Submission No 19

INQUIRY INTO HEALTH IMPACTS OF AIR POLLUTION IN THE SYDNEY BASIN

Organisation: Lane Cove Tunnel Action Group

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Position: Chairman

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Date Received: 4/08/2006

Subject:

Summary

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The intent of this Submission is to highlight:

1. The growing body of international and Australian scientific evidence of the risks posed to the public in the Sydney Basin by traffic-related air pollution, especially coarse, fine and ultra-fine particles, gaseous irritants (e.g., O3 and NO2), and polycyclic aromatic hydrocarbons (PAH's) either alone or in combination, are known to be associated with, for example:

- inflammatory lung diseases e.g., asthma, bronchitis and alveolitis
- increased cardio-vascular disease
- increased risk of myocardial infarction in susceptible persons
- risk for exercise-induced heart damage
- limited blood flow and increased blood clotting
- increased mucous production and airway hyper-responsiveness
- 1/5 lung cancer deaths (USA) and accelerated tumour growth
- premature death
- symptoms of anaemia e.g., tiredness, headaches, fatigue and shortness of breath.
- low birth weight and small head circumference of neonate.
- intra-uterine growth retardation (for each 10 nanograms PAH's /M3 increase)
- certain leukaemias e.g., from exposure to benzene.
- loss in productivity, absenteeism from work and school.
- Increased sensitivity to bacterial products in airways
- more severe common viral asthma
- reduced male fertility
- significant risk of ovarian cancer from exposure to vehicle pollution
- adverse effects on lung development from the age of 10 to 18 years

The effect is a major increase in sickness-care costs to the State/nation's health budget. Costs of health impacts vary according to the value placed on a human life and in Australia ranges from $1.3 million to $7 million. Overseas studies place a value on a human life of Aus$ 8.16 million.

For Sydney, the cost varies according to many factors, including the density of population. In the CBD of Sydney, the total cost of all pollutants from industrial, commercial, domestic and mobile sources is in excess of $ 8 billion annually. Of this, between $2-3 billion is attributed to the impacts of vehicle pollution in Sydney.

Overseas reports showed 2/3 of health care costs due to pollution results when levels of pollution were below the Australian national standard for Particulate Matter (PM), less than 10 micrometre in diameter, i.e., PM10 of <50µg/M3/24 hours.

In drawing these life-threatening risks to the attention of the NSW Government, the author, as a matter of urgency, urges the Government to put in place clear policies in support of measures that immediately address and substantially reduce these risks to human health. These would include:

- Support for the increased production, distribution and use of ethanol as a clean renewable energy fuel;
- Active replacement of toxic fossil fuels (diesel and petrol) with ethanol or ethanol-blended petrol, liquid petroleum gas (LPG) and biodiesel.
- NSW Government urgently implement a policy to change its diesel-powered bus-fleet to LPG which is cleaner than even low-sulphur diesel with a pollution cost 10-fold less than dirty diesel.
- Reduction in highly toxic aromatics e.g., benzene and PAH's, in existing petrol and diesel.
- Installation of in-tunnel filtration and gas-detoxification systems in vehicular tunnels in heavily populated areas of Sydney

2. Review and address the inappropriateness of National air quality standards regarding Weighing particulates. Numbers and surface area of particles, rather than weight, are more accurate indicators of toxicity of fine particles in the emissions of motor vehicles powered by

petrol or diesel.

3. Address the financial impacts of air pollution on the NSW health system and why they are unsustainable.

4. Respond to the ineffectiveness of current laws and programs for mitigating air pollution that encourage unsustained economic growth with a trade-off in sickness and death.

5. Address the failure of the NSW Regulatory Authorities to apply or to enforce the relevant sections of the Environment Protection Act.

6. Implement meaningful strategies to reduce toxic pollution, mainly from the combustion of fossil fuels such as petrol and diesel.

7. Investigate how the bureaucracies form alliances with the corporate stakeholders to augment wealth and profit by shifting sickness-care costs to the community?

8. Examine how the NSW DEC (formerly the EPA) cheats on air-quality measures by not incorporating correction factors in the continuous readings of particulate pollution and by manipulating data e.g., by the removal of a monitoring station in the Sydney CBD, prior to the opening of the Cross City Tunnel?

9. Condemnation of the NSW Roads and Traffic Authority (RTA) who persistently trash community rights and entitlements, and for its callous disregard for community amenity, public health, and its general mismanagement of the road system.

10. Investigate why NSW Health fail to consider the known health significance of short-term (15 min.) peak exposures to toxic PM2.5 when on many days there are numerous short-term (15-min. average) excursions that are 3-4 times the measured 24 hr. average concentration.

Among the solutions, Government is urged to adopt a clear, non-partisan policy, in support of the expanded use of renewable and alternative fuels such as ethanol, biodiesel, and liquid petroleum gas (LPG) and compressed natural gas (CNG) would represent the introduction of known and proven measures to reduce these risks posed to human health by petrol and diesel fuels. Ethanol is particularly suitable as it is renewable and is not a carrier of toxic particles etc found in petroleum fuels such as petrol and diesel.

Evidence confirms that the oil cartels are unwilling to share the market of fossil fuels with cleaner alternative energy by a voluntary agreement. Therefore, a mandate requiring by 2020 that 20% of the fossil fuel sold at the pump be replaced with ethanol-blended petrol, of at least 10% and up to 85%, be made available. The need is urgent for many reasons.

LANE COVE TUNNEL ACTION GROUP INC.
ABN 89 787 371 833

SUBMISSION

to

NSW PARLIAMENTARY INQUIRY INTO THE HEALTH IMPACTS OF AIR POLLUTION IN THE SYDNEY BASIN

by

Dr. Ray KEARNEY

The University of Sydney, 2006

3 August, 2006

Contents

Terms of Reference of the Inquiry

(a) Changes in the emissions of various air pollutants and the impact of those changes on air quality in the Sydney basin over the past three decades, including any 'hot-spots' where pollution is concentrated

(b) Changes in the emissions of various air pollutants and the impact of those changes on air quality in the Sydney basin over the past three decades, including any 'hot-spots' where pollution is concentrated

(c) The causes of air pollution in the Sydney basin over the past three decades

(d) The health impacts of air pollution on any 'at risk' groups

(e) The financial impacts of air pollution on the NSW Health system

(f) The effectiveness of current laws and programmes for mitigating air pollution

(g) Strategies to reduce the health impacts of air pollution; and

(h) Any other relevant matter

Summary

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General comments:

Unfortunately, what we see in NSW is the art of perpetuating a health hazard where "denial" of the hazard by an expert may not imply – *"the truth, the whole truth and nothing but the truth"*. The general topic is the subject of an excellent paper by M. Greenberg (*J. Occup. & Environ. Med.* 2005;Vol 47: 137-144).

To date, the NSW Health, RTA, EPA (now DEC) and Planning (now DIPNR) appear to adopt the **same strategies** used successfully to support the use of white asbestos (chrysotile) as a safe product.
The same techniques are being used to subvert the community e.g., by the RTA Public Relations Unit, into thinking exposure to vehicle pollutants e.g., in the M5 East tunnel, is without risk to health and well-being. It can be readily inferred from the highly critical Katestone Review of the NSW Health Report on the M5 East Health Study that, as with the asbestos scandal, a "denial" of the hazard of an agent by its protagonists, no matter how distinguished, may not correspond with *"the truth, the whole truth and nothing but the truth."*

The conclusion of NSW Health's findings seems consistent with a popular form of "denial" used by the advocates of asbestos and runs like*: "We did not find the evidence for a causal association between an agent and its alleged effects"* when the evidence is based on such factors as:

- Unsound "negative" results derived from flawed data, methodology and study-design.
- Concealment of data that effectively removes scientific rigour and renders a reviewer powerless.
- Sampling (or questionnaire) is not properly conducted in the true exposure and breathing zones.
- Subverting the thinking of people by the release of false information, rather than a disclosure of the true facts publicly.
- Deliberately avoiding definitive answers to a number of important questions by failing to establish and operate a long-term sampling strategy for determining the qualitative and quantitative measures of hazard exposure of subjects in the study.

- Keeping opinions to themselves, when confronted with the facts, allowing government or industry agents to effectively operate a policy of concealment by silence in the face of error while evidence of proven causal effects is kept confidential by agreement with management.
- Early denial is given authority when made by government or industry medical officers or by some medical consultants and others, often with 'conflicts of interest'. The significance of the hazard is down-played with a "so what?" attitude.
- Claiming to adopt "world's best practice" to imply, falsely, there are no risks to health.
- Omitting significant numbers of workers (receptors) and thereby introducing a 'negative' bias.
- Applying inappropriate standards or methods to effectively minimize the concentration of the hazardous agent in the exposure.
- By initiating an 'epidemiological survey', as a ploy, when faced with a health problem, or to simply ignore the problem. It buys time, similar to RTA's hoax 'filtration trials.'
- Deliberately terminating studies at a stage when findings are suggestive.
- Failing to adopt Precautionary Principles to contain the toxic agent by not installing adequate environmental control technology.
- Suppressing highly critical 'audits of performance' for political expediency.

There have been too many studies world-wide which directly link vehicle emissions with mortality and morbidity for NSW Health to engage in a study where they would not be able to find the associations between stack emission and community health It is now evident that these strategies used to hide the public health hazards of asbestos for over a century also feature in the techniques adopted by NSW Health to perpetuate the **myth** to the NSW Government and its bureaucrats that the exhausting of vehicle pollutants from tunnel stacks, in residential areas, poses no health risk, either short or long term, for anyone.

In a recent document headed, *'Fuel Taxation Inquiry: The Air Pollution Costs of Transport in Australia'*, by Watkiss (2002) submitted to the Federal Government, data shows that for the inner parts of Sydney (covering 2.5 million), the annual health 'pollution' cost is about $342,000 per tonne of particles and $1750 per tonne for oxides of nitrogen (NOx). When this information is applied to current stack pollution from the **unfiltered** M5 East tunnel, the annual health-costs are about $6 million. For the Lane Cove Tunnel (LCT) and using under-reported LCT EIS stack-pollution data for 2006, the annual health-costs alone are about $5 million.

It is high time lessons from asbestos, tobacco, exposure to radiation and the like are learnt and as the Hon Ms Sandra Nori, a Government Minister in the NSW Government and Member for Port Jackson said recently that action *"must be taken to protect our communities from the impact of car emissions by using the latest and best tunnel filtration technology available".* Ms Nori should know the health impacts of vehicle emissions as she is Secretary of the ALP's Air Pollution Task Force.

An opinion and a call for action:

If we were assembled to deliberate on such life and death issues as **capital punishment** and **abortion** the process of discourse and the elements of reason would be different. The issues under discussion in this Submission are also about life and death.

Indeed, it is perhaps quite ironic in this so called 'age of science and materialism' that probably never before have ordinary men and women, including scientists, as well as politicians, been confronted with so many moral and ethical problems. Scientists, on the one hand, stress and seek objectivity whilst in the arts, religion and philosophy, by contrast, the emphasis is upon subjectivity, i.e., the experience through the individual conscious.

Thus, whether or not something is good or beautiful or right in a moral sense, for example, cannot be determined by the scientific method. Science is thus limited to what is **observable and**

measurable. Theories about precisely **how** pollution affects health and well-being maybe shattered, but with additional knowledge, new theories are found. However, the recorded observations endure. Moreover, such observations are used over and over again. The extensive scientific/medical literature documents the numerous observations that episodes of air pollution are positively associated with enhanced mortality and acute/chronic illness in urban populations.

Because of this emphasis on **OJBECTIVITY**, value judgments cannot be made in science in the way such judgments are made in religion, philosophy and the arts.

However, in a **moral sense** whether it is right to install filtration systems in a stack – any stack – or to replace toxic fossil fuels with clean renewable fuels is a problem solved not only by a **value judgment** but also these decisions can be greatly assisted in this case by the **scientific method**.

Therefore, the problem that now confronts us in this forum, in my view, can and should be solved **both** by **value judgments** AND by the **scientific method**. This issue then is one that transcends party politics.

There is no difficulty, as one familiar with the air-pollution and stack-filtration issues for over a decade for any person of integrity in telling the "good guys" from the "bad guys" especially in the saga of events recounted in recent years.

The regulatory authorities and in particular the RTA have isolated and quarantined new filtration systems and technologies which have arisen abroad, especially when such technologies have been contrary to the prevailing political point of view.

These technology decisions, it would appear, have also been assisted by comments, without proof of evidence, from the associated regulatory authority – the NSW Health Department. Despite all the posturing about new engine designs and better fuels, the combustion engine continues to belch out pollution and fill the air with noxious particles and chemicals.

It is clear that for some time there has been an organised campaign, orchestrated principally through the Roads Ministers Office and the RTA against filtration technology. This was evident in the Report of the 2003 Parliamentary Inquiry on the Ventilation of the M5 East Stack when the Chairman, the Hon. Richard Jones wrote in the Forward:

"The single stack as currently planned will concentrate the tunnel emission into one source and add to the pollutant load of the valley. The adverse health effects of this increased pollution on the surrounding community must be acknowledged, but the RTA fails to do so."

The predominant culture of any society or political system is **not a conspiracy**, it is a taken-for-granted acceptance of many spoken and unspoken precepts. The RTA states, without objective proof, filtration systems have not been shown to be effective. The NSW Health declares – there is no risk to health or well-being – again, without objective evidence of proof. The EPA oversees a monitoring program that fails to disclose the size and number of ultra-fine particles generated locally but resorts to irrelevant averages of Sydney's regional airshed. This all helps to under-estimate exposure by up to 35%.

<u>Such a prevailing political and patronising culture is more powerful than any conspiracy.</u>
A conspiracy can be tracked down, found out, divided and broken. The deep weave of cultural and political patronage that currently exists among the regulatory authorities is difficult to unpick. No single individual or group of individuals can be easily brought to book or held responsible for the collective representation. No single statement or set of statements outlines their code. This cultural and political patronage does not have an easily identifiable beginning or an end. It simply is: engrained within the consciousness of each individual – the senior advisor, the project manager, the political spokesperson or even the Consultant. It goes on largely unquestioned, however bizarre its consequences and with an indifference to the objective analysis of scientific and medical evidence.

Observations confirm that the RTA-managed Workshop in June, 2000 had little to do with a search for the truth and a great deal to do with the confirmation of prejudice i.e., *"tunnel stacks do not require filtration – the technologies do not work and there is no risk to health."* Nothing could be further from the truth!

In addressing these issues of toxic fossil fuels, air pollution and the question of filtration in stacks and tunnels there is clearly a need for Government to apply **honesty** as well as **objectivity** to the appraisal of the technologies as well as to the scientific and medical evidence for health risk.

This should not be, as it is at the moment, a dickering, by the NSW Government, its Ministers and the statutory authorities, **on the margins of a duty of care**, and an endless and irrelevant discussion about whether or not more research is needed to be undertaken.

The **totality of the evidence** is, beyond reasonable doubt, in favour of replacing harmful fossil fuels with clean re-newable fuels. Equal to this is the proven evidence that confirms installing filtration is obligatory for Government, to unanimously endorse the installation of filtration technology in tunnels and/or in stacks as a responsible '**duty of care**'.

Key words: vehicle exhaust pollution, adverse health effects, acute lung injury, air-pollution monitoring, health-risk flaws, alcohol and other biofuels.

Declaration

This Response has been undertaken with the best of my ability and knowledge, based on materials, documents available and current information, as well as over 15 years involvement with the Lane Cove Tunnel Project from its earliest beginnings.
During the past 11 years, I have been Chairman of the Lane Cove Tunnel Action Group Inc (LCTAG).
Its presentation is true and does not, by its presentation of information or omission of information, materially mislead or intend to materially mislead.

Ray Kearney

The Lane Cove Tunnel Action Group Inc (LCTAG)

LCTAG is a coalition of 16 groups drawn from the residential, commercial and industrial sectors of Lane Cove. The Action Group was formed over ten years ago to seek the construction of a 3.7km, bore-driven, twin three-lane tunnel with the installation of air-cleaning technologies (electrostatic precipitators and activated carbon beds) that also negate the need for unsightly ventilation stacks.

A corner stone of the Lane Cove Tunnel Action Group's (LCTAG) mission is to achieve the construction of a twin three-lane 3.7km continuous tunnel with the installation and operation of air-pollution treatment systems to reduce adverse health impacts. This objective is based upon independent expert advice from consultants in tunnelling design and construction as well as from air-filtration consultants at both national and international levels. Our commitment to this mission is also based upon the inescapable fact that vehicle emissions are injurious to the health of everyone exposed to them. "At risk" persons such as children, asthmatics, the elderly and pregnant women are especially affected by both short and cumulative exposures, even below prescribed National standards.

It was indeed the LCTAG who **proposed**:

- The continuous long bore-driven tunnel as the most acceptable alternative to RTA's proposal to widen Epping Road or to construct a short 'cut and cover' tunnel.
- The current route of the present LCT.
- The most suitable and recommended site for the western tunnel exit/entrance or portal being on the RTA land at the intersection of Mowbray Road and Epping Road. However, in typical spite, to thwart the proposal, the RTA sold its land and in arrangements with the City of Willoughby Council had the site re-zoned to 'residential' to make way for a chosen developer to build townhouses on the site. The naivety of the RTA in its fit of pique was realized when the LCTAG tunnel proposal was independently published, by us, in the *North Shore Times* that forced the former Roads Minister Carl Scully to finally accept the LCTAG proposal. Today, the RTA is left with the only option to carve into the middle of Epping Road to build the western portal. By doing so the RTA had to compromise a needed 3-lane entry with only 2-lanes as they had already sold their land for townhouses. Thus, the RTA today has compromised surface traffic on Epping road by destroying more lanes from the surface road. Therein is the truth to the background of why the east-bound tunnel carriage-way begins with a compromised 2-lane entry, rather than the 3-lane entry, originally recommended by LCTAG on expert engineering advice?

The LCTAG is also part of a coalition with the M5-East Residents Against Polluting Stacks (RAPS) as well as those affected by the Cross City Tunnel i.e., Sydneysiders Against Polluting Stacks (SAPS) and the Cross City Tunnel Action Group (CCTAG). This coalition of concerned communities continue to campaign under the name of Groups Against Stack Pollution (GASP).

Background to Submission Title:

Issues of air pollution related to health impacts

- Traffic-related air pollution remains a key target for public-health action overseas including Europe, Britain, USA and India. Australia has been slow to act e.g. unlike overseas, Australia

does not have health standards for a number of pollutants derived from the combustion of fossil fuel, e.g., carcinogenic 1,3-butadiene and toxic acetaldehyde. Only recently, it introduced a benzene standard (3ppb) while UK has a benzene standard for years that is now 1ppb. However, the States are given jurisdiction over the interpretation and application of the NEPC air-quality standards. For example, the NEPC state that the NEPM for PM_{10} do not apply to canyons between tall buildings or to point sources, yet, NSW EPA and RTA, Health and DIPNR (Planning) apply them to point sources such as pollution from tunnel stacks.

- In a major study in Austria, France and Switzerland by Kunzli et al (2000), air pollution caused 6% of total mortality or more than 40,000 attributable cases per year. Traffic pollution accounted for more than 25,000 new cases of chronic bronchitis (adults); more than 290,000 episodes of bronchitis (children); more than 0.5 million asthma attacks; and more than 16 million person-days of restricted activities.
- Toxic diesel particulates account for at least 21,000 deaths annually in USA (CATF Report, Feb '05).
- In Sydney, twice as many people die from vehicle exhaust than die from road accidents. Cost of morbidity and mortality due to vehicle pollution in Sydney alone exceeds $2 billion annually.
- Vehicle emissions account for up to 65 % of urban air pollution. Most of the particulate pollution is in the fine mode $PM_{2.5}$ fractions that are soluble in the lungs and have PAH's adsorbed to their surfaces.
- In most States of Australia, the toxic fine particles in the soluble PM2.5 mode are EXCEEDING the air-quality standards/guidelines set by the National Environment Protection Council (NEPC) and are generally increasing annually.
- Weight-for-weight, fine particles in fossil fuel combustion have an enormous surface area. For example, one billion 0.01 µm particles are equivalent to one PM_{10} (10µm) particle but have 1000-times the surface area, hence an enormous carrying capacity for their carcinogenic cargo of PAH's. International experts - Professors Lidia Morawska and Michael Moore - of Queensland, discuss fine and ultra-fine particles in their excellent review found at the following link:

http://www.deh.gov.au/atmosphere/airquality/publications/health-impacts/index.html

It is highly relevant that in Section 1.3 under 'Toxicology' they make the following profound statement:

"All of the studies available to us demonstrate that the primary determinant of the effect of ultrafine particles is their number and their surface area and not the weight of particles present. This means that the traditional use of PM weight measures is inappropriate in evaluation of the likely biological effects of ultrafine particles."

Fine and ultra-fine particles are the predominent particles in the tail-pipe of vehicles powered by the combustion of petrol and diesel.

Seaton (1996) states:

> *". . . particles generated by combustion the size distribution in terms of numbers of particles are predominantly very small, with two peaks at around 30nm (0.03µm) and 100nm (0.1µm). The ultra-fine particles below about 50nm have a relatively short life, condensing into larger (but still very small) ones"*

Kittelson (1998) shows that the bulk of the **alveolar deposition** is in the range below 0.1µm i.e., 90% of the number and 30% of the mass is formed during exhaust dilution from particle percursors that are in the vapour phase of the tail-pipe. New particles are formed by nucleation. This is likely to be the source of most of the respirable ultra-fine and

nano-particles (and particle number) associated with engine exhaust .

The point is that weighing particulate pollution, as currently done, is inappropriate for determination of health risk. The other factor is the coarse particles are mainly insoluble. In contrast, fine particles are soluble in the respiratory tract and release chemical carcinogens.

- Does TEOM PM_{10} monitoring include 'secondary particles' formed from vehicle emissions?

  (a) "*Such particles cannot be correctly measured by the TEOM method*" (Filliger *et al*, 1999).

  (b) In France (Filliger *et al*, 1999) when TEOM PM_{10} measurements are used for background, to introduce the missing secondary particles into the analytical model, a constant correction value of $9.5\mu g/M^3$ is added to all grid points. The value of $9.5\mu g/M^3$ corresponds to regional background secondary particles as estimated for France by the European scale EMEP model (EMEP, 1997). This is a conservative correction factor, because the urban-scale portion of the secondary particles is ignored. As a consequence, the PM_{10} levels (by TEOM) in studies in France indicate a **minimum level of PM_{10}.**

  Note: NSW Regulatory Authorities FAIL to incorporate any correction factor and hence underestimate levels of particulate pollution by continuous monitoring TEOM method.

