant dermatitis, yellowing – teeth & tongue, renal symptoms, liver failure & death</p> <p>Chronic: Skin ulcers, mouth lesions, eye lesions, nasal septum perforation, respiratory tract symptoms, immunosuppression, lung Ca, Gastrointestinal Ca: esophagus, stomach, intestinal, pancreatic</p>	<p>Toxic substance, common sources of pollution: mining and related industry North West province, known contaminant of cement, essential element in low concentrations,</p>
<p>Cobalt (Co)</p>	<p>Chronic: Occupational asthma, interstitial fibrosis, dermatitis, cardiac symptoms, Sarcoma(?) / Mutagenesis.</p>	<p>Exposure symptoms unlikely from stack emission sources</p>
<p>Copper (Cu)</p>	<p>Acute. Only with very high exposures.</p> <ul style="list-style-type: none"> • Chronic: Granulomatous/fibrotic lung lesions, liver granulomas. 	<p>Common in living environment, Exposure effects uncommon</p>
<p>Lead (Pb)</p>	<p>Inorganic Lead:Chronic: wrist drop, anaemia, fatigue and asthenia, myalgia / arthralgia, neurobehavioural disturbances, chronic encephalopathy, chronic renal failure</p> <p>Organic Lead: Fatigue & lassitude, psychiatric manifestations – insomnia, hyperexcitability, mania, memory loss, delirium, seizures, coma.</p>	<p>Common pollutant in living environment, seldom acute symptoms, chronic symptoms probably much more common that evident.</p>
<p>Manganese (Mn)</p>	<p>Chronic:Increased risk of pulmonary infection, headaches, asthenia, hypersomnia.</p> <p>Mental disturbances:</p> <p>Stage1: Anorexia, Asthenia, Apathy, Spasms, Arthralgias, Irritability</p> <p>Stage 2: psychic & psychomotor disturbances: Dysarthria, Gait disturbances, Salivation</p> <p>Stage 3 – Parkinson’s disease</p>	<p>Common use in living environment, exposure symptoms very seldom diagnosed</p>
<p>Mercury (Hg)</p>	<p>Chronic.: Neuropsychological symptoms (Mercurial erethism): Tremor of lips, eyelids, fingers, tongue, Severe behavioral changes, Memory loss, Increased excitability, Delirium, Hallucinations. Kidney symptoms.</p> <p>Inorganic Mercury:</p> <p>Acute:ulceration & necrosis of GI mucosa, Acute renal failure</p> <p>Chronic: renal disease/failure, Children:</p>	<p>Various sources of pollution in living environment, occupational exposures diagnosed (Thor Chemicals), usually chronic symptoms, present in amalgam (teeth fillings)</p>

Substance name.	Health Impacts – salient points.	Notes.
	'Pink disease'/acrodynia: salivation; swelling of hands & feet; pink, peeling skin; hypotonia of limbs	
Nickel (Ni)	Chronic: Nasal & nasal sinus cancer, Laryngeal cancer, , stomach cancer, Sarcoma, Lung cancer, Contact dermatitis, Allergies, Asthma, Immuno-suppression/toxicity , Kidney & lung accumulation, Embryotoxic and teratogenic, Coronary vasoconstriction & stroke	Ni carbonyl highly toxic in low doses – 8 ppm., Ni metal very common in living environment, seldom acute exposure.
Thallium (Tl)	Acute: GI symptoms, neurological symptoms, Peripheral neuritis, pain, Asthenia, Alopecia Chronic: neurological symptoms, Mental abnormalities, Pulmonary oedema	Extremely toxic, Safe exposure levels adjusted downwards
Tin (Sn)	Acute:irritation of eyes, mucosa, skin, Cerebral oedema Chronic: Benign pneumoconiosis (Stannosis), Hepatic necrosis, Renal failure, Neurological effects, Pulmonary oedema, Death	Low toxicity metallic compounds, Fungicidal, Bacteriocidal, Some organic compounds severely toxic
Vanadium (V)	Acute: irritation eyes, respiratory system, skin, allergy Chronic: Green discoloration of tongue, fingers, thighs, scrotum , lung disease(?)	Symptoms generally reversible
Zinc (Zn)	Acute: Food poisoning effects – nausea, vomiting, diarrhea, Metal fume fever.	Low toxicity
Dioxins and Furans (Polychlorinated Hydrocabons)	Acute: Skin, eye, respiratory tract irritation, neurological symptoms, dyspnea, liver symptoms, Clotting abnormalities Chronic: Teratogenic, Chloracne, Foetotoxic, Fatigue, Weight loss, Insomnia, irritability, Immunotoxic , Soft tissue sarcoma, Non-Hodgkins lymphoma, Hodgkins lymphoma	Extremely toxic, Persists many years in body, Bind to particulates, bioaccumulates readily, Soil levels of 0.05 ppb makes soil unusable for agriculture, US Federal regulations limit emission to 0.2 ng - 0.4 ng TEQocm

