

SINGLETON **S**HIRE **H**EALTHY **E**NVIRONMENT **G**ROUP

“Rocky Hill Mine Gloucester SSHEG Submission”



A community-based group looking to address environmental issues affecting Singleton Shire residents

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SSHEG is Not Anti Mining or Anti Power Stations, we seek identification of What is making our Children and Community Sick so they can be mitigated by OH&S Compliance Orders.

SSHEG Focus On Health

This Singleton Shire Healthy Environment Group submission does not support the Rocky Hill Mine Project for four reasons;-

- (1) Recent breakthrough identification says:- short and long term Air Pollution and Particulates Human Exposure (without any lower limit value) are Group1 Carcinogens in 2013 Epidemiology Studies and World Health Organisation announcements. When added to the WHO 2012 same Diesel Exhaust Fume classification, then only much lower Pollution Levels, and not more Mines in Valleys near Residents is acceptable.**
- (2) Singleton Shire Resident's Disease experiences in the last 10 years, especially for Children provides a valuable lesson for Governments, Authorities, Mining Companies when Open Cut Mines are approved to operate beside Residences, villages and towns. Gloucester town, Forbesdale, Thunderbolt and Avon River estates, and Farmlets will all begin to report Diseases just as Singleton began to report in Camberwell, Maison Deiu, Bulga, Warkworth, Farming Families and Ravensworth the town that is no more.**

- (3) **Experience in the Hunter Valley shows Mine Blasting poses the greatest Air Pollution risk for Residents, since from time to time Mine Blast Plumes remain as drifting Hot Gas Bubbles and seem drawn down to ground by rivers and creeks Gassing Residents. Recent incidents in Camberwell, in SE Qld, at Jerrys Plains, and Bulga Mine all tend to require Hospitalisation. However Blast Hole Plastic Stemming Plugs would not only stop the huge Dust and Toxic Gas Plumes from being released, but in the Hunter Valley would dramatically reduce the Brown Smog build up that hangs over the Valley.**
- (4) **Specifically the Health Risk Assessment Part 2B of the EIS needs a rethink in the light of the WHO announcement regarding Air Pollution and Air Particulates. With no lower limit of exposure now identified, all previous EIS and Safe Limit Standards need to be significantly lowered.**

SPECIALIST CONSULTANT STUDIES
Part 2B: Health Risk Assessment

GLOUCESTER RESOURCES LIMITED
Rocky Hill Coal Project
Report No. 806/04

EXECUTIVE SUMMARY

Gloucester Resources Limited (GRL) proposes to develop and operate a small scale open cut coal mine, approximately 3.5km to 7km southeast of the Gloucester urban area, which is located approximately 120km north of Newcastle, New South Wales (NSW) in the Gloucester Basin (the Proposal).

Around 150 Receptors (Gloucester Resident's Homes) are within 700 plus metres of the Mine as evaluated by Toxikos Consultants, not as stated above.

2. AIR QUALITY HEALTH RISK ASSESSMENT METHODOLOGY

The methodology adopted in the conduct of this HRA is consistent with the protocols and guidelines recommended by the enHealth Council. These are detailed in the document "Environmental Health Risk Assessment: Guidelines for assessing human health risks from environmental hazards" (enHealth, 2012).

In the light of October 2013 WHO announcements, these enHealth 2012 guidelines need a rethink, as does this Toxikos Consultancy Report.

This SSHEG Submission has thus been prepared to reinforce the confirmation, that Air Pollution is a Human Disease and Death Risk (Group 1 Carcinogen) for exposed Resident's as released by the World Health Organisation on 17th October 2013.

Most NSW Government Senior Authorities were present, or represented, at the 5 day Symposia and Lectures in Sydney and Newcastle 19th to 25th September 2013, "CAR Air Quality and Health Community Scientific Engagement Forum" where details of Epidemiology and Toxicity Population Studies over many decades were detailed by International Researchers.

The consequential impact of this Pollution – Disease, short and long term associations, with no threshold lower limits is far reaching for Government Authorities, Industry – especially Mining and Power Stations, Diesel Exhaust emissions, and Residents – with a proximity to Air Pollution Emission Sources.

Specifically, the Alarm bells should now be ringing in Government and Industry Circles, just as they have been ringing loud and clear in Communities over the last decade in the Hunter Valley as the Black Coal Industrial Expansion overrun Resident's Health "Checks and Balances".

Recent Government initiatives while focused on the reduction of "Mining Dust" is seen as an attempt to reduce the overall Pollution Dust so new Mines can open and the cumulative impact still fall under the existing inadequate NEPM National Standards.

It is that Coal Mines were approved to operate with Residents as "Near Neighbours", that Occupational Diseases such as "Dusted Lungs and Cardiovascular Diseases" are now being confirmed in Singleton Shire Residents; while not surprisingly Mine Workers with OH&S protection seem immune.

The truth is evident everywhere that Air Pollution is both “Life Shortener and in some cases Life Threatening”.

The WHO Disease associations from Human exposure to Air Pollution and Particulates, reinforces the need to separate Residents from Pollution Emission Sources; Coexistence is to be replaced by Buffer Zone separation, as it was in the past.

The proposition that the Rocky Hill Mine and AGL Coal Seam Gas are proposed to coexist with Residents without any consideration for Buffer Exclusion Zones is exposing the Authorities and Industry leaders to litigation damages as a result of proceeding in full knowledge of the Disease consequences to Residents: a repeat of the Hardy’s Asbestos damages cases.

The Cumulative Pollution Impact on Residents at Gloucester would also include:-

- Disturbing Noise and Machinery Ground Vibrations**
- Mine Blasting Toxic Plumes,**
- Valley Inversion layer concentrations of Blast Dust & Fumes, Mine Dust, Fugitive Coal emissions, Diesel and Gas Exhaust Fumes.**
- and a background of Pollution from expanded Open Cut Mines at Duralie and Strathford just to the south,**
- as well as the AGL Gas Power Unit Exhaust Stack virtually beside the Mine**
- which would combine with any Gas wells leakage as has already been reported in 2007.**

Does the Hunter Valley Air Pollution story provide an insight into the Gloucester Valley impending Demise ?

Four years of investigations into the Disease impact of Air Pollution in the Hunter Valley by the NSW Government Authorities has now unearthed the extent to which Resident's Health is recognised by medical authorities as being affected.

The Gloucester Valley story is beginning to unfold just as the Hunter Valley Open Cut Mining did. Firstly, small, often privately owned mines that limited the "dust" impact on local residents and with an open dialogue approach. Once a foothold for mining was established; enter the multinational Corporations who Upscaled the mining operations to claim the Coal resource no matter what was in the way; usually farming enterprises, villages and towns and their Residents including their livelihoods.

Now in 2013 the Gloucester Valley Coal Resource is up for grabs, but the question is; Will this resource be quarantined as a Rural food bowl or not ?

There is no doubt that the Disease Impact on Residents, especially Children will mirror those reported in the Hunter Valley. After the Epidemiology Study 2013 revelations and the WHO announcements perhaps the communities of the Gloucester Valley will withdraw the "Social Licence that has allowed Mining and CSG Projects to develop to this Stage".

This submission then outlines the experiences in the Hunter Valley as they are considered to relate directly to the Gloucester Valley should these Projects proceed.

The following extract from the SSHEG Document 2010 pages 9 to 13 being Attachment A10 in the SSHEG 2013 Senate submission on Air Quality in Australia” outlines the Impact on Residents from Open Cut Mining.

Why is it that Authorities, Mining Companies and Mine Explosive Companies apparently refuse to use “Blast Hole Plastic Stemming Plugs” that mostly eliminate Dust and Blast Toxic Plumes spewing into the atmosphere ?

Would this be Best Practice Blasting for the Hunter valley?

1.0 SSHEG Review of Major Community Concern

Recent reviews by SSHEG of Camberwell Community Blasting Plume “Gassing” of Residents (Attachment 1), Photographs (Attachment 2) and Videos (Attachment 3) demands immediate cessation of Aston Mining Blasting Practices that release Flame, Gases and Dust, as Blast Plume into the atmosphere; to be by Government Order to change Blasting Practices to the non-release to atmosphere method.

On many occasions in the period 2004 to 2010 the Community Complaints Hotline has recorded numerous Blasting incidents mainly by Aston Mining that has damaged Homes, shaken and displaced Homes on their foundations, “ it knocked tiles off the bathroom walls, glass fell from cupboards and pictures were knocked right off the walls”, moved items on shelves inside the houses, showered Homes with debris, and to date no repairs to these homes of the damages has been carried out by the Mining Companies that surround the Camberwell Village. A particular extreme series of Blasts are recorded on or around 9.20am on 4th July 2007 (perhaps as an Independence Day prank or Licence # 11879).



Photo 1 Camberwell Gassing Incident 2010 type viewing West
*Mine Blast over the Ridge is sucked down to Glennies Creek
and drifts over Camberwell Residents in Bridge Street*

To the casual observer it would appear that in particular Aston Mining set about in 2004 to clear the Residents from this Historic Colonial Village which dates back to the mid 1800's by making the lives of the Residents so jittery by their excessive mining practices of Blasting Dust and Smelly clouds, and unbearable noisy 24 hour operations, that the inhabitants would be driven out. We trust that the Authorities were not an active participant in this Plan. Indeed the Expert Advisory Committee should investigate these Incident reports of the Mining Companies and the Authorities Response to appreciate the extent of the "Residents Environmental Attack".

The much more serious Health concern is of the continuous Airborne Dust Residents have been exposed to, while the frequency at which that the Community Complaints (Attachment 4) record the presence of Mine Blasting Plumes that descend upon the Residents with regular monotony. On the 4th complaint for the same series of Blasts on 4th July 2007, the Residents report " It shook the House, it smells like sulphur and covered us in dirt" – caller is only 600-700 metres from the coal mine.

Others Blast reports; "two Blasts" on a Sunday; "Big yellowish dust cloud and also a strong smell of sulphur affecting caller"; "huge mushroom of dust resulted which then settled over Camberwell Village"; "A rotten nauseating smell gunpowder type smell resident feel sick"; "has left caller nauseous and very shaken"; "dairy farm and cows are affected by dust and noise from blasting"; "the blast shock the house & the front door came open due to the impact of the Vibration"; "overpressure level 123 decibels"; "House shook for 11 seconds. Large rocks were blasted from the mine (about 700 metres away) and landed in the paddock on the north side of the creek- just 100 metres from houses".