- What are the implications of the failure to incorporate correction factors for continuous TEOM PM_{10} monitoring for Planning NSW?

  The failure of NSW EPA and RTA to incorporate correction factors into the continuous TEOM PM_{10} measurements has implications of conditions of Planning NSW for exceedances because TEOM PM_{10} data under-estimate the pollution. Planning NSW state:

  "An exceedance is the occurrence of a 24 hours running average ground-level concentration (at any location) of fine particulates (PM_{10}) over the threshold of $50\mu g/M^3$.

  Generating a reliable **24-hour running average** requires continuous monitoring With logging of concentrations at a fine time resolution (better than one hour and preferably 5-10 minutes).

- Alternative fuel use will continue to be driven by concerns over clean air and legislation to reduce exhaust emissions. However, only lip-service is paid as illustrated by the NSW Government's decision to replace its cleaner LPG-powered bus fleet with a new bus fleet powered by dirty diesel.
- Regarding low-sulphur diesel (not petrol), it is important to note that whilst removing sulphur reduces the pollutants generated in combustion, the lubricant qualities are also removed with the sulphur. See the follow links:

  http://www.mbm.net.au/b100/lsd.html
  http://www.abc.net.au/rural/vic/stories/s232316.htm
  http://www.bebioenergy.com/impact.htm
  http://en.wikipedia.org/wiki/Ultra-low_sulfur_diesel

  The experience in USA is that engine wear significantly shortens the engine life, when powered by ultra-low sulphur diesel. The problem that has arisen is when conventional lubricants are added back to the low-sulphur diesel it negates the benefits of lower pollution. Therefore, there needs to be legislation to control the type of lubricant added to low-sulphur

diesel. Canola oil is a good additive to low-sulphur diesel.

The alleged claim that within a few years there will be little difference between conventional and alternative fuels in terms of emissions, does need to be qualified. There is no question that natural gas and LPG are among the cleanest conventional fuels. The gases burn far more cleanly than petrol or diesel.

- In USA, but not yet in Australia, diesel is classified as an air toxic.
- Diesel exhaust poses a cancer risk that is 7.5 times higher than the combined risk from all other air toxics (CATF Report, Feb '05).
- The risk of lung cancer for people living in urban areas is three-times that for those living in rural areas (CATF Report, Feb '05).
- A recent Case Study (June, 2005) established that the reduction of particle pollution in Tokyo was accompanied by a significant cost-reduction of over $A40 billion in mortality and morbidity.
- Ethanol as a 10% addition to petrol can reduce toxic particulates by up to a qualified 50%, more in older cars (G. Whitten). See following links:
  http://www.ethanolrfa.org/white991.html
  http://www.ethanolrfa.org/NEC-Whitten.pdf
  http://www.ethanolrfa.org/pubs.shtml
  See also P. Mulawa etal. *Env.Sci. & Technology* Vol 31 p 1302 (1997)

Term of Reference (a)
Changes in the emissions of various air pollutants and the impact of those changes on air quality in the Sydney basin over the past three decades, including any 'hot-spots' where pollution is concentrated

The following is copied from the NEPC website to illustrate the Summary of pollutants reported by NSW EPA to the NEPC. The link is:
http://www.ephc.gov.au/pdf/annrep_01_02/124-135_Jur_Rep_AAQ_NSW.pdf

Report to the NEPC on the implementation of the National Environment Protection (Ambient Air Quality) Measure for New South Wales by the Hon Bob Debus MP, Minister for the Environment for the reporting year ended 30 June 2002

Note the usual excuse to explain NSW particle exceedances that bushfires are mentioned. In reality such is not the case for all exceedances when bushfires are excluded from the PM 2.5 data.

Summary

The results show all regions are in compliance with the Air NEPM goal (prior to the established timeframe of 2008) for the pollutants carbon monoxide, sulfur dioxide, nitrogen dioxide and lead. The goal for photochemical oxidants (measured as ozone) was not met at eight sites in the Sydney region and one site in the Illawarra region. The goal for particles (as PM_{10}) was not met at one site in the Sydney region. Severe bushfires in December 2001 were a major influence on particle levels in the reporting period.

Analysis of the extent to which standards are, or are not met

Photochemical oxidants (as ozone) and particles (as PM_{10}) are the only pollutants for which exceedences of the standards were recorded in 2001. Under the NEPM goal for these standards maximum allowable exceedences are respectively one day per year for ozone and 5 days a year for particles (as PM_{10}).

Ozone standards

Both the 1–hour and 4–hour ozone standards were exceeded at all nine operational NEPM sites in the Sydney region, with only one site being limited to the one allowable exceedence day under the NEPM goal. The conditions associated with the bushfires in December 2001 gave rise to a number of ozone events. If the bushfire days are excluded, there were seven sites at which the goal was not met. The highest recorded 1–hour and 4–hour averages for the period were 175 and 170 per cent of the standard respectively. These did not occur during the bushfire period. At one site, there were nine days on which the 1–hour standard was exceeded and twelve days on which the 4–hour standard was exceeded. Three of these days were during the bushfire period.

In the Illawarra region, the three NEPM sites all experienced exceedences of either the 1–hour or 4–hour standard. At two of these sites there was only one day on which the standard was exceeded. Hence there was only one site where the NEPM goal was not met and the exceedences at this site occurred during the bushfire period. The maximum values were about 15 to 20 percent above the standards. There were no exceedences recorded in the lower Hunter region or at the Bathurst site, with maximum levels about 90 and 75 percent of the 4–hour standard respectively.

Particles standard

While the bushfires had only a minor impact on the results for ozone, they were a major influence on particle levels, with all recorded exceedences of the PM_{10} standard in the Sydney region occurring during the late December bushfires. All six operational NEPM sites in the Sydney region recorded exceedences. However, all sites were within the NEPM goal of five allowable exceedences. The maximum recorded value during the period was two and a half times the standard. For the non–bushfire period, the maximum value was about 83 per cent of the standard.

Both the Illawarra and lower Hunter regions recorded exceedences of the PM_{10} standard, some of these occurring during the December bushfires. The NEPM goal was met at all sites in these two regions. In regional NSW, monitoring of PM_{10} was carried out at four sites during 2001. The standard was exceeded on two days at Wagga Wagga, while there were no exceedences recorded at Tamworth, Bathurst or Albury. The NEPM goal was met at Tamworth, however, compliance with the goal cannot be demonstrated at the other three sites because technical difficulties with instruments resulted in a low availability of valid data.

Other pollutants

Levels of all other pollutants are well below the standards. Averages of sulfur dioxide are generally less than 20 per cent of the standards, with the exception of one site in the Illawarra region where a 1–hour average about 80 per cent of the standard was recorded on one occasion. The highest 8–hour average for carbon monoxide recorded at the peak Sydney CBD site was about 58 per cent of the standard. All other sites recorded lower levels. For nitrogen dioxide, the highest 1–hour average was about 58 per cent of the standard, while annual averages were generally less than 50 per cent of the standard.

Ambient levels of lead were low at all sites where monitoring was undertaken, less than 10 per cent of the standard. With the banning of leaded petrol, which came into force in January 2002, the primary source of lead in air, on the regional scale, has now been eliminated. Ambient levels of lead are now frequently below detection limits and it is envisaged that in the near future, there will no longer be a need to routinely monitor lead.

Diesel Vehicle Fleet Profile

NSW RTA registration data shows that diesel vehicles constituted 8.5% of the NSW fleet at 30 June 2002.
Table 1 below gives detail. Light commercial vehicles (LCVs) constitute the largest sector of the diesel fleet at 48%. Heavy trucks (rigid diesel vehicles) account for 27% of the diesel fleet. Together these categories account for 75% of the total diesel fleet in NSW
Copied from NEPC link at:
http://www.ephc.gov.au/pdf/annrep_01_02/244-248_Jur_Rep_Diesel_NSW.pdf

Table 1: Diesel vehicles by category as proportion of total fleet and diesel fleet

	Diesel Vehicles							
NSW – June 2002	**Passenger Vehicles**	**Off-road Passenger Vehicles**	**Light Commercial Vehicles**	**Heavy Trucks**	**Prime Mover**	**Small Buses**	**Buses**	**Total**
% of category – NSW fleet	0.21	16.57	27.73	87.64	97.99	15.02	91.04	**8.48**
% NSW diesel fleet	1.75	13.84	48.33	26.72	4.34	1.73	3.29	**100**

Source: NSW RTA registration data June 2002.

Motor Vehicles Emissions Estimates

Motor vehicles contribute approximately 82% of oxides of nitrogen (NO_x), to the atmosphere in the Sydney Region and 31% of particles (as PM_{10}). Diesel vehicles make a disproportionately high contribution to air pollution from the transport sector even though they constitute only approximately 8.5% of the vehicle fleet in NSW and 7% of the fleet both in the Sydney region and in the GMR. EPA modelling of motor vehicles emissions estimates that in the year 2000, diesel vehicles contributed approximately 22% of NO_x and 51% of PM_{10} emissions from all vehicles in the GMR.

Emissions from diesel vehicles are predicted to fall from 2005 to 2010 as the more stringent Euro diesel vehicle emissions standards start to apply from 2002 and low sulphur and ultra–low sulphur diesel become available from January 2003 and January 2006 respectively.

The following is extracted from the Executive Summary of the NEPC Report, April 2004, found at:
http://www.deh.gov.au/atmosphere/airquality/publications/status/index.html

PM_{10}

- The 2001 peak levels are above the NEPM standard at most sites. But, only Launceston records more than the five allowable exceedences.
- Due to relatively short data periods, trend results are not conclusive. No significant trends are detected, but the tendencies appear to be mostly downward.

$PM_{2.5}$

- The 2001 peak $PM_{2.5}$ levels are above the advisory reporting standards at the four jurisdictions (NSW, Victoria, Queensland and WA) that provided data.
- Due to monitoring at a limited number of sites and the short data periods at most of these sites, trends cannot be estimated with confidence. Nevertheless, data indicate a statistically non-significant upward tendency at most of the NSW sites and at the two Queensland sites, and mostly a downward tendency in WA and Victoria.

Note: The Report highlights the upward tendency for PM2.5 in NSW (Sydney Region) to exceed the reporting standard.

The following 'Compliance with Reporting Standard for PM2.5' has been cut/pasted from 2004 NSW Annual Compliance Report at the following link:
http://www.ephc.gov.au/pdf/Air_Quality_NEPM/Monitoring2004/nsw_compliance_rpt_2004_final.pdf

Note: Confirmation of the Annual Average PM2.5 standard ($8\mu g/M^3$) is NOT MET for each of the monitoring sites in the Sydney Basin, despite the explanation that it was due to a 'closure of monitoring station'.
Remember, these are based on 24-hour averages that hide peak levels that in a.m. and p.m. peak traffic periods and can exceed the 24-hour average by 3-4 fold. Overseas 15 minute high excursions have been shown to impact adversely on health.

Compliance with reporting standards

The variation to the Ambient Air Quality NEPM (2003) states that values obtained using the TEOM method for $PM_{2.5}$ "cannot be used for comparison with the advisory reporting standards until the outcomes of the $PM_{2.5}$ Equivalence Program have been formally included in the Principal Measure."

This report only presents data obtained by TEOM monitors. These data are compared to the advisory reporting standard for $PM_{2.5}$ purely for interest.

Table A3: Summary of compliance with ARS for $PM_{2.5}$ in New South Wales - 2004

Advisory Reporting Standard
25 µg/m³ (24-hour average)
8 µg/m³ (Annual average)

Region/ Performance monitoring Station	Data availability rates (% of hours)					Number of Exceed-ences (days)	Annual mean (µg/m³)	Performance against the standard	
	Q1	Q2	Q3	Q4	Annual			24-hour	1-year
Sydney									
Chullora	85.7	89.0	92.4	89.1	89.1	0	**8.7**	Met	Not met
Earlwood	95.6	97.8	91.3	100.0	96.2	0	**10.8**	Met	Not met
Liverpool	85.7	92.3	62.0	100.0	85.0	10	**12.5**	Not met	Not met
Richmond	100.0	96.7	100.0	90.2	96.7	2	**9.6**	Not met	Not met
Westmead	100.0	92.3	29.3	00.0	55.2	8	**13.8**	Not met	Not met
Woolooware	92.3	98.9	65.2	00.0	63.9	0	**9.9**	ND	Not met
Illawarra									
Warrawong	100.0	94.5	94.6	87.0	94.0	2	**11.4**	Not met	Not met
Wollongong	98.9	91.2	100.0	98.9	97.3	2	**9.8**	Not met	Not met
Lower Hunter									
Beresfield	95.6	98.9	93.5	72.8	90.2	1	**11.0**	ND	Not met
Wallsend	91.2	63.7	94.6	100.0	87.4	1	**9.9**	ND	Not met

Bold font indicates values that exceed the AAQ NEPM advisory reporting standard

All stations above recorded exceedences of the advisory reporting standard annual standards during 2004. Seven stations exceeded the 24-hour advisory reporting standard: Liverpool, Richmond, Westmead, Warrawong, Wollongong, Beresfield and Wallsend. Liverpool recorded ten days that exceeded the 24-hour standard and also recorded the highest annual average of 12.5µg/m³. The closure of the Westmead and Woolooware stations meant that the data availability criteria were not met at these stations, hence the annual averages given for these stations are not truly representative of the annual average.

Data analysis

Table A4: Summary for $PM_{2.5}$ - Daily maximum 24-hour average concentrations (2004)

Region/ Performance monitoring Station	Data availability rates (%)	Number of valid days	Maximum values ($\mu g/m^3$)			
			Highest Value	Highest Date	2nd Highest Value	2nd Highest Date
Sydney						
Chullora	89.1	326	24.5	10-Jan	22.1	07-May
Earlwood	96.2	352	24.4	01-Jun	23.3	26-Mar
Liverpool	85.0	311	**41.8**	09-Jan	**39.8**	08-Jun
Richmond	96.7	354	**26.9**	24-May	**26.8**	23-May
Westmead	55.2	202	**36.2**	09-Jan	**31.5**	10-Jan
Woolooware	63.9	234	21.5	16-May	20.8	02-Jun
Illawarra						
Warrawong	94.0	344	**26.8**	10-Jan	**25.8**	26-Mar
Wollongong	97.3	356	**26.7**	10-Jan	**26.2**	21-Feb
Lower Hunter						
Beresfield	90.2	330	**31.7**	08-May	24.0	14-May
Wallsend	87.4	320	**26.9**	08-May	21.9	07-May

AAQ NEPM advisory reporting standard - 25µg/m³ (24-hour average)

Bold font indicates values that exceed the AAQ NEPM advisory reporting standard

Table A6: Statistical summary for $PM_{2.5}$ - Daily 24-hour average concentrations (2004)

Region/ Performance monitoring Station	Data availability rates (%)	Maximum conc. ($\mu g/m^3$)	Percentiles (ppm)						
			99th	98th	95th	90th	75th	50th	25th
Sydney									
Chullora	89.1	24.5	19.4	18.2	16.1	14.2	10.9	7.9	5.8
Earlwood	96.2	24.4	22.2	21.3	18.6	16.5	13.3	10.0	7.5
Liverpool	85.0	**41.8**	**29.2**	**25.8**	20.8	19.3	15.1	11.7	8.5
Richmond	96.7	**26.9**	23.5	20.4	17.5	15.0	11.7	8.9	6.5
Westmead	55.2	**36.2**	**26.6**	**26.2**	23.7	20.0	16.5	12.9	10.0
Woolooware	63.9	21.5	19.7	18.8	16.5	15.4	12.2	9.4	7.0
Illawarra									
Warrawong	94.0	**26.8**	23.6	22.1	20.7	17.9	14.2	10.4	8.0
Wollongong	97.3	**26.7**	21.7	20.6	16.9	15.8	12.2	9.1	6.7
Lower Hunter									
Beresfield	90.2	**31.7**	23.6	23.0	19.9	16.5	12.9	10.3	7.8
Wallsend	87.4	**26.9**	20.1	18.3	16.4	14.3	11.6	9.0	7.3

AAQ NEPM advisory reporting standard - 25µg/m³ (24-hour average)

Bold font indicates values that exceed the AAQ NEPM advisory reporting standard

Trend data

Annual averages and annual maximum 24-hour averages for all stations are given below.

Table A7: Maximum 24-hour average concentrations for $PM_{2.5}$ ($\mu g/m^3$)

Region/ Performance monitoring Station	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004
Sydney										
Chullora									81.0	24.5
Earlwood		22.6	**39.3**	**33.4**	**27.6**	**35.4**	**81.7**	**56.1**	**39.4**	24.4
Liverpool				**26.5**	**25.4**	**45.1**	**118.6**	**89.2**	**50.1**	**41.8**
Richmond		17.2	**51.3**	**40.3**	**33.1**	17.0	**101.3**	**98.2**	**61.9**	**26.9**
Westmead				**29.6**	**25.3**	**31.4**	**91.6**	**59.1**	**67.8**	**36.2**
Woolooware				20.5	23.2	**33.2**	**81.9**	**87.3**	**67.7**	21.5
Illawarra										
Warrawong		**31.7**	**37.1**	**27.0**	19.9	**32.6**	23.2	**85.1**	**160.3**	**26.8**
Wollongong				18.8	19.4	**31.1**	**53.4**	**93.8**	**112.5**	**26.7**
Lower Hunter										
Beresfield				19.2	21.4	**34.1**	**66.4**	**50.4**	**40.9**	**31.7**
Wallsend		14.1	**43.4**	**38.2**	21.9	**61.5**	**56.4**	**59.6**	**34.1**	**26.9**

AAQ NEPM advisory reporting standard - 25µg/m³ (24-hour average)

Bold font indicates values that exceed the AAQ NEPM advisory reporting standard

Table A8: Annual average concentrations for $PM_{2.5}$ ($\mu g/m^3$)

Region/ Performance monitoring Station	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004
Sydney										
Chullora									11.2	8.7
Earlwood		**9.1**	**10.2**	**10.3**	**10.2**	**10.3**	**11.6**	**12.8**	**11.0**	**10.8**
Liverpool				**10.1**	**9.6**	**10.4**	**11.8**	**15.2**	**13.6**	**12.5**
Richmond		6.8	7.9	6.4	6.5	7.0	**10.7**	**11.5**	**10.0**	**9.6**
Westmead				**10.3**	**9.9**	**9.9**	**12.2**	**13.1**	**11.4**	**13.8**
Woolooware				7.9	**8.1**	**9.5**	**11.1**	**11.7**	**10.7**	**9.9**
Illawarra										
Warrawong		7.6	**8.7**	**8.8**	**8.3**	**9.1**	**9.9**	**12.7**	**12.0**	**11.4**
Wollongong				7.7	8.0	**8.3**	**9.4**	**11.5**	**10.5**	**9.8**
Lower Hunter										
Beresfield				**8.2**	**8.8**	**8.8**	**12.4**	**13.6**	**9.4**	**11.0**
Wallsend		7.3	**9.6**	**8.5**	**8.0**	**8.4**	**10.2**	**11.3**	**9.8**	**9.9**

AAQ NEPM advisory reporting standard - 8µg/m³ (annual average)

Bold font indicates values that exceed the AAQ NEPM advisory reporting standard

The following Tables also illustrate exceedances of PM2.5, 24 hour and Annual Averages, in Sydney, and are copied from the Final Report (September, 2002) titled *'Exposure Risk and Exposure Standard for the Development of a PM2.5 Standard'* by M. Burgers and S. Walsh

Table E1c. 24-Hour $PM_{2.5}$ results for Sydney, 2000.

	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
Maximum 24-hour PM2.5 (µg/m3)	45.1	45.1	33.2	*	*	35.4	37.6
# Days > 20 µg/m3	12	3	7	*	*	6	6
# Days > 25 µg/m3	5	2	3	*	*	3	3
# Days > 30 µg/m3	2	1	2	*	*	2	2
# Days > 35 µg/m3	2	1	0	*	*	1	1

(*) insufficient data (< 75% of the year) available

Table E1d(1). 24-Hour $PM_{2.5}$ results for Sydney, 2001 (December 2001 bushfires excluded).

	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
Maximum 24-hour PM2.5 (µg/m3)	24.4	24.8	23.7	*	*	26.6	23.3
# Days > 20 µg/m3	9	2	7	*	*	19	3
# Days > 25 µg/m3	0	0	0	*	*	2	0
# Days > 30 µg/m3	0	0	0	*	*	0	0
# Days > 35 µg/m3	0	0	0	*	*	0	0

(*) insufficient data (< 75% of the year) available

Table E1d(2). 24-Hour $PM_{2.5}$ results for Sydney, 2001 (December 2001 bushfires included).

	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
Maximum 24-hour PM2.5 (µg/m3)	118.6	82.9	81.9	*	*	81.7	93.7
# Days > 20 µg/m3	15	7	9	*	*	26	10
# Days > 25 µg/m3	6	4	2	*	*	8	7
# Days > 30 µg/m3	6	4	1	*	*	6	7
# Days > 35 µg/m3	6	4	1	*	*	5	6

(*) insufficient data (< 75% of the year) available

Table E1e(1). 24-Hour $PM_{2.5}$ results for Sydney, 1999-2001 (December 2001 bushfires excluded).

3 Year Period	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
Maximum 24-hour PM2.5 (µg/m3)	45.1	45.1	33.2	*	*	35.4	37.6
# Days > 20 µg/m3	26	12	15	*	*	35	10
# Days > 25 µg/m3	6	3	3	*	*	9	4
# Days > 30 µg/m3	2	1	2	*	*	2	2
# Days > 35 µg/m3	2	1	0	*	*	1	1

(*) insufficient data (< 75% of the year) available

Table E1e(2). 24-Hour $PM_{2.5}$ results for Sydney, 1999-2001 (December 2001 bushfires included).

3 Year Period	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
Maximum 24-hour PM2.5 (µg/m3)	118.6	82.9	81.9	*	*	81.7	93.7
# Days > 20 µg/m3	32	17	17	*	*	42	17
# Days > 25 µg/m3	12	7	5	*	*	15	11
# Days > 30 µg/m3	8	5	3	*	*	8	9
# Days > 35 µg/m3	8	5	1	*	*	6	7

(*) insufficient data (< 75% of the year) available

The annual average $PM_{2.5}$ concentrations derived from hourly data at each monitoring site and the composite annual average concentrations are shown in Tables E1f(1) and E1f(2).

The supplied annual averages included the bushfire-affected data, and are shown in Table E1f(2).

To estimate the annual averages excluding the bushfire, daily averages without the 25-31 December 2001 data were averaged. Then a correction factor was applied to account for the difference between 1-hour derived annual averages and 24-hour derived annual averages. The results are shown in Table E1f(1).

