Clean and Healthy Air for Gladstone

Key pollutant inventory

and

Suite of health outcomes to be assessed

Population Health Services

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Contents

1. Introduction

This discussion paper outlines the key pollutants and health outcomes to be investigated as part of the Health Risk Assessment component of the Clean & Healthy Air for Gladstone Project. Information on the health effects of each pollutant is presented together with the reasons for its inclusion in the study.

Note that the list of actual pollutants to be monitored by the Air Monitoring Plan is more extensive than that presented here and the reader is referred to that document for the complete listing.

This document also provides:

- descriptions of the health outcomes to be investigated;
- some background information on their prevalence in Australia and known risk factors associated with their occurrence; and
- a discussion on the limitations and expectations for the study.

A draft of this discussion paper was presented to the community and industry reference groups in February 2008. This document was also made available to the public via the EPA project website.

As a result of this consultation, additional information has been included regarding coal dust and particulates. In addition, coal dust has been included as a stand alone item in the pollutant inventory. The health outcomes outlined in the draft discussion paper have been accepted as being appropriate.

2. Developing the key pollutant inventory

2.1 Criteria

The project seeks to achieve the following three health-related objectives:

- to assess the potential risks to human health associated with air emissions from industrial sources
- to assess whether the community has experienced adverse health outcomes as a result of those emissions
- to identify whether further health monitoring or evaluation is required.

The study population is the residents within the geographical boundaries of Gladstone City and Calliope Shire prior to the local government amalgamations to take place in March 2008. This area is referred to throughout the project as the Gladstone area.

The health survey component of the project has identified “key” air pollutants; substances for which significant exposure could be associated with adverse health outcomes. The breadth of substances identified as key pollutants is designed to assess multiple aspects of the air quality of the Gladstone area and encompass a wide range of potential adverse health outcomes.

During the course of the project, information will be obtained through the expanded air monitoring program regarding actual ambient air levels of the key pollutants. This will enable an appropriate assessment of actual exposure to these pollutants within the community and enable a good health risk assessment for the inhabitants of the area regarding air quality.

The Environmental Protection Agency (EPA) prepared a list of emissions to air in the Gladstone area. Fifty-four substances from the 19 major industrial sources were identified through the National Pollutant Inventory (NPI); a further 133 substances were identified as emissions or potential emissions from other industrial activity. The total list of 187 emissions included 22 metals and metal compounds, 145 organic substances and 20 inorganic substances.

An expert group was formed to review this list, with expertise in the following areas:

- environmental analysis and monitoring
- analytical chemistry
- environmental health and toxicology
- public health medicine
- health risk assessment

2.2 Prioritisation

Pollutants were assessed and prioritised according to the following criteria:

1) The availability of good quality toxicological and scientific evidence. The study will be informed by ongoing review of scientific literature and toxicological data identifying potential adverse health outcomes associated with each relevant pollutant. The evidence is weighted according to its source (see Figure 1 below).

2) The potential for significant exposure. This was based on existing sources of emission data (via the National Pollutant Inventory [NPI] and other sources). The study will progressively be informed by data from the enhanced air monitoring program at 6 and 12 month points.

3) The availability of ambient air standards against which results of monitoring could be compared. These standards are based on extensive reviews of available toxicological and exposure data and provide an independent risk assessment. Priority was given to Australian standards, then in order of preference: international (WHO), USA, Canadian and Californian standards. However, the absence of a standard was not a criterion for exclusion of a substance from the key pollutant list.

4) The potential for significant variations in exposure, which would include elevated exposures that may be of public health concern. This study focuses on the variety of operating conditions under which the Gladstone industries usually function. The health risks associated with emissions from industrial accidents and major departures from normal operating procedures have already been raised as a separate parallel issue.

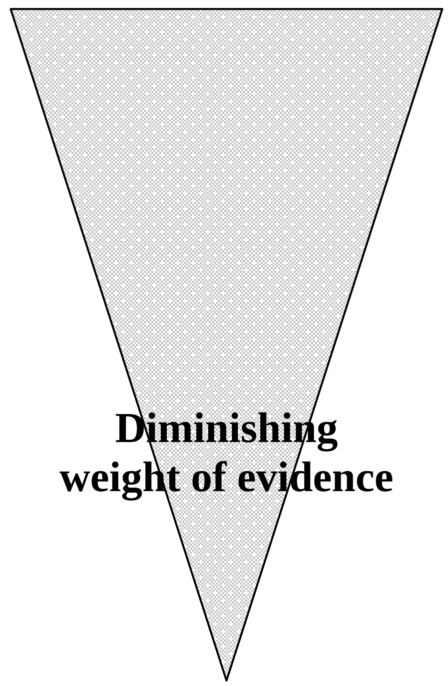
5) Whether the substance may be a representative marker for exposure to a range of other substances. Example 1 - ozone as an indicator of photochemical oxidants; example 2 – if heavy metals were detected in ambient air in unusual quantities, it would raise the possibility of other pollutants being present.

6) The ready availability of validated analytical techniques for measurement.

7) The potential level of community concern.

Figure 1. Weighing the evidence: how toxicological and scientific sources were ranked as sources of evidence to inform the project

Evidence hierarchy utilised throughout the project
Reviews, standards, guidelines and publications by World Health Organization
Reviews, standards, guidelines and publications by Australian agencies such as National Health and Medical Research Council, National Industrial Chemicals Notification and Assessment Scheme
Reviews, standards, guidelines and publications by overseas agencies especially those in North America and Europe
Multiple peer-reviewed published studies
Single peer-reviewed published studies
Peer-reviewed expert opinion and anecdote
Non-peer-reviewed documents



Some substances were excluded because of lack of credible toxicological data and an absence of air standards to enable a useful interpretation of results. Some substances were excluded because of their normal role in human health (eg as essential trace elements) and because there was no evidence to suggest they were likely to be present in high levels (eg copper). However, to ensure broad representation, some essential trace elements (eg zinc) were retained in the list. Note that many substances not included in the key pollutant inventory will be included in the air monitoring program – refer to the full list in the EPA proposed monitoring document.

The study will consider the broader context of exposure to pollutants, retaining the focus on the potential impact of air emissions on public health. As discussed above, actual exposure of the community to the key pollutants through ambient air will be assessed through:

- validation of the NPI data
- direct measurement of most of the identified key pollutants through the EPA's enhanced Gladstone air monitoring program
- modelling of the Gladstone airshed.

For many of the substances included in the key pollutant inventory, at emission levels reported in the NPI, the risk of adverse health effects in the community due to exposure through ambient air is likely to be negligible. Actual data, as collected by the enhanced monitoring program, will enable better understanding of the potential for actual exposure to the key pollutants. Mid-term results from the air monitoring program will be reviewed, and if these indicate a requirement to undertake further health assessments or changes to the monitoring or list of key pollutants, this will be done.

2.3 Air quality standards

The intent of the National Environment Protection (Ambient Air Quality) Measure (NEPM) was to establish standards for air quality that is protective of population health. The air quality standards in the NEPM provide nationally consistent benchmarks to assess air quality. Consistent with international trends, the NEPM is currently under review: this review includes consideration of the exposure-reduction approach to air quality as well as broadening and refining the actual measures.

2.4 Process for review

The proposed key pollutant inventory and preliminary health measures will be presented to the steering groups on 6 February 2008, and forwarded to the external independent reviewer Professor Brian Priestley. There will be a six week period for review, with adjustments as necessary during that time. Discussion on feedback and endorsement of the health measures will be sought at a reference group meeting in March.

3. Key Pollutants

3.1 CRITERIA GASEOUS POLLUTANTS

Oxides of nitrogen

Oxides of nitrogen are gases composed of nitrogen and oxygen. This group includes nitric oxide (NO), nitrogen dioxide (NO_2), nitrogen trioxide (NO_3), dinitrogen trioxide (N_2O_3), dinitrogen tetroxide (N_2O_4) and dinitrogen pentoxide (N_2O_5) . Oxides of nitrogen are part of the continuous cycling of nitrogen and are found in air, soil and water. In the air, oxides of nitrogen are rapidly converted to nitrogen dioxide (NO_2), a pungent gas. Sunlight enables conversion of nitrogen dioxide to nitric acid; excessive levels of nitric acid can lower the pH of rain to form acid rain. Photochemical reactions between nitrogen dioxide, volatile organic compounds and oxygen in the presence of sunlight lead to the formation of ozone and other photochemical oxidants.

Oxides of nitrogen are produced during combustion of wood, fossil fuels and tobacco products. Oxides of nitrogen are present in the exhausts of all vehicles. Nitrogen dioxide is produced for the manufacture of nitric acid, used in the manufacture of fertilisers and explosives.

Low levels of oxides of nitrogen can irritate the eyes and respiratory tract. Nitrogen dioxide can exacerbate chronic obstructive pulmonary disease and asthma, especially in children. Large population-based studies comparing health outcomes with continuously monitored criteria gaseous pollutants indicate a probable contribution of increases in oxides of nitrogen with increases in daily mortality, increases in hospital admission and emergency room attendance for both respiratory and cardiovascular disease. Nitrogen dioxide may sensitise individuals to the effects of other pollutants and allergens and may increase susceptibility to respiratory infections.