Please note that these are mortality rates, i.e. measurements of the causes of death in the National population. These figures are based on death certificates' diagnosis. These diagnoses will list the primary cause of death, and separately 'Additional/contributing causes'. Burden of disease (measurements of the incidence and prevalence of diseases) cannot readily be determined from these figures since the latter measure disease and the former deaths only.

Table 2: Percentage of cancer deaths by cause, South Africa 2000 - Revised

All Persons			Males			Females		
Rank	Cause of death	%	Rank	Cause of death	%	Rank	Cause of death	%
1	Trachea/bronchi/ lung cancer	16.5	1	Trachea/bronchi/ lung cancer	21.9	1	Cervix cancer	17.2
2	Oesophageal cancer	13.4	2	Oesophageal cancer	16.7	2	Breast cancer	15.6
3	Cervix cancer	8.4	3	Prostate cancer	11.8	3	Trachea/bronchi/ lung cancer	10.9
4	Breast cancer	7.7	4	Liver cancer	7.8	4	Oesophageal cancer	9.9
5	Liver cancer	6.4	5	Stomach cancer	6.5	5	Colo-rectal cancer	6.9
6	Colo-rectal cancer	6.2	6	Colo-rectal cancer	5.4	6	Liver cancer	4.9
7	Prostate cancer	6.1	7	Mouth and oropharynx cancer	4.6	7	Stomach cancer	4.7
8	Stomach cancer	5.6	8	Leukaemia	3.8	8	Pancreas cancer	3.7
9	Pancreas cancer	3.7	9	Pancreas cancer	3.7	9	Ovary cancer	3.5
10	Leukaemia	3.5	10	Larynx cancer	3.0	10	Leukaemia	3.2
11	Mouth and oropharynx cancer	3.3	11	Lymphoma	2.8	11	Corpus uteri cancer	3.1
12	Lymphoma	2.5	12	Bladder cancer	2.2	12	Lymphoma	2.1
13	Larynx cancer	1.8	13	Bone and connective tissue cancer	1.7	13	Mouth and oropharynx cancer	2.0
14	Bone and connective tissue cancer	1.7	14	Brain cancer	1.3	14	Bone and connective tissue cancer	1.6
15	Ovary cancer	1.7	15	Kidney cancer	1.2	15	Brain cancer	1.2
16	Bladder cancer	1.6	16	Melanoma	1.1	16	Bladder cancer	1.0
17	Corpus uteri cancer	1.5	17	Non-melanoma skin cancers	0.7	17	Melanoma	1.0
18	Brain cancer	1.3	18	Breast cancer	0.2	18	Kidney cancer	0.9
19	Melanoma	1.0	19			19	Larynx cancer	0.6
20	Kidney cancer	1.0	20			20	Non-melanoma skin cancers	0.5
	All cancers	100.0		All cancers	100.0		All cancers	100.0

In a very hypothetical way, it would be perhaps only ranks 2, 3, 15, and 17 that could, with any form of certainty be excluded as not having co-factors from environmental pollution from all sources. All the other types of cancer could have an industrial connection. Proof of such can only be determined with very specific epidemiological cohort studies.