Table E1f(1). Estimated annual average PM2.5 results for Sydney (Dec 2001 bushfire excluded)

Year	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
1999	9.7	10.0	8.1	6.7	9.9	10.2	9.1
2000	10.4	9.9	9.6	*	*	10.3	10.1
2001	10.8	10.5	10.9	*	*	10.7	10.4

Table E1f(2). Annual average $PM_{2.5}$ results for Sydney (Dec 2001 bushfires included)

Year	Liverpool	Lidcombe	Woolooware	Richmond	Westmead	Earlwood	Composite
1999	9.7	10.0	8.1	6.7	9.9	10.2	9.1
2000	10.4	9.9	9.6	*	*	10.3	10.1
2001	11.8	11.1	11.1	*	*	11.6	11.4

These Reports confirm that the PM2.5 levels in Sydney are not showing a downward trend and are already exceeding the National Reporting Standards (even accounting for bushfires) for the Annual Average PM2.5 levels of 8µg/ M^3.

The following 'Trend analysis' for PM10 has been cut/pasted from 2004 NSW Annual Compliance Report at the following link:
http://www.ephc.gov.au/pdf/Air_Quality_NEPM/Monitoring2004/nsw_compliance_rpt_2004_final.pdf

Trend analysis

Table 102: Maximum 24-hour average concentrations for PM_{10} ($\mu g/m^3$)

Region/ Performance monitoring Station	1995	1996	1997	1998	1999	2000	2001	2002	2003	2004
Sydney										
Blacktown	38.6	39.2	**57.3**	**66.9**	37.5	36.2	**127.1**	**122.0**	**186.8**	42.6
Bringelly	47.0	**92.0**	**68.2**	45.9	33.9	36.5	**99.4**	**120.2**	**274.7**	**60.3**
Chullora									**212.8**	**57.5**
Liverpool	40.0	37.3	**58.7**	45.7	46.0	**64.1**	**61.4**	**127.6**	**282.6**	**60.5**
Macarthur										**59.1**
Oakdale										41.3
Richmond	**53.6**	**85.8**	**71.5**	**55.6**	44.4	43.2	**119.9**	**126.4**	**194.3**	46.2
Rozelle									36.8	**51.4**
Wooloowware	**70.6**	**82.0**	**62.7**	42.3	39.0	46.1	**90.7**	**109.5**	**102.5**	40.8
Illawarra										
Albion Park			**61.6**	**63.6**	48.7	**62.5**	**58.7**	**88.3**	**281.0**	**51.5**
Kembla Grange										**57.6**
Wollongong	**61.0**	**69.6**	**64.8**	**56.9**	40.2	**58.1**	**68.2**	**76.7**	**280.5**	48.1
Lower Hunter										
Beresfield	**66.2**	**100.6**	**71.8**	46.1	48.0	**53.6**	**81.0**	**166.4**	**88.0**	**55.7**
Newcastle										46.9
Regional										
Albury							28.8	**81.3**	**921.4**	**55.6**
Bathurst						35.2	35.6	**258.2**	**621.7**	**72.9**
Tamworth						21.1	34.6	**189.8**	**243.3**	**55.7**
Wagga Wagga							**69.8**	**178.2**	**837.0**	**105.9**

AAQ NEPM Standard – 50 µg/m³ (24-hour average

Bold font indicates values that exceed the AAQ NEPM standard

NOTE:

Detailed trend data will not be provided until 3 full years of data have been collected for the following newly instrumented or commissioned sites – Rozelle, Oakdale, Macarthur, Kembla Grange and Newcastle.

A number of Australian and International studies have assessed the emissions from motor vehicles. Within Australia, a number of recent studies have undertaken extensive emission testing to provide emission factors (usually expressed as g/vehicle km) for the domestic fleet.

Examples of changes in the emissions factors, derived from results in NEPM study Project 2.2 are shown in theTable below. The results are the average values for diesel vehicles under the Composite Urban Emissions Drive Cycle (CUEDC). For most categories, there is a clear trend of lower particulate and CO emissions for more modern vehicles (e.g. for vehicles made in 1996 – 1999). However, for these pollutants, there does not appear to be a clear trend of higher emissions with larger vehicles. In contrast, emissions of NOx do increase with vehicle size, though are slightly irregular with respect to vehicle age (Watkiss, 2002). The Table indicates uniformity of CO and PM_{10} emissions for different vehicle sizes, but show NOx emission increase significantly with size.

Drive Cycle (CUEDC) Emissions Results Summary (NEPM 2.2).

Vehicle	g/km						Litres
(age group)	NOx	CO	HC	PM_{10}	$PM_{2.5}$	CO_2	/100km
Diesel Passenger Car and off road							
1980-89	1.72	3.25	0.46	0.78	0.75	454	19.5
1990-95	1.09	2.84	0.11	0.42	0.41	437	17.9
1996-99	1.27	1.18	0.19	0.27	0.26	465	20.5
Light commercial <3.5 t GVM							
1980-89	1.26	3.23	0.08	0.64	0.63	442	17.1
1990-95	1.04	3.28	0.11	0.37	0.36	410	16.3
1996-99	1.84	3.41	0.11	0.32	0.31	438	17.6
Medium goods vehicle3.5 – 12 t GVM							
1980-89	3.07	3.47	1.11	0.71	0.69	523	22.6
1990-95	3.02	3.92	0.56	0.97	0.94	491	20.8
1996-99	4.18	1.75	0.47	0.25	0.23	496	21.3
Heavy bus							
1980-89	16.59	5.38	0.93	1.10	1.07	1039	41.9
1990-95	-	-	-	-	-	-	-
1996-99	9.2	2.32	0.47	0.70	0.69	1085	44.1
Goods vehicle 12 – 25 t GVM or GCM							
1980-89	9.1	2.26	0.92	0.73	0.67	767	32.3
1990-95	7.9	3.56	1.01	0.72	0.67	798	32.9
1996-99	5.75	1.72	0.66	0.40	0.40	814	34.0
Heavy goods vehicle >25 t GVM or GCM							
1980-89	13.29	7.6	0.85	-	-	1187	51.1
1990-95	15.36	5.23	0.48	0.38	0.36	1176	48.3
1996-99	8.01	2.03	0.64	0.69	0.66	1107	45.4

Source: Project 2. Phase 2. Vehicle Testing and Supplementary Work. Parsons Australia Pty Ltd. GCM (gross combination mass) rest GVM (gross vehicle mass). Fuel sulfur content 0.17%. Note, diesel vehicles manufactured 1996 or later are Euro 1.

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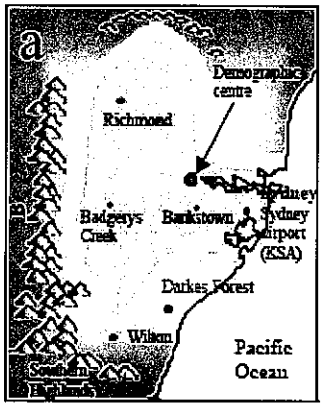
The following figure is cut and pasted from an article titled 'Sydney Basin: Air Toxic Emissions & Health Update' by J. A. Barros (2001) accessed at link:
http://www.areco.org/SYDNEY%20BASIN.pdf

Note the location of the lung cancer 'hot spots' in relation to the movement of air pollution in the Sydney Basin.

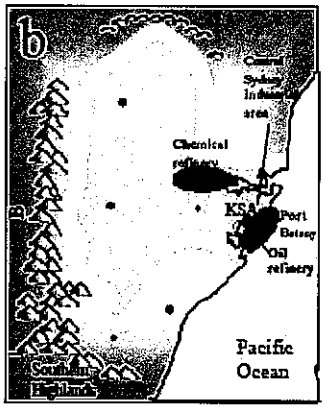
Sydney Basin: trapped air pollution circulation & lung cancer occurrence

SYDNEY: population 4m



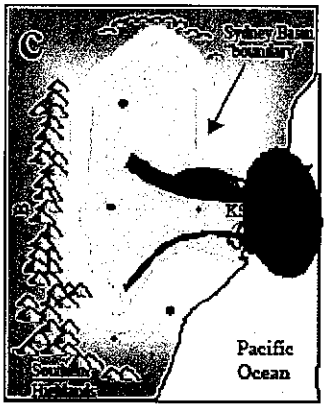
Sydney is in a classic, closed 'smog' basin. Its boundaries are mountains to W & S, high ground to N, and land/ocean temperature differential to the E. The boundary is stippled to indicate the 'slopping' nature of trapped air moving around inside the basin.

PRIMARY POLLUTION SOURCES



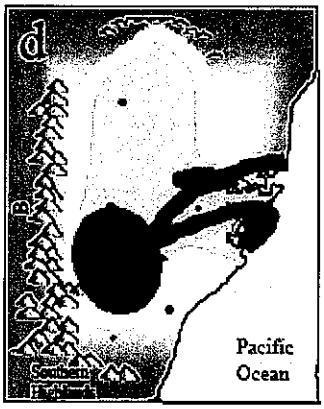
From early morning pollution is generated from primary sources: i.e. the airport, seaport, other industrial sources such as petroleum refinery, chemical works, and basin-wide road traffic.

MID MORNING



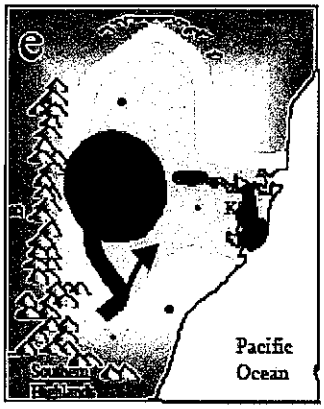
At about 10-11am, the offshore breeze blows morning air pollution about 7-8 km offshore, where it is blocked by the temperature differential between colder sea and warmer land.

AFTERNOON



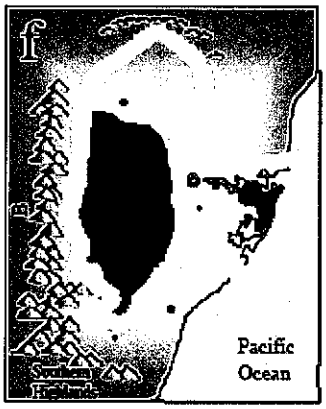
In the afternoon, sea breezes bring the morning pollution back onshore, picking up afternoon emissions along the way, and concentrate it in the south-west corner of the basin. Note the pollution cloud comes back onshore in a different direction because of the coriolis effect.

NIGHT TIME



At night, cold air moving downhill from the south and southwest pushes the main bulk of accumulated pollution north and then east, over the most populous parts of Sydney. Smog may be re-circulated for several days, in a 'figure 8' pattern, until a strong wind flushes out the basin.

LUNG CANCER HOT SPOTS



Dark red depicts areas of lung cancer occurrences that are higher than the NSW average. The pattern corresponds with Sydney's primary pollution source areas in the east and the air toxic pollution trap in the west. *(map after NSW Cancer Council 1991-1995 report)*

Term of Reference (b)
The impact of NSW air pollution laws (including the Clean Air Act 1961, the Protection of the Environment Operations Act 1997 and any regulations made under those Acts) on air quality over the past three decades

Community residents have a number of expectations of NSW Health, RTA, DEC, DIPNR and the Corporate Stakeholders. We expect these agents will:

- Understand the public's point of view and that our concerns will be the agency's top priority.
- Ensure these concerns will be scientifically investigated, researched, studied, documented and addressed.
- Explain or find the reason for the illnesses e.g., of M5 East community residents and prevent further exposures as well as to learn from the agency's own mistakes.
- Adopt proper and effective enforcement of the Minister's Conditions of Approval and not to corrupt enforcement by delegating that responsibility to the RTA.
- Maintain a permanent documentation database of records and information.
- Validate the concerns of the residents.
- Implement all these duties in a timely manner.

LCTAG's expectations, however, have been dashed by bureaucratic dishonesty, incompetence, indifference and tardiness in each of the respective departmental portfolios. Concealment of data, overlooking published evidence, or the deliberate termination of studies at a stage where findings were suggestive have caused LCTAG to be mistrustful of government officials and suspicious of the activities they conduct with corporate clients.

Why then is the 'Precautionary Approach' not taken? Because the risk-based approach to public health is adopted instead i.e., wait until the dead bodies can be counted. Whilst diesel fumes are a known cause of lung cancer, health bureaucrats state they are "not yet sure" how big the problem is and "we have not identified the extent of the problem".

This is the classic risk-based approach. Ignore the evidence so long as it is not 100% watertight. Use uncertainty as an excuse to delay. Wait for the dead bodies to pile and then slowly acknowledge the need for action. Remember asbestosis? Precaution is not (yet) fashionable while risk-assessment is!

The risk-based approach to unfiltered tunnels, as it also is for diesel and petrol, is to adopt the principle 'business as usual'. This has the backing of powerful special-interest corporate groups harnessing governments to deflect and stymie the search for least harmful alternatives. So long as the exact size of the problem is uncertain, risk-assessors call for delay and more study. It is now clearly evident that RTA's insidious delaying tactic of implementing a 'filtration trial', as endorsed by former Roads Minister Scully, is to buy time politically as the tunnel projects continue. Tabled internal documents show because consultants can be 'bought' or 'hired' to reinterpret old data to cast doubt on the nature of a problem, action can be stalled for decades. Maybe DEC will be more forceful in going public with its concerns about the trial estimated to cost tens of millions of dollars and their reluctance to be part of the review panel. They know the trial is a waste of time and is under-rated to about 10% of the airflows in the LCT.

The following 'Introduction' has been cut/pasted from 2004 NSW Annual Compliance Report at the following link:
http://www.ephc.gov.au/pdf/Air_Quality_NEPM/Monitoring2004/nsw_compliance_rpt_2004_final.pdf

Noteworthy is the disclosure that meeting both the PM10 and PM2.5 standards are a challenge for NSW. The PM2.5 reporting standards for Sydney were not met in 2004.

Introduction

The goal of the National Environment Protection Measure for Ambient Air Quality (AAQ NEPM) is to meet the NEPM standards (within the maximum number of allowable exceedences) by 2008.

This report, required under Clause (18) of the Ambient Air Quality NEPM, demonstrates that in 2004 NSW has met the requirements of the Ambient Air Quality NEPM for most pollutants. Non-compliance has been demonstrated for ozone in Sydney, and the Illawarra region, and for particles as PM_{10} at Wagga Wagga.

In 1998, the Government released Action for Air, its comprehensive long-term plan to protect and improve regional air quality in the Sydney Greater Metropolitan Region (GMR). The plan tackles regional air quality issues such as particle pollution and photochemical smog (ozone). The NSW air quality management plan, *Action for Air*, outlines a broad range of strategies used to manage air quality in NSW such as integrating air quality goals and urban transport planning; providing more and better transport choices; making cars, trucks and buses cleaner; promoting cleaner homes and business; and managing the impact of open burning. Action for Air is a 25-year plan that is reviewed regularly to assess achievements and the need for adaptation of control strategies. Action for Air is currently under review, following on from a stakeholder consultation Clean Air Forum held in November 2004.

Meeting the Ambient Air Quality NEPM goal for ozone will be a challenge for the major urban areas of NSW given pressures from a growing population, urban expansion and associated increase in motor vehicle use. However, NSW has a broad range of strategies to reduce precursor pollutants in place, and being developed, under Action for Air. These include the requirement for Stage 1 vapour controls at service stations in Sydney, the NSW Cleaner Vehicles Action Plan as well as initiatives under the Cleaner Industries Program and the Clean Air Fund. The latter two focus on reducing precursor emissions from smaller, commercial/industrial sources and, in the case of the Clean Air Fund, also domestic sources. A regulatory framework, which restricts emissions from larger industry through license limits and load-based fees, is in place. These measures, together with stricter motor vehicle emission standards, tighter fuel regulations, including the introduction of regulated limits on summer petrol volatility in Sydney, and NSW Diesel NEPM programs will help move NSW towards meeting the NEPM goal for ozone in the longer term.

Over and above the impacts of drought, bushfires and dust storms, meeting the goal of the Ambient Air Quality NEPM for particles, measured as PM_{10}, presents a challenge for NSW. This is particularly the case in rural population centres where a combination of topography, climate and relatively high use of solid fuel heaters, combine to produce elevated levels of particles in winter. Similarly, bringing $PM_{2.5}$ levels in line with the $PM_{2.5}$ advisory reporting standards is an area of difficulty for NSW with all $PM_{2.5}$ reporting stations exceeding the annual average reporting standard during 2004.

As is the case for ozone, Action for Air includes a broad range of strategies for managing particle emissions (both PM_{10} and $PM_{2.5}$) across mobile, industry and domestic sources. Some of the more significant initiatives are:

- National vehicle emission and fuel quality standards;
- Actions under the Diesel National Environment Protection Measure which requires jurisdictions to assess the impact of emissions from in service diesel vehicles and where necessary to implement programs to reduce them. NSW programs include the Smoky Vehicle Enforcement program;
- Particle emissions limits for industrial combustion processes under the Clean Air Plant and Equipment Regulation (currently in the process of renewal);
- Environmental Impact Assessment processes for new developments;
- Emission limits for particles from solid fuel heaters; and
- the Woodsmoke Reduction Program that was run from 2002 to 2004 to reduce woodsmoke from solid fuel heaters in regional areas.

Table 1: Sydney region Ambient Air Quality NEPM monitoring network

Station	Station type [1]	Number of parameters	Ozone	Nitrogen dioxide	PM_{10}	Carbon monoxide	Sulfur dioxide	Lead
Blacktown	T	5	X	X	X	X	X	
Bringelly	T	4	X	X	X		X	
Central Coast [2]	C	4	X	X	X		X	
Chullora [3]	T	5	X	X	X	X	X [6]	
Liverpool [4]	C	5	X	X	X	X		
Macarthur	T	5	X	X	X	X [6]	X [6]	
Oakdale	P	2	X		X			
Richmond	T	4	X	X	X		X	
Rozelle	T	5	X	X	X	X		X
St Marys	P	1	X					
Woolooware	T	4	X	X	X		X	
CBD [5]	P	2				X		X

(1) P denotes performance; T denotes trend; C denotes campaign.
(2) Scheduled to begin operation in 2005.
(3) Replaced the Lidcombe trend station.
(4) Data from the Liverpool station will be reported at least until the Macarthur station is fully established.
(5) Peak station.
(6) Instrument to be installed in 2005.

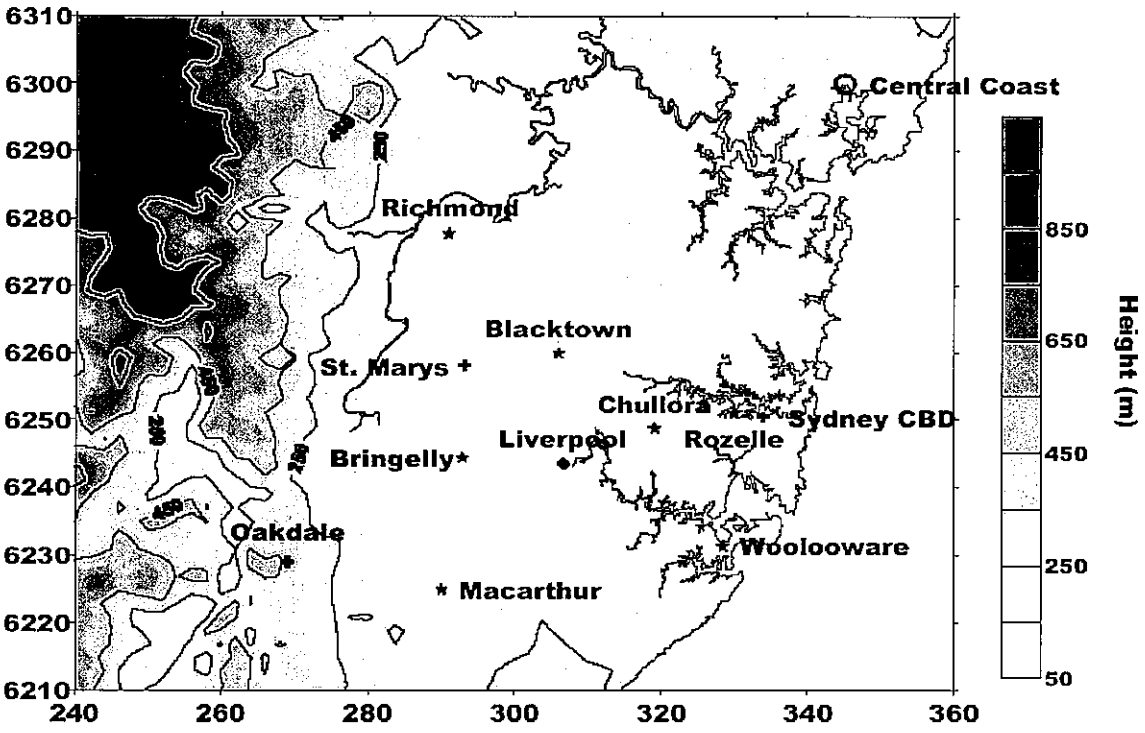


Figure 1: Ambient Air Quality NEPM Monitoring in the Sydney region (AMG co-ordinates)

★ trend station; + performance station; ◆ campaign station; O proposed station;

The following 'Conclusion' has been cut/pasted from 2004 NSW Annual Compliance Report at the following link:
http://www.ephc.gov.au/pdf/Air_Quality_NEPM/Monitoring2004/nsw_compliance_rpt_2004_final.pdf

Conclusions

The data presented in this report demonstrate that NSW achieved compliance with the Ambient Air Quality NEPM goals for carbon monoxide, nitrogen dioxide, sulfur dioxide and lead. Levels of these pollutants continue to be well below Ambient Air Quality NEPM standards.

Compliance with Ambient Air Quality NEPM goals for photochemical smog and fine particles was not demonstrated. Bushfires and severe drought conditions experienced throughout NSW during 2004 have at times contributed to elevated ozone and particle pollution events. However, for ozone in particular, anthropogenic emissions are sufficient to generate exceedences of the Ambient Air Quality NEPM standards and meeting the Ambient Air Quality NEPM goal for photochemical oxidants will be a challenge for NSW.

As has been outlined here, the Action for Air outlines a broad range of strategies to manage air quality in the Sydney Metropolitan Region. A review of Action for Air that is currently underway will examine ways in which the current air quality management framework can be improved to more effectively address the problem areas of ozone and particles.

Harming People by Government and Corporate Corruption

That corporate wealth buys broad influence in law and public policy is well documented and widely acknowledged. Holders of high political office themselves frequently have significant influence, ownership or representation in large corporations. Often working behind a wall of secrecy to protect corporate interests, can these arrangements serve not the interests of humanity but to enrich the few at the expense of the many?