The EPA currently monitors oxides of nitrogen at Targinie and South Gladstone, with results available at www.epa.qld.gov.au/projects/air/

Sulfur dioxide

Sulfur dioxide (SO_2) is a colourless irritant gas with a strong, suffocating odour. Sulfur dioxide is oxidised to sulfur trioxide which reacts with water to produce sulfuric acid, lowering the pH of rain to form acid rain. Sulfur oxides are usually adsorbed onto atmospheric particles, particularly carbonaceous (eg soot) particles, on which these reactions occur.

Sulfur dioxide is released into the atmosphere in large quantities by natural processes, such as the natural decay (by anaerobic bacteria) of vegetation in peat bogs and tidal marshes. It is also produced from the combustion of fossil fuel and wood fuels. It is a major contributor to the production of fine atmospheric particles.

At concentrations of 10 to 50 ppm, sulfur dioxide causes irritation of the eyes and respiratory tract. Prolonged exposure may cause inflammation of the respiratory tract and lung damage. Sulfur dioxide causes muscular constriction of the large airways. This is most noticeable in asthmatics when they exercise, and may be observed in such circumstances at concentrations of 0.2ppm or possibly less. Non-asthmatics are usually not sensitive to concentrations of 1ppm or below.

The EPA currently monitors sulfur dioxide at Targinie and South Gladstone, with results available at www.epa.qld.gov.au/projects/air/

Ozone

Photochemical oxidants are a group of air pollutants formed when strong sunlight produces chemical reactions between organic chemicals and oxides of nitrogen in the air. Ozone accounts for about 90% of the total photochemical oxidants in the air and is used as an index of exposure to this group of pollutants: it is the photochemical of greatest concern to health; it is also the most studied. Other photochemical oxidants produced include aldehydes (eg formaldehyde and acrolein), peroxyacetyl nitrates (PAN), and a myriad of aromatic hydrocarbons, alkenes and reactive radicals. Gaseous hydrocarbons do not have strong health associations. PAN is one of the better known of the minor components of photochemical smog; it is thought to be responsible for much of the eye irritation associated with smog.

In the stratosphere (the higher layers of the earth's atmosphere), ozone has beneficial effects as it helps filter out ultraviolet rays. In the lower atmosphere (troposphere) it is usually found at very low levels (less than 0.05 ppm).

The major organic chemicals that lead to the formation of ozone are volatile organic compounds produces by motor vehicles, industry and bushfires.

Ozone can irritate the lining of the throat and lungs, causing breathing difficulty. It can increase the risk of respiratory infections. Workers occupationally exposed to elevated levels of ozone showed decreases in lung function and chest pain on breathing in. These acute effects, stronger in workers with a history of asthma or wheeze, are reversible. Large population-based studies comparing health outcomes with continuously monitored criteria gaseous pollutants indicate a probable contribution of increases in ozone with increases in daily mortality and hospital admission for both respiratory and cardiovascular disease. Long term exposure to elevated levels of ozone is associated with reduced lung function in adults and children.

Past EPA monitoring of ozone levels indicate extremely low levels in ambient air in Gladstone; at these levels, adverse health effects in the community would not be expected.

Carbon monoxide

Carbon monoxide (CO) is produced by combustion of carbon containing fuels such as petrol, gas, oil, coal and tobacco products. Vehicle exhaust is a major source of carbon monoxide.

Carbon monoxide has no irritant effect on the lungs, but is absorbed directly into the bloodstream where it combines with haemoglobin in red blood cells, impairing the uptake of oxygen in the lungs and the release of oxygen at the tissues. Toxic effects of carbon monoxide are first demonstrated in organs with a high oxygen demand, such as the brain, heart and exercising muscle. Maternal smoking is recognised as a significant cause of reduced birth weight and delays in fetal and neonatal development; much of this impact is through the effects of carbon monoxide.

Inhalation of 200ppm for 2 to 3 hours can cause headache, dizziness and fatigue. Exposure to higher concentrations can cause drowsiness, loss of consciousness and death. Large population-based studies comparing health outcomes with continuously monitored criteria gaseous pollutants indicate a probable contribution of increases in carbon monoxide with increases in daily mortality and hospital admissions due to cardiovascular disease.

Past EPA monitoring of carbon monoxide levels indicate extremely low levels in ambient air. The risk of adverse health effects in the community due to exposure to ozone in ambient air is likely to be negligible.

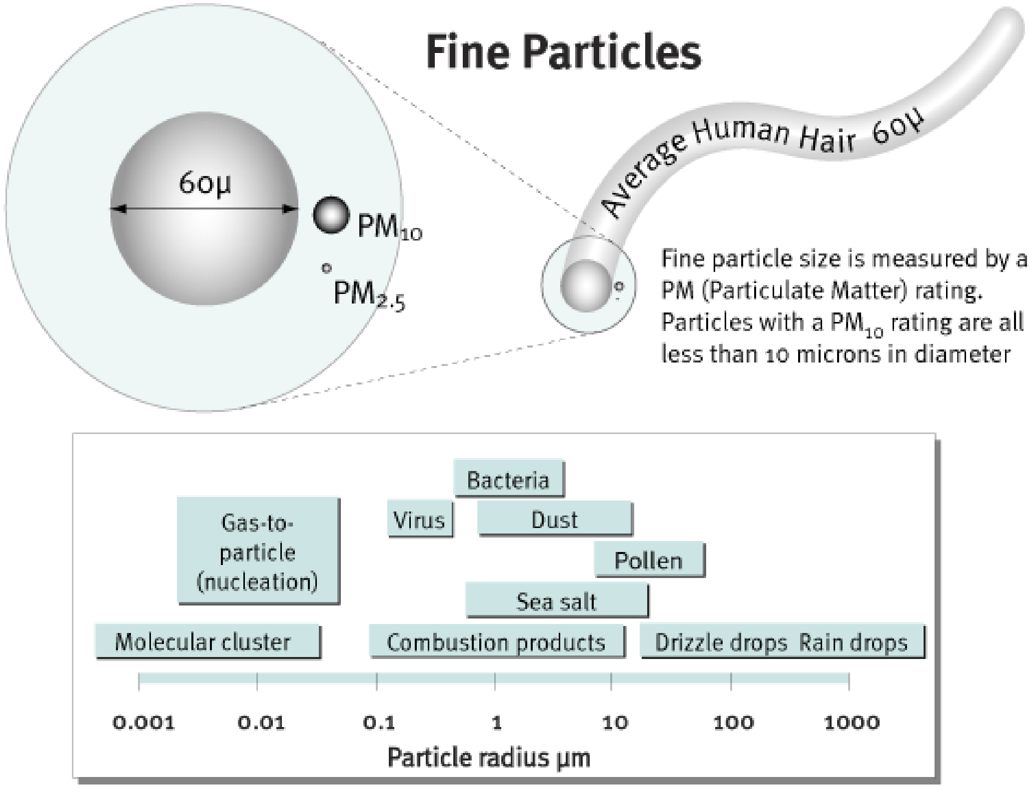
3.2 PARTICULATE MATTER

Particulate matter refers to solid and liquid particles that may be suspended in the air (ie they are not gases). They vary in size, shape and chemical components and are found everywhere in the atmosphere. Particulates are classified by 'mass median aerodynamic diameter' (MMAD), which is a measure of density and shape. Particulates include:

- visibility reducing particles
- total suspended particles (TSP)
- PM_{10}, which is particulate matter with MMAD 10 micrometres or less;
- $PM_{2.5}$, which is particulate matter with MMAD 2.5 micrometres or less (also known as fine particles; and
- PM_1, which is particulate matter with MMAD 1 micrometre or less (also known as ultrafine particles.

Estimated size ranges for various particles are shown in Figure 2. For reference, 1 micrometer (µm) is 1 millionth of a meter or 1 thousandth of a millimetre.

Figure 2. Estimated size ranges for various particles



Source: Environmental Protection Agency

Large particles tend to settle out of the air quicker than smaller particles; rain helps remove particles from the air. The composition of particulate matter is complex and variable. Particles include dust, smoke, combustion products, plant spores, micro-organisms, metals and salt. Composition varies in place and time. The particles may vary in shape, surface area, electrical charge and light scattering properties. PM_{10} are sufficiently small to penetrate to the large airways of the lungs; $PM_{2.5}$ have a high probability of deposition in the smaller conducting airways and alveoli.

Natural sources of particles include bush-fires, dust storms, pollens and sea-spray. Particles are emitted during the burning of fossil fuels (especially diesel-powered vehicles), lawn mowing, and use of wood stoves. Industrial processes that may produce particulate matter include bulk material handling, refineries, cement works and fossil fuel power plants. PM_{10} typically include dust from roads and industrial activities and biological material such as pollen grains and bacterial fragments; $PM_{2.5}$ are largely formed from gases but combustion processes may also generate primary particles in this size range.

A range of studies have linked PM or specific PM components to a range of significant health outcomes:

- increased mortality and hospital admissions for people with chronic obstructive pulmonary disease
- exacerbation of asthma symptoms and increased use of asthma treatments

- increased mortality and hospital admissions for people with cardiovascular disease
- increased mortality and hospital admissions for people with diabetes (which may perhaps be because of cardiovascular effects)
- increased risk for heart attack
- lung inflammation

Most susceptible are children and the elderly, and people with existing conditions such as asthma, chronic obstructive pulmonary disease, cardiovascular disease and pneumonia.