Table 3. Twenty leading specific causes of death in persons 60 years and older South Africa, 2000 (13)

Rank	Cause of death	Deaths	% of total
1	Ischaemic heart disease	26575	16.5
2	Stroke	24291	15.1
3	Hypertensive disease	12400	7.7
4	Chronic obstructive pulmonary disease	9665	6.0
5	Diabetes mellitus	8915	5.5
6	Lower respiratory infections	8610	5.4
7	Tuberculosis	6622	4.1
8	Trachea/bronchi/lung cancer	4298	2.7
9	Nephritis/nephrosis	4012	2.5
10	Asthma	3808	2.4
11	Oesophageal cancer	3139	2.0
12	Inflammatory heart disease	2968	1.8
13	Septicaemia	2429	1.5
14	Diarrhoeal diseases	2357	1.5
15	Prostate cancer	2348	1.5
16	Cirrhosis of liver	2069	1.3
17	Colorectal cancer	1886	1.2
18	Road traffic accidents	1794	1.1
19	Breast cancer	1660	1.0
20	Interpersonal violence	1603	1.0
	All causes among 60+	160639	

From this table, it is perhaps again only 12, 13, 18 and 19 that probably has no causality from industrial pollution. Once again, as with the cancer data, determination of links to any specific industry is not possible.

Table 4. Data from Victoria Hospital, near Slurry, for the year June 06 – March 07

<u>Category</u>	<u>Number of patients</u>	<u>% of Total</u>
A. Infectious diseases	616	4.97
B. Viral infections / infections / infestations.	78	0.63
C. Neoplasms.	67	0.54
D. In situ neoplasms, Blood disorders	265	2.14
E. Endocrine, Nutritional & Metabolic Disease	279	2.25
F. Mental & Behavioural Disorders.	616	4.97
G. Nervous System	393	3.17
H. Ear & Mastoid Process	225	1.81
I. Circulation System	485	3.91
J. Respiratory Diseases	1857	14.97
K. Digestive System	1031	8.31
L. Skin & Subcut.	282	2.27
M. Musculoskeletal & Connective Tissue	628	5.06
N. Genito-urinary tract	1075	8.67
O Pregnancy, Birth, Puerperium	1071	8.64
P. Perinatal period.	68	0.55
Q. Congenital malformation, deformations, genetic abnormality.	24	0.19
R. Other Symptoms and Signs	1128	9.10
S. Fractures, limb injuries	1003	8.09
T. Other injuries, accidents	326	2.63
V. Transport accidents/injuries	7	0.06
W. Other accidental injury	18	0.15
X. Exposure to Smoke, fire, substances	13	0.10
Y. Events of undetermined intent.	5	0.04
Z. General Medical procedures, examinations	842	6.78
Total	12402	100%

Respiratory conditions make up 14.97% of patients seen at the Victoria facility (Table 4). The primary cause of pneumonia could be Viral, Bacterial, Fungal, SO_x, NO_x, HCL, Cd, Cr, Cu and V. There may also be secondary causes, ie the pneumonia follows injury to the lung due to other medical conditions such as neoplasms, other infections, notably HIV, TB, aspiration, injuries etc.

Data from the PPC Slurry Clinic does not reflect community health data, but rather site specific data. This data should be utilized for the determination of Occupational Health evaluation and not be used as an indicator of community health status. The Victoria Hospital data reflects a mainly rural population group of different socioeconomic stratifications and includes Pediatric, Obstetrics and Gynaecology data that is absent in the Slurry clinic data. See also discussion below.