Occupational and environmental diseases are often viewed as isolated and unique failures of science, the government, or industry to protect the best interest of the public. However, they are in fact an outcome of a pervasive system of corporate priority setting, decision making, and influence with political and bureaucratic stakeholders. This 'structure of harm' is based on corporations compelled to maximize profitability while costs to society such as from pollution are largely ignored.

The system in NSW, revealed recently in 'privileged' documents released in NSW Parliament, produces disease because political, economic, regulatory and ideological norms prioritize values of wealth and profit over human health and environmental well-being. In other words, the current economic and political system in NSW privileges corporate actors and actually provides incentives for the production of injury and disease rather than its prevention.

These documents revealed the NSW Government and certain of its bureaucracies appear to have forfeited a legislative and constitutional role as servants of the public and have aligned themselves with corporate stakeholders in the design, construction and operation of traffic tunnels in Sydney. What is now clear is the social and environmental costs have been ignored by externalizing them, or shifting costs to the government (taxpayers), residents, neighbours, motorists and workers.

Thus, these closely-knit alliances become even more profitable to the extent the financial deals make other people pay for the bills for the impacts on the health of society. For the M5 East, Cross City and the Lane Cove tunnels, the respective companies appear to externalise the cost of air pollution and its associated health and environmental damage onto the taxpayers who get sick from unfiltered tunnel pollutants exhausted into the local precincts. Aided by the alleged abuse of legislative power by the RTA, these companies, in secret contract deeds, avoid paying these true costs. By adopting incredible craft and deceit, the RTA through its influence and management in these profit-earning co-ventures have determined not to install protective technology such as particle filtration and gas detoxification systems thereby enhancing corporate profit in exchange for financial paybacks.

Even more deplorable is that the documents disclose the RTA has agreed to indemnify their bedfellow companies for costs, charges and expenses or for claims or losses should a court find that environmental assessment or determination of the 'Tunnel' including the Ministers Condition of Approval fails to comply with the Law or is invalid in any respect. The RTA has indemnified the respective company in relation to any investigation or 'legal challenge'. It is noteworthy that in September 2004, the NSW Government closed their air-quality monitoring station near the Cross City Tunnel thereby removing evidence of local high pollution levels and thwarting potential litigation by residents affected by pollution from the toxic tunnel exhausts together with that generated by gridlocked surface traffic. The NSW Government's reason that it was unaffordable to maintain the monitor is hardly credible when in June, 2002 the RTA paid $9,110,375 to acquire the land with a market value of $4,520,250 to build the eastern stack for the Lane Cove Tunnel.

The 'secret' Deeds of Contract appear to protect the companies from paying 'restitution' of the injured through the payment of unenforceable compensatory fines, capped by the RTA for the Cross City Motorway Company at $5million, rather than criminal penalties. The failure to impose fines is the experience with the M5 East tunnel debacle where numerous breaches of the Ministers Conditions of Approval are on record. Thus, the costs never approach the economic advantage that accrues to the respective companies that perpetuate these injuries and escape liability. In other words the RTA has made it cheaper for the companies not to install proper filtration and thereby inflict sickness and potential death on the community exposed to toxic stack pollution.

It seems reckless to wilfully discharge additional toxic hazards into Sydney's air-shed that is already exceeding National Standards for harmful fine particles. The interlinked RTA-Corporate goal of profit maximization exceeds any future compensation cost. Twice as many people die from exposure to vehicle exhaust in Sydney than from road accidents. Total health impacts cost $2-3billion annually for Sydney alone.

In girding up the profits of its corporate co-partners, the RTA has over the past few years embarked on an utterly misleading campaign to discredit tunnel filtration. So effective has this campaign been that former Roads Minister Carl Scully has reinforced the RTA propaganda by his refrain that "Tunnel filtration is unproven technology and is only a high-tech placebo". Such false and delusional claims only helped his RTA bureaucrats to embark on a 'structure of harm' with corporate stakeholders to maximize wealth, corporate profit at cost to human health and environmental well-being. The conduct of the RTA, with government and ministerial fanfare, in hoodwinking the community with two hoax 'filtration trials' to appease community anger is unconscionable.

Lane Cove Tunnel Action Group (LCTAG) believes there is not only an obligation of 'due diligence' by the Regulatory Authorities (RTA, DEC, Health and DIPNR) but also on the respective tunnel consortia to implement proven measures to clean and detoxify the polluted tunnel airstream where the poisonous components are derived almost entirely from the combustion of fossil fuels, mainly petrol and diesel. Such measures would be consistent with the Precautionary Principles.

LCTAG also believes that to date, the NSW RTA and the respective tunnel corporate stakeholders, have failed to exercise such care, skill and foresight that would be expected of a reasonable corporation and helps to remove a defence of 'due diligence' by ignoring such facts. Indeed, the failure of the RTA and the respective companies could be interpreted now as a wilful and pre-meditated decision not to adopt preventive or precautionary measures. Such a decision implies a deliberate intention to discharge untreated toxic waste, knowing it has the potential to harm or be likely to harm the environment, including those 'most at risk' in a community who are already described in documents, known to the RTA and to the Consortia as the ***"most affected receptors."***

The RTA seem not to exercise 'due diligence' by knowingly and negligently intending to discharge higher levels of toxic waste from the re-designed Lane Cove tunnel in a manner likely to cause harm. To date, LCTAG alleges that neither the RTA nor the Lane Cove Tunnel Company has volunteered the truth about traffic volumes or the real pollution levels. LCTAG also understands that a defence of 'due diligence' is established if a company commissions the offence due to causes over which they had no control; and that they took reasonable precautions and exercised due diligence to prevent the offence.

The law seems unable to provide relief except under certain prescribed conditions. The only relief, it seems, that may be granted by a Court is if **"...it can be demonstrated that an actual impact will be experienced as a consequence of the venting of these pollutants to the environment."** (Memorandum of Advice, Point 17, p. 9; 3.2.02). This provision for future action may allow a Court to provide relief to our grievances.

Sydney community groups have grave concerns about the standards and frameworks used to regulate the increasing number of road tunnels in our city. Of equal concern is the RTA's and regulatory authorities' approach to dealing with serious health and safety risks these tunnels pose to drivers and residents alike. Tail-pipe emissions in tunnels and especially from exhaust stacks are more concentrated and more toxic than background pollution.
For the safe administration of road tunnels, the public is entitled to expect that:

- the design and regulations of these tunnels would reflect international best practice, especially in terms of their safety, ventilation systems, value for money and accepted precautionary principles
- the standards and regulations would be strictly enforced
- mechanisms would be in place to promptly and adequately respond to any foreseen or unforeseen problems, including the emergence of new knowledge and the development of new guidelines
- all public agencies involved in planning and regulation, and especially those proposing and developing new projects, would be held accountable for their performance and that mistakes made would be rectified in both current and later projects.

Lack of transparency in NSW bureaucratic processes

Internal papers tabled in 2003 revealed that the RTA's Representations Report (RR) for the LCT project was submitted to the Department of Infrastructure Planning & Natural Resources (DIPNR), without RTA addressing the serious questions raised by the EPA regarding air quality issues in the EIS. Papers disclosed that the RTA merely treated such questions as 'correspondence'. Director General of EPA, Lisa Corbyn, sent a protest letter (also tabled) to Paul Forward, CEO of the RTA, to admonish and remind him that such conduct "did not make the process transparent to the community."

One of the fundamental flaws in the EIS process is the lack of independent, rigorous assessment of traffic volumes, impact on public transport, health, the environment, costs, amenity or even property values.

Tabled papers also showed that commissioned 'expert advice' is often edited by the RTA to add 'spin' and conform to a party line, or even be suppressed.

Internal papers confirm that the EPA initially determined at least a 10-fold increase in cancer risk for most **"affected receptors"** (individuals) exposed to toxic stack exhaust from the LCT, but nevertheless allowed a reworking of the data by RTA consultants to include discounts for future vehicle technology improvements and average the results over a 70 year lifetime. It then became an acceptable risk. Recently tabled documents disclose that the levels of pollution are much higher than levels submitted to DIPNR for approval by the Minister. Who fudged the data to get Ministerial approval? LCTAG raised vigorous concerns and concluded that the levels in the EIS were seriously underestimated. This was confirmed independently, with pollution levels underestimated by 11-40%, but were kept hidden during the EIS. RTA engaged their consultant who responded with an alleged flawed and patronising defence.

Planning Minister Sartor's Answer to Cancer – Blow it in the Wind!

It is time we the people came together and took back our State of NSW and our country. The community at large, of different political alliances, have ALL been alienated, manipulated, ridiculed and ignored by ruling elite at both political and bureaucratic levels whose only ideology it seems, is to serve money and power.

The real question is "Whom does this NSW Government serve?" We need a government committed to serving the people and big enough and strong enough to do the job.

The Government has also handed over its regulatory powers to the lobbyists and ex-politicians representing the corporations supposedly being regulated. We now have the biggest government that we have ever had, but one that is totally ineffectual in protecting us from the big money interests exploiting us.

What has emerged in the M5 East, CCT and the LCT projects is a scandalous dereliction of duty. Privileged and public documents disclose a litany of 'corrupt conduct'. The Government, through the RTA, has formed alliances with the corporate stakeholders to augment profit by externalizing costs to the community and environmental well-being.

This pre-election year is set to become a tsunami of documented recklessness by the NSW Government. Contributing to these scandals, Planning Minister Frank Sartor seems set to expunge a strict Condition of Approval of the M5 East tunnel that allows toxic fumes to be discharged from the tunnel portals (entries and exits) ONLY under emergency conditions such as a fire. At present such cancer-causing fumes are normally exhausted from the single tunnel stack.

The truth of the debacle is the ventilation system in the M5 East tunnel cannot cope with the high levels of toxic fumes by blowing them out the stack and to concurrently maintain lower toxic levels in the tunnel. However, what Minister Sartor also failed to disclose is that the levels of pollution in the residential precinct around the stack are now close to exceeding the prescribed national air-quality standards because the RTA grossly underestimated the traffic volumes, now over 110,000 vehicles a day. Should there be one proven exceedance of the air-quality standard outside the stack, another Condition of Approval requires the RTA to install filtration.

The demonstrably inept NSW DEC (formerly EPA) appears also to aid the RTA by refusing to incorporate an appropriate correction factor into the monitoring data, thus underestimating the real levels of toxic pollution – another scandal? The RTA is now faced with a major dilemma. How can the RTA save the Tunnel Company the expense of installing retro-fitted in-tunnel filtration? Simple, rather than install filtration, the cancer, my friend, is blown in the wind. The resultant costs of health impacts are passed onto the community by legalizing portal emissions at ground level. Such has been regularly undertaken illegally and disclosed by the Operator for over two years in full knowledge of NSW Planning who did nothing to enforce the Conditions of Approval. This was one of the several reasons why the NSW Health's study of sicknesses among M5 East residents was flawed. NSW Health understood there were no portal emissions and so the adjacent community was believed to be in a 'low impact zone'. How wrong NSW Health was who still refuse to withdraw their invalid findings of "no adverse impacts" – another looming scandal?

What is equally abhorrent is the executive of the RTA media/propaganda unit has hoodwinked the community into believing the RTA was serious about tunnel filtration by undertaking a 'filtration trial'. But the RTA was only kidding! The hoax 'filtration trial' was a ploy to appease the community's anger and allow the CCT and LCT construction to reach a stage where in-tunnel installation could only be an expensive retrofit.

A tabled internal audit by NSW Planning reveals the RTA has failed to comply with a litany of other Conditions of Approval regarding the M5 East. Whom does this Government serve?

What seems unconscionable about Minister Sartor's desire to blow known cancer-causing pollution into the lungs of taxpayers for commercial, bureaucratic and political expediency is that in June, 2003, as Minister for Science and Medical Research, he launched the $205 million NSW Cancer Institute. Among its aims is "To reduce the incidence of cancer in NSW". See the following link:
http://www.cancercouncil.com.au/editorial.asp?pageid=1124

As former Lord Mayor of the Sydney City Council, he was one of the architects of the 'Gateways Works' in which the narrowed William Street was undertaken in secret and proven now to be integral to the commercial viability of the CCT. See the following link:

http://www.smh.com.au/cgi-bin/common/popupPrintArticle.pl?path=/articles/2003/02/14/1044927802247.html

The air pollution levels in William Street that were already exceeding national standards were acknowledged by NSW Planning will be even higher after the CCT opens!

The focus of current community anger, however, at what the CCT really means to the community, is the coercion written into the tunnel deal. The contract is clear: the Government must keep a long list of alternative routes closed or narrowed and provide dedicated lanes feeding the tunnel. The roads cannot be re-opened without penalty to the motorist and taxpayer who in turn also meets the cost of the CCMotorway Company in any legal dispute. That means the outcome of the tunnel boycott is continuing congestion. And the frustration is not just local; traffic piling up around Macquarie Street and the Eastern Distributor is slowing the travel times from Sydney's north. For all this anger, motorists are left with two unpalatable choices: a $3.56 one-way toll for 2kms or a very slow trip.

Despite all the current Government posturing about reducing the toll, it was the RTA that amended the tunnel contract only late in 2004 to load another 15 cents into the toll to cover $35 million in extra construction costs. It was the NSW Government which signed a flawed deal. And it is government which is responsible for the ensuing mess - be it exorbitant tolls, gross impacts on local traffic amenity, portal emissions, no in-tunnel filtration, disgraced, wishy-washy go-along-to-get-along 'Community Consultative Meetings' coupled with a litany of bureaucratic incompetencies at immense cost to the taxpayer.

The secret 'Contract Deeds' have eroded the protection of Human Rights and moved us toward an autocratic state. Indeed, it seems, in these contract deeds the corporate co-partners are given veto power over many of our laws and regulations – even those of states and municipalities. Where is the Crown Solicitor in all this? Licenses are not issued for these tunnels, putting them outside existing NSW EPA (DEC) Legislation.

Government should concern itself with morality in the board room. Morality has a great deal to do with money and power. Minister Sartor, it is to do with how we treat one another. It is immoral for the big money interests to force government to serve their greed instead of serving the people's need.

In the end it comes down to just three principles

1. support for the Constitution and Human Rights
2. commitment to honesty and openness in government, and
3. independence to serve the needs of people according to their individual judgment and conscience.

The NSW Government is reminded of the Occupational Health and Safety Act, 2001 (Chapter 1, Clause 5 and 136 (3n)) where there is an obligation to **control** a risk to health and safety. With respect to the latter Clause, reference to an employer extends to an owner of the plant (tunnel/stack) affecting public safety. The RTA has not to date, exercised all 'due diligence' by not allowing the prospective 'owner' of the respective tunnels to incorporate proven filtration and detoxification systems.

NSW EPA (DEC) fails to adopt national standards and Guidelines.

NSW Health agrees the NEPM air-quality standards do not apply to a point source (stack) pollution. However RTA and NSW EPA ignore the restriction and breach the guidelines.

NEPM states:

> *"Conversely the air quality of some localised areas within major air sheds are dominated by local activities such as that experienced in a road tunnel or a heavily trafficked canyon street. Air quality management in these areas is complex and needs a different approach to that directed at meeting ambient standards intended to reflect the general air quality in the air shed".*
> (NEPM (1998) p.13).

Note the directive, in this context of monitoring, from NEPC that air quality in tunnels and canyons **"needs a different approach"** NSW RTA and EPA endorsed by NSW Planning show contempt for the NEPM Guidelines and set their own standards.

Term of Reference (c)
The causes of air pollution in the Sydney basin over the past three decades

The following Table is cut and pasted from the Report dealing with pollution in the Sydney Greater Metropolitan Region (GMR) by the NSW DEC titled 'Air Pollution Economics' found at the following link: http://www.environment.nsw.gov.au/resources/airpollution05623.pdf
They show the principal sources of air opollution being industrial, commercial, domestic and motor vehicles as well as other mobile sources.

Table 2.3: Sources of PM_{10} emissions in GMR region, 2002

Sources	Annual tonnes (and % contribution)		
	Sydney	Wollongong Illawarra	Newcastle Hunter
Domestic fuel combustion	5 982 (23%)	220 (2%)	404 (1%)
Domestic lawn mowing	155 (1%)	8 (<1%)	15 (<1%)
Domestic natural gas combustion	41 (<1%)	0	0
Domestic waste combustion	21 (<1%)	922 (7%)	1 082 (4%)
Other (commercial & small industrial)	3 316 (13%)	324 (2%)	324 (1%)
Industrial facilities and power stations	11 896 (47%)	9 136 (65%)	22 278 (79%)
Motor vehicles	2 318 (9%)	107 (1%)	251 (1%)
Other mobile sources	1 739 (7%)	3 233 (23%)	3 795 (13%)
Total	25 467	13 951	28 149

Source: DEC Atmospheric Science emissions data, 2003.

Table 2.4: Sources of NO_x emissions in GMR region, 2002

Sources	Annual tonnes (and % contribution)		
	Sydney	Wollongong Illawarra	Newcastle Hunter
Domestic fuel combustion	642 (<1%)	23 (<1%)	43 (<1%)
Domestic lawn mowing	76 (<1%)	4 (<1%)	7 (<1%)
Domestic natural gas combustion	381 (<1%)	3 (<1%)	5 (<1%)
Domestic waste combustion	11 (<1%)	365 (2%)	428 (<1%)
Other (commercial & small industrial)	4 122 (4%)	220 (1%)	403 (<1%)
Industrial facilities and power stations	37 622 (35%)	8 126 (53%)	111 408 (90%)
Motor vehicles	62 806 (58%)	3 536 (23%)	7 246 (6%)
Other mobile sources	3 088 (3%)	2 988 (20%)	3 572 (3%)
Total	108 747	15 265	123 112

Source: DEC Atmospheric Science emissions data, 2003.

Table 2.5: Sources of VOC emissions in GMR region, 2002

Sources	Annual tonnes (and % contribution)		
	Sydney	Wollongong Illawarra	Newcastle Hunter
Domestic/commercial	51 591 (41%)	4 479 (50%)	6 998 (42%)
Industrial facilities and power stations	19 511 (15%)	804 (9%)	2 520 (15%)
Motor vehicles	48 632 (38%)	2 564 (29%)	5 705 (34%)
Other mobile sources	6 663 (5%)	1 103 (12%)	1 344 (8%)
Total	126 397	8 950	16 567

Source: DEC Atmospheric Science emissions data, 2003.
Note: Columns do not sum to total owing to rounding.

The following Table dealing with the main air toxics is cut and pasted from the Report dealing with pollution in the Sydney Greater Metropolitan Region (GMR) by the NSW DEC titled 'Air Pollution Economics' found at the following link:
http://www.environment.nsw.gov.au/resources/airpollution05623.pdf

Health endpoints associated with selected air pollutants

Particulates	Nitrogen dioxide	Carbon monoxide	Ozone	Air toxics	Air toxics (PAHs)
• Increase in cardiac and respiratory mortality • Admissions to respiratory and cardiovascular casualty room and hospital • Increased incidence of acute bronchitis in adults and children. Increased prevalence and exacerbations of COPD in adults and children • Asthma attacks in adults and children • Cough • Restricted activity days • Reduced lung function	• Increased mortality • Impaired lung function • Impaired respiratory defence mechanisms, leading to increased susceptibility to infections • Increased respiratory disease in children	• Mortality, especially those with cardiovascular disease • Aggravation of cardiovascular disease & chest pain • Nausea • Headache • Fatigue	• Mortality • Acute respiratory problems • Chest constriction & pain • Increase in incidence and severity of asthma attacks • Increase in asthma and respiratory-related casualty room visits and hospitalisations • Coughing and wheezing • Eye irritation • Headache	Benzene: • Leukaemia • Long-term harm to immune system • Skin and eye irritations • Drowsiness • Dizziness • Headaches **Toluene:** • CNS dysfunction (often reversible) • Narcosis • Light-headedness **Xylene:** • Irritation of respiratory tract • Eye irritation • Headaches, dizziness, fatigue, tremors, coordination difficulties • Impaired pulmonary function **1,3-butadiene:** • Cancer • Eye, nose, throat irritation	• Cancer • Kidney & liver damage • Respiratory irritation • Exacerbation of asthma • Chronic bronchitis • Coughing & throat irritation

Secondary Pollution – Nitrates and Ozone

Nitrates are secondary particulates, formed from the reaction of NO_x emissions with other species. The mechanisms associated with particulate nitrate are rather complex and only partially understood. Nitrogen oxides emitted in urban and rural areas are oxidised to nitric acid (HNO_3) vapour by hydroxyl (OH) radicals during the daytime, and by ozone (O_3) to dinitrogen pentoxide (N_2O_5) at night. Sulphuric acid produced by oxidation of SO_2 can readily self-nucleate to form new particles or can be adsorbed to pre-existing particles to produce accumulation mode aerosol. However, nitric acid vapour does not have this property of self-nucleation. Particulate nitrate is instead formed from: the adsorption of HNO_3 on existing particles; the adsorption and reaction of N_2O_5 on particles; and the reaction of NO_2 with water to form nitrous acid (HONO) which is subsequently adsorbed by particles. In addition there are reactions of oxidised N species with sea-salts. The only way particulate nitrate can be formed directly is the reaction of HNO_3 with NH_3, which is temperature, humidity dependent, and reversible.

Under certain conditions secondary particulates may have significant impacts locally (close to the emission source). An example is during summer time smog episodes, when low wind speeds and large amounts of radiation can result in the formation of secondary particulates within urban areas. Measurements made in the Los Angeles basin show that particulate nitrate is a major inorganic contributor to urban PM_{10} levels in the city. However, an analysis within ExternE has shown that in general, local nitrate effects are small. An assessment was undertaken within ExternE to investigate the potential impact of local-scale damages from nitrate particulate formation in urban areas. This analysis calculated the impacts over distance using a steady state model (assuming uniform vertical mixing), together with the particulate dose-response functions for human health. The resulting fractional damage (as a percentage of the total damage) arising from nitrates within the local area (i.e. within 50 km) was estimated to be less than 10% of the total nitrate damages, even in large urban areas.

Within Australia, nitrate formation will be extremely site-specific, with significant variations between different states or even cities. Indeed, in order to evaluate the role of nitrates, a detailed assessment is needed to understand the levels of particulate nitrate aerosol in urban PM_{10} levels, the types of aerosol species present, the background concentrations of other pollutants involved (e.g. ammonia) and the regional scale photochemical production of particulate nitrate. It should be stressed that these factors are not well understood in Europe.