Estimates of the impact of PM_{10} on mortality range from 0.2 to 1.0% increase in all-cause mortality per 10 $\mu g/m^3$ increase in PM_{10}.

Long-term exposure to particulates has also been shown to be associated with reduced lung function. Acute changes are likely to be reversible; long term there is a contribution to decline in lung function.

The pathways by which these occur is not understood. Several hypotheses are under investigation including examination of the roles of metals, organic compounds, oxidants and actual particle size. The roles of particle composition and size are unclear, with adverse effects attributed to both particles originating from the surface of the earth (known as crustal particles) and particles arising from combustion.

In the process of the CHAG project, concerns have been raised about particles smaller than one micrometre, including nanoparticles, which are defined as particles less than 100 nanometres in at least one dimension (ie 0.1 micrometres, or 100 billionths of a metre). There are very limited studies into the human health effects of particles smaller than 1 micrometre.

Studies of nanoparticles have demonstrated that nanoparticles can penetrate deeply into the lung and cause inflammation. This is consistent with current understanding of particulates, although as stated above, the contribution of particles of various sizes to adverse health outcomes of the respiratory and cardiovascular systems is unclear. Some studies of specific substances of nanoparticle size have demonstrated entry into the bloodstream; the mechanism by which the body deals with these substances is the subject of further research. It is also unclear whether toxicologic effects of nanoparticles are related to the number of particles, or the mass concentration of particles of this size. Modelling studies also suggest the human placenta could allow passage of substances of nanoparticle size; this is also an area for further research.

In the past decade, there has been significant growth in technology to design, produce and use a whole range of different substances of nanoparticle size for a range of applications from information technology, cosmetics and energy production, to electronics and medicines. Nanoparticles also include combustion-derived nanoparticles from internal combustion engines, diesel engines, industrial furnaces, and naturally occurring nanoparticles.

Understanding of the potential risks to human health from manufactured nanomaterials has not kept pace with product development. There are significant gaps in knowledge about potential occupational risks associated with nanoparticle manufacture. Globally, many governments have put in place strategies for targeted research and strategy development pertaining to health and safety issues relating to nanoparticles. In Australia, the National Health and Medical Research Council has established an Advisory Committee on Nanotechnology & Health (and Brian Priestly is the invited Chair).

The development of appropriate instruments and methods that can measure nanomaterials and differentiate between different nanomaterials is the subject of research in many countries globally. In addition, there is a widespread lack of evidence of research on human health aspects of nanomaterials – so there is a lack of basic understanding of which characteristics of nanomaterials are toxic to humans and hence what risks may be associated with exposure to nanoparticles.

In summary, whilst particles less than one micrometre in size are of interest, current knowledge hampers the assessment of health risks associated with these particles in ambient air in Gladstone.

The EPA currently monitors PM_{10} at Targinie, Clinton and South Gladstone, with results available at www.epa.qld.gov.au/projects/air/.

Under the National Environment Protection Measure for Ambient Air Quality, standards have been set for PM10. Since 2003, advisory reporting standards have also been set for PM2.5. Currently, there are no ambient air standards anywhere in the world for PM_1 or smaller.

3.3 COAL AND COAL DUST

Coal is the generic term for a diverse group of rock of varying composition and characteristics made from carbonised plant matter. Major constituents are carbon and moisture; with variable amounts of volatile matter including polycyclic aromatic hydrocarbons (PAHs); sulfur; and mineral matter, including quartz. There are numerous forms e.g. soft and hard, brown and black. Typical Bowen Basin coal is hard coal with a carbon content of 86-90%.

Major exposure to coal and coal dust occurs in occupational settings during mining and processing of coal. Exposure occurs also during bulk loading and transfer, and at sites where it is stored and used, such as power stations. Combustion of coal releases oxides of nitrogen, sulfur dioxide, particulate matter, polycyclic aromatic hydrocarbons, volatile organic compounds (VOCs) and a range of metals reflecting the metal constituents of the coal.

In exposed workers, coal dust has long been known to cause coal workers' pneumoconiosis (CWP) and progressive massive fibrosis of the lungs. Key factors that influence the risk of these outcomes include intensity of exposure (dust level inhaled) and duration (years) of exposure. CWP can be simple or complicated. In simple CWP, there is collection of dust in the lungs which shows as small opaque spots (typically less than 1 cm diameter) on chest X-ray. In complicated CWP, these opaque spots are much larger and can coalesce such that there appears a mass of black tissue in the lungs. This may be adherent to the chest wall. Complicated CWP is associated with decreased lung function, causing breathlessness. These types of effect have been associated with premature death. Occupational exposure to coal dust also has been associated with chronic bronchitis (chronic cough with excess sputum production) and focal emphysema. Coal dust with a quartz content of more than 5% is thought to be toxicologically similar to quartz exposure in other mining and similar situations e.g. hard rock mining. Substantial exposure to respirable quartz is associated with the form of lung fibrosis termed silicosis.

The International Agency for Research on Cancer (IARC) has classed coal dust in Group 3, which means it cannot be classified as to its carcinogenicity in humans. There is inadequate evidence from human epidemiological studies and experimental animal studies to implicate coal dust as a cancer-causing agent. Most studies have focussed on cancers of the lung and stomach, but also other sites such as the urinary bladder. There is no consistent evidence of increased risk of cancer among coal-exposed workers, and studies have not been able to demonstrate a consistent dose-response relationship.

The adverse health effects described above have been associated with long-term occupational exposure to respirable coal dust at dust concentrations in inhaled air of 1 to 10 mg/m^3. The respirable fraction of any dust is that with a mass median aerodynamic diameter of less than 4.25 µm (micrometers). The respirable fraction of dust (that which can penetrate deep into the lungs) is captured within the PM_{10} fraction, which is a routine component of ambient air quality monitoring in Gladstone and elsewhere. However, in the ambient environment of Gladstone, it can be anticipated that coal dust would constitute only a proportion of the PM_{10} fraction given other contributing sources described elsewhere in this paper. Excluding dust storms, ambient PM_{10} levels in Gladstone are reported by EPA to be typically in the 10-20 $\mu g/ m^3$ range. These levels are similar to other locations in Queensland where monitoring is conducted. Thus, it can be anticipated that ambient exposure to respirable coal dust in Gladstone would be in the order of at least 2 orders of magnitude (100 times) below levels associated with known adverse effects from occupational studies. At such levels, you would not expect the development of lung diseases that are seen in occupationally exposed coal workers.

Studies of health impacts of community exposure to coal dust appear to be few in number. A study published in 2001 (Pless-Mulloli, Howel & Prince) into the prevalence of asthma and respiratory symptoms in children living near opencast mining sites in Northern England was reviewed. It showed little evidence of an association between residential proximity to coal mining sites and cumulative or period prevalence of respiratory illness or asthma severity. Dust exposure was measured as PM_{10} with levels in the order of 17.0 $\mu g/ m^3$ in communities close to opencast mines and 14.9 $\mu g/ m^3$ in communities distant from such mines. These PM_{10} levels are comparable to those reported in Gladstone by the EPA.

Potential adverse health impacts of coal dust on the community in Gladstone will be assessed in three ways. First, as coal dust will contribute to particulate matter in ambient air, effects will be assessed through assessment of potential adverse health impacts associated with particulates. Secondly, as combustion of coal will contribute to pollutants including the criteria pollutants (oxides of nitrogen, sulfur dioxide and particulate matter), PAHs, VOCs and metals, effects will be assessed through assessment of potential adverse health impacts associated with those pollutants. Thirdly, for completeness, the study will include data on coal workers' pneumoconiosis (CWP) and other forms of pneumoconiosis in the Gladstone area.

3.4 METALS

Arsenic and Compounds

Arsenic is an element that occurs naturally in the earth's crust in ores and minerals. It is usually found in combination with oxygen, chlorine and sulphur as inorganic arsenic compounds. Organic arsenic compounds are formed when arsenic combines with carbon and hydrogen.

Industrial sources of arsenic in Gladstone include power generation and alumina and cement production. Arsenic is also used in some timber treatments, wood preservatives and pesticides.

No acute exposure effects are expected for communities exposed to ambient air levels. Exposure to high levels of arsenic can cause multiple organ damage and death. Occupational inhalational exposure (eg smelter work) is associated with increased risk of lung cancer. Inhalation of low levels of arsenic over long periods of time can reduce intellectual development in children, cause skin discolouration and lesions, restrict fetal growth and cause fetal malformations or death.

Beryllium and compounds

Beryllium is a naturally occurring element present as compounds in mineral rocks, soil, coal, and oil. It has a lot of uses in production of metals, electronic and ceramic materials and in chemical manufacture. Industrial sources of beryllium in Gladstone include chemical manufacture and power generation. Combustion of petrol may emit beryllium into the air. Beryllium may also be present in tobacco smoke.

No acute exposure effects are expected for communities exposed to ambient air levels. Beryllium may irritate the eyes, and cause acute lung damage (pneumonitis).
Some people exposed to beryllium through occupational exposure become sensitised to it and develop cough, weight loss and weakness. Beryllium is considered to be a probable human carcinogen; occupational exposure appears to be associated with increased risk of lung cancer.