It is surprising that the Authorities did not prohibit the Blasting in the entire period 2004 to 2010 that allowed the Blast Plumes to descend onto the Camberwell Residents; in this case at the Glennies Creek end of Dawson Street within 500 metres of Aston Mine over the Ridge to the west in Photo 1.

The Camberwell Community rightly recorded in the Complaints Hotline (Attachment 4) Why is it that Mines Compliance Monitoring and Recording Equipment do not register the intensity experienced by Residents?

SSHEG review has confirmed that all three types of Equipment, Blast intensity and Vibration, Noise, Dust and Blast Gases Levels have been completely ineffective as a day to day means of controlling Mining Operations within acceptable Community standards.

SSHEG have previously identified that significant changes are needed to the Real Time Monitoring Technology used to understand the Pollution Sources and their Health Impact on the Community. Specifically, the PM10 24 Hr Real Time Monitoring current reference needs to not only record PM4, PM2.5, PM1 but also record three 8 Hr Maximum & Minimum, and the 15 Minute running averages.

Similarly, Noise methodology needs to change to recognize:-

- (1) low frequency "rumbling sounds" that are felt also as vibrations through the ground particularly at night, and*
- (2) Metal on metal "clanging sounds",*

Both of which cause the most annoyance and mental anguish for Residents.



Photo 2

Blast Plume sucked down slope to Glennies Creek Slow dissipation over Camberwell



Photo 3

Camberwell Gassing Incident 2004 Lethridge St.area viewing North

The drifting of Blast Plumes in 2004 identified to the Aston Mining Company and the Authorities that the Plumes instead of rising and dissipating, were sucked over the ridge and down into the creek gully where it lingered and spread over the Residents of Lethridge Street as in Photos 2 & 3. A different Blast Plume drifting pattern also in 2004 is shown in Photos 4 & 5.



Photo 4



Photo 5

Blast Plume drifting at ground level over Camberwell Village Residents in 2004

SSHEG also feel that urgent progress is needed towards Major changes to all Hunter Valley Mine Operations and Compliance Standards to counter the authority's complacency that is allowing mine generated Dust Clouds and Blast Plumes to often drift at high concentrations over "Near Neighbors" and "Village Residents Properties". Camberwell Residents as close as 500 to 800 metres from Mine Operations experience choking Dust and Gases for varying periods from some minutes upwards to 30 minutes at high concentration, and often days at a time.

Recent Videos have been provided to Dr David Durrheim of HNEAH detailing these types of occurrences where Blast Plumes float over the ridge and for some reason sucked down to the Glennies Creek level and then drifts generally at ground level over the Camberwell Residents area. Camberwell Residents have reported and photographed these events since 2004, only to be let down by the Authorities; however it should be noted that this does not release any of the persons involved who allowed these events to continue unchecked as they remain both culpable and liable to consequential damages that result from these events, then or in the future.

Just as the Camberwell Residents have been under "Environmental Attack", spare a thought for the farmer whose livelihood is threatened by the Mines creeping ever closer.

One such Farmers story is told in Attachment 14.

The impact of Mining on the rich agricultural land along the environs of the Hunter River and creeks is twofold.

Firstly the mining damage to rock strata and the so called accidental leakage or spills are now responsible for the River water contamination, as upstream of Mining the water is crystal clear while throughout the Mining areas the Rivers and Creeks are now always contaminated. The Community demands restitution by mining of these breakthroughs and discharges, no matter the cost to Mining. The Contaminated Hunter River water downstream of Mining (just above Singleton) now supplies water to the Pokolbin-Broke Vineyard areas with the accompanying Health Risks. *The time has come to recind the Salinity Trading Scheme and force Mining to use zero discharge practices.*

Secondly, the rich alluvial flats so prized by farmers are increasingly under threat from mining, to the degree that one such Mined area is being used as a test site by Authorities and Mining Companies to claim the apparent success in rehabilitation back to rich alluvial flats in three years. Nothing is further from the truth, as parts of the reclaimed and rehabilitated soil is continually sinking, and so far the Hunter River levee wall protecting mining is still in place. The land was cropped only long enough to satisfy mining lease conditions. The Authorities sponsored trial will need years of farm trials and a number of Hunter River flood events over the land to understand the extent of the permanent damage and the mine void contamination that will result.

It would appear that the very Flood Plain gravel presence that allows drainage so necessary for lucerne cultivation is now being used by the Authorities and Mining Companies to downgrade the rich productive River Flats from Class 1 to Class 2 or even Class 3, and therefore falsely creating an argument to allow mining to occur.

The Alluvial Flats prior to Mining had demonstrated over 160 years since Colonial times the superior Agricultural growing quality, especially for lucerne irrigated for Hay production. These Alluvial Hunter River Flats are the best Lucerne production areas in the Hunter Valley. Agricultural experts confirm that the best Lucerne production occurs on deep, well drained soils, with neutral to mildly alkaline, good native soil levels of P, K, S and Zn.

The Authorities apparently think that a 90 metre void filled with mining spoil covered by 1000mm of subsoil with a 400mm covering of reclaimed topsoil will reinstate the Rich Alluvial Flood Plains land that is over the centuries compacted in a way that lucerne which is known to send its roots up to twenty (20) feet will thrive.

Presently now in 2010 some 4 to 5 years later the “Mythical Picture” of perfect Lucerne trials of some 4 years ago is now clear for all to see with the sunken water laden land in parts not even fit to grow lucerne and will need topsoil relevening from wherever they can obtain suitable Topsoil from elsewhere. (not Alluvial Topsoil)

More importantly is the presence of disease and Pest Infestation of Whitefringed Weevil (that severely damage the root system and the productive life of Lucerne plants) that apparently resulted from the heaping of the Topsoil during Mining, which after spreading over the site infested the entire Lucerne crop and needed additional management. Why was this infestation not discovered before the heaped Topsoil was spread ?

Much was made of the Weevil infestation causing the accelerated decline in Lucerne persistence and production, and it now appears that the disturbed soil allowed the weevils to flourish in a way not seen in the other undisturbed Alluvial River Flats of the Hunter Valley.

Perhaps in the future, as the mine void fills with water and begins to leach into the Hunter River, the next episode and litany of Pollution from these misguided judgments will emerge.

Both the Authorities and the Mining Companies apparently see this earlier reported Lucerne Production Rates as a green light to repeat the mining practice elsewhere with a degraded Class classification being employed to achieve approvals.

The most graphic of Owner Farmers rights being “stolen” by stealth is outlined in the Attachment 15 where without any consequential damages financial compensation even to this day, the Mining Company with the Authorities consent destroyed the Farmers income and property, and in so doing polluted the creeks and the Hunter River.

The Mining Companies Stealth, aided and abetted by NSW Government approvals saw creeks that before mining were the breeding habitat for Platypus become so polluted that both Animal health was dramatically affected and agricultural crops wilted from the irrigation water from the once crystal clear waters.

The Australian Public are beginning to wake up to the plight of Farmers who are being pushed out in the name of progress, but at what cost to the future generations. For example, most people would believe the Rehabilitated Mine sites have been returned for farming, nothing is further from the truth.

Veterinary investigations (Attachment 20) in 1997 identified that Cattle on rehabilitated land soon became sick and since then only untouched mine lease land is being used for Cattle grazing.

The best that seems to be achieved for rehabilitated land pastures is cattle grazing for a short spell on land after 20 years left fallow.

Continuous Cattle Stocking has the risk of damaging the Mine Rehabilitation on one hand and exposing the Dust Pollution problem, or Cattle becoming affected by the pasture deficiencies or toxic contaminants leaching into the pastures from the mine spoil material on the other hand.

Do Epidemiology and PM2.5 Particle Characterisation Studies provide an insight into the Singleton Shire Residents Health concerns ?

The Singleton Shire Healthy Environment Group (SSHEG) focus is on Community Health where since 2008 the perception is that Coal Mining and Power Stations are progressively making Residents sick in the Hunter Valley Environs.

The Recent September 2013 “Centre of Air quality health Research (CAR)” Symposia in Sydney and Newcastle and the CSIRO PM2.5 Particle Characterisation Study in the Upper Hunter has begun to confirm the relationship between Air Pollution and Residents perceived detrimental health effects.

Traditionally, Epidemiology Studies form the body of Medical Opinion to quantify the relationships between Air Pollution and Population Health effects. The fact that populations are exposed to Air pollutants is largely beyond the control of individuals and requires action by public authorities at the national, regional and international levels.

While SSHEG contend that the Environment in the Hunter Valley, especially the Air Quality contains Gases, Particulates, Vapours, Fumes, Aerosols, Pollens, etc; and if the Composition, Toxicity, Exposure and related Health effects were understood, then better Mitigation steps could be taken to reduce the Health Risk.

The reality at this stage in 2013 is somewhat different, as outlined at CAR recently, clearly establishing that. International Epidemiology Cohort and Toxicology Studies have now in 2013 further quantified since 1993 the range of Mortality and Morbidity related Human Diseases associated with specific Air pollution Particulates Fractions.

World Health Organisation in the “Review of evidence on health aspects of air pollution – REVIHAAP” 2013 via its Scientific Advisory Committee and Expert Reviewers outlined much of the Scientific Expert deliberations associating Particulate Fractions to Health effects, both short and long term.

Most significantly is:-

“There is no safe threshold for Particulate Air Pollution”, with indications from Canada not only confirming long term exposure associations between cardiovascular mortality, with the strongest association with ischemic heart disease with concentrations of PM2.5 as low as only a few microgram per cubic metre.

“There is a linear Dose-Response Relationship between Particle Levels and Disease, with No Threshold that is Safe”.

This report then outlines the Community Health impact of recent Hunter Valley Air Pollution study developments attended by Singleton residents.

By way of explanation, back in 2009 Singleton Shire Residents made a submission to NSW Government calling for an Independent Health Study, especially related to Near Neighbours and children impacted by Mines and Power Stations.

Last month 4 Years later in 2013 details of these investigations are being released, both in the Hunter Valley Particle Characterisation PM2.5 Study and the International Epidemiology & World Health Organisation Research (WHO) 2013; both detailed below.