A similar problem emerges for ozone. The assessment of the effect of emission reductions on ozone concentrations at the local scale probably represents the most difficult problem within atmospheric transport modelling for air pollution impact assessment. The difficulties are caused by very site-specific nature of ozone formation, and the strong non-linearities associated with ozone formation, particularly concerning NOx emissions. A release of NOx within an urban area may well cause an immediate *decrease* in ozone levels near the source (because of the titration reaction, $NO+O_3$ to NO_2). Slightly downwind (for example in suburban areas), this NOx release may still cause an ozone decrease in most cases, but in some photochemically very active cities (typically Mediterranean cities such as Milan or Athens) chemical reaction processes may enhance ozone levels. Further downwind, e.g. in rural areas, this NOx release can cause increased ozone in other parts of Europe, with the exception of those rural areas still heavily influenced by anthropogenic NOx sources. In general, the effects of VOC emissions are much more straightforward, although still rather variable in magnitude. Increased VOC emissions almost always give rise to increased ozone levels. In high-NOx areas, ozone levels are strongly affected by VOC emissions. In low-NOx areas, ozone levels are less sensitive to VOC emissions.

Whether local ozone increases or decreases as a result of NOx and VOC emission changes depends heavily on a range of factors, including the distribution of emissions near the source, the ambient NOx level (high or low-NOx), and the meteorological conditions. The same release may cause an increase in ozone one day and a decrease in ozone on the next day. Evaluation of the effects therefore requires either detailed modelling for each case of interest, or extrapolation from a sufficiently large number of studies where there is some confidence in the reliability of predictions. Unfortunately, there is no systematic assessment of the effect of local emission-control measures on ozone concentrations in and around urban areas. Many studies have been conducted of course, but the over-riding problem has always been that studies have covered only very short time periods and specific conditions. Given these considerations, it is not possible to recommend a simple reliable method of estimating the effects of local ozone. Detailed studies are required, either with full photochemical models, applied over long-periods, or with appropriate detailed dispersion modelling. More information is available on the effects of NOx and VOC emission abatement on *regional* ozone levels in Europe (i.e. between countries). These studies clearly suggest that road-traffic emissions have a significant role to play in ozone formation over Europe. (Watkiss, 2002)

Ozone

Local ozone is certainly an issue in Australia. NEPM (Ambient Air Quality 1998) states. *'In general, high levels of ozone are only a problem for major cities where emissions from concentrated urban activities can accumulate to high levels if the meteorology is favourable for pollution build up and for smog formation. Melbourne, Sydney, Perth, Brisbane and Adelaide, are the main Australian cities of sufficiently large size and favourable meteorology for significant ozone formation. Most rural areas and other cities have either populations which are too small and dispersed and/or meteorology that does not favour ozone production'.*

The following Tables have been cut/pasted from 2004 NSW Annual Compliance Report at the following link:
http://www.ephc.gov.au/pdf/Air_Quality_NEPM/Monitoring2004/nsw_compliance_rpt_2004_final.pdf

The document the failure to meet the National Standards for ozone

Ozone

Table 10: 2004 compliance summary for O_3 in New South Wales

Region/ Performance monitoring Station	Data availability rates (% of hours)					AAQ NEPM standard 0.10 ppm (1-hour average) 0.08 ppm (4-hour average) Number of exceedences (days)		Performance against the standards and goal	
	Q1	Q2	Q3	Q4	Annual	1-hour	4-hour	1-hour	4-hour
Sydney									
Blacktown	92.8	66.0	00.0	00.0	39.5	**2**	**4**	Not met	Not met
Bringelly	94.1	95.1	87.0	88.3	91.1	**6**	**7**	Not met	Not met
Chullora	84.7	88.5	91.8	84.1	87.2	1	**4**	Met	Not met
Liverpool	94.0	91.4	89.1	94.7	92.3	**3**	**5**	Not met	Not met
Macarthur [1]									
Oakdale	84.9	66.9	95.1	95.2	85.6	**7**	**7**	Not met	Not met
Richmond	83.9	90.9	93.1	90.1	89.5	0	1	Met	Met
Rozelle	90.4	93.6	81.6	90.0	88.9	0	1	Met	Met
St Marys	95.1	94.3	95.4	89.5	93.6	**3**	**4**	Not met	Not met
Wooloowaré	87.6	91.9	61.7	00.0	60.2	1	1	ND [#]	ND [#]
Central Coast [2]									
Illawarra									
Albion Park	94.9	92.0	94.6	92.7	93.5	1	1	Met	Met
Kembla Grange	88.8	92.1	92.1	92.3	91.3	**3**	**3**	Not met	Not met
Wollongong	92.0	91.8	94.6	91.6	92.5	1	**2**	Met	Not met
Lower Hunter									
Maitland [3]									
Newcastle	92.2	95.5	95.1	86.5	92.3	1	0	Met	Met
Wallsend	86.6	93.9	85.3	87.1	88.2	1	0	Met	Met
Regional									
Bathurst	95.7	80.9	94.8	88.3	89.9	0	0	Met	Met

ND Not demonstrated.

Bold font indicates values that exceed the AAQ NEPM standard

(1) Station established November 2004. Data reported from Liverpool until station fully operational.

(2) Station to be established

(3) Station to be established. Data reported from Wallsend in the interim.

(#) Site newly commissioned or de-commissioned in 2004.

Both the 1-hour and 4-hour standards for ozone were exceeded in NSW during 2004. Sydney and the Illawarra region did not comply with the Ambient Air Quality NEPM goal. Compliance was demonstrated in the lower Hunter and Bathurst.

Table 50: Statistical summary for O_3 - Annual daily maximum 1-hour average concentrations

Station: Blacktown

Year	Data availability rates (%)	Number of Exceedences (days)	Maximum value (ppm)	Percentiles (ppm)						
				99th	98th	95th	90th	75th	50th	25th
1995	95.3	0	0.059	0.054	0.052	0.048	0.042	0.032	0.023	0.017
1996	85.7	0	0.082	0.065	0.060	0.052	0.046	0.033	0.024	0.018
1997	93.7	**4**	**0.149**	0.088	0.075	0.064	0.053	0.036	0.026	0.021
1998	83.8	**3**	**0.109**	0.093	0.083	0.063	0.052	0.038	0.024	0.018
1999	95.1	0	0.091	0.079	0.075	0.063	0.050	0.035	0.026	0.020
2000	91.5	**2**	**0.113**	0.088	0.075	0.061	0.051	0.037	0.028	0.024
2001	93.6	**5**	**0.153**	**0.107**	0.088	0.075	0.054	0.040	0.030	0.024
2002	91.7	**2**	**0.130**	0.093	0.083	0.068	0.059	0.043	0.033	0.026
2003	90.3	**3**	**0.181**	0.085	0.073	0.061	0.050	0.037	0.029	0.025
2004	39.5	**2**	**0.123**	0.093	0.089	0.080	0.066	0.050	0.036	0.028

AAQ NEPM Standard - 0.10 ppm (1-hour average)

Bold font indicates values that exceed the AAQ NEPM standard

Table 51: Statistical summary for O_3 - Annual daily maximum 1-hour average concentrations

Station: Bringelly

Year	Data availability rates (%)	Number of Exceedences (days)	Maximum value (ppm)	Percentiles (ppm)						
				99th	98th	95th	90th	75th	50th	25th
1995	94.6	0	0.081	0.075	0.064	0.057	0.050	0.036	0.026	0.022
1996	94.2	0	0.098	0.077	0.071	0.057	0.049	0.036	0.027	0.022
1997	93.7	**5**	**0.135**	**0.102**	0.087	0.069	0.058	0.044	0.029	0.024
1998	74.5	**4**	**0.113**	**0.101**	0.098	0.078	0.066	0.044	0.029	0.024
1999	92.1	**3**	**0.114**	0.100	0.094	0.073	0.055	0.037	0.029	0.024
2000	94.9	**3**	**0.130**	0.096	0.092	0.070	0.059	0.039	0.032	0.027
2001	91.5	**9**	**0.175**	**0.115**	**0.102**	0.074	0.059	0.042	0.033	0.027
2002	93.0	**2**	**0.118**	0.098	0.090	0.074	0.064	0.045	0.034	0.028
2003	91.3	**3**	**0.155**	0.095	0.076	0.065	0.056	0.041	0.032	0.028
2004	91.1	**6**	**0.122**	**0.104**	0.091	0.074	0.060	0.044	0.033	0.029

AAQ NEPM Standard - 0.10 ppm (1-hour average)

Bold font indicates values that exceed the AAQ NEPM standard

Term of Reference (d)
The health impacts of air pollution on any 'at risk' groups

Studies of air pollution episodes have shown that very high levels of ambient air pollution are associated with strong increases in adverse health effects. Recent studies also reveal increases in adverse health effects, especially in 'at risk' persons, at the current levels of ambient air pollution typically present in urban areas.

These health effects include a range of endpoints, such as premature mortality (deaths brought forward), respiratory and cardio-vascular hospital admissions, and exacerbation of asthma, other respiratory symptoms and loss of lung function. The evidence for these effects is strongest for the pollutants PM_{10} , SO_2 and ozone and the relationships are widely accepted as causal. Recent studies also suggest that long-term exposure to these pollutants, especially particles, also damages health and that these effects may be substantially greater than the acute effects described above.

At the national level, these health effects are important. It is estimated some 2,400 people die each year in Australia from air pollution, and some 10-15% of the population display respiratory symptoms (NEPC, 1998). These health impacts have major economic costs, estimated at around $A18 billion/year[1]. Transport is a major source of these atmospheric pollutants and therefore can be assumed to be a major cause of these health effects (Watkiss, 2002).

The association between exposure to vehicle pollution and ill-health is not new. The relationship between air pollution, death and disease has been studied for decades, leading to the recent conclusion that combustion of diesel and petrol is among the most toxic sources of emissions today (USA Clean Air Task Force Report, Feb. '05) viewed at the link:
http://www.catf.us/publications/view.php?id=83

These exhausts contain numerous dangerous compounds, ranging from respiratory irritants to carcinogens including a host of air toxics, particulate matter, carbon monoxide and nitrogen oxides.

The very fine particles adsorb toxic gases and liquids onto their surfaces. On a weight basis, a billion ultra-fine particles are about equivalent to one coarse particle 10 micrometres in diameter, but have 1000 times the surface area. The fine particles are mainly soluble and penetrate deep into the lungs. Health research indicates that the invisible exhaust may be the most dangerous of all. Technology exists right now to clean up emissions from these engines and to remove such toxics from road tunnels. The only thing that stands between us and dramatically healthy air is the political will to require these reductions.

Former NSW Health Minister and now Premier Morris Iemma knows that in Sydney, twice as many people die from exposure to vehicle pollution than from road accidents. Yet, despite NSW Health's protestations and legal advice, as disclosed in tabled internal documents, RTA deliberately refuses to install signs warning motorists to close their windows on entering the toxic M5 East tunnel.

In the USA, fine particle pollution kills 21,000 people each year. Diesel exhaust poses a cancer risk that is 7.5 times higher than the combined risk from all other air toxics. The risk of lung cancer for

[1] Note this estimate (NEPC, 1998) is considered high for the effects quantified, as it applies a full Value of Statistical Life (VoSL) of $A7million to value acute mortality (deaths). The literature now suggests that these deaths only reduce life expectancy by a relatively short period of time and more recent studies factor this into account with the use of a very much lower VoSL.

people living in urban areas is three times that for those living in rural areas (CATF Report, Feb. '05). Air pollution was not on the agenda of the NSW Government in deciding to power its new bus fleet with toxic diesel that replaced clean LPG (See comparative costs of pollution in Tables below).

Children are more susceptible than adults (except the elderly) to the adverse effects of air pollution because:

- Children are more active and breathe more rapidly.
- They have more lung surface area compared to their body weight and inhale more air kgm-for-kgm than adults.
- They have higher lung volume to body size, higher respiration rates and spend more active time in the polluted outdoor environment.
- When exposed to fine particles, children have slowed lung function growth, increased emergency room visits, increased incidence of asthma, bronchitis and crib death (CATF Report, Feb. '05).

The Health and Non-Heath Impacts of Different Pollutants

Pollutant	Health Effects	Non-Health Effects
PM_{10}/ $PM_{2.5}$	Substantial epidemiological evidence of adverse acute health effects of particulate air pollution in Europe and US; and strong, but much less widespread, epidemiological evidence of chronic health effects including life expectancy (US only). ExternE approach: Quantify acute mortality (deaths brought forward), chronic mortality (life expectancy), a number of acute morbidity (from respiratory hospital admissions through to minor restricted activity days) and chronic morbidity impacts. Valuation based on willingness to pay. Mortality based on Value of Statistical Life (Australian $6M) but adjusted to reflect years of life lost.	Dominant cause of building soiling. Reduces visual range (visibility).
SO_2	European results have established an association of SO_2 with acute mortality, and probably with hospital admissions. However, evidence from epidemiological studies carried out in the USA is less convincing. SO_2 leads to formation of secondary particles over regional range. ExternE approach: Quantify acute mortality (deaths brought forward), and Respiratory Hospital Admissions (RHA) from SO_2. Quantify sulphates assuming similar to $PM_{10}/PM_{2.5}$.	Material damage (SO_2 and 2^{ndary} pollutants) Effects on crop yield. Ecosystem damage SO_2 and 2^{ndary} pollutants including acidification (though potential benefits through fertilisation). Sulphates reduces visual range (visibility).

CO	Severe health effects at high levels. Relatively little epidemiological evidence concerning ambient CO, but a number of (well-conducted) studies that report positive associations. ExternE approach: Quantify acute hospital admissions (congestive heart failure). Other studies including associations with mortality currently discounted because of the problems separating CO from other components of the air pollution mixture, though information on CO is accumulating and may change in future.	-
NO_2	Effects at very high concentrations. Some studies report ambient NO_2 effects, however, consensus that NO_2 not causal, but acting as a surrogate for mixture (e.g. effects disappear when correct for particles). Possible relationship for respiratory hospital admissions. ExternE approach: Quantify direct effects not quantified. NOx leads to formation of secondary particles over regional range. NOx is an ozone pre-cursor. ExternE approach: Quantify nitrates assuming similar to $PM_{10}/PM_{2.5}$. Ozone pre-cursor.	Ecosystem damage through 2^{ndary} pollutants including N deposition (eutrophication) and acidification (though potential benefits through fertilisation). Ozone pre-cursor. Reduces visual range (visibility).
NmVOC	Ozone pre-cursor..	Ozone pre-cursor
Ozone	Strong evidence of a relationship of between ambient ozone and acute mortality and hospital admissions. ExternE approach: Quantify both mortality and RHA.	Damage to materials (paints, polymers and rubbers). Effects on crop yield.
Benzene	Possible carcinogen. ExternE approach: Quantify mortality.	-
PAHs	Possible carcinogen. ExternE approach: Quantify mortality.	-

For conservative (non-reactive) pollutants, the single most important factor in determining air pollution costs is the population exposed. For local impacts, the population density near the emission source is the major parameter of interest. For regional effects, the population density and the total population are both important.

Population distribution in Sydney

Population distribution in Sydney. (based on 1995 figures)

City Area	Total Population** within Area	Area** (approx) km^2	Average Density** (person/km^2)	Highest Density ** (person/km^2)
Central Sydney	2,394,722	346	6,928	23,861
Outer Sydney	1,244,553	1,602	777	
Total Sydney	3,639,275	1,947	1,869	-

Exposure to outdoor air pollution is associated with a broad spectrum of acute and chronic health effects ringing from irritant effects to death.

Box 1. HEALTH OUTCOMES POTENTIALLY RELEVANT FOR HEALTH IMPACT ASSESSMENT OF AIR POLLUTION

Acute outcomes

- Daily mortality
- Respiratory hospital admissions
- Cardiovascular hospital admissions
- Emergency room visits for respiratory and cardiac problems
- Primary care visits for respiratory and cardiac conditions
- Use of respiratory and cardiovascular medications
- Days of restricted activity
- Work absenteeism
- School days missed
- Self-medication
- Avoidance behaviour
- Acute symptoms
- Physiologic changes, e.g. in lung function

Chronic disease outcomes

- Mortality (in infants and adults) from chronic cardio-respiratory disease
- Chronic respiratory disease incidence and prevalence (including asthma, COPD, chronic pathological changes)
- Chronic change in physiologic function
- Lung cancer
- Chronic CVD

Reproductive outcomes

- Pregnancy complications (including fetal death)
- Low birth weight
- Pre-term delivery

(From WHO, Quantification of the Health Effects of Exposure to Air Pollution 20-22 November, 2000).

The frequency of occurrence, of the health outcomes universally related to its severity (see Figs. 1 and 2 below).

Fig. 1. Air pollution health effects pyramid (adapted from ATS 2000)

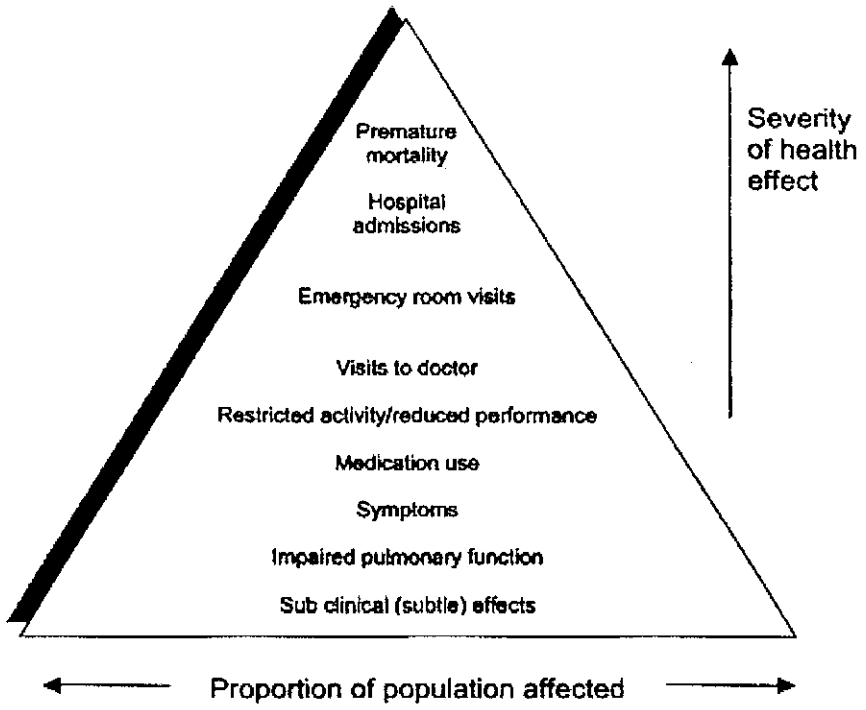
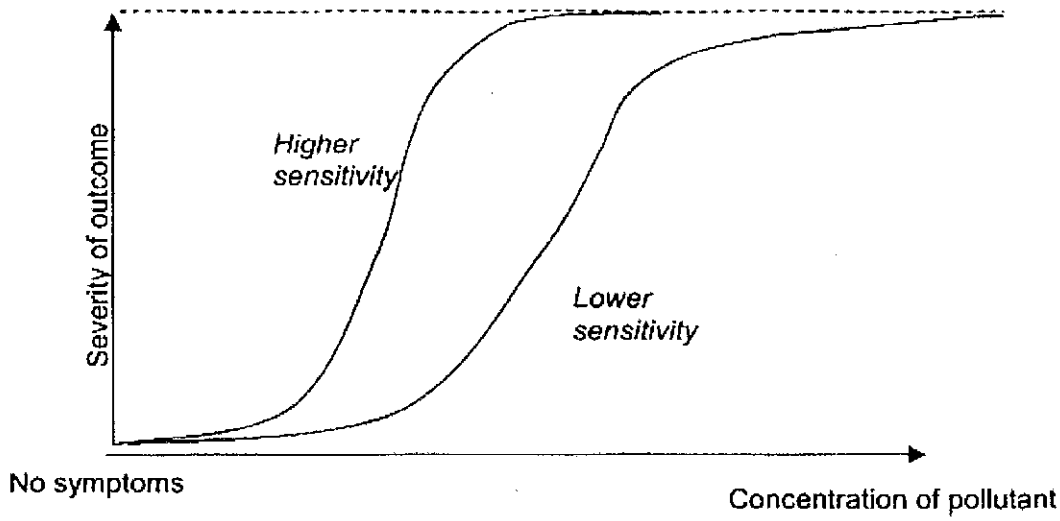


Fig. 2. Severity of health response to air pollutant in relation to subject's sensitivity



(From WHO, Quantification of the Health Effects of Exposure to Air Pollution 20-22 November, 2000).

This suggests that the total impact is likely to exceed that contributed by the less frequent, more severe outcomes, and, in some cases, may be dominated by the less severe, but more frequent, ones (Fig 1.)

As an individual's sensitivity to pollutant exposure increases, so the severity of the response will increase for a given pollutant exposure. Thus, a response resulting in a specific outcome (e.g., hospital admission) will occur at a lower concentration in a more sensitive individual. See Fig. 2 which is a model of two hypothetical individuals with differing sensitivities.

Many deaths caused by air pollution occur among those who are frail due to either chronic disease, or to some transient condition.

In evidence presented by Dr. Stephen Corbett (Acting Director, Health Protection Branch, NSW Health Department) to the NSW Inquiry on the Protection of the Environment Operations (Clean Air) Regulations 2002, (Report November, 2002) he informed the Committee that:

> *"The documented effects of motor vehicle emissions on health are increased death rates from heart disease and lung disease, especially lung cancer, increased admissions to hospital for heart and lung diseases, and increased rates of asthma attacks, days off work and respiratory complaints in the general population. I would like you to note that that is a mixture of acute effects that happen immediately after or soon after exposure and long-term effects, such as lung cancer which results from months and years, a lifetime of exposure to pollutants. That is an important issue. . . Numerous studies, including those undertaken in Sydney, have noted a dose and response relationship between fine particle pollution and many of these health outcomes. That is, quite simply, the more you are exposed to, the greater the risk."*

Dr. Corbett stated further:

> *". . .We are not just concerned about lung cancer here, but lung cancer has been an important bench-mark, I think, because the thing about lung cancer is it is much more related to chronic long-term lifetime exposure. That is what is relevant in terms of lung cancer risk, rather than what you get today or the next day; it is an accumulated risk over a lifetime."* (Report November, 2002, p. 23)

It is especially noteworthy that. . . . reported from a study involving 500,000 Americans that **one fifth** of lung cancer cases was associated with exposure to **fine** particles of vehicle exhaust. See link:

http://www.newscientist.com/hottopics/pollution/pollution.jsp?id=23331100

Also highly relevant is confirmation by Nigel Routh (Director, Air Policy, NSW Environment Protection Authority) in evidence to the NSW Inquiry on Protection of the Environment Operations (clean Air, Regulation, November 2002, - that production of **'fine particles' are likely to be worse from vehicle exhaust in the future"**

"Diesel exhaust contains high levels of particulate emissions and oxides of nitrogen. Emerging research is indicating that while new vehicle standards will reduce these emissions, when it comes to particles there may well be an increase in those particles which are suspected of having the greatest impact on health – i.e., those ultra-fine particles below $PM_{2.5}$ microns in size. Given the findings of current research it is important that there is a precautionary approach to policy making in this area. . . Diesel emissions are projected to fall as new vehicles enter the market and diesel sulphur is reduced. On the other hand, the EPA is increasingly aware of the ***health impacts of fine particles and that the situation of these may worsen in the future****. Australia will soon have a national air quality standard for fine particles. . . The percentage of vehicles that emit higher levels of pollutants increases with vehicle age. Certainly, experience to date is that if diesel vehicles are not properly and regularly maintained their emissions quickly increase and often dramatically."*
(EPA Response to Committee by Nigel Routh, EPA, 8 November, p. 15).