Cadmium and compounds

Cadmium is a naturally occurring element in the crust of the earth. Coal and other fossil fuels contain cadmium and their combustion releases the element into the atmosphere. The main industrial sources of cadmium in Gladstone are alumina refining and coal-fired power generation. Combustion of petrol may emit cadmium into the air. Cadmium is also found in many domestic products including tobacco products, some fertilisers, polyvinyl chloride (PVC) products, textiles, and heating elements.

No acute exposure effects are expected for communities exposed to ambient air levels. Acute exposure to cadmium may be toxic to the kidneys (renal tubular and glomerular dysfunction). Cadmium is considered to be a probable human carcinogen, with evidence of it causing lung and possibly prostate cancer in humans. Large inhaled doses damage the respiratory tract; lower doses can cause renal damage, fetal abnormalities and anaemia.

Chromium III and chromium VI compounds

Chromium is a common, naturally occurring element in rocks, soil, plants, animals, dust, gases, oil and coal. In nature, chromium is mostly in the form of chromium (III) and rarely as chromium (VI). Chromium has a wide range of uses in industry including as an alloy to harden steel, in chrome plating, in dyes and pigments, in fungicides and wood preservatives and as a chemical catalyst. Chromium may be emitted to air on combustion of oil and coal.

The major sources of chromium emissions to air in Gladstone are alumina production and electricity generation.

Chromium III is essential for health, but exposure to high levels for prolonged periods may aggravate asthma and cause skin and respiratory tract irritation.

Inhalation of chromium VI compounds may cause irritation to the respiratory tract and aggravate asthma and cause kidney and liver damage. Chromium VI is a recognised human carcinogen, with occupational exposure associated with an increased risk of lung cancer.

Potentially harmful exposure to chromium VI can occur in occupational exposure in welding and chrome plating industries.

Lead and compounds

Lead occurs naturally in the earth's crust and is found in combined form in minerals such as galena. Lead adheres to soil. Lead is used in the production of electronic parts, plastics, rubbers and metals, batteries, metal products and ceramics, and was used in leaded fuels in Queensland until 1 January 2002. The lead contents in paints in Queensland was reduced to 0.1% in December 1977, except for some industrial coatings and specialised paints.

The major industry sources of lead in Gladstone are chemical manufacture, and cement and alumina production. Lead may also be released from bushfires and on combustion of fuels.

Lead can affect most organs in the human body. Exposure to significant levels of lead can affect the brain and kidneys, cause miscarriage, reduce fertility in men, affect fetal development, reduce intellectual development in infants and children and cause learning difficulties and reduced growth.

Manganese and compounds

Manganese is a metal that occurs naturally in the environment in a wide range of forms. It is mainly used in the production of steel and non-ferrous alloys and has a wide range of applications including porcelains, ceramics, dyes, fungicides, disinfectants and food additives. The major industry source of manganese in Gladstone is alumina refining.

Manganese is essential for health. Chronic inhalation of significant quantities (usually as an occupational exposure) can cause reduced fertility in males, inflammation of the respiratory tract and disorders of the nervous system including hallucinations and behavioural disturbances.

Mercury and compounds

Mercury is a naturally occurring element found in rocks and ores. Mercury is released into the atmosphere by evaporation from soils and through the burning of fossil fuels (coal, oil, petrol, asphalt etc). Mercury is used in batteries and lights, especially outdoor or powerful lights. It is used to assist ore extraction, and in the chemical manufacturing industry. The major industry source of mercury in Gladstone is alumina production.

Exposure to high levels of mercury can cause eye and respiratory irritation, damage the brain and kidneys and reduce fertility.

Nickel and compounds

Nickel is an abundant element found in soils, waters and foods and in the seabed. It is used in metal alloys for a wide range of purposes including coin and jewellery, batteries and electrodes. Combustion of coal and other fossil fuels leads to the release of nickel into the atmosphere. Nickel is also present in tobacco smoke. The major industry source of nickel in Gladstone is associated with alumina production.

Nickel is essential for health as an essential trace element. Skin sensitivity to nickel is common (jewellery and watches); respiratory sensitivity to nickel is less common and may cause asthma attacks. Nickel dust (seen in occupational exposures) is an irritant to eyes and respiratory tract and is a probable carcinogen (cancer of the nose and lungs).

Zinc and compounds

Zinc is an abundant metal that occurs naturally in the environment in a wide range of forms. It is present in foods. It is widely used as a protective coating for iron and steel, and for the production of zinc alloys eg brass. It is also used in a range of other products including rubber, chemicals, paints, floor coverings, fabrics, plastics and pharmaceuticals. The major industry sources of zinc in Gladstone are alumina and cement production.

Zinc is essential for health. Zinc dust is an irritant to the eyes and respiratory tract. Inhalation of zinc fumes in occupational settings can cause mild to severe respiratory tract irritation (metal fume fever, a transient flu-like illness). Some zinc compounds are harmful because of other harmful constituents eg chromium, cyanide.

3.5 VOLATILE ORGANIC COMPOUNDS (VOCs)

Volatile organic compounds are a wide range of organic chemical compounds which, because of their volatility, are usually present at ambient temperatures as a vapour or gas. VOCs are produced by a wide range of biological and industrial processes. VOCs are a known cause of photochemical smog and they react with oxides of nitrogen to produce ozone.

Benzene

Benzene (C_6H_6) is a colourless aromatic chemical. It occurs naturally in fossil fuels and is released as vapour from fuels. It is released during combustion of organic matter such as wood, coal, tobacco and petroleum products. Petrol vehicles are a significant source of benzene. Some furnishings, solvents and adhesives also contain benzene and can contribute to exposure when used indoors. The major industry sources of benzene in Gladstone are alumina and cement production.

Benzene may irritate the eyes, skin and respiratory tract. In the past, several groups of workers in the shoemaking, leather, rubber, adhesive and chemical industries were exposed to high concentrations of benzene. Occupational studies of these groups identified that benzene depresses the bone marrow (causing reduced bone marrow production). Benzene is a recognised human carcinogen, associated with an increased risk of acute myeloid leukaemia. Benzene has been linked with birth defects and low birth weight.

Carbon tetrachloride

Carbon tetrachloride is a manufactured chemical, produced as a clear liquid that evaporates very easily. Most carbon tetrachloride that escapes to the environment is found as a sweet-smelling gas. It does not occur naturally in the environment.

Carbon tetrachloride was produced in large quantities to make refrigeration fluid and propellants for aerosol cans. Since many refrigerants and aerosol propellants have been found to affect the earth's ozone layer, the production of these chemicals is being phased out. Consequently, the manufacture and use of carbon tetrachloride has declined a great deal. Carbon tetrachloride was also widely used as a cleaning fluid, in fire extinguishers and as a fumigant. Most of these uses were discontinued in the mid-1960s.

Most information on the health effects of carbon tetrachloride in humans comes from studies where people have been exposed to relatively high levels of carbon tetrachloride, either only once or for a short period, for example, by accidental poisoning or by working with the chemical in a confined space without ventilation. Experiments have not been performed on the effects of long-term exposure of humans to low levels of carbon tetrachloride, so the human health effects of such exposures are not known.

Exposure to relatively high levels of carbon tetrachloride can cause damage or destruction of liver and kidney cells, leading to a decrease in liver and kidney function. Such effects are reversible unless exposure is very high. Exposure to high levels of carbon tetrachloride can also impair the brain and cause headache, dizziness, and drowsiness, leading to coma or permanent damage to nerve cells.

Ethylbenzene

Ethylbenzene (C_8H_{10}) is a colourless chemical. It occurs naturally in fossil fuels and is released as a vapour from fuels. It is released during combustion of organic matter such as tobacco, coal and petroleum products. It is used as a solvent; is also present in a range of household products including paints, cleaning products, herbicides, sealants and flooring. The major industry source of ethylbenzene in Gladstone is petroleum handling.

Ethylbenzene may irritate the eyes, skin and respiratory tract. High concentrations cause loss of consciousness; chronic exposure can cause damage to the liver.

Methyl ethyl ketone

Methyl ethyl ketone (C_4H_8O) is a colourless chemical with a faint sweet odour. It is released during bush fires and during biological degradation, is present in tobacco smoke and in some foods. It is used or produced in the chemical industry, rubber manufacture, pharmaceutical industry, and in the manufacture of wood products, paints, inks and varnishes. The major industry source of methyl ethyl ketone in Gladstone is waste disposal.

Short term exposure causes drowsiness and can lead to loss of consciousness. Methyl ethyl ketone may irritate the eyes, skin and respiratory tract.

According to the NPI, small quantities of methyl ethyl ketone are emitted into the Gladstone airshed. The risk of adverse health effects in the community due to exposure through the airshed is likely to be negligible.

Methyl isobutyl ketone

Methyl isobutyl ketone ($C_6H_{12}O$) is a colourless chemical with a faint camphor-like odour. In air it breaks down quickly into acetone, formaldehyde and 2-methylpropanal.

Methyl isobutyl ketone is used or produced in the chemical, rubber and pharmaceutical industries, and in the manufacture of wood products and varnishes. It may be present in paints, dry cleaning products and other consumer products. It occurs naturally in some fruits and is present in vinegar. The major industry source of methyl isobutyl ketone in Gladstone is waste disposal.

Human exposures tend to occur through use of products containing methyl isobutyl ketone in confined spaces (eg painting in a small enclosed area). The effects include drowsiness and dizziness. Methyl isobutyl ketone may irritate the eyes, skin and respiratory tract.