7.0 Discussion.

- 7.1 Comparability of figures: All figures obtained from Statistics SA (StatSA) reflect mortality rates (death rates) rather than morbidity rates (disease rates). The MRC could not provide any specific morbidity studies at this moment in time. The demographic information obtained from StatSA and other sources, (7 – 12) on health utilizes indicators such as immunization, sanitation, birth rates, perceived health status questionnaires etc. These national figures cannot be utilized to make any specific prediction as to specific health impacts such as the effect of any one industry on the morbidity profile on the health of its surrounding communities. Provincial figures similarly are a compilation and reworking of data from all sources throughout the relevant province. This by necessity includes rural, as well as urban data.
- 7.2 The PPC clinic data is for reasons below, not suitable for the determination of community health impacts as it addresses a very selective population. Similarly, the Victoria hospital figures *may* contain information as to the community health impact of PPC Slurry, but since no connection can be made, the data cannot be utilized to make inferences as to the community health impact of PPC Slurry. If any such connections is needed, then highly sophisticated epidemiological studies is needed that would probably have low specificity.
- 7.3 Data from the site clinics, such as Slurry, is significantly skewed in an number of ways:
- 7.3.1 The Slurry population is almost exclusively male.
 - 7.3.2 The Slurry population, being employed, would exhibit a far better lifestyle than most of the other rural population groupings. This would hold true to an extent in the urban settings as well.
 - 7.3.3 Well worker effect – on persons well enough to work is included. Only persons that are healthy are selected to work.
 - 7.3.4 Advanced disease tends to fall away from the figures through retrenchment, dismissal, resignation etc.
 - 7.3.5 Clinic data reflects mainly salaried or wage personnel, whereas hospital data would reflect large numbers of the socio-economic disadvantaged population.
 - 7.3.6 Almost all medical aid persons would tend to visit private practitioners and private hospitals rather that the public health service. Data from these service providers is not available.
- 7.4 Clinically it is practically impossible to ascribe a specific person's pneumonia to e.g. Vanadium releases from industry. Only with specific investigations for Vanadium once it is suspected, can it be postulated that Vanadium may be a *contributing* cause. The laboratory results may only come days to weeks later

and could still be inconclusive. The data for this case would be logged as pneumonia of undetermined cause. This would group it with a multitude of other causes.

- 7.5 The situation for malignancies is even worse in that a malignancy may appear only 20- 30 years after exposure. The connection would only be made with retrospective cohort studies. (Asbestosis / Mesothelioma is the exception to the rule) It is therefore not possible to predict to what extent the increase in atmospheric Vanadium would increase the incidence of pneumonia. It is only after various special tests and investigations that one can arrive at a final diagnosis. In the majority of cases the person would be treated empirically and in most cases healing would be effected. In the case of clustering of diagnoses, associations will be sought and aetiology investigated if the manpower and funds are available
- 7.6 The interaction between HIV and Occupational Diseases (OD) is not clear. As a rule of thumb one might expect that HIV will increase susceptibility to other diseases. However, no current data either confirm or deny this. In the assessment of the possible constituents of the emissions (Table 1), it is clear that some of them are known to be immunosuppressors, and therefore will have a negative influence on the morbidity and mortality of HIV positive persons. The magnitude of this influence is indeterminate
- 7.7 The diagnosis of Occupational Diseases (OD) in South Africa is notoriously poor. (13) While Occupational Injuries are usually obvious and immediate, Occupational Diseases normally present after long periods from months to years. Asbestosis for instance, and cancers, will only manifest after 20 or more years. Occupational Diseases usually have an insidious onset and may be disguised as many common illnesses. Only in special cases where accidents or incidents occur, will exposures be related to symptoms – as is the case for the majority of investigations worldwide. This means that determination of environmental or occupational causation of disease is limited and flawed in our current system.
- 7.8 Individual responses to similar exposure also cause a variation in the presenting symptoms. Only if the suspecting doctor will actually do the appropriate tests (if available in the prevailing clinical setting) would environmental causation be suspected. Thus diagnosis of exposures and its consequences remain difficult.
- 7.9 Once the clinical staff has been alerted at to the possibility of certain diseases occurring, then clusters of certain exposure diseases may be identified. This would then lead to further epidemiological investigation.

8.0 Conclusion.

In the current situation, there is no information or study that demonstrates that PPC, in itself, has any negative effect on its surrounding communities. The available data cannot be utilized to make any conclusive decision as to whether PPC has a negative effect at any one of its sites. In order to do this, extensive epidemiological studies is needed. Even with such a study, clear association would be doubtful, as these studies often has fairly low specificity. This is evident in the myriad of publications in Occupational Medical literature 'suggesting' association. It is only H₂SO₄, (Sulphuric acid) that has been classified as a human carcinogen on the basis of epidemiology alone.

Determination of adverse effect is indeterminate at this point in time.

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Appendix 1.