Recent evidence has emerged that PM10 might not be the measurement most representative of the fraction of the ambient aerosol that is responsible for its harmful effects on health. Evidence has accumulated that this toxicity may lie in a finer fraction of the particles, perhaps below 2.5 µm or smaller. Moreover, it has become apparent that PM10 measurements may include an amount of resuspended dust of probable low toxicity, leading to difficulties in compliance with the Standard in areas where coarse particles, such as wind-blown dust or sea spray, may make an important contribution.

Levels of fine particle matter, PM2.5, in Sydney's air-shed already exceed the National annual average for PM2.5 reporting standards.

The Bureau of Transport and Regional Economics (BTRE) in Canberra estimated that in 2000, twice as many people in Sydney died from exposure to vehicle exhaust pollutants than died from road accidents

In 2005, NSW Health published two papers - one reporting adverse effects on the developing fetus of mothers exposed to Sydney's pollution and the second, the increased risk of heart attack in the elderly, confirming overseas findings.

I was in Canberra (Thursday 15 June,2006) as an invited speaker at the Bureau of Transport and Regional Economics Colloquium to speak about 'Health Impacts of Fossil Fuels'. During discussion I was advised by Dr David Cosgrove (BTRE) that he had for years been trying to make the standards for particulates more stringent i.e., to incorporate numbers and surface area of fine particles as a measure, rather than by weight.
Similarly, Mr Robin Seeley (Assistant Director –Air Quality Section, Dept of the Environment and Heritage) expressed major concerns about the irrelevance of the current standards for particles in terms of health risk.

According to international experts - Professors Lidia Morawska and Michael Moore - of Queensland, the current PM10 standard is inappropriate for assessing health risk. The following link deals with their excellent review of fine and ultrafine particle pollution:

http://www.deh.gov.au/atmosphere/airquality/publications/health-impacts/index.html

It is highly relevant that in Section 1.3 under 'Toxicology' they make the following profound statement:

> *"All of the studies available to us demonstrate that the primary determinant of the effect of ultrafine particles is their number and their surface area and not the weight of particles present. This means that the traditional use of PM weight measures is inappropriate in evaluation of the likely biological effects of ultrafine particles."*

The point is that weighing particulate pollution, as currently done, is inappropriate for determination of health risk. For example, one coarse PM10 particle is equivalent in weight to one BILLION PM0.01 particles but the latter has x1000 times the surface area of ONE coarse PM10. The other factor is the coarse particles are insoluble. In contrast, fine particles are soluble in the respiratory tract and release chemical carcinogens.

It is not entirely clear why particles are so dangerous, although a recent research has shown that the system may be reacting to fine particles as if they were invaded by bacteria. The response is to greatly increase receptors on normal cells to respond to exhaust particles that simulate molecules on bacteria. The effect is to hyper-respond to natural loads of bacteria that would usually be handled by normal clearance mechanisms. Thus, this hyper immune response inflammation of tissues is similar to the allergic reaction of an asthmatic or hay-fever sufferer but when ultra-fine particles enter deep into the lungs and provoke 'non-allergen' inflammation. It is estimated that in Britain more than 10,000 die prematurely each year and is Britain's greatest environmental problem. Australia has the same serious environmental problem.

In view of this evidence, the Government should immediately investigate the introduction of alcohol fuels along with filtration technology as well as an additional (or perhaps alternative) more stringent PM_{10} Standard for particulate matter. Also taking into account that the PM_{10} measure provides no useful information about particles in exhaust of vehicles. The smaller respirable size range $PM_{2.5}$ appears to be responsible also for toxic effects. Only now has Australia introduced 'guidelines' for $PM_{2.5}$ but are not yet enforceable. This report briefly reviews the evidence and gives conclusions consistent with those documented elsewhere.

Size Distribution and Chemical Nature of Airborne Particles

The likelihood of a particle remaining suspended in the air depends upon its size, shape and density, and that those particles most likely to be inhaled into the lung are usually below about 10 µm in diameter. Below this size, particles differ in their chances of being deposited on the inner surface of the lung's tissue and, therefore, in their potential to cause harm.

The size distribution of particles in the urban air is conventionally characterised by three modes. The smallest of these, below 0.1 µm in diameter, is called the **nucleation** mode and is formed by **condensation** of hot vapour from combustion sources and from chemical conversion of gases to particles in the atmosphere. Particles of this size have a high chance of deposition in the gas-exchanging (alveolar) part of the lung; they are relatively short-lived and grow into larger particles between 0.1 and about 1 µm in diameter, known as the **accumulation** mode. These particles remain suspended for up to several weeks in the air, and are not readily removed by rain.

The third, **coarse**, mode comprises particles greater than about 2 µm in diameter. These are generally formed by break-up of larger matter, and include wind-blown dust and soil,

particles from construction and sea spray. Their size means that they remain in the air for relatively short periods, but they make (in relation to their numbers) a disproportionate contribution to PM10 mass when this is measured close to a source.

The chemical composition of ambient airborne particles varies both in time and space, depending on the activity of local and distant sources and meteorological conditions. Combustion processes produce particles based on carbon, carrying a variety of metal compounds and organic chemicals derived from the fuel burnt. Photochemical reactions produce mainly ammonium sulphate and nitrate particles, derived in part from gases produced by combustion sources and in part from ammonia derived from animal sources. Distinct differences in the proportions of these components occur in different seasons. In addition to this fine component, the coarser fraction may contain a wide variety of chemical substances including salt, silicates and biological particles, depending on local sources.

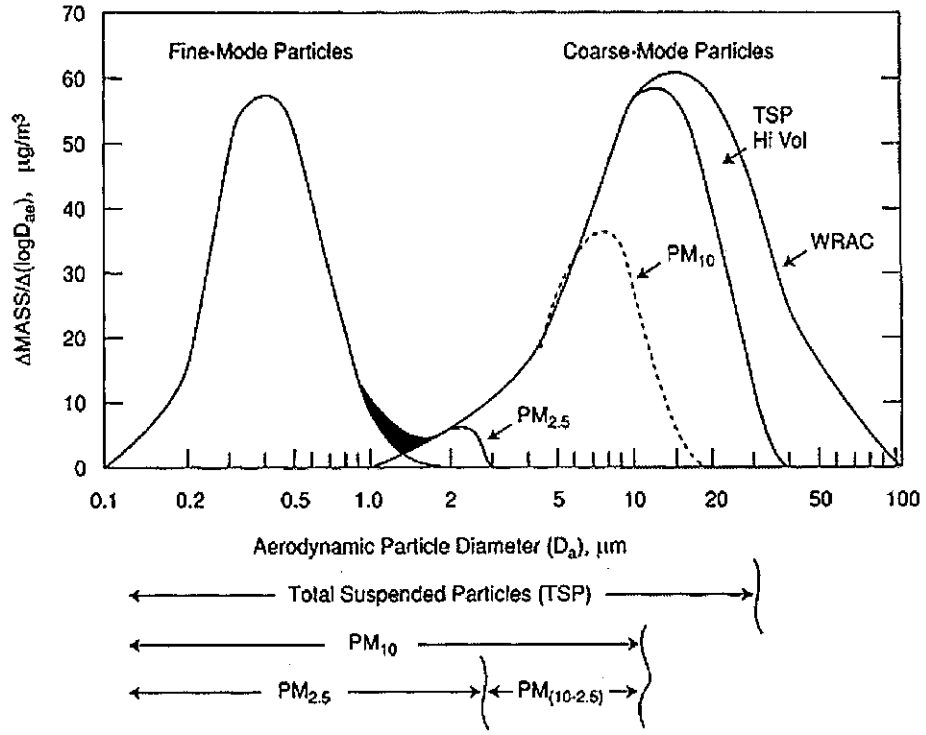
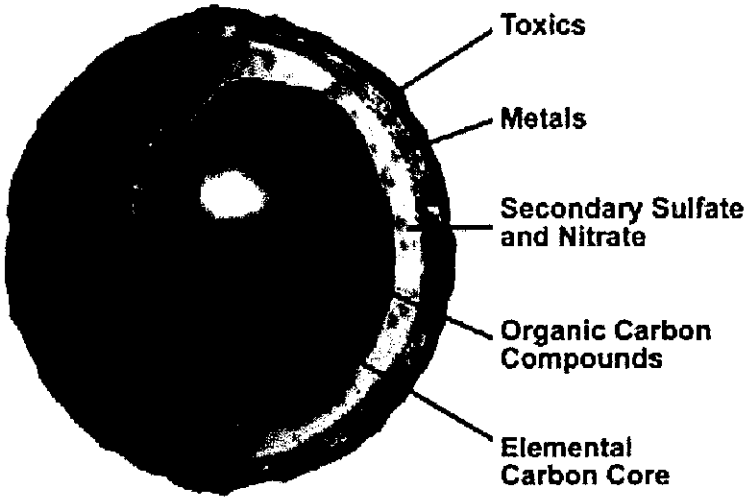


Figure 3. Representative example of a mass distribution of ambient PM as function of aerodynamic particle diameter. A wide-ranging aerosol collector (WRAC) provides an estimate of the full coarse mode distribution. Inlet restrictions of the high volume sampler for TSP, the PM_{10} sampler, and the $PM_{2.5}$ sampler reduce the total mass reaching the sampling filter.
Below is depicted a diesel particle with adsorbed PAH's on the surface

http://www.who.int/environmental_information/Air/Guidelines/Chapter2.htm

> Diesel particles are carbon at their core with toxics and carcinogenic substances attached to their surfaces.



http://www.catf.us/publications/reports/Diesel_Health_in_America.pdf

Table 1. Comparisons of ambient fine and coarse mode particles

	Fine Mode	Coarse Mode
Formed from:	Gases	Large solids/droplets
Formed by:	Chemical reaction; nucleation; condensation; coagulation; evaporation of fog and cloud droplets in which gases have dissolved and reacted.	Mechanical disruption (e.g. crushing, grinding, abrasion of surfaces); evaporation of sprays; suspension of dusts.
Composed of:	Sulphate, $SO_4^=$; nitrate NO_3^-; ammonium, NH_4^+; hydrogen ion, H^+; elemental carbon; organic compounds (e.g., PAHs); metals (e.g. Pb, Cd, V, Ni, Cu, Zn, Mn, Fe); particle-bound water.	Resuspended dusts (e.g., soil dusts, street dust); coal and oil fly ash, metal oxides of crustal elements (Si, Al, Ti, Fe); $CaCO_3$, NaCl, sea salt; pollen, mould spores; plant/animal fragments; tire wear debris
Solubility	Largely soluble, hygroscopic and deliquescent	Largely insoluble and non-hygroscopic

Sources	Combustion of coal, oil, gasoline, diesel, wood; atmospheric transformation products of NO_x, SO_2 and organic compounds including biogenic species (e.g. terpenes) high temperature processes, smelters, steel mills, etc.	Resuspension of industrial dust and soil tracked onto roads; suspension from disturbed soil (e.g. farming, mining, unpaved roads); biological sources; construction and demolition; coal and oil combustion; ocean spray
Lifetimes	Days to weeks	Minutes to hours
Travel Distance	100s to 1000s of kilometres	< 1 to 10s of kilometres

http://www.who.int/environmental_information/Air/Guidelines/Chapter2.htm

Particle Sizes

http://www.defra.gov.uk/environment/airquality/aqs/air_measure/index.htm

Three curves (above) showing the number-, area-, and volume-size distributions, taken from Whitby (1978). Figure 3 shows the fraction of the number of particles in each size range in the idealised urban aerosol. The curve shows a predominant single peak with a shoulder towards higher particle sizes. This number distribution peaks at about 0.013 µm (13 nm) with a total number density of about 100,000 particles per cm3. Particles in this size range below about 0.1 µm are said to be in the **nucleation mode.**

Nucleation mode particles have been emitted into the atmosphere as primary particles by combustion sources (primary particulate material), both stationary and mobile. Some nucleation mode particles are formed by condensation of gaseous material through gas-particle conversion processes (secondary particulate material). Few vapours can readily form totally new nucleation mode particles directly, but sulphuric acid vapour, formed by the photochemical oxidation of sulphur dioxide, has this capacity. In urban areas, nucleation mode particles are the most numerous of all the particles due to constant replenishment by fresh emissions.

Urban aerosol pollution shows a strong single peak with shoulders to both higher and lower particle sizes. The area distribution peaks at about 0.1 µm.
Particles in the size range 0.1 to 1 µm are said to be in the accumulation mode. Few accumulation mode particles are actually emitted into the atmosphere in this particle size range, rather they form by coagulation. Because this mode contains the bulk of the surface area of the particles, these particles offer the largest target area for adsorbing gaseous material. The process of **coagulation** reduces the number of particles.

The process of **adsorption** of gaseous material from the atmosphere onto pre-existing nucleation or accumulation mode particles leaves the number of particles unchanged but causes the area, volume and mass of the suspended particulate material to grow rapidly. The accumulation mode is therefore the ageing region for all small particles because growth is rapid and loss processes are at a minimum in this size range. Typically there might be about 10,000 particles per cm^3 in the accumulation mode in an urban area. Coarse and nucleation mode particles make a relatively small contribution to the total surface area of the urban aerosol.

Total volume (and hence mass) of the particles in each size range in the same urban aerosol pollutionas shows a **bimodal** distribution and the almost complete absence of a shoulder to lower particle sizes. The volume (or mass) distribution **peaks at particle sizes of about 0.3 and 6 µm**. Thus the volume (or mass) of the urban aerosol appears to be in two modes: the **accumulation** mode and the **coarse** mode, with the latter containing particles in the size range 2 µm and above.

The **nucleation** mode particles, although the **greatest in number**, contain a negligible fraction of the aerosol volume and mass.

Most of the particles in the **coarse** mode are formed by the frictional processes of comminution, such as wind-suspended soil dust or sea spray from breaking waves (primary suspended particulate material) and from the slow growth of particles from the accumulation mode (secondary particulate material). Typically, there might be a few tens or hundreds of particles per cm^3 in the coarse mode in an urban area. However, not all of the coarse mode particles pass through the PM10 sampling and monitoring system and are measured, as some are too large.

Coarse mode particles generally account for about 20-50% of the urban background PM10 mass.

Figure 4

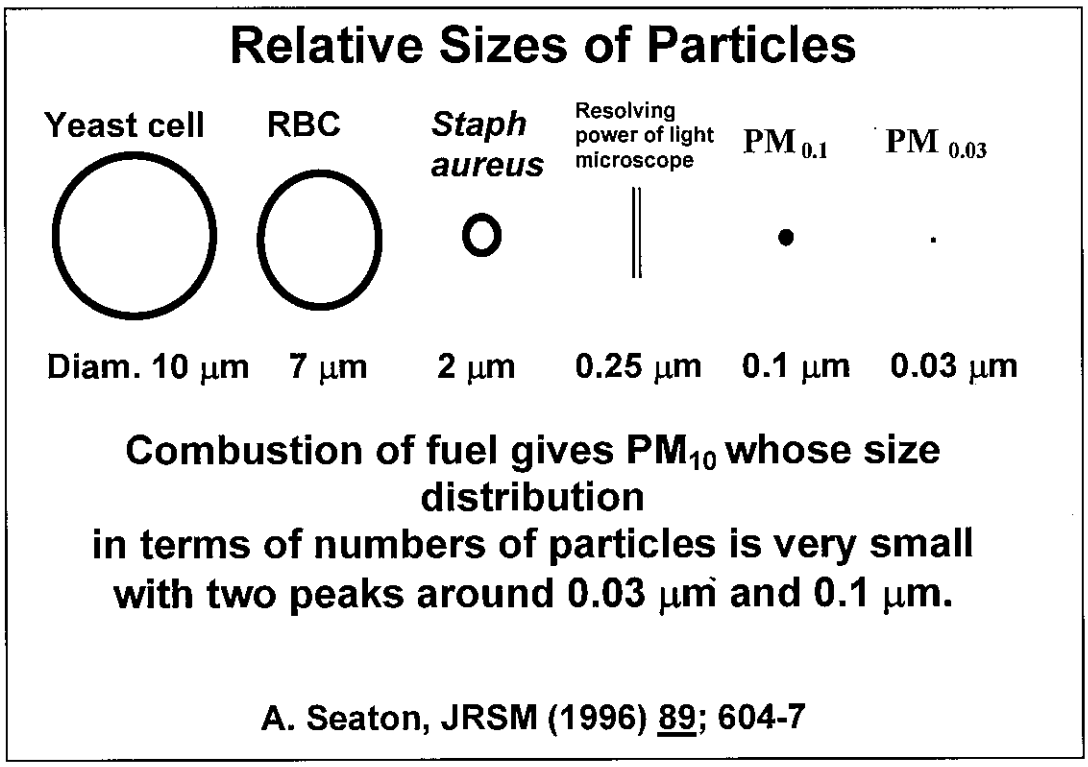
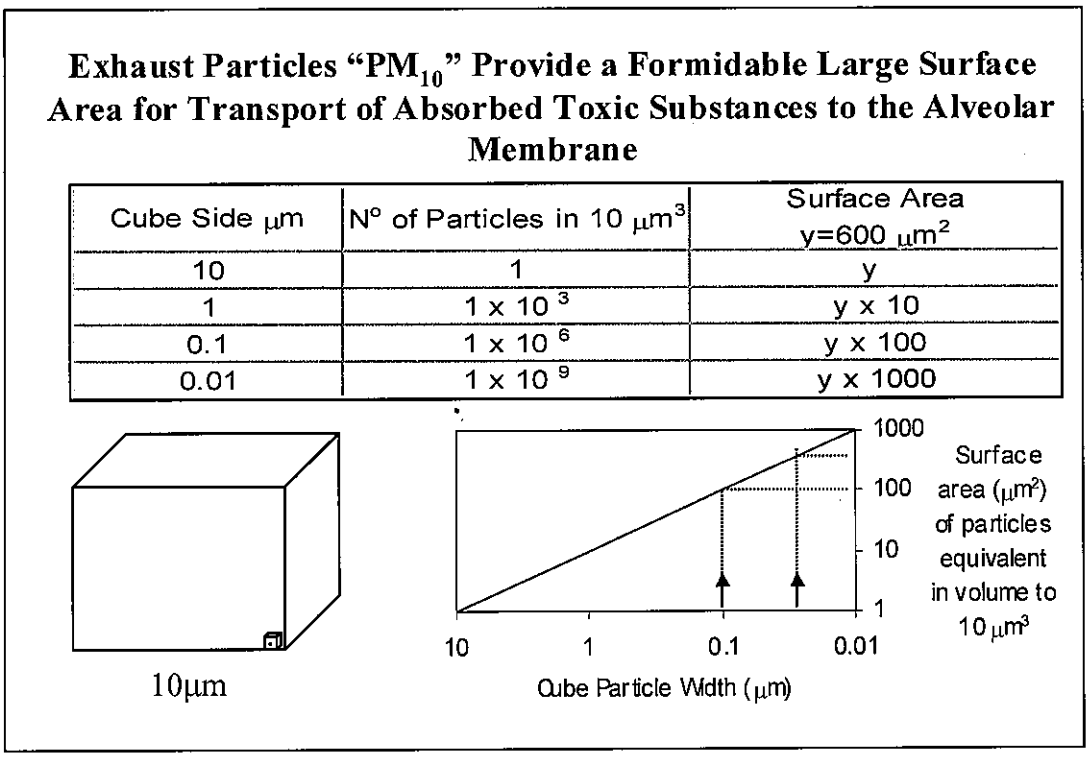


Figure 5



Importance of Particle Size

A serious and fundamental defect in monitoring air pollution, is the failure to take account of the fact that the relevant size of particles from combustion of fuel is within two peaks[2] - 0.03μm and 0.1μm. Neither of these particle sizes (mass or number) is measured in any of the EIS Air Quality surveys.

Morawska &Thomas[3] report that only 3% of combustion particles between 0.1μm and 1μm are present in PM_{10} measurements. Despite this research conducted for the Queensland Environment Protection Agency, the RTA continues to claim that PM_{10} measures all particles less than 10μm in size. Monitoring by NSW EPA excludes most particles 1μm and less. About 90% (by mass) of diesel particulates[4] are less than 1μm and are missing in the PM_{10}/M^3 measurements in most EIS Reports. As a consequence, PM_{10} measurements of air quality and those of air-streams vented from tunnel stacks are seriously under-estimated.

Morawska & Thomas[5] concluded: "*PM_{10} measurements provide information almost entirely on particles generated from mechanical processes. In an urban environment, this could mean particles re-suspended by vehicular traffic and mechanical wear and tear of tyres,* ***but not on emissions of motor vehicles."***

Therefore, if the PM_{10} measurement is not an instrument for evaluating traffic emissions, the air pollution data and the calculated health-risks are also underestimated. The above authors conclude that PM_1 measurement provides very good information about contributions from the combustion engine, and distinguishes it from suburban background. The fact that PM_1 contributions are about two-fold greater in traffic aerosol than in ambient air[6] is a serious deficiency in pollution monitoring by the RTA. Use of PM_{10} data serves to hide exceedances of air quality standards. By using PM_{10} measurements, the bulk of tail-pipe particulates are excluded and what is analysed is mainly re-suspended dust from the road surface[7]. The exclusion of combustion particles suppresses data of the enormous surface area (> 100 fold) of toxic respirable particles missing from PM_{10} measurements. Several overseas countries have a standard for PM_{10} and $PM_{2.5}$. Australia has only now introduced a 'guideline' for $PM_{2.5}$ additional to a standard for PM_{10}. It is misleading to imply PM_{10} is stringent and includes all particles as fine as $PM_{2.5}$ and PM_1.

Ultrafine and Nanoparticles

A major omission in current EIS assessments of the atmospheric air quality and of the air-stream from the tunnels, is the failure to understand that, unlike the carbonaceous soot particles associated with older diesel engines, many of the particles are ultra-fines and nanoparticles[8] [9].