Toluene

Toluene (C_7H_8) is a clear chemical with a sweet smell. It breaks down in the air to other chemicals including benzaldehyde and cresol.

Toluene is a component of petrol. Toluene is present in tobacco smoke and the vapours of petrol, some glues and paints and nail varnishes. Toluene is released to the air from bush fires. According to the NPI, the major industrial source of toluene is alumina production.

Inhalation of toluene can produce light-headedness and euphoria, dizziness, drowsiness and loss of consciousness. Long term exposure to high quantities can lead to brain damage. Long term exposures to low levels can damage the kidneys.

1,1,2-Trichloroethane

1,1,2 – trichloroethane ($C_2H_3Cl_3$) is a colourless chemical with a sweet smell. It is used as a solvent, and in the production of adhesives, lacquers and coatings. It does not occur naturally in the environment.

1,1,2 – trichloroethane may irritate the eyes, skin and respiratory tract. Exposures in confined or poorly ventilated spaces can lead to dizziness, drowsiness and loss of consciousness. Long term exposures can cause liver and kidney problems.

Trichloroethylene

Trichloroethylene (C_2HCl_3) is a colourless chemical with a sweet smell. It is used as a solvent and in the production of pharmaceuticals, flame retardants and insecticides. It does not occur naturally in the environment.

Trichloroethylene may irritate the eyes, skin and respiratory tract. Exposures in confined or poorly ventilated spaces can lead to dizziness, drowsiness and loss of consciousness. Long term exposures can cause kidney problems and immune system depression. The effects of trichloroethylene are enhanced by consumption of alcohol. Trichloroethylene is classified as a probable human carcinogen (liver and lungs).

Xylenes

Xylene (C_8H_{10}) is a colourless chemical with a strong sweet smell. There are three xylenes, with the same chemical formula but slightly different chemical structure. Xylene is used as a solvent, in petrol manufacture and to make dyes, paints and insecticides. Xylene occurs naturally in petroleum and is released from the environment in bush fires.

Xylene may irritate the eyes, skin and respiratory tract. Exposures in confined or poorly ventilated spaces can lead to dizziness, drowsiness and loss of consciousness. Long term exposures can damage the bone marrow. Xylene may damage a developing fetus.

3.6 CARBONYL COMPOUNDS

Acetaldehyde

Acetaldehyde (C_2H_4O) is a colourless compound with a fruity odour. It has widespread natural occurrence, occurring in the respiration of plants and the ripening of fruit, and is present in tobacco, some vegetables and herbs. It is also a product of the fermentation or metabolism in the body of alcohol and sugars. It is released into the environment during bush fires and agricultural burning and on combustion of fossil fuels. Acetaldehyde is also produced by photochemical oxidation of other compounds in the air. According to the NPI, the major industrial source of acetaldehyde in Gladstone is associated with alumina production.

Acetaldehyde is an irritant of the skin, eyes and respiratory tract.

Acrolein

Acrolein (C_3H_4O) is a colourless chemical with a sweet pungent odour. It is considered a volatile organic compound by the NPI. Acrolein can enter the environment as a result of burning trees, fuel or tobacco. It is also produced by photochemical oxidation of hydrocarbons in the atmosphere. Acrolein decomposes quickly in the air by reacting with other chemicals or exposure to sunlight.

Acrolein may irritate the eyes, skin and respiratory tract. Inhalation can contribute to chronic respiratory disease.

Formaldehyde

Formaldehyde (CH_2O) is a colourless chemical with a pungent odour that is detectable at very low levels (1 ppm). It is released during combustion of organic matter such as tobacco, coal and petroleum products, and it is produced by photochemical oxidation of hydrocarbons in the atmosphere. It is used in a range of industries including wood-products, adhesives and carpets and is present in a wide range of personal care and consumer products. It is present in many indoor environments as a result of "gassing off" from materials such as plywood, furniture and insulation. According to the NPI, the major industrial sources of formaldehyde in Gladstone are associated with alumina production and chemical manufacture.

Formaldehyde can also be formed in the atmosphere as a result of the photochemical oxidation of reactive organic gases; formaldehyde breaks down in air to form formic acid and carbon monoxide.

Formaldehyde may irritate the eyes, skin and respiratory tract and can cause an asthma-like respiratory allergy. It is considered a carcinogen, with evidence it is associated with cancer of the nose and pharynx.

3.7 POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

Polycyclic aromatic hydrocarbons are a group of more than 100 carbon-based chemicals which contain at least two benzene (six sided) rings. PAHs are formed by the incomplete combustion of coal, oil, petrol, wood, tobacco products, garbage and other organic materials. PAHs are also produced by char-grilling meats. A few PAHs are used in medicines and to make dyes, plastics and pesticides (eg naphthalene). Anthracene is used in dyes, insecticides and wood preservatives. Most PAHs have no known use. According to the NPI, the major industrial source of PAHs is aluminium smelting.

Benzo[a]pyrene is the best studied and one of the most toxic of the PAHs. However, benzo[a]pyrene is usually found in combination with a large number of other PAHs and can be used as an indicator for this group of air pollutants. There is little information on human exposure to any single, pure PAH.

PAHs may irritate the eyes, skin and respiratory tract. Acute exposure to high levels may cause headaches, nausea, vomiting and abdominal pain. A number of PAHs are classified as probably and possibly carcinogenic to humans and benzo[a]pyrene is classified as a carcinogen. It is associated with lung cancers, with the risk increasing in the presence of other substances such as tobacco smoke.

3.8 ACIDIC / CAUSTIC AEROSOLS

Acidic gases

As there have been complaints about acidic and caustic burns in the South Gladstone area in particular, air monitoring will include measurement of the pH of gases.

Ammonia

Ammonia refers to the mixture of ammonia (NH_3) and the ionised form (NH_4^+). Ammonia is found naturally in air, soil, water, plants and animals and it is released to the air during bush fires. Bacteria in human and animal intestines produce ammonia. Ammonia is present in tobacco smoke. It is used in many household and industrial cleaning and disinfection agents. Ammonia is emitted during the production of chemicals, metals, cement, mining, electricity supply and petroleum refining. According to the NPI, industrial sources of ammonia in Gladstone include power generation, cement and chemical production and aluminium smelting.

Exposure to typical environmental concentrations of ammonia will not affect humans. Exposure to high levels can cause irritation and burns to the skin, eyes and respiratory tract.

Hydrogen sulfide

Hydrogen sulfide (H_2S) is a colourless gas with a characteristic smell of rotten eggs. It can be smelt at low levels. It occurs naturally in crude petroleum, natural gas, volcanic gases, and hot springs. It can also result from bacterial breakdown of organic matter. It is also produced by human and animal wastes. Hydrogen sulfide can also result from industrial activities, such as food processing, paper mills, tanneries, and petroleum refineries. According to the NPI, the major source of hydrogen sulphide in Gladstone is waste disposal.

Low concentrations of hydrogen sulphide may irritate the eyes, nose and throat (5 to 50 ppm).

Sodium hydroxide

Sodium hydroxide (NaOH, also known as caustic soda or lye) is a manufactured alkaline odourless substance. Sodium hydroxide is very corrosive. Sodium hydroxide is used in metal cleaning and processing, oxide coating, electroplating, and electrolytic extracting, and is used to manufacture soaps, some fabrics and petroleum products. It is used in laundering and bleaching and is commonly present in commercial drain and oven cleaners.

Sodium hydroxide separates in water to positively charged sodium atoms and negatively charged oxygen and hydrogen atoms, which decrease the acidity of water.

Sodium hydroxide is very corrosive and can cause severe burns in all tissues that come in contact with it. Inhalation of low levels of sodium hydroxide as dusts, mists or aerosols may cause irritation of the nose, throat, and respiratory airways. Inhalation of higher levels can produce swelling or spasms of the upper airway and inflammation of the lungs.

Skin contact with sodium hydroxide can cause severe burns with deep ulcerations. Pain and irritation are evident within 3 minutes, but contact with dilute solutions may not cause symptoms for several hours. Long-term exposure to sodium hydroxide in the air may lead to ulceration of the nasal passages and chronic skin irritation.

3.9 FLUORIDES

Fluoride compounds all contain fluorine, a naturally occurring element in the earth. Fluorine is a yellow-green gas with a strong, sharp odour. Hydrogen fluoride is colourless with a strong irritating odour.

Hydrogen fluoride arises in the production of aluminium and it is used in the petroleum, chemical and plastics industries.

Small amounts of sodium fluoride help prevent tooth decay. Fluorine or hydrogen fluoride gas can irritate the eyes, skin and respiratory tract. Long term exposures to high levels can damage the kidneys and liver.

3.10 POLYCHLORINATED DIOXINS AND FURANS

Dioxins and furans are a large group of substances belonging to a group of chemicals called halogenated aromatic hydrocarbons. The most well studied member of the group is 2,3,7,8-tetrachloro-*p*-dibenzodioxin which is usually referred to simply as dioxin or 2,3,7,8-TCDD. The principal sources of dioxins and furans are combustion of solid waste, rubber, coal and wood petroleum products; steel manufacture, smelting operations, and the manufacture of chemicals especially those containing chlorines. Dioxins and furans are also produced during chlorinated bleaching processes. Dioxins and furans persist and accumulate in the environment; they can be found in very small quantities in many foods, especially those of animal origin.