Substance name	Health Impacts	Notes	Exposure levels and criteria
SO_x as SO₂	Acute: <ul style="list-style-type: none"> ○ mucous membrane irritant, ○ pulmonary edema, death. Chronic: <ul style="list-style-type: none"> ○ decrease in smell & taste ability. ○ chronic bronchitis. ○ corneal ulceration and scarring. ○ chronic bronchitis ○ conjunctivitis, ○ corneal ulceration 	Extremely common pollutant Acid rain Multiple sources of exposure throughout the country.	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3.
NO_x	Acute: <ul style="list-style-type: none"> ○ Insidious ○ Pulmonary irritation ○ Pulmonary edema ○ Death Chronic: <ul style="list-style-type: none"> ○ Brown discoloration teeth. ○ Transient patchy-lung opacities on CXR ○ Dental complaints – brown discoloration of teeth. 	Common pollutant in living environment, multiple sources of exposure Cause acid rain	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3.
CO	Acute: <ul style="list-style-type: none"> ○ Insidious, ○ Giddiness, ○ Headache, ○ Chest tightness, ○ Nausea, myocardial infarction, ischemia, arrhythmia ○ Unconsciousness, ○ Brain damage, ○ Death. Chronic: <ul style="list-style-type: none"> ○ Headache. 		Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3.
HCL	Acute: <ul style="list-style-type: none"> ○ Mucous membrane irritation ○ Cough ○ Stridor (bronchospasm) ○ Dyspnea. ○ Laryngeal edema. ○ Lower resp tract injury ○ Pulmonary edema. ○ ARDS (Adult Respiratory Distress Syndrome) ○ Lacrimation. ○ Rhinorhea. ○ Burning of mouth and throat. 	Common in living environment – pool acid	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 US: 3 ppm (c) NIOSH: 5 ppm (C)
HF	Acute: <ul style="list-style-type: none"> ○ Laryngeal spasm ○ Cough. ○ Hemoptysis, epistaxis 	Extremely toxic / hazardous Rare in living	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3

Substance name	Health Impacts	Notes	Exposure levels and criteria
	<ul style="list-style-type: none"> ○ Severe skin burns, penetrating. ○ Prolonged hoarseness ○ Prolonged tracheo-bronchitis +20 infection. ○ Pulmonary fibrosis. ○ Osteofluorosis / Osteosclerosis ○ Cardiac arrhythmia (plasma Ca and Mg disturbance) 	environment	US: 3 ppm TWA NIOSH: 3 ppm TWA, 6 ppm (C) UK: 1 ppm STEL (1.5 mg/m ³)
H₂SO₄	<p>Acute:</p> <ul style="list-style-type: none"> ○ Mucosal irritation ○ Lacrimation ○ Conjunctivitis. ○ Bronchospasm ○ Hemoptysis. ○ Burns – 20, 30, charring. ○ Chronic: ○ Dental erosion. ○ Bronchospasm, asthma. ○ Emphysema. ○ Pulmonary fibrosis. ○ Laryngeal cancer ○ Lung cancer 	<p>Strong acid</p> <p>Common in living environment</p>	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 NIOSH: 1 mg/m ³ US: 1 mg/m ³
Arsenic(As), various forms. Arsine gas (AsH₃)	<p>Acute:</p> <ul style="list-style-type: none"> ○ Respiratory irritation ++ ○ Headache. ○ Abdominal pain, hemorrhage ○ Cardiogenic shock, ↓ BP ○ Nausea & vomiting → shock ○ Skin irritation, allergy. ○ Massive hemolysis, hemolytic anaemia, jaundice. ○ Acute oliguric renal failure, acute tubular necrosis. ○ Stupor, convulsions. coma, death. <p>Chronic:</p> <ul style="list-style-type: none"> ● GI symptoms ● Symmetric peripheral neuropathy, stocking-glove pattern ● Skin <ul style="list-style-type: none"> ○ Dermatitis, +/-depigmentation. ○ Hyperpigmentation (scars, axillae, groin, nipples, neck) ○ Mees' lines (Nails) ○ Desquamation palms & soles ○ Cancer. ● Liver <ul style="list-style-type: none"> ○ Hepatomegaly. ○ Cirrhosis. ○ Angiosarcoma. ● Cardiovascular <ul style="list-style-type: none"> ○ Peripheral vascular disease. <ul style="list-style-type: none"> ▪ Acrocyanosis → hyperpigmentation, hyperkeratosis. ▪ Raynaud. ○ Endarteritis obliterans → gangrene. 	<p>Uncommon in living environment</p> <p>Associated with gold mining.</p>	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 UK OES: 0.2mgm ⁻³ , 0.05 ppm