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[2] Seaton Anthony, *Particles in the air: the enigma of urban air pollution*, Journal of the Royal Society of Medicine, Vol 89(11), pp 604-607, November 1996.

[3] Morawska Lidia, Thomas Stephen, *Modality of ambient particle distribution as a basis for developing air quality regulations.* 15th International Clean Air & Environment Conference, Sydney, Nov. 2000, Vol 1 pp 432-437.

[4] Brown S. et al, *Emission profile of the Australian diesel fleet project 2-2 of DNEPM preparatory work* 15th International *Clean Air & Environment* Conference Sydney, November 2000, Vol. 2.

[5] Morawska. Op cit.

[6] Morawska. Op cit.

[7] Ibid

[8] Kittleson D.B. *Engine & Nanoparticles: A Review*, J. Aerosol Sci., Vol. 29, No 5/6 pp 578-88, 1998.

Many of these particles are not formed in the engine but by the gas-to-particle conversion processes from vapour phase particle precursors as the exhaust dilutes and cools in the atmosphere. As Kittelson[10] has shown, these processes are extremely non-linear and difficult to simulate in the laboratory.
Recent measurements of such particles, and the problems associated with their measurement, were described by Kittelson[11]. The final measure of merit of a dilution and sampling system is whether such particles form exhaust particle size distributions that are similar to those produced by atmospheric dilution. Current EIS Reports assume that particle size distribution from vehicle exhaust is similar to atmospheric dilution.
Kittelson[12] stated:" *....that a sampling and dilution scheme capable of simulating atmospheric dilution has not yet been identified. In fact, it may not be possible to come up with a single robust approach that is appropriate to all situations".*
Kittelson concluded from studies of nanoparticles that: *a significant amount of particulate matter (e.g., 90% of the number and 30% of the mass) is formed during exhaust dilution from particle precursors that are in the vapour phase in the tailpipe (e.g., sulphuric acid, fuel and oil residues). New particles are formed by nucleation. This is likely to be the source of most of the ultrafine and nanoparticles (and particle number) associated with engine exhaust. Pre-existing particles grow by adsorption or condensation.*
Currently, most of the particles in the nanoparticle size range are volatile. However, as engines become cleaner, metallic ash particles from the lubricating oil (or fuel if metallic additives are present) may become more important.
Nanoparticle measurements are very strongly influenced by the sampling and dilution techniques employed. Nucleation, adsorption, absorption, and coagulation during sampling and dilution depend upon many variables, including dilution rate, (or residence time at intermediate dilution ratio), humidity, temperature, and relative concentrations of carbon and volatile matter[13].
Sampling and dilution systems should be designed to mimic atmospheric dilution as closely as possible in order to obtain samples representative of the tailpipe to nose process, and thus of what we breathe. **The design of such systems remains a great challenge.**[14]

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[9] Kittleson D.B. *Recent Measurements of Nanoparticle Emissions from Engines,* Conference Current Research on Diesel Exhaust Particles – Japan Assoc. of Aerosol Science & Technology, Tokyo Japan, 9 Jan. 2001.

[10] Kittleson D.B. *Engine & Nanoparticles: A Review.* Op cit

[11] Kittleson, D.B. *Recent Measurements of Nanoparticle Emissions from Engines.* Op cit

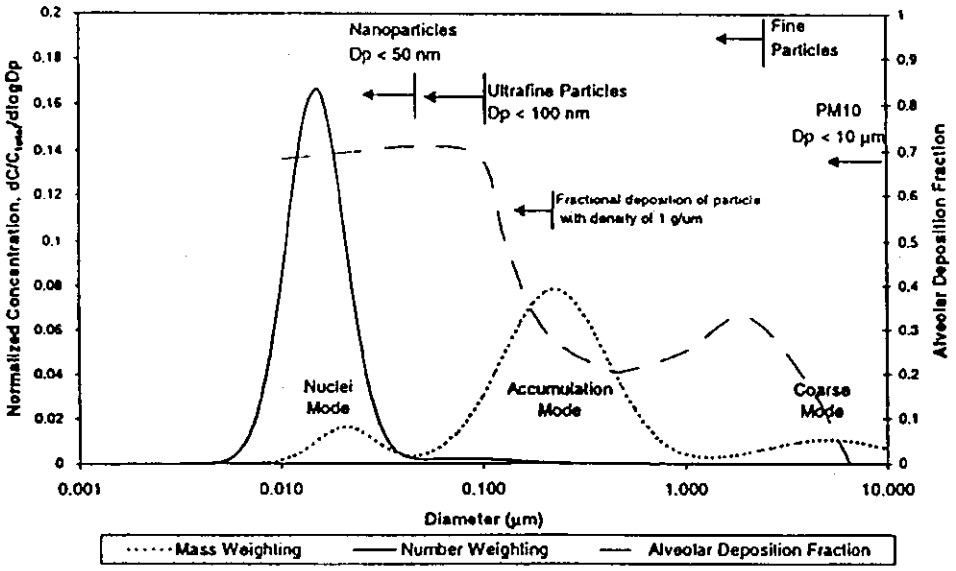
[12] Ibid

[13] Kittleson D. B. *Recent Measurements of Nanoparticle Emissions from Engines.* Op cit

[14] Ibid

The figure reproduced below (from Kittelson, 1998) illustrates number and mass weighted size as well as the alveolar deposition curve for diesel particular matter.

Figure 6



Typical engine exhaust mass and number weighted size distributions shown with alveolar deposition.

Do PM_{10} Measurements Apply to Emissions of Motor Vehicles?

Morawska and Thomas (2000) in their studies used the trimodal nature of atmospheric aerosol number sizes and consistency as follows:

(a) **Nuclei**, N, (<0.1µm). These originate from the condensation of hot, highly supersaturated vapours released during combustion.

(b) **Accumulation**, A, (<1.0µm but >0.1µm). These arise from combustion through coagulation and heterogenous nucleation to accumulate the submicrometre aerosol mass in the accumulation mode

(c) **Coarse**, C, particle (>1.0µm). Almost all particles in the coarse particle mode originatefrom natural and anthropogenic mechanical processes.

Conclusions made by Morawska & Thomas (2000) in their study of the relation between fractional contribution of volume from different modes in the particle size distributions (and thus from different sources) to PM_{10}, $PM_{2.5}$ and $PM_{1,}$ are as follows:

*"**PM_{10} measurements** provide information almost entirely on particles generated from mechanical process. In an urban environment this could also mean particles resuspended by the vehicular traffic and mechanical wear and tear of the tyres, **but not on the emissions from motor vehicles.***

***$PM_{2.5}$ measurements** also provide information mainly on particles generated by mechanical process, but the contribution from combustion process (N + A) modes becomes significant. Thus interpretation of $PM_{2.5}$ data could become very complex in order to distinguish the contribution from different type sources. It follows that the*

application of this parameter as bases for standards may not adequately facilitate control of particle emissions and concentrations.

***PM_1 measurements** provide very good information about contributions from **combustion processes** and enable a much better distinction between combustion and **mechanically** generated aerosols. It would thus appear that the existence of PM_{10} and PM_1 standards would be most desirable from the legislation point of view."* (Morawska & Thomas, 2000).

Table 2 Fractional contribution of N+A and C modes to the volumes of PM_1, $PM_{2.5}$ and PM_{10}

Environment type	PM_1 % contribution (by mass)		$PM_{2.5}$ % contribution (by mass)		PM_{10} % contribution (by mass)	
	N+A	C	N+A	C	N+A	C
Traffic Influenced	82	18	14	86	3	97
Urban Influenced	47	53	10	90	< 1	> 99
Vegetation burning	100	0	84	16	37	63
Marine influenced	6	95	< 1	> 99	0	100
Modified background	84	16	12	88	< 1	> 99
Suburban background	38	62	1	99	< 1	> 99

From L. Morawska and S. Thomas (2000)

(a) **Nuclei**, N. (<0.1μm) These originate from the condensation and coagulation of hot, highly suspersaturated vapours released during **combustion.**

(b) **Accumulation**, A (<1.0μm but >0.1μm). These arise from combustion through coagulation and heterogenous nucleation to accumulate the submicrometre aerosol mass in the accumulation mode.

(c) **Coarse**, C, particle (>1.0μm). Almost all particles in the coarse particle mode originate from natural and anthropogenic mechanical processes.

The implication of such studies, that confirm reports of other research groups, is that the NSW Regulatory Authorities and some air-quality consultants use PM_{10} monitoring data that do not relate to emissions from motor vehicles. Only PM_1 measurements provide very good information about contributions from the combustion process of vehicles, but are not done routinely. In the recently opened 4km, twin M5 East tunnel, carrying over 85,000 vehicles per day, emissions are vented from a single, unfiltered stack in a valley within a residential area.

In its assessment of health risks of residents exposed to these emissions *"The Department of Health is of the view . . that the stack* (M5 East) . . *will result in minimal changes to the local air quality, with no detectable health impacts."* (NSW Health Submission to the M5 East Parliamentary Inquiry, 3 June, 2001). Six months after the opening of the M5 East tunnel, independent health assessment concludes, on behalf of the local resident action group: *"During the past six months since the opening of the M5 East tunnel, 430 complaints from 24 household and one industrial site within 700m of the stack. . . The impacts recorded so far by the residents point to a significant problem of odour annoyance and adverse health impacts. Several of these residents have obtained medical certificates from their doctors confirming that their physical health has deteriorated over this period, for no apparent physiological reason."* (Katestone Environmental, 8 August,2002).

Air Quality Monitoring and Assessment

The decline in the domestic and industrial burning of fossil fuels and the associated pollutants has been replaced by a steady increase in traffic-associated pollutants such as finer, respirable particulates, oxides of nitrogen (NO_x), ozone (O_3), and toxic volatile organic compounds (VOCs).
For particulate matter (PM), monitored in many countries as PM_{10} (less than an aerodynamic diameter of 10µm), no safe threshold for exposure has been identified (Dockery *et al.,* 1994). This is different to NO_x, O_3 and most VOC's.

Flaws in Monitoring of Air Pollution

Ayers *et al.*, (CSIRO, 2001) confirmed that a routinely used method of continuous sampling and monitoring PM_{10} known as the Tapered Element Oscillating Microbalance (TEOM) under-estimated PM_{10} values, below temperatures of about 15 - 17°C, by as much as 25%.
The national automatic air quality monitoring networks employ mainly TEOM PM10 samplers, together with some TEOM PM2.5 instruments.
The TEOM instruments were adopted because of their ability to provide measurement data in real time, as compared to measurements from traditional gravimetric methods, which are available only after the period required for collection and weighing. TEOM measurements, therefore, allow the provision of real time information to the public and input into research to identify sources of particulate matter.

In the TEOM sampler, particles are collected on a filter that is attached to the vibrating element of a tapered element oscillating microbalance. The element vibrates at a precise frequency that changes according to the mass of particles on the filter, allowing a direct determination of collected mass. In theory such measurements should be directly

equivalent to those of a gravimetric procedure using the same inlet characteristics, but in practice divergences occur in the presence of semi-volatile components of the particulate matter.

This is because the TEOM instrument, as used in the national networks, pre-heats the air to 50 °C prior to particle collection in order to drive off associated water and other components which might lead to inconsistent and variable mass measurements. This causes loss of some semi-volatile particulate components (e.g. ammonium nitrate and some organic compounds). TEOM instruments give lower measurements of particle mass than both high and low volume samplers that do not involve pre-heating the sampled air, although these too will lose some volatile material (APEG 1999).

Because the differences depend upon the chemical composition of the particulate matter, they are dependent on both site and season, and no universally applicable correction is available. In measuring PM10 the divergence is typically of the order of 20%, but rarely exceeds 40%.

In addition, NSW air quality monitoring allegedly does not incorporate an additional correction factor for 'secondary particles.' In air quality studies e.g., in France (Filliger, 1999) when TEOM measurements are used for background, to introduce the missing secondary particles into the analytical model, a constant correction value of 9.5µg/M^3 is added to all grid points. This value of 9.5µg/M^3 corresponds to regional background of secondary particles that appear to be excluded from all air quality analyses in NSW. Such omissions would appear to invalidate regular health-risk assessments.

Failure to incorporate a correction factor UNDERESTIMATES particulate pollution

The following is a copy of the Executive Summary of a
REVIEW OF LANE COVE TUNNEL PM_{10} IMPACT ISSUES

FROM KATESTONE ENVIRONMENTAL
April, 2003

Note how the failure by the NSW RTA and NSW EPA to incorporate the prescribed correction factor for TEOM PM10 continuous measurements can UNDERESTIMATE the particulate pollution levels by 11-40%.

Executive summary

Katestone Environmental was requested to investigate the suitability of techniques used in determining background concentrations of fine particulate matter in the Lane Cove Tunnel Environmental Impact Statement (EIS) and Representations Report. The Lane Cove Council is concerned that estimates using continuous (TEOM) monitors may lead to significant underestimates of true values and may not include properly the presence of secondary particles (very fine aerosols such as organic compounds, nitrates and sulphates) that are formed in the atmosphere after being emitted from combustion sources such as vehicles. The EIS used TEOM measurements from Lindfield. Information on secondary particle composition is not mentioned but is available for some sites in Sydney (e.g. Mascot, Liverpool). A literature search has shown that

concerns on the underestimation of PM_{10} by automatic monitors are widespread in other countries especially where transport of pollutants between airsheds is significant.

Current techniques for measuring particulate air quality are known to have deficiencies and may require correction factors to measurements, prior to being used for health assessments. Many air quality managers in city councils in the United Kingdom, Europe and elsewhere use factors that can depend on climate, air quality and type of instrumentation. Correction factors for continuous automatic PM_{10} measurements vary from 0.8 – 2.0, with larger values recorded in wintertime near major road networks for some European countries. The correction factors are thought to be required because some inorganic and organic aerosols are inefficiently captured by filters and may be volatised in subsequent measurement processes. At this stage, international studies have yet to confirm fully this assertion and better instrumentation to capture all primary and secondary particles is not widely available.

For the Lane Cove EIS, 1997 information from Lindfield was used for background air quality but no correction factors were applied to the TEOM measurements. For this report, comparisons of TEOM and high-volume air sampler (HVAS) information for 1997 show that the TEOM underpredicts HVAS by 11 ± 7% for all seasons but winter. Winter correction factors are in the range 29 ± 7%. Similar but smaller seasonal variations were found recently for the M5 East network. Corrections based on air temperatures alone do not appear reasonable for most Sydney sites - of the past recommendations of a CSIRO report, preference should be given to using site-specific values, not the default values.
The variation is thought to reflect the differing amounts of the more volatile ammonium nitrate, organic compounds and primary nitrogen oxides at the various sites under different air quality conditions. Long-term measurements at Mascot by ANSTO using low-volume samplers show a ten-fold variation throughout the year in the mass of organic aerosols, a two-fold variation in ammonium sulphate and a two-fold variation in very fine particulates ($PM_{2.5}$) loading. Nitrate levels appear to be low and we believe that the variability of organic compounds is probably the main cause of TEOM variability for inner Sydney sites. Even HVAS sampling may result in some loss of nitrates and organics, depending on the laboratory procedures. Recent international studies show that the "true" values of PM_{10} can be estimated in various independent ways and give reasonable agreement with corrected measurements.

We have applied the seasonal correction factors to Lindfield data for 1997 to determine the variability of "true" background levels of PM_{10}. The EIS choice of worst-case background 24 hour average PM_{10} of 39.8 $\mu g/m^3$ is likely to be too low on several days.

Whether the underestimation of background levels has a major significance depends on the potential errors involved in various other stages of the EIS assessment and the choice between air quality goals or risk assessment procedures to evaluate the health consequences of tunnel emissions. The accuracy of the predictions of the EIS and subsequent evaluations are discussed with respect to:

(a) The choice of background PM_{10} values.

(b) The likely accuracy of emission rates of PM_{10} from vehicle exhaust and vehicle tyre wear.

(c) The need to include road dust in emission estimates, especially for confined situations such as tunnels and street canyons.

(d) The applicability and accuracy of dispersion modelling.

(e) The basis of health guidelines for PM_{10}.

(f) The importance of secondary aerosols for health assessments near roadways.

The conclusions from these considerations are:

- *Background levels of PM_{10} have been underestimated by 11–40%, dependent on meteorological and air quality conditions.*
- *The emission rates from the tunnel have probably been overestimated if road dust is neglected, and underestimated by up to 50% if road dust is included.*
- *The dispersion modelling is likely to underestimate worst-case PM_{10} concentrations on nearby buildings unless the ventilation air temperatures are well above ambient temperature (as for the M5 East tunnel). The degree of underestimation has not been quantified in this report, and will depend on final design configurations*
- *Health guidelines have generally been based on various measurements (HVAS, TEOM, light scattering devices etc). British guidelines are based on uncorrected TEOM measurements. Australian guidelines are mainly based on uncorrected measurements of HVAS and light scattering instruments.*
- *Australian TEOM results should therefore be corrected to use with recommended guidelines.*
- *Some recent Australian research shows some health impacts at levels below the NSW PM_{10} goal of 50 $\mu g/m^3$ for a 24 hour average.*
- *Secondary aerosols are very small in size (and hence penetrate into the lung) and may be more chemically reactive than larger particles, although available medical evidence suggests that sulphates and nitrates may be less of a problem than organic compounds. They should be considered for locations close to major sources of road emissions.*
- *In a ventilation tunnel, secondary aerosol production and particle coagulation may occur and result in enhanced rates of emissions of the very fine fraction (PM_1) compared to surface roadways.*
- *If the TEOM correction factors based on site-specific measurements are used for background estimation, they should account for much of the presence of secondary aerosols. Adding in an extra regional component is probably only necessary on very urbanised continents where emissions from other states and countries can influence air quality in a surrounded location. Adding an extra component is hard to justify for Australian urban areas, if care is taken with the choice of TEOM correction factors.*

Regardless of the air quality guidelines chosen, the EIS may underestimate maximum daily PM_{10} exposures by up to 10 $\mu g/m^3$ on winter days. This could have a quantifiable health impact for sensitive people in potential hotspots, based on recent epidemiological studies in cities throughout the world.

It is recommended that further consideration of the PM_{10} impact should include:

- *Contact with the EPA and other experts to determine better estimates of PM_{10} emission rates for the tunnel (taking into account better information on expected traffic flows, the use of improved traffic emission factors and treatment of vehicle type, roadspeed and slope and the presence of resuspended road dust).*
- *Consideration of the health risks, due to the smaller particulate matter expected in the tunnel exhaust compared to ambient air.*
- *Further evaluation of the likelihood of ventilation stack plumes impacting on nearby buildings.*

Comment :

The correlation of TEOM and HVAS PM_{10} measurements has been of great concern to regulatory authorities in Australia and elsewhere. In Australia, following on work conducted by CSIRO on behalf of Environment Australia (CSIRO, 2001), the Peer Review Committee of NEPC has recommended that site-specific comparisons should be used in order to provide suitable correction factors for use in air quality evaluations. A European Community Working Group (Williams and Bruckmann 2001) have also formulated various procedures and have collated information from a variety of European and urban studies. Both groups have produced procedures against which acceptability or otherwise of the various types of measures can be judged.

NSW RTA and NSW EPA, however, FAIL to comply.

The Katestone Environmental Review by Dr Peter Best highlights the consequence of such errors as follows:

Error analysis for worst-case 24 hour average PM_{10} exposure

The above values can be used to estimate the worst-case exposures for the most-affected receptors – more detailed modelling is required to look at the community exposure.

The resulting concentrations are shown in the following Table:

Error estimates for worst-case PM_{10} exposure due to ventilation stack emissions

Quantity	Estimated error (%)
Emission rates	± 50
Dispersion modelling	Up to 100
Background	11-29

Hence:

- Worst-case background concentrations (stated as 39.5 $\mu g/m^3$) should be increased to 51 $\mu g/m^3$.
- Stack contribution of 3 $\mu g/m^3$ for ground-level and 8 $\mu g/m^3$ for elevated level receptors should be increased to 5 $\mu g/m^3$ and 24 $\mu g/m^3$ respectively, for worst-case emission factors and dispersion estimates
- Should these occur at the same time as high backgrounds, there will be additional exceedances of the NSW goal of 50$\mu g/m^3$.
- The frequency of such high events does depend on traffic and stack characteristics as well as pertaining meteorological conditions.

Comment (RK):

What this means is the NSW EPA and NSW RTA have deliberately and recklessly abused proper procedure in monitoring by the TEOM PM10 so as to underestimate particulate air pollution and thereby corruptly lower the risk of demonstrating an exceedance of the National Air Quality Standard.

Dr. Peter Best (Katestone Environmental) states:

The failure by Holmes Air Sciences to make the upward adjustments for background TEOM PM_{10} can lead to the following:

(a) the local air quality may not meet NEPM guidelines even without the ventilation stacks in operation.

(b) the existing PM_{10} levels may exceed 50µg/M^3 as a 24 hour average for more than 5 days per year in most recent years.

(c) the modelling assessment of the stack impact is fundamentally flawed.

EIS estimates of workers exposure in the Lane Cove West Industrial/commercial area are too low by a factor of 2 to 5 times.

(Dr. Peter Best, Katestone Environmental, 13.2.02 – Parliamentary Order).

Dr Best then commented on the significance of this error factor in terms of health risk as follows:

Increase in health risk

The health risk evaluation in the EIS used a largest maximal increase of 3.0 µg/m^3 for 24 hour average PM_{10} concentration (and disregarded the exposure of people in nearby buildings). It used relative risk factors for mortality and some morbidity measure based on pre-1997 epidemiological studies. There has been considerable work conducted in the past 10 years that give an idea of seasonal risk factors for different sections of the community.

In many ways, the value of a background concentration being in the range 30-50 µg/m^3 is only needed to justify the use of risk factors from other cities since, if the exposure-effect relationship is linear, the exact value does not matter much. The above considerations suggest that an increment of over 10 µg/m^3 for a 24 hour PM_{10} concentration is quite possible for receptors near to the stack. Recent Australian work on hospital admissions for asthmatics would imply that this may lead to a significant risk for nearby sensitive people, especially if background values of PM_{10} exceed 40 µg/m^3. A better definition of the make-up of these local communities is required to give an assessment of the likely risks caused by such exposures.

Using Air Quality Goals as a License to Pollute

The national pollution guidelines do not apply PM_{10} standards to point source pollution such as road tunnel vent stacks. This flaw in the standards enables the Regulatory Authorities to pump emissions from vent stacks in a concentrated, toxic plume that would far exceed the standards if it were measured at source. However, when these poisons are dispersed, they appear to have minimal effect on air measured several kilometers away (the regional airshed). The problem is that the Regulatory Authorities cannot guarantee that the poisons disperse before they fall to the ground. Modelling shows that certain wind conditions can cause them to fall to ground level quite close to the stack, well before "dilution" can occur.