STOP PRESS RELEASE World Health Organisation 17th October 2013

“ IARC: Outdoor air pollution a leading environmental cause of cancer deaths

Lyon/Geneva, 17 October 2013 – The specialized cancer agency of the World Health Organization, the International Agency for Research on Cancer (IARC), announced today that it has **classified outdoor air pollution as carcinogenic to humans (Group 1)**. ¹

After thoroughly reviewing the latest available scientific literature, the world's leading experts convened by the IARC Monographs Programme concluded that there is *sufficient evidence* that exposure to outdoor air pollution causes lung cancer (Group 1). They also noted a positive association with an increased risk of bladder cancer.

Particulate matter, a major component of outdoor air pollution, was evaluated separately and was also classified as carcinogenic to humans (Group 1)."

"The predominant sources of outdoor air pollution are transportation, stationary power generation, industrial and agricultural emissions, and residential heating and cooking. Some air pollutants have natural sources, as well".

Authorities and Communities attended 5 Days Symposia & Lectures, 2 days in Sydney and 3 days in Newcastle, where the Singleton Community perception about both short and long term Air Pollution making the Community sick, now has a medical explanation, and is confirmed.

The "Pandora's Box of Air Pollution Human Diseases" has been opened, and this will now give traction to new Occupational Health and Safety Regulations related to the Density of Industry activity and the interface with Community Populations.

These Particulate Matter based Health Implications follow on from the WHO's 30 year investigations that identified in 2012 Diesel Exhaust Emissions as Group 1 Carcinogens, and changes in this

regard are now also expected to address the dangers of PM10, PM2.5(Fine), and PM10-2.5 (Coarse) Particulate Matter.

Categorically, “ *Air pollution exposure is Life shortening and can be Life Threatening*”

However there is a word of Caution – that like any other Human Health Risk it needs to be seen in perspective.

Such an understanding is outlined in the 2011 Global Burden of Disease report where Risks such as High Blood Pressure, Obesity, Smoking and Diabetes are generally more serious than Air Pollution issues. This is not to say that Pollution areas in the Hunter Valley are not the Highest Health Risk Factor for some residents.

The difference is; the Community has no choice but to breathe the Air in the Environment, while a choice exists for the others.

Issues at the Mt Thorley Industrial Estate, while a high profile Population is no different to “Near Neighbours” to Mines, Power Stations and Smelters that the Singleton Community have previously identified.

But it is especially all children, Pre & Post Natal and under 8 years of age that are now being confirmed as one Population Group vulnerable to Air Pollution; confirming the Singleton Community reports .

It is clear that in the future it will be the collaboration between the Authorities, Community and Industry that will establish a more acceptable balance, provided honest assessments based on clear understanding of the Health Risks are acknowledged and recognised by all.

In summary, with the Authorities and the Mines already with programmes focused of the reduction of Operational Air Pollution, a further focus on the reduction of short term Pollutant Emissions (2-4

hours) would appear to be a good step in the right direction for improvement.

“In particular a focus on eliminating Mine Blast Plumes with the use of “Plastic Stemming Plugs”, combined with reductions in Mine Diesel Exhaust Emissions should substantially reduce the Brown Smog haze that daily builds up and hangs over the Hunter Valley”.

Obviously, Authorities will need to play its key role in prepare media and educational resources to disseminate the balance of opinion of the way forward with respect to the Air particulates and Human Diseases.

Will Air Pollution take its place in the Climate Change Debate ?

Previous in 2007 Medical Expert Opinion to Mining was:-

“ No convincing evidence for an association between Residents Proximity to mines and Asthma, or Asthma Severity, or daily Diary Symptoms” and

“ Unlikely to have significant Health Impact”

By 2009 Dr Au and Singleton Residents associated disproportionate disease occurrences with increased Coal Industry behaviour in a Health Survey conducted by SSHEG .

The many Products of Combustion emissions from Coal Mining and Coal Fired Power Stations that enter into the Atmosphere we breath in the Hunter Valley contains Particulates and Gases, which by both Weight and Volume collectively outweigh the Climate Change Carbon Dioxide targets.

With Air Particulates now in 2013 being confirmed significantly associated with a range of Human Diseases, the obvious emphasis is expected to shift towards **Global Population Air Purity**, incorporating the full range of Gases and Particulate Man Made Pollution Emission Sources that are considered to Globally affect Climate Change.

Part A Epidemiology – What Insight in 2013

Epidemiology is the study (or the science of the study) of the patterns, [causes](#), and effects of [health](#) and [disease](#) conditions in defined [populations](#). It is the cornerstone of [public health](#), and informs policy decisions and [evidence-based medicine](#) by identifying [risk factors](#) for disease and targets for [preventive medicine](#). Epidemiologists help with study design, collection and [statistical analysis](#) of data, and interpretation and dissemination of results (including [peer review](#) and occasional [systematic review](#)). Epidemiology has helped develop [methodology](#) used in [clinical research](#), [public health](#) studies and, to a lesser extent, [basic research](#) in the biological sciences.^[1] [Wikipedia](#),

***@Epidemiology in 2013 outlines Health Impacts Released in 5Days
Lectures & Workshops, Symposia in Sydney and Newcastle.***

Latest European and WHO Epidemiology studies now have opened “*A Pandora’s Box*” with respect to the Human Diseases now being confirmed as associated with Air Pollution.

***@Three separate Particulate Fractions now exhibit
quite separate Health effects.***

1. PM10 fraction also includes PM4, PM2.5, PM1.0, PM0.1; and now PM10 is understood to have no threshold and differing Health effects to the other fractions.
2. PM10 to 2.5 fractions are designated PM (Coarse) Health effects.
3. PM2.5 fraction are designated PM (Fine) Health Effects

@ Since 2005, the evidence for a biological mechanism, derived from both epidemiological and toxicological studies, has also increased and indicates that exposure to PM2.5 is associated with systemic inflammation, oxidative stress and alteration of the electrical processes of the heart (Brook et al., 2010).

@ “Review of evidence on health aspects of air pollution – REVIHAAP”

This 2013 REVIHAAP document presents answers to 24 questions relevant to reviewing European policies on air pollution and to addressing health aspects of these policies. The answers were developed by a large group of scientists engaged in the WHO project “Review of evidence on health aspects of air pollution – REVIHAAP”. The experts reviewed and discussed the newly accumulated scientific evidence on the adverse effects on health of air pollution, formulating science-based answers to the 24 questions.

Three important components or metrics – black carbon, secondary organic aerosols, and secondary inorganic aerosols – have substantial exposure and health research finding associations and effects. They each may provide valuable metrics for the effects of mixtures of pollutants from a variety of sources.

a. New evidence links black carbon particles with cardiovascular health effects and premature mortality, for both short-term (24 hours) and long-term (annual) exposures. In studies taking black carbon and PM_{2.5} into account simultaneously, associations remained robust for black carbon. Even when black carbon may not be the causal agent, black carbon particles are a valuable additional air quality metric for evaluating the health risks of primary combustion particles from traffic, including organic particles, not fully taken into account with PM_{2.5} mass.

b. No new toxicological evidence has been presented to support a causal role for such inorganic secondary aerosols as ammonium, sulfates and nitrates. However, epidemiological studies continue to report associations between sulfates or nitrates and human health. Neither the role of the cations (for example, ammonium), nor the interactions with metals or absorbed components (for example, organic particles) have been well documented in epidemiological studies (see Answer C8). Even when secondary inorganic particles (especially sulphate particles) may not be the causal agents, they are a valuable additional air quality metric for evaluating health risks.

c. There is growing information on the associations of organic carbon with health effects, and carbonaceous primary emissions are one of the important contributors to the formation of secondary organic aerosols (a significant component of the PM_{2.5} mass). The evidence is insufficient to distinguish between the toxicity of primary and secondary organic aerosols.

3. The new evidence suggests that short-term exposures to coarse particles (including crustal material) are associated with adverse respiratory and cardiovascular effects on health, including premature mortality. Data from clinical studies are scarce; toxicological studies report that coarse particles can be as toxic as PM_{2.5} on a mass basis. The difference in risk between coarse and fine PM can, at least partially, be explained by differences in intake and different biological mechanisms.

4. There is increasing, though as yet limited, epidemiological evidence on the association between short-term exposures to ultrafine (smaller than 0.1 µm) particles and cardiorespiratory health, as well as the health of the central nervous system. Clinical and toxicological studies have shown that ultrafine particles (in part) act through mechanisms not shared with larger particles that dominate mass-based metrics, such as PM_{2.5} or PM₁₀.

A. Health effects of PM

Question A1

What new evidence on health effects has emerged since the review work done for the WHO air quality guidelines published in 2006, particularly with regard to the strength of the evidence on the health impacts associated with exposure to PM_{2.5}? Based on this new information, do the scientific conclusions given in 2005 require revision?

Answer

Since the 2005 global update of the WHO air quality guidelines (WHO Regional Office for Europe, 2006) were issued, many new studies from Europe and elsewhere on both short- and long-term exposure to PM with an aerodynamic diameter smaller than 2.5 µm (PM_{2.5}) have been published. These studies provide considerable support for the scientific conclusions in the 2005 global update of the WHO air quality guidelines and suggest additional health outcomes to be associated with PM_{2.5}. Among the major findings to date are the following:

1. additional support for the effects of short-term exposure to PM_{2.5} on both mortality and morbidity, based on several multicity epidemiological studies;
2. additional support for the effects of long-term exposures to PM_{2.5} on mortality and morbidity, based on several studies of long-term exposure conducted on large cohorts in Europe and North America;
3. an authoritative review of the evidence for cardiovascular effects, conducted by cardiologists, epidemiologists, toxicologists and other public health experts, concluded that long-term exposure to PM_{2.5} is a cause of both cardiovascular mortality and morbidity;
4. significantly more insight has been gained into physiological effects and plausible biological mechanisms that link short- and long-term PM_{2.5} exposure with mortality and morbidity, as observed in epidemiological, clinical and toxicological studies;
5. additional studies linking long-term exposure to PM_{2.5} to several new health outcomes, including atherosclerosis, adverse birth outcomes and childhood respiratory disease; and
6. emerging evidence that also suggests possible links between long-term PM_{2.5} exposure and neurodevelopment and cognitive function, as well as other chronic disease conditions, such as diabetes.