Substance name	Health Impacts	Notes	Exposure levels and criteria
	<ul style="list-style-type: none"> ○ Myocarditis, MI. ● Respiratory system ○ Nasal septum perforation. ○ Lung carcinogen ● Immunosuppressant / immunotoxic. ● Anemia ● Kidney failure 		
Cadmium (Cd)	<p>Acute:</p> <ul style="list-style-type: none"> ○ Salivation, nausea, vomiting → shock (after ingestion) ○ Fumes: chemical pneumonitis, pulmonary edema, death. ○ Fever, chills, dyspnea (metal fume fever) ○ Mucous membrane irritation. ○ Renal failure. ○ Gastrointestinal irritation. <p>Chronic:</p> <ul style="list-style-type: none"> ○ Non-specific: GI disturbances, yellow rings around teeth, ○ Anosmia. ○ Severe focal emphysema ○ Osteomalacia. ○ Anemia. ○ Kidney damage: proteinuria, glycosuria, aminoaciduria. ○ Hypertension. ○ Lung carcinoma. ○ Prostate carcinoma. 	<p>Toxic substance</p> <p>Common pollutant in living environment</p>	<p>Refer OHS Act, 85/93</p> <p>Haz Chem Regs, Table 1-3</p> <p>UK MEL (max exp lim): 0.05 mgm⁻³</p> <p>OHSA: 2.5µg/m³ 8h TWA</p> <p>ACGIH TLV 2.0µg/m³ 8h TWA</p>
Chromium (Cr), as Cr III and Cr VI (Hexavalent)	<p>Acute:</p> <ul style="list-style-type: none"> ○ Respiratory tract irritation ○ Allergic & irritant dermatitis ○ Gastric distress ○ Dysosmia ○ Yellowing – teeth & tongue ○ Renal chromate toxicosis, ○ Liver failure & death ○ Nasal septum perforation <p>Chronic:</p> <ul style="list-style-type: none"> ○ Skin ulcers ○ Gingivitis & periodontitis ○ Eye lesions, conjunctivitis & keratitis ○ Sinusitis ○ Bronchitis ○ Asthma. ○ Rhinitis ○ Nasal mucosal polyps ○ Immunosuppression ○ Chemical pneumonitis ○ Lung Ca ○ Gastrointestinal Ca: esophagus, stomach, intestinal, pancreatic 	<p>Toxic substance</p> <p>Common sources of pollution: mining and related industry North West province</p> <p>Known contaminant of Portland cement</p>	<p>Refer OHS Act, 85/93</p> <p>Haz Chem Regs, Table 1-3</p> <p>HSE OES = 0.5 mg m⁻³ (Cr III)</p> <p>HSE OES = 0.05 mg m⁻³ (Cr VI)</p> <p>ACGIH TLV = 0.5 mg m⁻³ TWA (Cr metal)</p> <p>ACGIH TLV = 0.05 mg m⁻³ TWA (Cr VI)</p> <p>NIOSH = 1µg m⁻³ TWA</p>
Cobalt (Co)	Chronic	Exposure	Refer OHS Act,