Exposure to large amounts of 2,3,7,8-TCDD causes a severe acne-like skin disease (chloracne), liver damage and changes in the metabolism of sugar. 2,3,7,8-TCDD is a recognised human carcinogen (respiratory cancers, non-Hodgkins lymphoma and soft tissue sarcoma).

According to the NPI, minute quantities of 2,3,7,8-TCDD are emitted into the Gladstone airshed. The risk of community exposure through the airshed is likely to be negligible.

3.11 CYANIDE COMPOUNDS

Cyanides are a group of compounds based on a carbon-nitrogen structure. Cyanides are usually present as combinations with other chemicals. Hydrogen cyanide and the common cyanide salts (calcium cyanide, potassium cyanide and sodium cyanide) have a bitter almond odour, but only about 50% of people can smell it.

Vitamin B12, essential for human health, contains very small amounts of cyanide. Exposure to high levels can cause irritation and burns to the skin, eyes and respiratory tract and can cause harm to the central nervous system, respiratory system, heart and circulation. Long term effects to low levels may cause problems with the thyroid gland and problems with hearing and vision.

3.12 CARBONYL SULFIDE

Carbonyl sulfide (OCS) is a colourless gas with an unpleasant odour. It is the major sulfur group that occurs naturally in the atmosphere (from volcanoes, deep sea vents, swamps and marshes) and has an important role in the global sulfur cycle. It is present is foodstuffs such as cheese, the cabbage family of vegetables, seeds and grains. It is produced by the combustion of fossil-based fuels, plastics and refuse and during the manufacture of rubber.

Carbonyl sulfide is broken down to hydrogen sulphide and carbon dioxide.

Carbonyl sulfide can cause irritation to the skin, eyes and respiratory tract. High levels of inhalational exposure can cause respiratory paralysis.

3.13 RADIONUCLIDES

Radionuclides will be included in the air monitoring plan for Gladstone for completeness. Exposure to radionuclides from ambient air leading to toxic effects is considered remote.

4. Health outcomes discussion paper

Having established a list of key pollutants, the next step of the project is to review existing toxicological and scientific evidence to determine what adverse health outcomes may be associated with significant exposures to those pollutants. Evidence was reviewed according to the hierarchy in Figure 1. The list of proposed health outcomes for analysis in the study is included as Figure 4.

4.1 Review of the literature

Possible relationships between exposure to a pollutant and a health effect observed in an individual or a population can be studied in several ways. Many adverse health outcomes associated with exposure to a substance have been identified through studies of disease in occupational groups. Toxicological studies often use animals to investigate health effects from exposures to chemicals; these may or may not be appropriately extrapolated to humans. Occupational and toxicological literature is the foundation for available health risk assessments of pollutants. In recent years, studies called time-series studies have compared exposures to pollutants with measures of health outcomes, adjusting for other exposures such as tobacco smoking. Large populations (millions) are usually required. These studies try to determine the contribution of pollutants to adverse outcomes – for example, they may estimate that within a population, 0.5% of deaths may be due to peaks in particulates or criteria gaseous pollutants.

To help evaluate whether an exposure contributes to or causes a health outcome, the following factors are usually considered:

- Is there a strong association? If not, it can be very difficult to identify a true causal effect.
- Is the association consistent (ie made repeatedly in different settings)?
- Has the association been tested by a true experiment, or has the evidence been gained from observation alone? Evidence from true experiments is most compelling.
- Is there a dose effect? Do higher levels of exposure cause a greater level of effect? Again, this is compelling.
- Is there an association in time? Exposure to the environmental cause must precede the development of disease. In addition, for some diseases (eg many cancers) there is a long lead time before disease emerges.
- Is it plausible and does it fit with our understanding of disease?
- Is there similarity to a previously established relationship? For example, it is plausible that diesel particle pollution could cause lung cancer because it contains many of the same polycyclic aromatic hydrocarbons as cigarette smoke.

These factors have been considered in review of the literature to inform the selection of health outcomes.

4.2 What constitutes an "adverse health outcome"?

The World Health Organization defines health as "a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity". An adverse health effect can be surmised to mean anything than departs from this picture of health. For the purpose of the Clean and Healthy Air for Gladstone project, specific and measurable health effects need to be established, that focus on the objective of the study regarding assessment of potential adverse health outcomes associated with air quality in Gladstone.

Adverse health effects may include:

- Death. The 'pea-soup' fogs in London in 1952 convincingly demonstrated an increase in deaths associated with pollution.
- Clinical outcomes, such as emergency department visits or hospital admissions for heart attack, or for exacerbations of asthma or chronic obstructive pulmonary disease, or data on cancer incidence.
- Symptoms, such as eye irritation, sneezing, cough or shortness of breath.
- Impacts on functioning, for example, impairment of lung function has been associated with exposure to several air pollutants.
- Impact on quality of life. Validated questionnaires that are sensitive to the impact of environmental factors are available.
- Biomarkers, for example elevated lead levels in blood. These studies may be used to investigate high exposures.

From the outset, it is recognised that subtle effects of air pollution that may not have caused obvious disease are unlikely to be detected by the study. Furthermore, the relatively small population size in the Gladstone area may limit interpretation of some health data. However, the project team will work to draw the maximum amount of useful information from the study.

4.3 The complexity of disease causation

Many health outcomes of interest to the study have complex and multifactorial causal pathways, which are then impacted further by age, sex, diet, family traits, lifestyle, and state of health (see Figure 3). The study will identify and measure a range of health outcomes. Whilst these health outcomes have been selected on the basis that they may be potentially associated with exposure to pollutants, they also occur under a range of other conditions. The study will therefore have to establish whether these health outcomes occur at a higher rate in Gladstone compared with the Queensland average or a comparison city; and if so, whether pollutants from industry may be a contributory factor. The choice of comparison city needs to consider factors such as population size and characteristics, climate, proximity to the coast and sources of pollution. A city such as Bundaberg is proposed.

Air monitoring data, reported at 6 and 12 months after commencement of the expanded air monitoring program associated with this project, will assist with interpretation of health results. This process will take into account factors typically considered when determining possible adverse health outcomes of environmental exposures such as the dose (how much) and the duration (how long). As a further complication, data from existing datasets will not contain detailed histories of occupational exposure.

Figure 3. Determinants of individual and population health*

General background factors	**Socioeconomic characteristics**	**Health behaviours**	**Individual makeup**	**Biomedical factors**
Culture Resources	Education Employment Income Family Neighbourhood Access to services Population mobility	Diet Physical activity Tobacco use Alcohol consumption Vaccination Sexual practices Use of illicit drugs	Physical and psychological makeup Family influences on behaviour Genetics Ageing Life events	Body weight Blood pressure Blood cholesterol Immune status
Environmental factors		**Psychological effects**		
Landscape Climate Chemical Human-made				

* Adapted from *Australia's Health 2006*, Australian Institute of Health and Welfare

4.4 Addressing sensitive sub-populations

The presence of sensitive sub-populations within the general population will be addressed by considering the potential adverse health impacts on children, older persons and pregnant women. Outcomes in people with existing chronic conditions such as chronic obstructive pulmonary disease, asthma and cardiovascular disease will also be evaluated where possible.

4.5 Measures

Adverse health effects may include effects of short-term, high level exposures, or effects associated with long term exposures. The study aims to include measures of both, although the relatively small population may make interpretation difficult.

Data reflecting short term exposures may include day to day variations in mortality, hospital admissions, hospital emergency department visits, GP visits and variations in symptoms. If practicable, comparison will be made with air monitoring data.

Data reflecting long term exposures may include mortality (in comparison with other areas and the state and trends over time), prevalence of diseases (in comparison with other areas and with the state) or prevalence of symptoms (in comparison with other areas).

Figure 4. Proposed health outcomes for analysis

Condition	Events	Data Sources
Chronic Obstructive Pulmonary Disease	Mortality Emergency Department (ED) presentations Hospital Admissions Non-hospital exacerbations	Deaths Registry ED Information System Hospital separation data Community survey
Asthma	Mortality ED presentations Hospital Admissions Non-hospital exacerbations Lung function	Deaths Registry ED Information System Hospital separation data Community survey (frequency of symptoms, use of treatment) Not feasible
Cardiovascular Disease	Mortality ED presentations Hospital Admissions Angina	Deaths Registry ED Information System Hospital separation data Community survey (frequency of symptoms, use of treatment)
Diabetes	Mortality Hospital Admissions Prevalence	Deaths Registry Hospital separation data Community survey
Respiratory infections: pneumonia, influenza	Hospital Admissions	Hospital separation data
Pneumoconiosis - coal worker's - other forms	Hospital Admissions	Hospital separation data
Cancers - all cancers - lung - stomach - acute myeloid leukaemia - non-Hodgkins lymphoma - liver - prostate - upper respiratory tract cancers - soft tissue sarcoma	Diagnoses, incidence	Cancer Registry
Reproductive outcomes	- stillbirth - birth weight - fetal malformations	Perinatal Dataset
Chemical irritant / allergenic effects	- odour - eye irritation - skin irritation (e.g aggravation of eczema, dermatitis) - respiratory tract irritation	Community survey (frequency of symptoms, use of treatment) Hospital separation data (dermatitis & eczema)

5. Notes on selected conditions

The following has been prepared to provide background information on the health outcomes for analysis, to assist an understanding of what is proposed. It includes some information about how common the disease is, and our current knowledge of risk factors.