Question C8

Are there important interactions among air pollutants in the induction of adverse health effects that should be considered in developing air quality policy?

Answer

Note. This answer does not consider interactions with host susceptibility behaviour or other factors, with the exception of temperature.

Some interactions among air pollutants change the toxicity of the mixture. These occur as physicochemical interactions in air, as well as biological interactions. In developing air quality policies, the following issues can be considered.

There is very little evidence from health studies that the mixture of air pollutants results in significantly more health effects (synergy) than would be expected based on the information for single pollutants. However, this is largely due to a lack of data and methodological limitations.

Very few epidemiologic studies have examined the potential of pollutants to interact. This is likely due to their moderate to high correlations. The existence of such pollutant mixtures makes it often difficult, in an uncontrolled setting, to determine either independent or synergistic effects of ambient air pollutants.

Synergistic biological effects between ultrafine particles and transition metals and between particles and volatile organic compounds have been shown to indicate a larger combined impact on human health than would be expected from the separate entities.

A reduction of emissions of nitrogen oxides without an accompanying abatement of volatile organic compounds may result in no change, or even in an increase of ozone concentrations close to the source.

Airborne particles of any kind can carry aeroallergens or toxic condensed vapours, such that their impact can be substantially larger than without particles. There is a trend that the smaller the particles, the stronger the adjuvant effects. Limited evidence has been published suggesting that NO₂ can enhance allergic responses.

In general, reduction of one component will not result in a significant increase in the health risks associated with other components. The implications for reducing PM, on (semi)volatile organic compound formation, are not evident.

There is some evidence of interactions between pollutants and high temperature.

Changing the air pollution mixture due to changing fuels may, under certain conditions, lead to more harmful emissions.

Definitions of interactions

Interactions among air pollutants can be chemical, physical and biological. A chemical interaction would mean that two or more pollutants result in new components, based on the chemical composition. A well known example is nitrogen oxides and volatile organic compounds that result in the formation of ozone and other products in the presence of sunlight. In physical interactions, solid particles act as absorbers for organic compounds, affecting their transport through air and in the respiratory tract. Biologically, interactions are distinguished by the mode of action: dose addition (similar action), effect or response addition (dissimilar action), and complex interactions (synergistic, potentiating and antagonistic). Dose addition means that the chemicals in the mixture do not affect the toxicity of one another and that each component has different effects – for example, one component with produce lung inflammation, whereas a second component causes rhinitis only. Each of the chemicals in the mixture contributes to the toxicity of the mixture in proportion to its dose. In the case of response or effect addition, the components in a mixture have the same toxicological profile – for example, all lead to inflammation in the lung. Response addition is determined by summing the responses of each toxicant in a mixture. For interactions, compounds may interact with one another, modifying the magnitude and sometimes the nature of the toxic effect. This modification may make the composite effect stronger or weaker. An interaction might occur in the toxicokinetic phase (processes of uptake, distribution, metabolism and excretion) or in the toxicodynamic phase (effects of chemicals on the receptor, cellular target or organ). In the case of interaction, one cannot predict the toxicity based on exposure concentrations, and dose-response relationships are required to assess whether or not, for example, stronger responses occur than would be expected based on each of the pollutants alone.

In addition to (or even instead of) studies on single components or metrics, the *one-atmosphere* concept has been put forward as a novel way to investigate the effects on health of complex mixes. Advances in atmospheric modelling, in conjunction with validation studies that use targeted monitoring campaigns, will provide a more efficient way forward in research on health effects, rather than relying on increasing the number of components measured by routine monitoring networks.

@How then are Australian Residents currently Protected?

Ostensively, State Department of Health indirectly via EPA, OEH and Department of Planning collaboratively establish industry limits.

Presently the National Environmental Protection Measures (NEPM) Standard for Air Quality came out of the National Environmental Protection Act 1994 and is set for PM10 at 25ug/m3 Annual Average and 50ug/m3 as a 24Hr Average Value with no more than 5 exceedances permitted per year.(PM2.5 advisory only limits, 8ug/m3 annual average and 25ug/m3 for 24 Hours average). However this was only applied to selected Population areas nominated by the States, and with a guide Population limit of 25,000. This left Rural Populations such as the Singleton Shire in the Hunter Valley unrepresented.

The NEPM standard in part is based on 1993 Epidemiology 6 city USA Cohort Studies when it was considered that a Threshold of Particulate Exposure existed below which Human Health Effects were not evident at the time.(In 2013 No threshold is now evident)

Further, it is the Epidemiology Cohort Studies that have quantified Health Effects against an Annual Reference base for Particulate Matter Air Quality that underscores the significance of the Annual Average PM 10 NEPM.

With the 2013 WHO deliberations identifying many short term Health effects, it is evident the “Law of Averages” long time based Cohort distortion will in time be replaced by progressively shorted time based Reference Standards.

@What are the 2013 Epidemiological Particulate related Air Pollution Health associations, and How do these Risks compare ?

For Example:-

“Fine particulate and sulfur oxide–related pollution were associated with allcause, lung cancer, and cardiopulmonary mortality. Each 10-µg/m3 elevation in fine particulate PM 2.5 air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively,”

Regarding the European studies, the mortality risk estimated in the Dutch mortality cohort study for PM2.5 was 6% per 10 µg/m3 for natural-cause mortality (Beelen et al., 2008a), identical to the estimate from the American Cancer Society study (Pope et al., 2002).

@A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010

“We estimated deaths and disability-adjusted life years (DALYs; sum of years lived with disability [YLD] and years of life lost [YLL]) attributable to the independent effects of 67 risk factors and clusters of risk factors for 21 regions in 1990 and 2010. We estimated exposure distributions for each year, region, sex, and age group, and relative risks per unit of exposure by systematically reviewing and synthesising published and unpublished data”.

Risk factor	Ranking legend									
	1-5	6-10	11-15	16-20	21-25	26-30	31-35	36-40	>40	
	Global	High-income Asia Pacific	Western Europe	Australasia	High-income North America	Central Europe	Southern Latin America	Eastern Europe	East Asia	
High blood pressure	1	1	2	3	4	1	2	2	1	
Tobacco smoking, including second-hand smoke	2	2	1	2	1	3	3	3	2	
Alcohol use	3	3	4	4	3	2	4	1	6	
Household air pollution from solid fuels	4	42	14	23	20	5	
Diet low in fruits	5	5	7	7	7	5	6	5	3	
High body-mass index	6	8	3	1	2	4	1	4	9	
High fasting plasma glucose	7	7	6	6	5	7	5	10	8	
Childhood underweight	8	39	38	37	39	38	38	38	38	
Ambient particulate matter pollution	9	9	11	26	14	12	24	14	4	

Figure 5: Risk factors ranked by attributable burden of disease, 2010

Regions are ordered by mean life expectancy. No data=attributable disability-adjusted life-years were not quantified

www.thelancet.com Vol 380 December 15/22/29, 2012.

Interpretation These changes are related to the ageing population, decreased mortality among children younger than 5 years, changes in cause-of-death composition, and changes in risk factor exposures. New evidence has led to changes in the magnitude of key risks including unimproved water and sanitation, vitamin A and zinc deficiencies, and ambient particulate matter pollution.

Deaths Globally from household & ambient air pollution are around 7 million pa – more than from tobacco smoking.

@2007 Coal Mining advice by Medical Specialists at the time.

Existing Community Health Impact questioning of Open Cut Black Coal Mining has since November 2007 been along the lines that “ *No convincing evidence for an association between Residents Proximity to mines and Asthma, or Asthma Severity, or daily Diary Symptoms*”

@SSHEG Singleton Shire Health Perceptions 2009

Singleton Shire Community and Dr Au saw this differently, with the perception that Asthma and Air Quality are related, and SSHEG sought in 2009 an Independent Health Study to investigate what is in the Environment, its Composition, Toxicity, and Human Health impact, with a view to Mitigating any Health Risks identified.

@NSW Health Expert Advisory Committee 4 Years of investigations September 2013

- 2010 *Rural Rainwater Tanks Drinking Water & Cooking- treatment Guidelines*
- 2011 *Confirmation of the Dangers of Organic Growth Medium becoming Airborne.*
- 2012 *Confirmation of Mine Blasting Plumes “Gassing” possible some Kilometres away.*
- 2013 *Confirmation that Epidemiology is classically used worldwide to investigate for associations between Air Quality (Pollution) and Human Health impact. However these Studies are unsuitable as “A Health Study in NSW was unlikely to show a statistically significant Health effect because the impacted population is too small - .even when the Air pollutants under investigation are known to cause illness”*

@Summary Sept 2013

Latest European and WHO Epidemiology studies now have opened “A Pandora’s Box with respect to the Human Diseases now being confirmed as associated with Air Pollution.

Interestingly, the focus has shifted to:-

1. PM2.5 Exposure Response Functions, away from PM10.
2. Latest Health Risk Association is for all “Particulates in a PM2.5 Cocktail Mix”, Thus “ All PM Particulates are found to be equal in these Epidemiology Studies.”^(Note 1)
3. PM10 while remaining Health Risk significant; The Disease significance has shifted to the PM2.5 (PM Fine), and the PM10-2.5 as (PM Coarse) as distinct from the original PM10 Particulates fractions.
4. PM1.0 size fractions show lower order of significance in current Studies.
5. Interestingly, Total Suspended Particulates (TCP) correlations have emerged as a useful Health Risk guide methodology, including Dust Deposition Gauges to measure Total Suspended Particles to characterise the PM Coarse fractions.

@SSHEG contends that the “specific Pollutant Cocktail in the Hunter Valley” is one step closer to being understood, both in terms of the emerging Epidemiological Health implications, as well as the extent that “traditional Averaging methods” are disguising the real Toxic Health Impact from short term exposure to drifting Pollution Sources as being reported by Rural Residents.

**@Sept 2013 Centre for Air Quality, Health Research and Evaluation
5 Day Lectures, Workshops and Symposia
in Sydney and Newcastle.**

Topics include;-

1. *Exposure assessment Methods in Air Pollution Epidemiology
– current and emerging methods.*
2. *Review the latest evidence on the Health effects of Air pollution*
3. *How Exposure Response Functions are used in assessing Risk associated
with Air pollution*
4. *Review how Epidemiological evidence is used to quantify Air Pollution
Impacts including Global Burden of Disease.*

“The group of experts recognizes that air pollution exists as a complex mixture and that the effects attributed to individual air pollutants may be influenced by the underlying toxicity of the full mixture of all air pollutants”.