Substance name	Health Impacts	Notes	Exposure levels and criteria
	<ul style="list-style-type: none"> ○ Occupational asthma/ Hard Metal Asthma – (Cobalt hypersensitivity) ○ Interstitial fibrosis (Hard Metal Pneumoconiosis) ○ Allergic dermatitis. ○ Cardiomyopathy (?) ○ Sarcoma(?) / Mutagenesis. 	symptoms unlikely from stack emission sources	85/93 Haz Chem Regs, Table 1-3 TLV: 50 µg/m ³ .
Copper (Cu)	<p>Acute. (very high exposures)</p> <ul style="list-style-type: none"> ○ Upper respiratory tract irritation ○ Ulceration & perforation of nasal septum ○ Vomiting ○ Gastro-intestinal burns ○ Diarrhoea. ○ Metal fume fever. <p>Chronic.</p> <ul style="list-style-type: none"> ○ Granulomatous/fibrotic lung lesions ○ Liver granulomas. 	Common in living environment Exposure effects uncommon	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 ACGIH TLV-TWA 0.2 mg/m ³ .
Lead (Pb)	<p>Inorganic Lead.</p> <p>Acute.</p> <ul style="list-style-type: none"> ○ Encephalopathy ○ Hemolysis, anaemia ○ Acute renal failure ○ Abdominal cramps & constipation ○ Chronic. ○ Peripheral motor neuropathy (wrist drop) ○ Anaemia. ○ Fatigue and asthenia ○ Myalgia / arthralgia. ○ Neurobehavioural disturbances, chronic encephalopathy ○ Impaired fertility ○ Gout & gouty nephropathy ○ Chronic renal failure <p>Organic Lead.</p> <ul style="list-style-type: none"> ○ Fatigue & lassitude ○ Headache <ul style="list-style-type: none"> ○ Psychiatric manifestations – ○ insomnia, ○ hyperexcitability, ○ mania. ○ Memory loss ○ Delirium, seizures, coma. 	Common pollutant in living environment	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 See also Lead Regulations under the OHS Act, Act 85 of 1993. Inorganic lead: HSE MEL: 0.15 mg/m ³ . Organic lead: Tetraethyl lead as Pb: HSE MEL : 0.10 mg/m ³ .
Manganese (Mn)	<p>Acute.</p> <ul style="list-style-type: none"> ○ Respiratory mucous membrane irritant <p>Chronic.</p> <ul style="list-style-type: none"> ○ Increased risk of pulmonary infection – inflammatory response ○ Headaches ○ Asthenia ○ Hypersomnia ○ Mental disturbances: <p>Stage1</p> <ul style="list-style-type: none"> ○ Anorexia 	Common use in living environment	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 ACGIH TLV: 0.02 mg/m ³ .

Substance name	Health Impacts	Notes	Exposure levels and criteria
	<ul style="list-style-type: none"> ○ Asthenia ○ Apathy ○ Spasms ○ Arthralgias ○ Irritability <p>Stage 2 psychic & psychomotor disturbances.</p> <ul style="list-style-type: none"> ○ Dysarthria ○ Gait disturbances ○ Salivation <p>Stage 3 – Parkinson’s disease</p> <ul style="list-style-type: none"> ○ Rickets like bone changes 		
Mercury (Hg)	<p>Acute.</p> <ul style="list-style-type: none"> ○ Dyspnea ○ Weakness ○ Pleuritic chest pain <p>Chronic.</p> <ul style="list-style-type: none"> ○ Salivation ○ Gingivitis ○ Papular erythema with hyperkeratosis ○ Neuropsychological symptoms (Mercurial erethism): <ul style="list-style-type: none"> ○ Tremor of lips, eyelids, fingers, tongue. ○ Severe behavioral changes ○ Memory loss ○ Increased excitability ○ Delirium ○ Hallucinations ○ Kidneys: proteinuria to nephritic syndrome. <p>Inorganic Mercury:</p> <p>Acute.</p> <ul style="list-style-type: none"> ○ Corrosive ulceration & necrosis of GI mucosa. ○ Acute renal failure ○ Chronic ○ Proximal tubule necrosis ○ Membranous glomerulonephritis ○ Children: Pink disease/acrodynia: salivation; swelling of hands & feet; pink, peeling skin; hypotonia of limbs 	Various sources of pollution in living environment	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 HSE OES 0.05 mg/m ³ . HSE OES: 0,01 mg/m ³ , as Mercury Alkyls, Sk notation
Nickel (Ni)	<p>Acute.</p> <ul style="list-style-type: none"> ○ Sinusitis ○ Anosmia ○ Headache (Carbonyl) ○ Fatigue (Carbonyl) ○ Gastrointestinal symptoms (Carbonyl) ○ Interstitial pneumonitis (Carbonyl) ○ Delirium, coma, death (Carbonyl) <p>Chronic.</p> <ul style="list-style-type: none"> ○ Nasal cancer ○ Cancer of nasal sinuses ○ Laryngeal cancer 	Ni carbonyl highly toxic in low doses – 8 ppm.	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 ACGIH TLV: 0.05 mg/m ³ . OHS PEL: 1 mg/m ³ TWA (metal) OHS PEL: 0.1 mg/m ³ (soluble)