5.1 Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is described as limitation of the airflow in the lungs that is not entirely reversible. It includes emphysema, a condition where there is destruction and enlargement of the air sacs in the lungs (alveoli); chronic bronchitis, where there is chronic cough and phlegm; and small airways disease, where there is narrowing of the small airways in the lungs.

COPD is the 5th leading cause of death in males in Australia (2,986 deaths in 2004, representing 4.4% of all male deaths) and the 7th leading cause of deaths in females (2,213 deaths in 2004, representing 3.5% of all female deaths).

COPD is a serious long-term disease in which people experience progressively worse shortness of breath with activity. The main disease process underlying COPD is emphysema. This occurs as a result of the gradual destruction of lung tissue stimulated by inhaled irritants. The lungs become floppy and less able to move air in and out, thereby limiting the ability of the lungs to exchange gases. People with long-term cough and phlegm are regarded as having chronic bronchitis, a condition due to over-active mucus-producing glands in the large airways. Both emphysema and chronic bronchitis are caused mostly by tobacco smoking, so they often coexist.

Tobacco smoking is by far the most important risk factor for COPD. Worldwide, it is estimated that 82% of deaths due to COPD are attributable to smoking; in Australia this figure is likely to be as high as 95%. Other risk factors for COPD include exposure to passive (environmental) tobacco smoke, indoor and outdoor air pollution, occupational dusts and chemicals, and viral respiratory infections. These risk factors may worsen respiratory symptoms or may contribute to the risk of developing COPD, either independently or in conjunction with tobacco smoking. A small percentage of people with COPD have an inherited deficiency of a specific protein that normally inhibits the action of destructive enzymes in the lungs.

The number of people in Australia affected by COPD is difficult to determine. There are major differences in how the disease is defined (some definitions include asthma). The term COPD is not commonly used in health surveys, with the terms chronic bronchitis and emphysema often used instead. The symptoms of COPD can also be confused with asthma.

Based on self-reports to the 2004–05 National Health Survey, 3.0% of the Australian population, had bronchitis and/or emphysema. Aside from the limitations of self-reports, these numbers are probably underestimates because COPD is usually not diagnosed until it is moderately advanced and begins to restrict a person’s daily activities.

5.2 Asthma

Asthma is a chronic inflammatory disease of airways that is characterised by increased reactiveness of the airways to an array of stimuli. These lead to narrowing of the air passages due to contraction of the airway muscles and swelling of the lining of the airways. There is also increased mucus production. Symptoms of asthma include cough, wheeze and shortness of breath. Asthma is typically episodic, with acute exacerbations interspersed with symptom-free periods.

Asthma is a common condition. The National Health Survey 2004-2005 identified that 10.2% of Australians experience asthma; 9% in males, and 11.5% in females. It rarely causes death.

Allergic asthma is frequently associated with a positive family history of allergic conditions. Asthma has a strong familial component. Asthma can be stimulated by allergens, tobacco smoke, drugs, infections, exercise, emotions, occupational exposures and environmental factors. Air pollutants that can trigger asthma include sulfur dioxide, ozone, nitrogen dioxide and particulates.

Environmental causes are usually related to climatic conditions, such as thermal inversions (usually in winter), that promote the concentration of atmospheric pollutants and antigens. Under these conditions, patients with asthma and other respiratory diseases tend to be more severely affected. The air pollutants known to have this effect are ozone, nitrogen dioxide and sulfur dioxide. All produce greater effects during periods of increased breathing (eg exercise). Events such as bush-fires and pollen releases can also aggravate asthma.

5.3 Cardiovascular Disease

The term cardiovascular disease covers all diseases and conditions of the heart and blood vessels; coronary heart disease, stroke, hypertension, heart failure and peripheral vascular disease are all included in this definition. For the purposes of this study, cardiovascular disease will be limited to the outcomes of interest that have been associated with air pollutants: Ischaemic heart disease, angina and heart attack.

Ischaemic heart disease (IHD, otherwise known as coronary heart disease or CHD) refers to lack of oxygen to heart muscle due to inadequate blood flow. The main underlying causal mechanism in cardiovascular disease is atherosclerosis, a process marked by abnormal build-ups of fat, cholesterol and other substances in the inner lining of the arteries. It is most serious when it leads to a reduced or blocked blood supply to the heart (causing angina or heart attack) or to the brain (causing a stroke).

Ischaemic heart disease presents itself in two major forms: heart attack (often known as acute myocardial infarction or AMI) and angina. A heart attack is a life-threatening event that occurs when a blood vessel supplying the heart itself is suddenly blocked completely, threatening to damage the heart and its functions. Angina is a short episode of chest pain that occurs when the heart has a temporary deficiency in its blood supply due to a severe but incomplete blockage in one of its arteries.

The major preventable risk factors for ischaemic heart disease are:

- tobacco smoking
- high blood cholesterol
- high blood pressure
- diabetes
- insufficient physical activity
- overweight and obesity
- poor nutrition

Large population-based studies have made links between particulates in ambient air and the criteria gaseous pollutants (as described in the key pollutant inventory) and ischaemic heart events. Some international studies suggest the contribution of air pollution to deaths from heart attack could be between 0.6 and 1%.

In the 2004–05 National Health Survey, about 1.7% of respondents indicated they had IHD. Based on these responses, it is estimated that around 214,400 Australians experienced angina. The prevalence of CHD increases with age.

It is estimated that Australia-wide, there are about 50,000 heart attacks each year, with about half being fatal. Males have a higher risk than females.

Ischaemic heart disease is the leading cause of death for both males and females in Australia. In 2004, there were 13,152 deaths in males attributed to IHD (19.2% of all male deaths) and 11,424 deaths in females (17.8% of all female deaths).

5.4 Diabetes

Diabetes mellitus (diabetes) is a metabolic disease in which high blood glucose levels result from defective insulin secretion, insulin action or both. Insulin is a chemical produced by the pancreas controlling uptake of sugars by cells. There are three main types of diabetes. Type 1 diabetes is marked by a total or near-total lack of insulin. It usually develops during childhood. In Type 2 diabetes, insulin levels may be normal but the body is unable to use insulin properly. This type of diabetes usually develops in people aged 45 years and over. Gestational diabetes occurs during pregnancy.

Many factors contribute to the onset and development of diabetes. Type 1 diabetes is believed to be caused by particular biological interactions and exposure to environmental agents among people genetically predisposed to diabetes. Risk factors for Type 2 diabetes include obesity, physical inactivity and poor nutrition, genetic predisposition and ageing. The risk factors for gestational diabetes are mostly similar to those for Type 2 diabetes, especially if the pregnancy is late in the fertile years.

In 2004, 982 new cases of Type 1 diabetes in children aged under 15 years were recorded across Australia. This equates to an annual incidence of 24.6 per 100,000 population (around 1 in 4,000) in that age group. From self-reported data in the 2004–05 National Health Survey, about 699,600 Australians (3.5% of the population) had been diagnosed with diabetes. Of these, 13% reported having Type 1 diabetes and 83% reported having Type 2. Aboriginal and Torres Strait Islander peoples have a significantly higher risk for diabetes than non-Indigenous Australians.

Diabetes is a serious chronic condition which can lead to a variety of complications and premature death. Complications include heart attack, stroke, blockage of arteries in limbs requiring amputation, chronic kidney disease, nerve damage and loss of vision.

5.5 Pneumoconiosis

Coal workers' pneumoconiosis (CWP) is a lung disease generally associated with occupational exposure to coal dust over a long period of time. CWP can be simple or complicated. In simple CWP, there is collection of dust in the lungs which shows as small opaque spots (typically less than 1 cm diameter) on chest X-ray. In complicated CWP, these opaque spots are much larger and can coalesce such that there appears a mass of black tissue in the lungs. This may be adherent to the chest wall. Complicated CWP is associated with decreased lung function, causing breathlessness. These types of effect have been associated with premature death.

5.6 Cancer

Cancer is not one disease. Cancer is a broad term that describes hundreds of different diseases where there is uncontrolled growth and possible spread of abnormal cells throughout the body. Different types of cancer tend to have different known and/or suspected risk factors associated with them so are unlikely to have a common source. Known causes of come cancers are complex, involving interplay of genetics, tobacco smoke, diet, alcohol, sunlight, infections, industrial and environmental exposures and unknown factors. The majority of cancers have a long latency period, which is the period from the time of exposure to when disease becomes obvious.

Lung cancer

Lung cancer refers to cancers of a number of different cell types (eg small cell, squamous cell) that occur in the lungs. Cough, sometimes with bleeding, recurring chest infections and weight loss are usual features. Diagnosis is made through identification of cancer cells in sputum or tissue samples taken by bronchoscopy.

During the 19th century, an excess of lung cancer was observed among miners and some other occupational groups, but lung cancer was a rare disease until the beginning of the twentieth century. An epidemic increase in lung cancer began during the first half of the twentieth century, and its predominant cause, tobacco smoking, was identified in a series of landmark studies beginning in 1950.