“Since 2005, the evidence for a biological mechanism, derived from both epidemiological and toxicological studies, has also increased and indicates that exposure to PM_{2.5} is associated with systemic inflammation, oxidative stress and alteration of the electrical processes of the heart (Brook et al., 2010)”.

NOTE 1 *“An important issue regarding the present findings concerns the issue of pollutant-specific effects, that is, which (set of) pollutant(s) is responsible for the observed effects. As in our previous analyses (6, 19) it was not possible to disentangle the effects of the specific pollutants that were measured because of the high correlations among the different pollutants. In outdoor air, NO₂ often is highly correlated with other combustion products, in particular fine particulate matter (28). An expert panel reviewed the biological plausibility of epidemiological findings on criteria pollutants (29). Taking into account findings from epidemiological, human clinical, and toxicological studies, the experts concluded that NO₂ may be acting as a surrogate for a mixture of pollutants”.* AJRM Vol 181 Ulrike Gehring et al

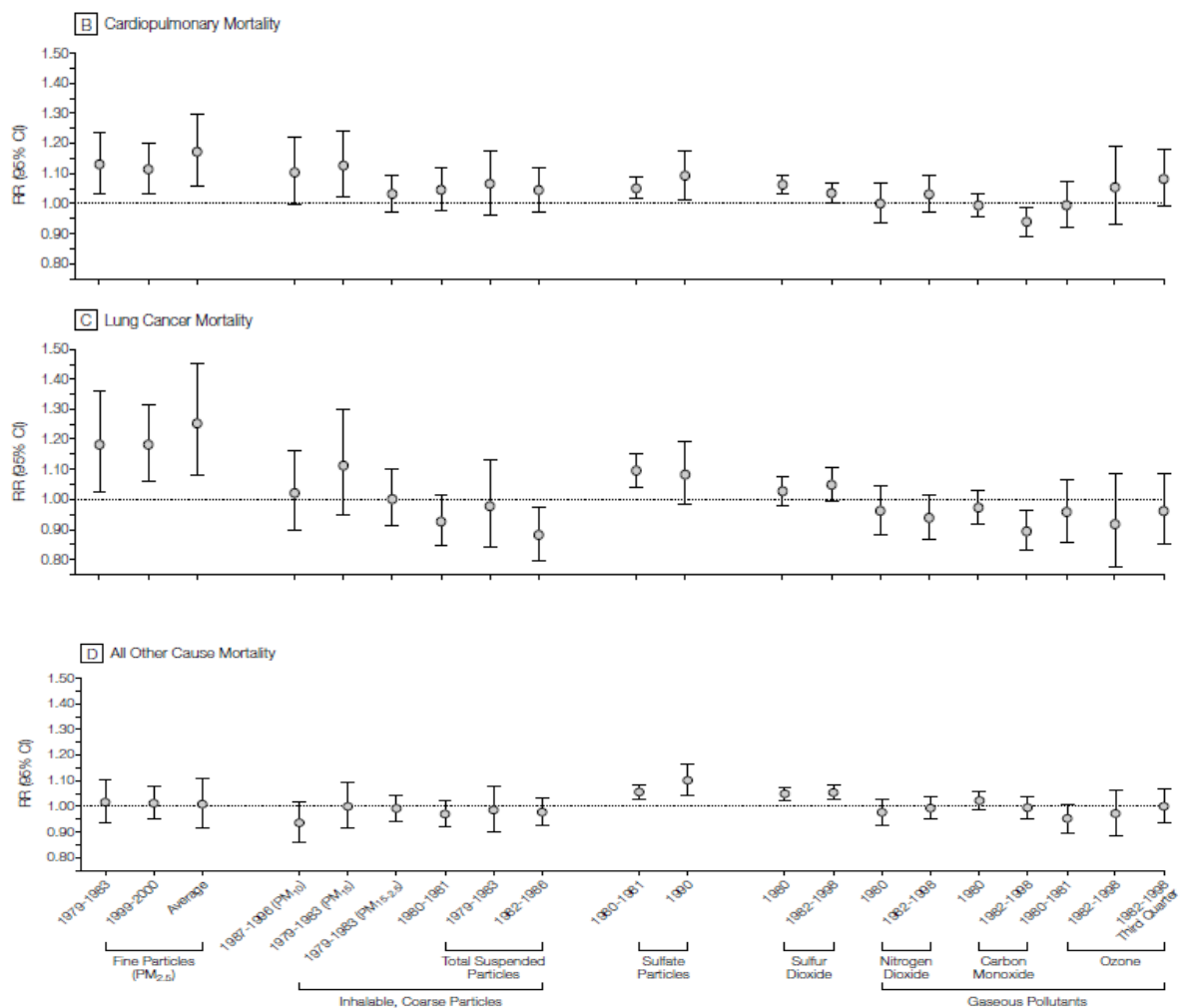
@Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution

C. Arden Pope III, PhD, Richard T. Burnett, PhD, Michael J. Thun, MD, Eugenia E. Calle, PhD
Daniel Krewski, PhD, Kazuhiko Ito, PhD, George D. Thurston, ScD.

“Fine particulate and sulfur oxide–related pollution were associated with all-cause, lung cancer, and cardiopulmonary mortality. Each 10- $\mu\text{g}/\text{m}^3$ elevation in fine particulate PM_{2.5} air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively, although the magnitude of the effect somewhat depended on the time frame of pollution monitoring.

Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality in this study”.

Figure 5. Adjusted Mortality Relative Risk (RR) Ratio Evaluated at Subject-Weighted Mean Concentrations



PM_{2.5} indicates particles measuring less than 2.5 μm in diameter; PM₁₀, particles measuring less than 10 μm in diameter; PM₁₅, particles measuring less than 15 μm in diameter; PM_{15-2.5}, particles measuring between 2.5 and 15 μm in diameter; and CI, confidence interval.

Conclusion Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.

JAMA. 2002;287:1132-1141 www.jama.com

@Risk of Nonaccidental and Cardiovascular Mortality in Relation to Long-term Exposure to Low Concentrations of Fine Particulate Matter: A Canadian National-Level Cohort Study

Dan L. Crouse,¹ Paul A. Peters,² Aaron van Donkelaar,³ Mark S. Goldberg,⁴ Paul J. Villeneuve,^{1,5} Orly Brion,¹ Saeeda Khan,² Dominic Odwa Atari,² Michael Jerrett,⁶ C. Arden Pope III,⁷ Michael Brauer,⁸ Jeffrey R. Brook,^{5,9} Randall V. Martin,^{3,10} David Stieb,¹ and Richard T. Burnett¹

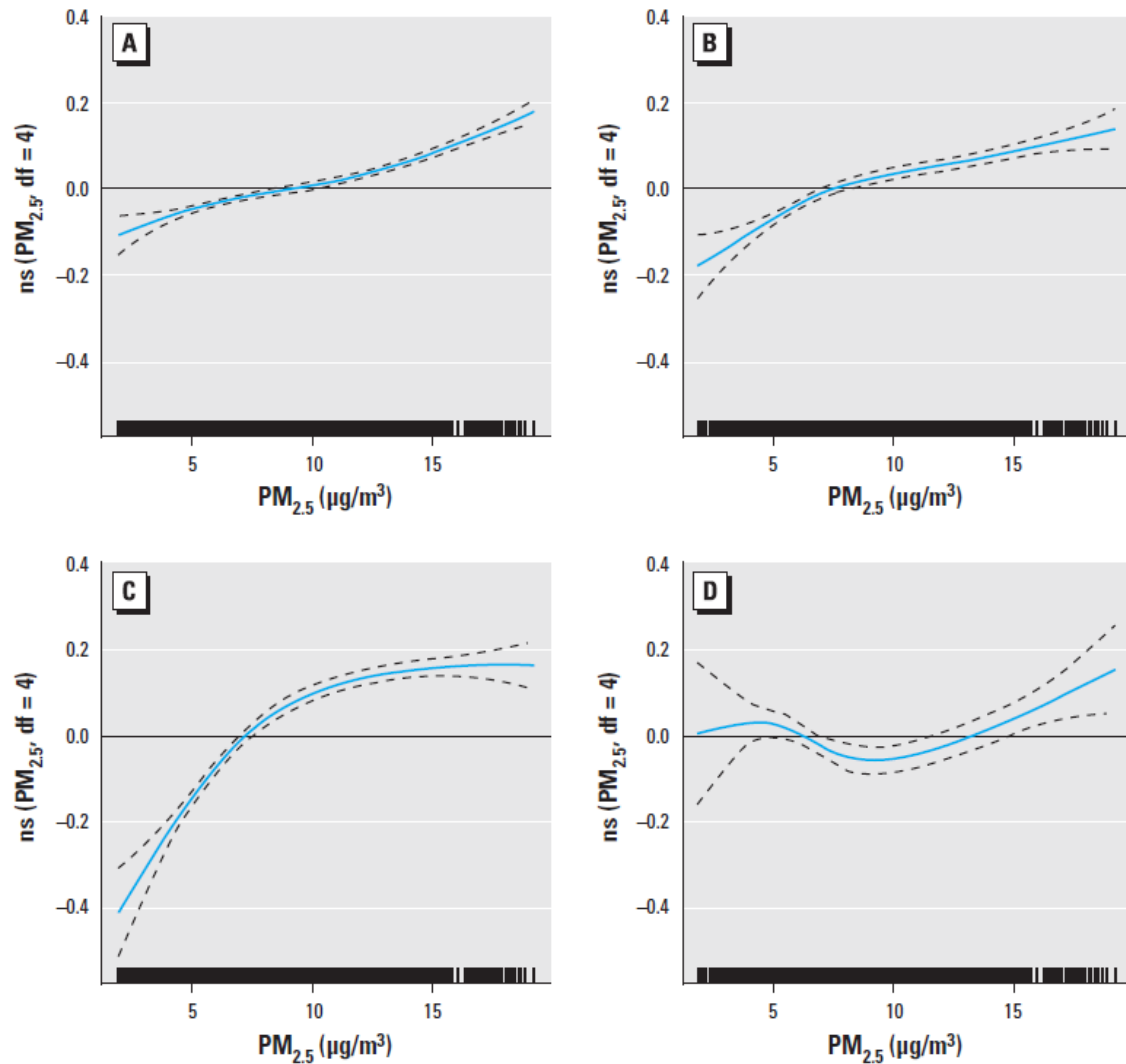
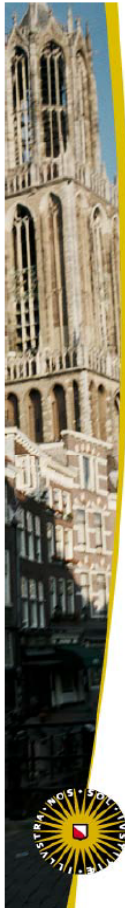


Figure 2. Concentration–response curves (solid lines) and 95% CIs (dashed lines) based on natural spline (ns) models with 4 df, standard Cox models stratified by age and sex, adjusted for all individual-level covariates, urban/rural indicator, and ecological covariates. (A) Nonaccidental causes. (B) Cardiovascular disease. (C) Ischemic heart disease. (D) Cerebrovascular disease. The tick marks on the x-axis identify the location of the $PM_{2.5}$ concentrations.