Substance name	Health Impacts	Notes	Exposure levels and criteria
	<ul style="list-style-type: none"> ○ Stomach cancer ○ Sarcoma ○ Lung cancer ○ Contact dermatitis ○ Allergies ○ Asthma ○ Immunosuppression/toxicity ○ Kidney & lung accumulation. ○ Embryotoxic and teratogenic ○ Coronary vasoconstriction & stroke 		
Thallium (Tl)	<p>Acute.</p> <ul style="list-style-type: none"> ○ Nausea ○ Vomiting ○ Diarrhea ○ GI hemorrhage ○ Strabismus ○ Peripheral neuritis ○ Pain ○ Asthenia ○ Parestesias in legs ○ Tremor ○ Retrosternal tightness ○ Alopecia <p>Chronic.</p> <ul style="list-style-type: none"> ○ Ataxia ○ Optic atrophy ○ Tremor ○ Mental abnormalities ○ Foot drop ○ Pulmonary oedema 	<p>Extremely toxic</p> <p>Safe exposure levels adjusted downwards</p>	<p>Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 ACGIH TLV TWA: 0.1 mg/m³</p>
Tin (Sn)	<p>Organotin.</p> <p>Acute</p> <ul style="list-style-type: none"> ○ Eye irritation ○ Mucous membrane irritation ○ Skin irritation ○ Cerebral oedema <p>Chronic</p> <ul style="list-style-type: none"> ○ Benign pneumconiosis (Stannosis) ○ Hepatic necrosis ○ Renal failure ○ Neurological effects ○ Pulmonary oedema ○ Death 	<p>Low toxicity metallic compounds</p> <p>Fungicidal</p> <p>Bacteriocidal</p> <p>Some organic compounds severely toxic</p>	<p>Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 Inorganic: ACGIH TLV TWA: 2 mg/m³ OHS PEL TWA: 2 mg/m³ Organic: ACGIH TLV TWA: 0.1 mg/m³ OHS PEL TWA: 0.1 mg/m³</p>
Vanadium (V)	<p>Acute</p> <ul style="list-style-type: none"> ○ Lacrimation, eye irritation ○ Epistaxis ○ Cough ○ Bronchitis ○ Pneumonia ○ Allergic asthma ○ Skin irritation, eczema 	<p>Symptoms generally reversible</p>	<p>Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 ACGIH TLV TWA: 0.05 mg/m³</p>

Substance name	Health Impacts	Notes	Exposure levels and criteria
	Chronic <ul style="list-style-type: none"> ○ Green discoloration of tongue, fingers, thighs, scrotum. ○ Obstructive lung disease(?) ○ Chronic bronchitis 		
Zinc (Zn)	Acute <ul style="list-style-type: none"> ○ Food poisoning effects – nausea, vomiting, diarrhea ○ Metal fume fever Chronic <ul style="list-style-type: none"> ○ None reported 	Low toxicity	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 ACGIH and OSHA values varies from 1.0 – 10 mg/m ³ , STEL, depending on compounds
Dioxins and Furans (Polychlorinated Hydrocabons)	Acute <ul style="list-style-type: none"> ○ Skin, eye, respiratory tract irritation ○ Headache ○ Vertigo ○ Nausea <ul style="list-style-type: none"> ● Chloracne ○ Severe myalgia ○ Fatigue ○ Nervousness & irritability ○ Dyspnea ○ Hepatomegaly ○ Peripheral neuritis ○ Clotting abnormalities Chronic <ul style="list-style-type: none"> ○ Teratogenic ○ Foetotoxic ○ Chloracne ○ Fatigue ○ Weight loss ○ Insomnia, irritability ○ Immunotoxic ○ Soft tissue sarcoma ○ Non-Hodgkins lymphoma ○ Hodgkins lymphoma 	Extremely toxic Persists many years in body Bind to particulates Bioaccumulates readily Soil levels of 0.05 ppb makes soil unusable for agriculture. US Federal regulations limit emission to 0.2 ng - 0.4 ng TEQocm	Refer OHS Act, 85/93 Haz Chem Regs, Table 1-3 NIOSH REL: lowest feasible level