In Australia in 2004, lung cancer was the third most common cause of death for men, with 4,733 deaths accounting for 6.9% of all male deaths. For females in that same year, there were 2,531 deaths, being 3.9% of all female deaths and making lung cancer the 6th most common cause of deaths in females. Lung cancer is uncommon in people aged less than 40 years.

The role of air pollution in lung cancer is difficult to assess because most lung cancers are associated with tobacco smoking.

Recognised risk factors for lung cancer include:

- Tobacco. Overall risk depends on average consumption, duration of smoking, time since quitting, age at start, type of tobacco product and inhalation pattern. Exposure to environmental tobacco smoke is identified as a risk factor for non-smokers. Tobacco smoke can also increase the effect of other cancer-producing agents eg asbestos. Tobacco smoke contains about 4,000 different chemicals including a number of known carcinogens (eg arsenic, radio-nuclides, cadmium, vinyl chloride monomer, and polycyclic aromatic hydrocarbons
- Ionising radiation. Atomic bomb survivors and patients treated with radiotherapy for spinal conditions or breast cancer are at moderately increased risk of lung cancer, as are underground miners who are exposed to significant levels of radon.
- Occupation. The risk of lung cancer is increased among workers employed in a number of industries. Asbestos, arsenic, beryllium, cadmium, chromium (VI), nickel, silica, dioxin, radon, vinyl chloride monomer and coal tars have been shown through occupational studies to increase risk of lung cancer.
- An increased risk of lung cancer has been described after tuberculosis.
- Air pollution. Studies of pollution in cities suggest a small increase in risk among people most highly exposed to pollution in ambient air. Indoor air pollution from passive smoking, coal-burning heating devices without proper exhaust emission, and high-temperature cooking using unrefined vegetable oils has been associated with increase in lung cancer risk. A number of known carcinogens, including arsenic, radionuclides, cadmium, vinyl chloride monomer, and polycyclic aromatic hydrocarbons are found in the emissions of any combustion source burning organic materials, including many industries, vehicle exhausts (especially those using diesel) and bushfires.

Upper respiratory tract squamous cell cancers (SCC)

Recognised risk factors include:

- Tobacco. Most cases of upper respiratory tract squamous cell cancers are attributable to tobacco smoking, alcohol drinking, and the interaction between these two factors.
- Occupation. Occupational exposure to mists of strong inorganic acids, sulfuric acid in particular, is an established risk factor for laryngeal cancer. A possible effect has been suggested for other occupation exposures including nickel, asbestos and ionising radiation, but the evidence is not conclusive.
- Infection. Laryngeal papillomatosis, a condition caused by infection with human papillomavirus (HPV) types 6 and 11, the same types that cause genital warts, is suspected to be a pre-cursor to laryngeal SCC.

Cancer of the liver (Hepatocellular carcinoma)

Liver cancers tend to occur in people with underlying chronic liver cell inflammation from a variety of causes. It is not among the leading cancers in Australia.

Risk factors include:

- chronic hepatitis B infection
- chronic hepatitis C infection
- alcoholic liver disease
- haemochromatosis (an iron storage disease)
- toxins (eg aflatoxin B1)

Acute myeloid leukaemia

Acute myeloid leukaemia (AML) is a cancer of the blood and bone marrow. It is the most common type of acute leukaemia in adults. In AML, white blood cell precursors are abnormal and do not develop into healthy white blood cells. Red blood cells and platelets may also be abnormal. Infection, anaemia and easy bleeding may occur. Leukaemia cells may also spread to other parts of the body.

Development of AML

- heredity, radiation, chemical and occupational exposures, and drugs have been implicated in the development of AML
- exposure to benzene is associated with an increased incidence of AML
- anticancer drugs (especially alkylating agents used to treat a variety of cancers) increase risk of AML. Risk peaks 4 to 6 years after exposure
- smoking, especially after the age of 60 years increases the risk of AML
- AML occurs more frequently in males than females

Diagnoses of AML in residents of the Gladstone area 1996 - 2004 was investigated during the investigation into Chronic Lymphocytic Leukaemia (CLL) in 2007. There were 5 people diagnosed with AML in the Gladstone area during the period 1996 - 2004. There was no elevation in comparison with numbers expected according to the state average. These figures will be reviewed in the process of obtaining Cancer Registry data for this study.

CLL is not associated with environmental exposures – refer to the full report available at http://www.health.qld.gov.au/healthieryou/gladstone_chronic.asp. Printed reports are also available in the Gladstone and Calliope libraries.

5.7 Reproductive outcomes

Stillbirth

WHO defines a stillbirth as a fetal death late in pregnancy, and individual countries define the gestational age at which a miscarriage becomes a stillbirth. The perinatal period is defined as 22 weeks or more of gestation (154 days since the first day of the last menstrual period) and ends 7 days after birth. If the gestational age is unknown, it includes infants with a birthweight of 500g or more. In Australia, stillbirths are defined as infants born showing no signs of life in the perinatal period. In developed countries, there is an average of about 1 stillbirth for every 190 deliveries.

In developed countries, maternal risk factors for and causes of stillbirth include:

- congenital or chromosomal abnormalities
- placental abnormalities
- maternal diseases including diabetes, systemic lupus erythematosus, renal disease, thyroid disorders, cholestasis of pregnancy
- hypertensive disease and pre-eclampsia

- congenitally acquired infections including Group B streptococcus, listeriosis, parvovirus B19
- maternal tobacco smoking
- multiple gestation
- lead exposure

Low birth weight

Long term exposures to benzene and particulates have been identified as possible contributory factors to low birth weight.

Low birth weight has been associated with maternal smoking, illicit drug use and alcohol use during pregnancy, poor nutrition, lower genital tract infections during pregnancy, a number of medical conditions and psychological stress.

5.8 Chemical irritant / allergenic effects

Many emissions to air may cause irritation of the eyes, skin and respiratory tract. Establishing the possible contribution of pollutants to irritant symptoms needs to consider the baseline prevalence of conditions that cause similar symptoms.

At least 40% of the population have what might be called an allergic tendency with variable periodic symptoms. In the 2004-2005 National Health Survey, 15% males and 17.2% females reported symptoms of hayfever and allergic rhinitis. The peak age group for these conditions was 15 to 34 years.

Allergic rhinitis is characterised by sneezing, watery discharge, blocked nose, itching of the eyes, nose and throat, and watering eyes, occurring at a time related to exposure to an allergen. Symptoms generally decline with age. Allergens may include weeds, grasses, pollens, animal dander, cockroach-derived proteins, mould spores and dust mites. In up to half of patients with perennial rhinitis, no clear-cut allergen can be demonstrated as causative. The ability of allergens to cause rhinitis rather than lower respiratory symptoms may be attributed to their size and retention within the nose.

Further Reading:

1. The National Pollutant Inventory has a of fact sheets on a large number of pollutants, available at: http://www.npi.gov.au/cgi-bin/npisubstance.pl
2. The EPA has a range of information available on air pollutants and hourly air quality data from monitoring stations in Queenlsand, available at: http://www.epa.qld.gov.au/projects/air/
3. The national standards for criteria air pollutants in Australia are available at: http://www.environment.gov.au/atmosphere/airquality/publications/standards.html#develop
4. For those who are interested in the process of setting standards for ambient air quality, a current (2006) report on an approach to health-based hazard assessment for ambient air quality standards setting is available at: http://www.nhmrc.gov.au/publications/synopses/eh40syn.htm
5. For more information of burden of disease in Australia, Australia's Health 2006 produced by the Australian Institute of Health and Welfare provides discussion on determinants of health and data on a range of health outcomes. Available at: http://www.aihw.gov.au/publications/index.cfm/title/10321

A list of references is available on request.

APPENDIX 1 – LIST OF KEY POLLUTANTS (in alphabetical order)

SUBSTANCE
Acidic/Caustic Aerosols
Ammonia
Acidic vapours
Hydrogen sulfide
Sodium hydroxide
Carbonyl Compounds – refer to monitoring plan for full list to be measured
Acetaldehyde
Acrolein
Formaldehyde
Coal & Coal Dust
Criteria Gaseous Pollutants
Carbon monoxide
Oxides of nitrogen
Ozone
Sulfur dioxide
Fluorides
Hydrogen fluoride and fluoride compounds
Metals – refer to monitoring plan for full list to be measured
Arsenic and compounds
Beryllium and compounds
Cadmium and compounds
Chromium (III) compounds
Chromium (VI) compounds
Lead compounds, inorganic
Lead compounds, organic
Manganese and compounds
Mercury and compounds
Nickel and compounds
Zinc and compounds
Particulate Matter
Total Particulate Matter
PM_{10}
$PM_{2.5}$
PM_1
Polychlorinated biphenyls
Carbonyl sulfide
Cyanides
Polycyclic Aromatic Hydrocarbons (PAHs) – refer to monitoring plan for full list to be measured
Anthracene
Benzo(a)pyrene
Benzo(g,h,i)perylene
Dibenzo(a,h)anthracene
Naphthalene
Phenanthrene
Radionuclides
Internally deposited radionuclides (Uranium/Thorium)
Volatile Organic Compounds (VOCs) – refer to monitoring plan for full list to be measured
Volatile Organic Compounds (total)
Benzene
Carbon tetrachloride
Ethylbenzene
Methyl ethyl ketone
Methyl isobutyl ketone
Toluene
1,1,1-Trichloroethane
Trichloroethylene
Xylenes