Conclusion

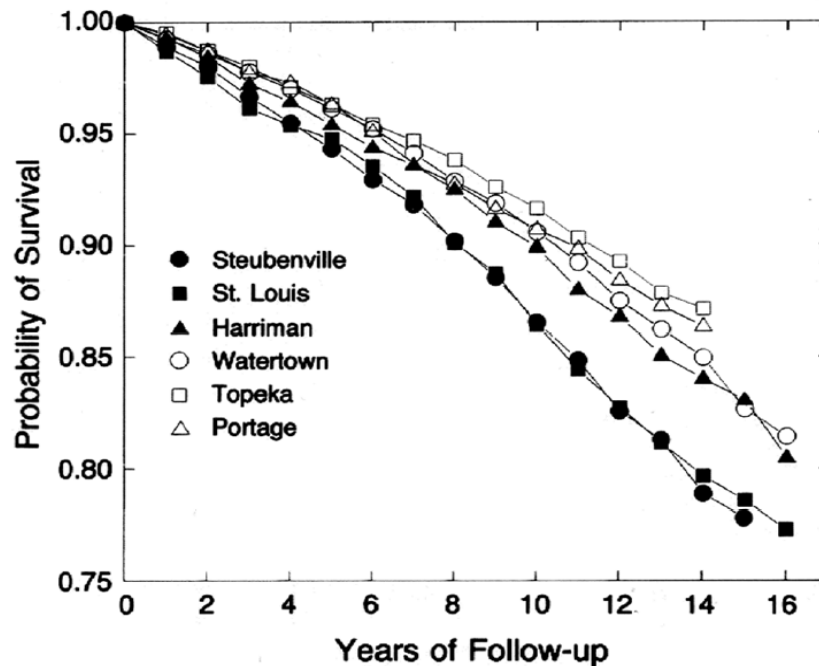
We identified in this national-level, population-based cohort study of nonimmigrant Canadians associations between cardiovascular mortality and long-term exposure to $PM_{2.5}$. We found the strongest association with ischemic heart disease, and our HRs were similar in magnitude to those reported in large cohort studies conducted elsewhere.

Our associations were reported with exposure to concentrations of $PM_{2.5}$ as low as only a few micrograms per cubic meter.



Universiteit Utrecht

Harvard Six Cities Study



Dockery, NEJM 1993

These Large USA Epidemiology Studies identified in 1993 and now Peer Reviewed in 2002 & 2006 identify the association between Air Quality and Mortality in the Populations.

Note: Epidemiology studies of Air Quality and Mortality, for example after 10 Years 87/100 survived in the Worst Polluted cities such as St. Louis (13/100 Mortality), while 92/100 survived in the cleanest cities such as Topeka (8/100 Mortality).

The difference 13-8 or 5 Deaths per 100 is associated with Air Quality.

The question remains; Globally Australasia ranks 26th with respect to “Ambient Particle Matter Pollution”, but is the Hunter Valley actually ranked first in the country with its concentration of Coal Mining , Power Stations and Aluminium Smelters in an Agricultural area surrounded by Forest Mountains ?

Traffic-related Air Pollution and the Development of Asthma and Allergies during the First 8 Years of Life

Ulrike Gehring¹, Alet H. Wijga², Michael Brauer³, Paul Fischer⁴, Johan C. de Jongste⁵, Marjan Kerkhof⁶, Marieke Oldenwening¹, Henriette A. Smit^{2,7}, and Bert Brunekreef^{1,7}

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AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE VOL 181 2010

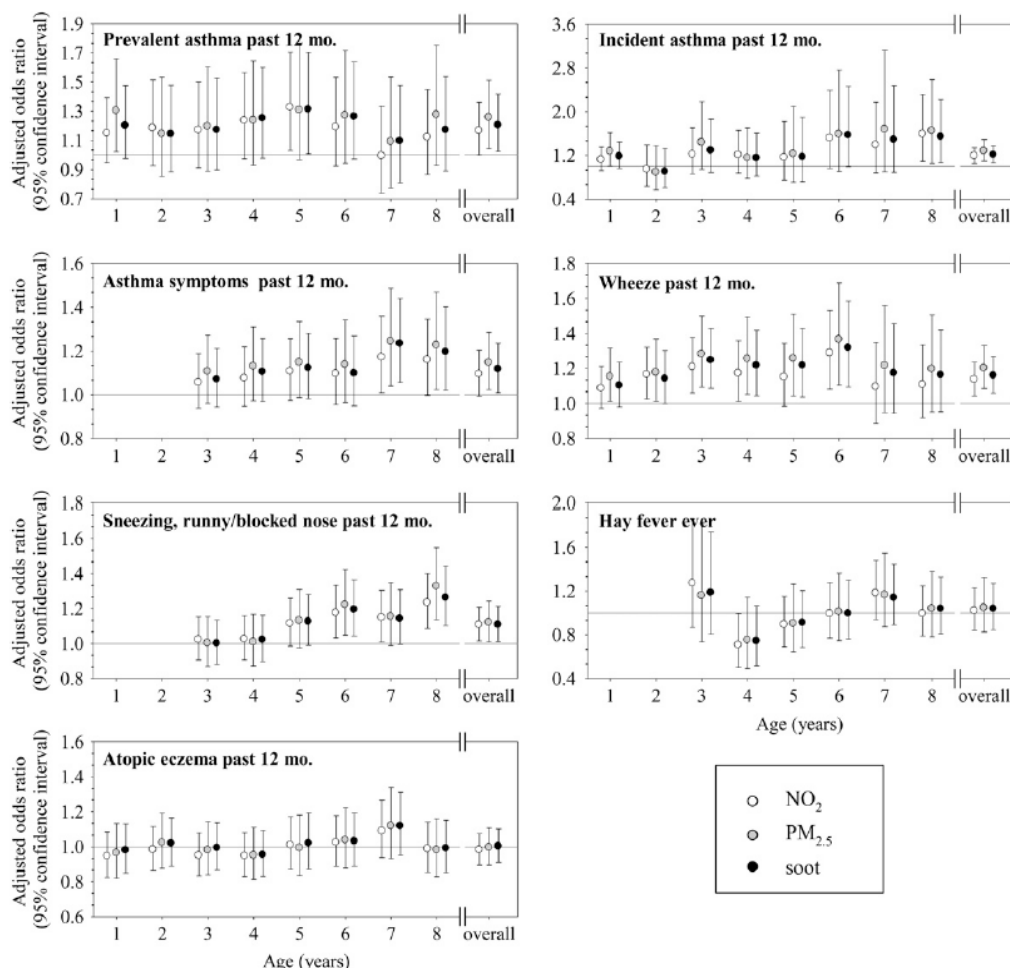


Figure 2. Adjusted overall and age-specific association between annual average levels of air pollution at the birth address and asthma, hay fever, atopic eczema, and related symptoms during the first 8 years of life. Results are presented as adjusted odds ratios (ORs) with 95% confidence intervals. We adjusted for all characteristics from Table 1 except study region. ORs were calculated for an interquartile range increase in air pollution levels ($10.4 \mu\text{g} \cdot \text{m}^{-3}$ for NO_2 , $3.2 \mu\text{g} \cdot \text{m}^{-3}$ for $\text{PM}_{2.5}$, and $0.57 \times 10^{-5} \text{ m}^{-1}$ for soot).

Measurements and Main Results:

Annual prevalence was 3 to 6% for asthma and 12 to 23% for asthma symptoms.

Annual incidence of asthma was 6% at age 1, and 1 to 2% at later ages.

$\text{PM}_{2.5}$ levels were associated with a significant increase in incidence of asthma (odds ratio [OR], 1.28; 95% confidence interval [CI], 1.10–1.49), prevalence of asthma (OR, 1.26; 95% CI, 1.04–1.51), and prevalence of asthma symptoms (OR, 1.15; 95% CI, 1.02–1.28).

Findings were similar for NO_2 and soot. Associations were stronger for children who had not moved since birth.

Positive associations with hay fever were found in nonmovers only.

No associations were found with atopic eczema, allergic sensitization, and bronchial hyperresponsiveness.

Conclusions: Exposure to traffic-related air pollution may cause asthma in children.

Part B PM2.5 Particle Characterisation Study

@ SSHEG On behalf of the Community sought a Health Study based upon PM2.5 Monitoring together with monitoring for other air toxins, at the very minimum Mercury, Lead, Chromium, VOCs, PAHs and Dioxins and National Pollution Index relevant Pollutants.

The Monitoring Network should set up a regime at Bayswater, Liddell and Rebank Power Stations and sample and analyse Gases and Particulate Matter that discharges from the various stacks to atmosphere, day and night in continuous Real Time. The monitoring must be long enough to capture all events and not be selective. It should be 24 hour monitoring and not averaged.

SSHEG believed that Air Quality would be both Monitored in Real Time and Sampled on appropriate Filter material, such as Teflon for ANSTO Analysis. UHAQMN TEOM PM10 and three OSIRIS Monitors were co-located along with Particulate Samplers.

SSHEG perception was that “PM2.5 particles should also be measured as they are of the size that reach the Human lungs and are thus more representative of the Community Asthma and Respiratory Diseases”.

After discussions, it was expected that PM10 TEOM & PM2.5 OSIRIS Real Time Monitoring (1 minute), and PM10 and PM2.5 Particulate Fractions would be sampled. However, some months later, SSHEG collaborating with the NSW Minerals Council discovered that a combination of CSIRO-ANSTO Analysis study, and a Macquarie University Size fractions study was underway.

Rather than designing for the Upper Hunter Study, the Expert Advisory Committee apparently Opted for Monitoring and Sampling Protocols used previously for the Mining Industry. As a consequence these latest PM2.5 Upper Hunter Study can be added to the numerous ANSTO Aerosol Data Sets available Hunter Valley studies in Mayfield (1998-2009), Muswellbrook (2001-2011), and Singleton and Grenville ACARP (2007).

Apparently, the shortcomings of the Upper Hunter PM2.5 Study are a lost opportunity, to correlate CAR Epidemiology Health effects recently identified for Monitoring and Sample Analysis for Pollutant Fractions PM10, PM2.5 (Fine), PM10to2.5 (Coarse), as well as Dust Deposition Gauges to measure Total Suspended Particles to characterize the Coarser Fractions.

The recently announced Newcastle Air Quality Monitoring System should now benefit from a better understanding of Air Pollutants and Community Health; and perhaps a further Characterisation Study in the Upper Hunter is now warranted, to judge the effectiveness of EPA and OEH Pollution reduction Programs such as the 2013 Mine “Dust Stop Program”.

@ What has the Upper Hunter Valley Particle Characterisation Study Identified ?

This one year 2012 study sampled PM_{2.5} Particulate Matter for composition, being “analysed for twenty elements, fourteen soluble ions, two anhydrous sugars, organic carbon, black carbon, as well as gravimetric mass”.

Eight identifying factors – “Fingerprints” which represent the mix of chemical elements as the most likely source of the emissions, that contribute to the measured PM_{2.5} concentrations.

In Singleton the dominant factors during the year were identified as:

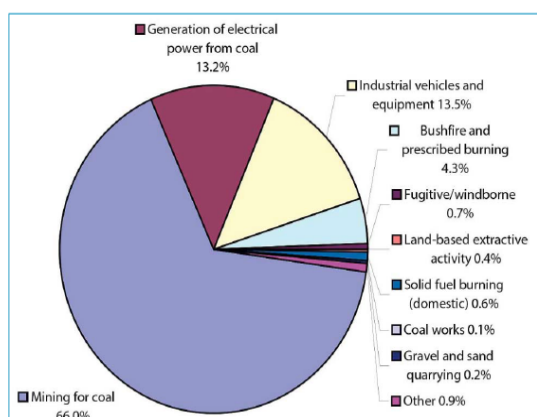
□ Factor 3 (Secondary Sulfate),	20 ± 2%
□ Factor 5 (Industry Aged Sea Salt),	18 ± 3%
□ Factor 2 (Vehicle/Industry),	17 ± 2%
□ Factor 1 (Woodsmoke),	14 ± 2%
□ Factor 6 (Soil),	12 ± 2%.

@

Background - issues

- ~10,000 tpa PM_{2.5} emitted in 2008
- doubled since 2003
- 20 coal mines in Upper Hunter
- Community concerns about mining, loss of amenity, health issues, ...
- Need improved understanding of fine particles in the Upper Hunter

Emissions inventory for PM_{2.5} in Upper Hunter



@ Yearly PM2.5 Variation

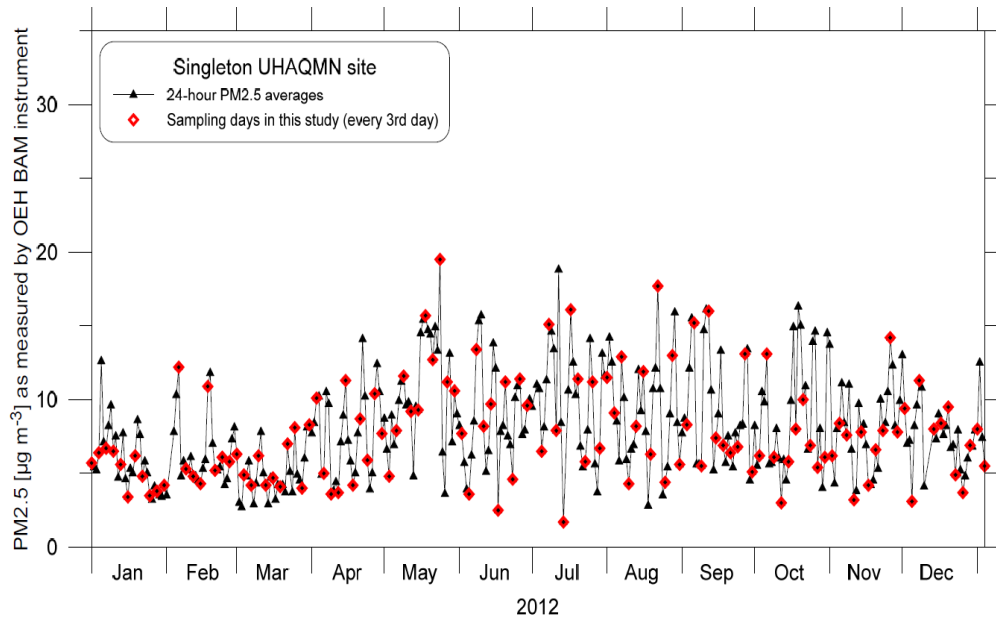
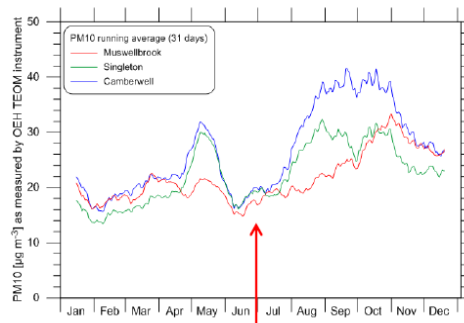


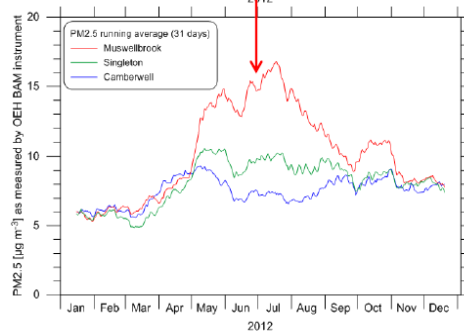
Figure 1 Time series of 24-hour average PM2.5 concentrations measured by the OEI BAM (Beta Attenuation Mass) monitor at Singleton. The red symbols show the days when sampling for the current study was carried out.

Background - PM10 vs PM2.5

**PM10
TEOM**



**PM2.5
BAM**



@ Potential PM_{2.5} Source Identification

Table 1 Summary of the PMF factors (from the EPA PMF 3.0 analysis), main species, contributions of these factors at each site and potential sources

Factor	Main Species in Factor	Contribution of the factor to total annual PM _{2.5} mass at:		Potential Sources
		Singleton	Muswellbrook	
Factor 1 Woodsmoke	levoglucosan, mannosan, OC1	14 ± 2%	30 ± 3%	Domestic woodheaters
Factor 2 Vehicle/Industry	BC, OC1, OC2, SO ₄ ²⁻ Fe, Zn, Mn, Cu	17 ± 2%	8 ± 1%	Vehicles, industry
Factor 3 Secondary Sulfate	NH ₄ ⁺ , SO ₄ ²⁻	20 ± 2%	17 ± 2%	Local and regional sources of SO ₂ such as power stations
Factor 4 Biomass Smoke	OC2, OC3, OC4, K, SO ₄ ²⁻ , Al, Si, Ti, BC	8 ± 2%	12 ± 2%	Wildfires, hazard reduction burns
Factor 5 Industry Aged Sea Salt	Na ⁺ , Mg ²⁺ , SO ₄ ²⁻ and with almost no Cl ⁻	18 ± 3%	13 ± 2%	Sea salt, local and regional sources of SO ₂ such as power stations
Factor 6 Soil	Al, Si, Ca, Ti and Fe	12 ± 2%	11 ± 1%	Soil dust, fugitive coal dust
Factor 7 Sea Salt	Na ⁺ , Cl ⁻ , and Mg ²⁺	8 ± 1%	3 ± 1%	Sea salt
Factor 8 Secondary Nitrate	NO ₃ ⁻ and includes some NH ₄ ⁺ , Cl ⁻ , Na ⁺ , OC	3 ± 2%	6 ± 1%	Motor vehicle NO ₂ , power station NO ₂

Notes: Al – aluminium; BC – black carbon; Ca – calcium; Cl⁻ – chloride; Cu – copper; Fe – iron; K – potassium; Mg²⁺ – magnesium; Mn – manganese; Na⁺ – sodium; NH₄⁺ – ammonium; NO₃⁻ – nitrate; OC1-OC4 – fractions of organic carbon distinguished by the volatility of the organic compounds, OC1 is the most volatile, as organic aerosol ages its OC becomes less volatile; Si – silicon; SO₄²⁻ – sulfate; Ti – titanium; Zn – zinc.

@ Seasonal Variation of “Fingerprints”

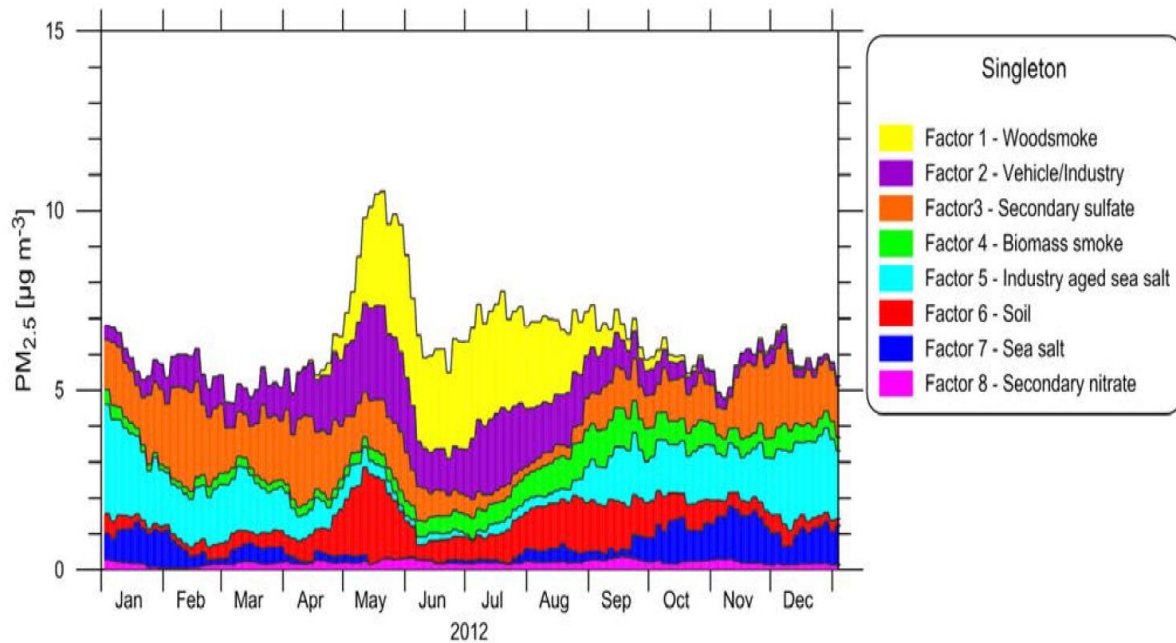


Figure 48. Time series (smoothed with 31-day running window) of the contribution of each factor to the total $PM_{2.5}$ in Singleton.

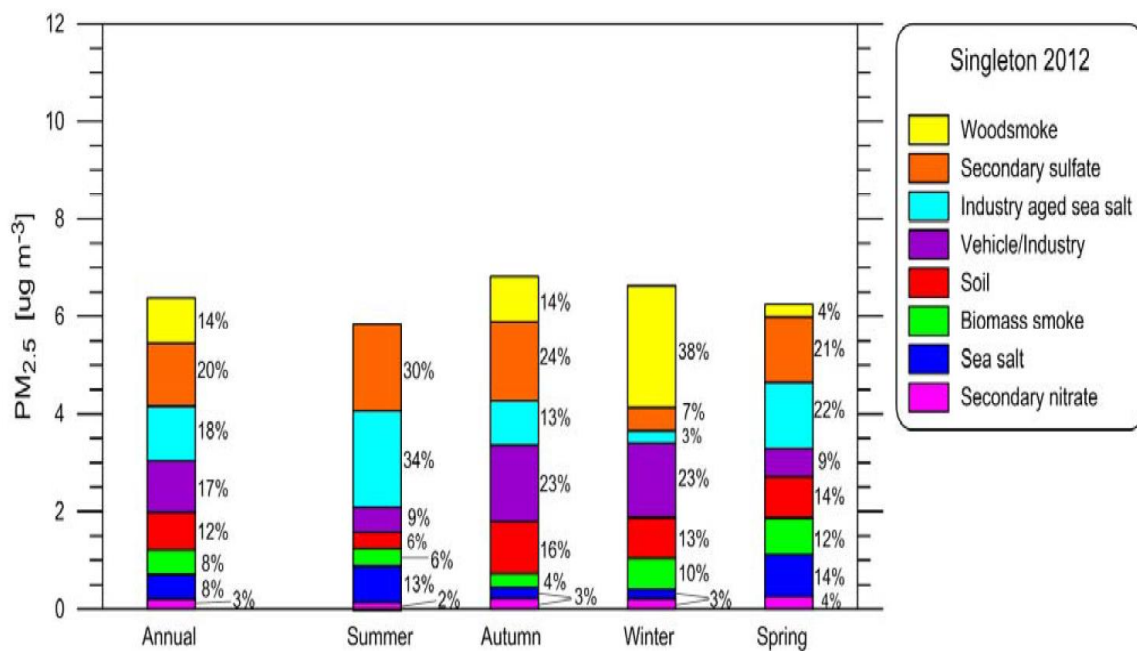


Figure 1 Annual and seasonal contributions of the PMF factors to $PM_{2.5}$ in Singleton

@ Singleton PM2.5 Speciation

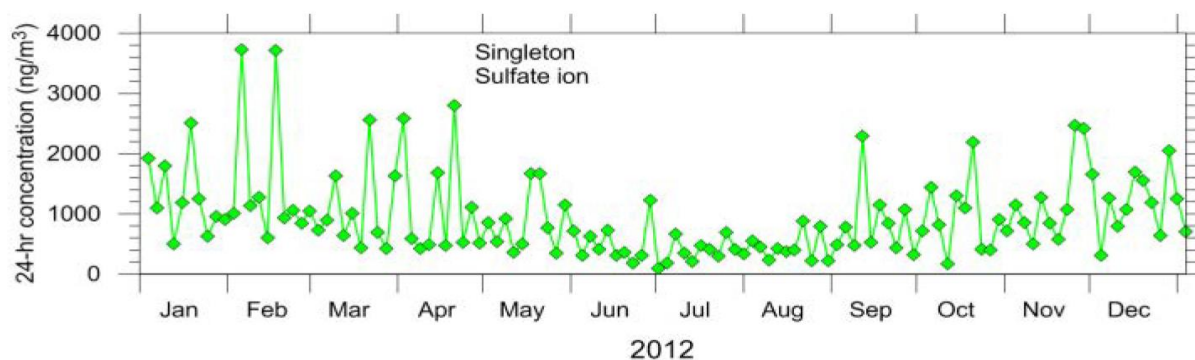
For example

Table 2 Median concentrations for species measured at Singleton using either ion chromatography(IC), ion beam analysis (IBA), LIPM (laser integrated plate method (LIPM), or thermal-optical carbon analyser (TA)

Species	Median Conc. (ng m ⁻³)	MDL (ng m ⁻³)	% of values <MDL	Uncert. (%)	Species	Median Conc. (ng m ⁻³)	MDL (ng m ⁻³)	% of values <MDL	Uncert. (%)
Na ⁺ (IC)	219	0.6	0%	9	OC1 (TA)	223	13.9	0%	10
Na (IBA)	200	77	27%	14	OC2 (TA)	397	36.1	0%	10
NH ₄ ⁺ (IC)	141	0.3	0%	8	OC3 (TA)	821	63.7	0%	10
Mg ²⁺ (IC)	26	0.14	0%	9	OC4 (TA)	409	1	0%	10
Cl ⁻ (IC)	46	0.7	0%	5	Mn (IBA)	1.1	0.4	6%	20
Cl (IBA)	52	1.9	8%	6	Cu (IBA)	0.6	0.4	31%	25
NO ₃ ⁻ (IC)	126	0.6	0%	6	Zn (IBA)	3.0	0.4	2%	15
SO ₄ ²⁻ (IC)	765	0.6	0%	7	Br (IBA)	2.0	1.4	25%	40
S (IBA)	255	1.5	0%	6	Pb (IBA)	1.4	2.6	77%	40
C ₂ O ₄ ²⁻ (IC)	53	0.3	0%	9	Mass	6108	160	0%	5
Levoglucosan	48.5	3	0%	5	NO ₂ ⁻ (IC)	0.4	0.8	100%	9
Mannosan	1.3	2	18%	5	Br ⁻ (IC)	0.3	0.5	94%	9
Al (IBA)	45	4	12%	7	PO ₄ ³⁻ (IC)	3.3	0.6	0%	9
Si (IBA)	143	2	0%	6	P (IBA)	0.2	2	92%	35
K (IBA)	37	1	0%	6	F ⁻ (IC)	0.1	0.2	58%	9
K ⁺ (IC)	35	0.35	0%	10	Acetic (IC)	3.2	6.4	77%	12
Ca (IBA)	23	1.2	0%	6	Formic (IC)	3.5	1.8	38%	9
Ca ²⁺ (IC)	25	1.4	0%	8	HCO ₃ ⁻ (IC)	20	0.2	0%	5
Ti (IBA)	3.4	0.7	15%	11	H (IBA)	161	6.6	0%	6
Fe (IBA)	53	0.5	0%	6	V (IBA)	0.3	0.7	88%	33
BC (LIPM)	857	29	0	8	Cr (IBA)	0.2	0.5	98%	30
EC (TA)	1273	5	0%	10	Co (IBA)	0.3	1.6	100%	-
MSA ⁻ (IC)	14	0.7	0%	19	Ni (IBA)	0.2	0.6	82%	18

Figure 15 shows the time series of the these species concentrations, many of which show a seasonal variation, some with peaks in winter such as levoglucosan, mannosan, and others with a minimum in winter such as sulfate, sodium and MSA.

Each of the Species Analysis exhibits a seasonal variation during the year, e.g.. Sulphate ion



@ Singleton PM_{2.5} Mass Balance

The identification of most of the Factors is reasonably clear-cut because of the use of either unique tracer species, e.g. levoglucosan for Factor 1 (Woodsmoke), or two or more species whose ratios are defined by a particular source, e.g. Si and Al in Factor 6 (Soil), Na⁺ and Mg²⁺ in Factor 7 (Sea Salt), and NH₄⁺ and SO₄²⁻ in Factor 3 (Secondary Sulfate).

However in the case of Factor 2 (Vehicle/Industry) and Factor 4 (Biomass Smoke), the identification of the source is less definitive. Figure 22 shows an example of the 'fingerprint' of the factor, so called because it shows a unique pattern of species concentrations.

The fingerprint shows the relative amounts of the various species in the factor. It does this in two ways. Firstly, the vertical blue bars show the species concentrations, e.g. in Factor 1 at Singleton, the levoglucosan concentration is 210 ng m⁻³ and the mass (second bar from the right) is 920 ng/m³ = 0.92 µg m⁻³. Secondly, the dark red squares show the percentage of the species that occurs in the factor, e.g. Factor 1 at Singleton includes 86% of the levoglucosan measured in the samples and 14% of the mass Figure 23 shows how the contribution of the factor to total PM_{2.5} varies during the year. Factor 1 only makes a significant contribution from May to August with almost no contribution from November to March. the cooler weather from May to August corresponds to the period when domestic woodheaters are used, and this agrees with the fingerprint analysis indicating woodsmoke.

Smoke is also produced by bushfires and hazard reduction burns, but the time series of these in Figure 34 is quite different from the time series for Factor 1.