PM_{10} 24-hour averages obscure or dilute the major unhealthy exceedances during peak or heavy traffic periods. The Regulatory Authorities seem to view air quality goals as a licence to pollute. If the standard is $50\mu g/M^3$, then they assume that 48 or to creep toward 50 is acceptable. In contrast, contemporary risk management principles demand that risk levels be reduced as far as possible. The Regulatory Authorities should not pollute up to a standard but work down to a risk. This principle is not adopted by the Regulatory Authorities generally.

Discharging tonnes of untreated toxic emissions into residential areas is intuitively illogical, especially when there is no safe threshold. In 1800, industry stacks belched clouds of pollution into the environment. Two hundred years later we cannot continue to use the atmosphere as a sink.

Secondary Particles

Secondary particles formed by gas phase chemical reactions from VOC (volatile organic chemicals) emissions are subject to long-range transport (e.g. Sydney's bushfire smoke has carried as far as N.Z.) The same is true of fine fraction of primary emitted particles.[15] Filliger et al (1999) found that typical residence time of fine particles ($<2.5\mu m$) in the atmosphere is of the order of a few days. Fine particles are therefore subject to long range transport. In view of the fact that little information is available on long-range transported primary PM_{10} it is difficult to place any validity on the plume dispersion data in the current Sydney tunnel EIS Reports. Common sense and observation predict that if a large component of bushfire smoke is ultrafine particles then ground level dispersion will be similar from vent stacks.

[15] Filliger et al. Op cit

Estimation of Primary and Secondary Particles

The estimation of road traffic related PM_{10} may be derived from emission inventories[16], receptor studies and dispersion models.
By definition, PM_{10} emission inventories specify the mass of emitted **primary** particles. However, for **secondary** particles, the traffic-related fraction must be estimated indirectly using the portion applying to the precursor emission. At present, EIS Reports provide no evidence that traffic-related secondary particles are included.
Furthermore, while the emissions from the stacks will be largely traffic-related as opposed to ambient levels of PM_{10}, in many emission inventories, re-suspended road dust is **NOT** yet included. To date, EIS Reports, unlike overseas studies, provide no evidence that for modelling of stack emissions, re-suspended road dust is included.[17] The only data used appears to be Hi Vol PM_{10} collections that represent mainly ambient levels, not road-traffic related PM_{10} as in a tunnel. Data generally used in the modelling and plume dispersions are not only unrepresentative but can be seriously under-estimated.

Failure to Correct for Secondary Particles

As already mentioned, in tunnels a substantial part of PM_{10} can arise from chemical gas to particle conversion (secondary particles). Such particles cannot be correctly measured by the TEOM method.[18] Thus the TEOM under-estimates PM_{10} concentration in comparison to that obtained from gravimetric (Hi Vol) filter samples.
In France,[19] when TEOM measurements are used for background, to introduce the missing secondary particles into the analytical model, a constant correction value of 9.5 $\mu g/M^3$ is added to all grid points. The value of 9.5 $\mu g/M^3$ corresponds to regional background of secondary particles as estimated for France by the European scale EMEP model (EMEP, 1997). This is a conservative correction factor, because the urban-scale portion of the secondary particles is ignored. As a consequence, the PM_{10} levels (by TEOM) in studies in France indicate a *minimum level for* PM_{10}.

Chemical Composition

When a site is influenced by many sources, as is the case with particulate matter, not only will the concentration of particles vary quite rapidly with time, the chemical composition will also fluctuate similarly.

Investigations of the health effects of exposure to airborne particles are most commonly based on either daily or annual average measurements categories

[16] Ibid

[17] Ibid

[18] Ibid

[19] Filliger et al. Op cit p. 44

Possible Mechanisms of Toxicity

Reduction of risks to health from inhaling particles depends upon control of the toxic components of the aerosol cloud. In contrast to other pollutants, usually single chemical substances, particles are of complex chemical, physical and biological composition, differing from place to place and from time to time.

The ambient atmosphere contains a mass of biological material in the form of bacteria, fungal spores and pollens, many of which are known to cause infectious and allergic disease. Almost all of this material is found in the larger fraction of particles collected as PM10, above 1 μm in aerodynamic diameter. In addition, ambient air also contains a large number of non-biological particles produced by human activity.
The smallest, **nucleation mode**, particles aggregate into larger, **accumulation mode** particles, resembling bunches of grapes, which thus have a greater total surface area than if they were a uniformly spherical particle. Both types of particle are less than 1μm in diameter.

Following inhalation, particles <10 μm in diameter may deposit in the conducting and gas exchanging areas of the lung where they may, if they override local defence mechanisms, initiate tissue injury and inflammation.

Figure 7

Respiratory Structures

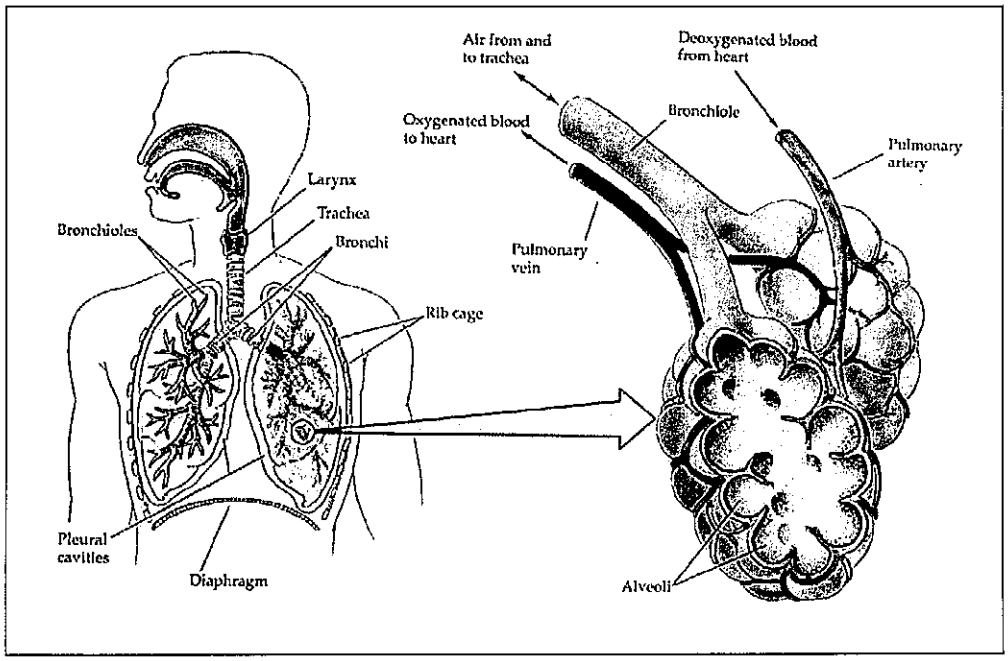


Figure 8

Gas Exchange Unit of Lung

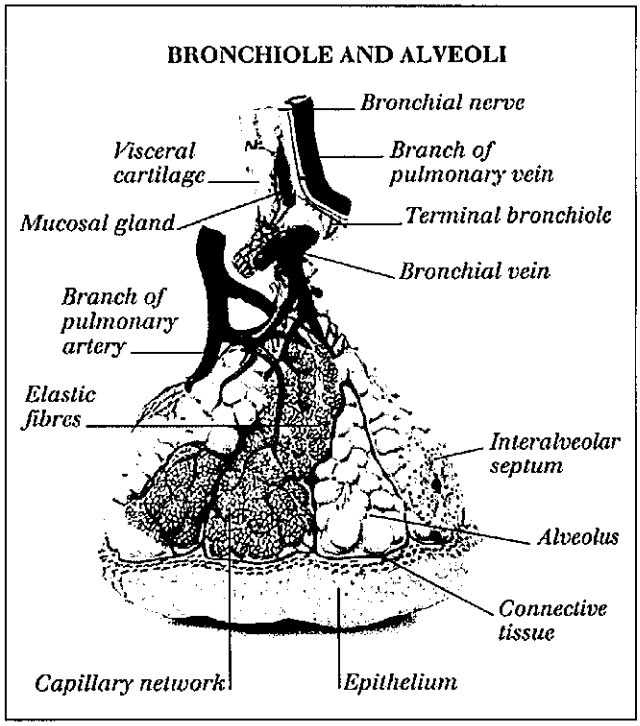


Figure 9

Gas Exchange Alveolar Unit

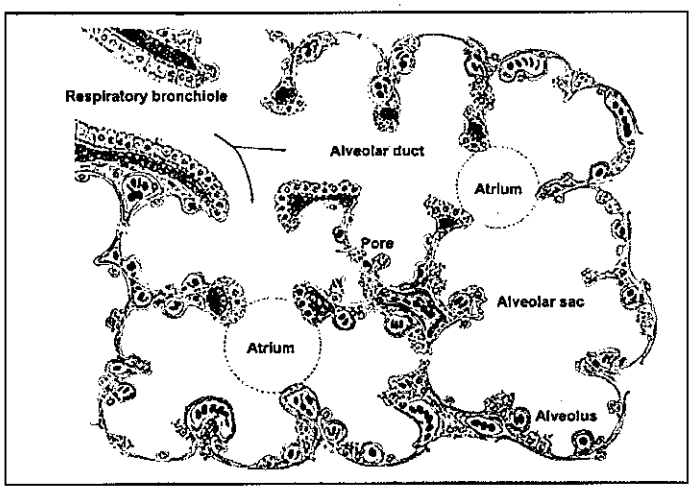
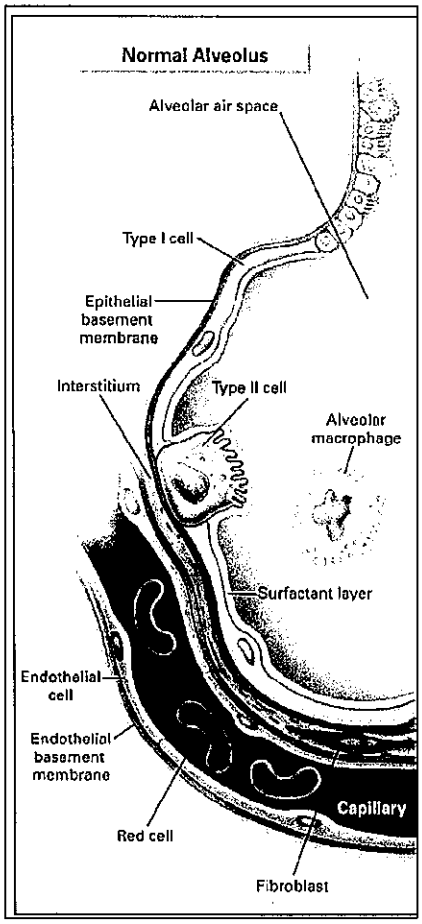


Figure 10

Relationship Between Alveolus and Blood Vessels



Once coming into contact with epithelial cells or phagocytic macrophages, toxic particles may cause the release of oxygen free radicals. This may be due to the effects of chemicals adsorbed onto the surface of the particles, including reactive metals, or to some property of the particle surface itself. These free radicals may initiate a series of biochemical reactions and molecular events, culminating in inflammation.

Experimental animal and limited human studies indicate that the **smallest**, <0.1 µm, particles cause more inflammation in the **periphery of the lung** than do larger particles. The inflammatory reaction caused by inhalation of toxic particles may lead to worsening of existing lung disease and enhance the sensitivity to allergens of people with hay fever and asthma. It may *also have the capacity to alter blood coagulability and circulating red cells and platelets*, a mechanism that could explain the adverse influence of inhaled particles on cardiovascular morbidity and mortality.

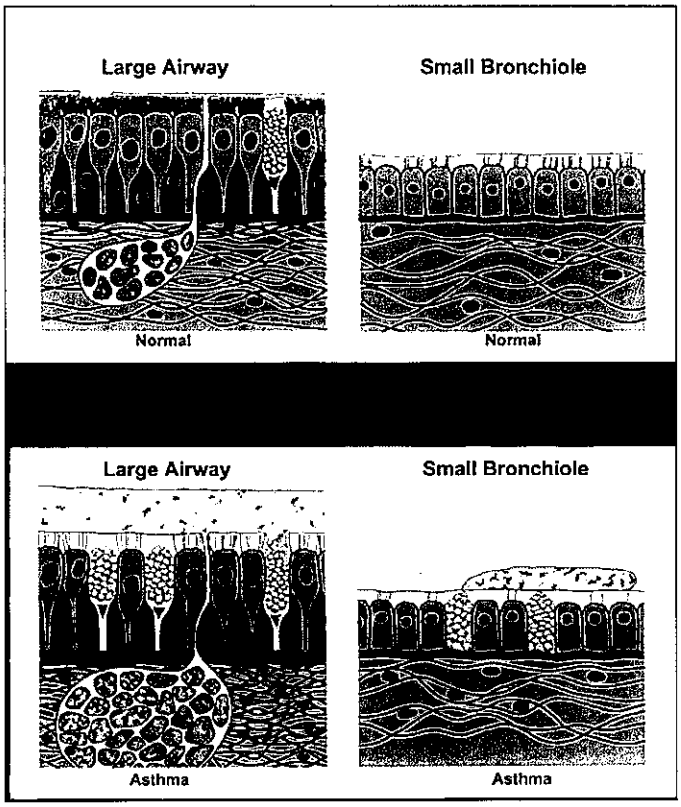
Experimental evidence and theoretical considerations suggest that small particles, <1 µm in diameter, may be the predominant source of the toxicity of ambient anthropogenic particles.

The Toxic Effects of Inhaled Particles

The toxic effects of any inhaled particle depend upon its capacity to elicit these responses and cause damage to the lung. Although the healthy lung is capable of dealing effectively with a large number of particles deposited onto its surface, there will come a point at which the defence mechanisms are overwhelmed either by particle numbers or by the inherent toxicity of the particle. In patients with compromised lung function, such as defective mucocilia clearance, the lung is likely to be less well equipped to deal with particle loads, and such people may succumb more readily to infections and notice effects of air pollution on their lungs that well people do not.

Figure 11

Mucocillary System of Respiratory Tract in Normal and Asthmatic Condition



Injury to the lungs may take many forms, but can be divided into damage to conducting airways and to the gas-exchanging parts. Larger biological particles deposited on airways may cause an allergic reaction, such as hay fever caused by very large (30 μm) grass pollen grains or asthma by particles derived from house dust mites. Other biological particles, bacteria and viruses, may lead to infections such as acute bronchitis. Most biological particles are in the size range >1 μm, an important point to note when considering the relative toxicities of different size fractions, since most allergic reactions are likely to be a response to particles in this relatively large fraction of the ambient aerosol. Non-biological particles may cause chemical bronchial irritation, rarely leading to a non-allergic form of asthma (sometimes called the reactive airways dysfunction syndrome), and may interfere with the airway defence mechanisms, leading to cough and sputum production.

Figure 12

Bronchial Constriction and Effect of Mucous in Inflammation of Lungs

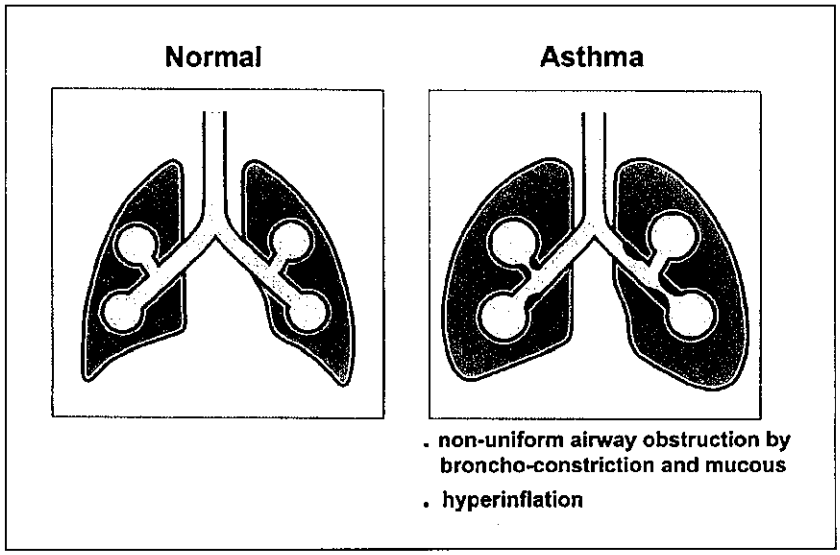


Figure 13

Postmortem of Asthmatic Lung showing Mucous Plug in Major Airways

Smaller particles, deposited in the alveolar region of the lung, may cause inflammation affecting the cells in the alveoli and of the capillary blood vessels within them. Such inflammation may have immediate effects, such as pneumonia, or longer-term effects causing scarring (fibrosis) and/or destruction (emphysema) of the alveoli. These effects are also familiar as causes of breathlessness and premature death in long-term cigarette smokers.

Figure 14

Normal Alveolus and Effect of Acute Inflammation

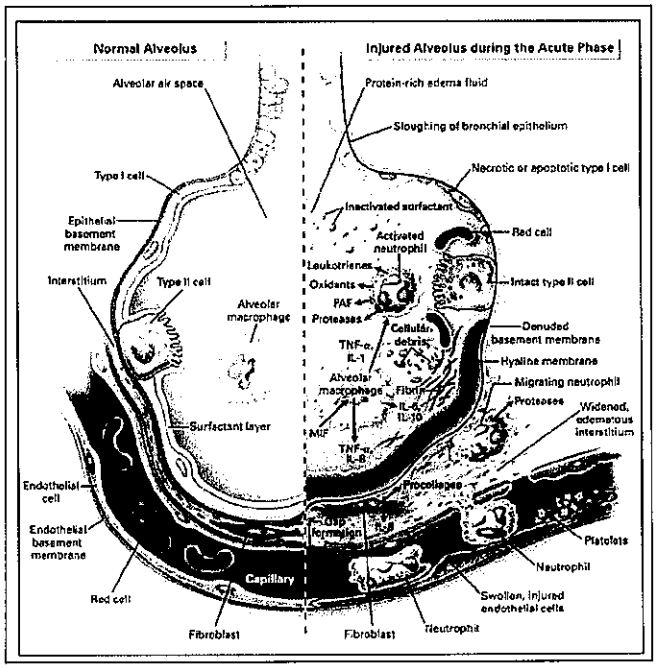
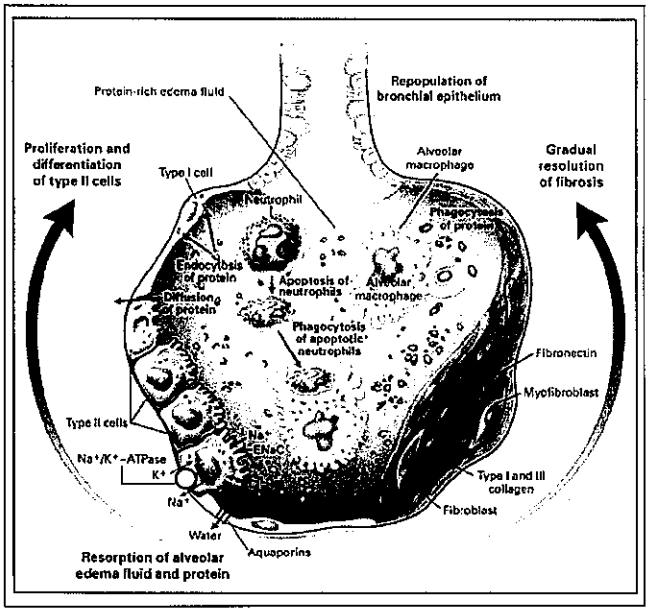


Figure 15

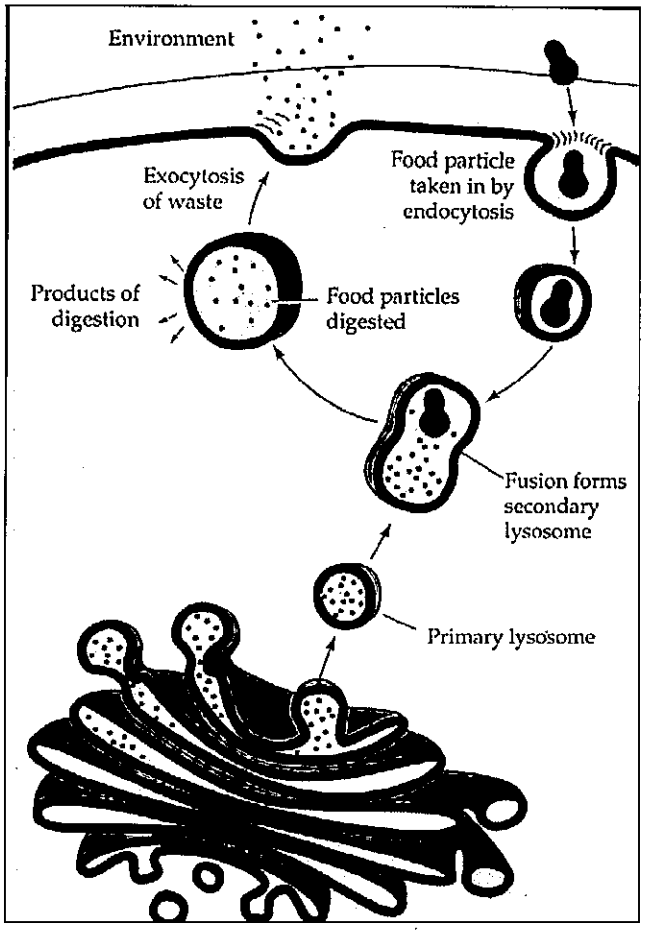
Chronic Inflammation of Alveolus



The toxicity of particles depends on both their aerodynamic size and shape, which determine where in the lung they are deposited, and their chemical reactivity, which determines their effect on the defensive cells of the lung and, probably, their ability to penetrate the epithelial lining of the alveoli. In general, particles larger than about 10 μm diameter are deposited in the nose and throat and those between about 4 and 10 μm deposit mainly within the airways; all these are likely to be removed by ciliary action. Particles of less than about 4 μm are able to reach the alveoli, from which they are cleared by macrophages.

Figure 16

Uptake of Particles by Alveolar Macrophages in Lungs



Generally, as the particle size and breathing rates increase, particles deposit closer to the throat and nose, whereas enhanced deposition in alveoli takes place with smaller sized particles and slower breathing rates (Heyder et al 1986, Iwasa et al 1970). Within the conducting airways, particle deposition tends to be greatest where airways divide, especially for those particles >1 μm in diameter (Kim and Hu, 1998).

In patients with diseased airways, the pattern of deposition varies and becomes more irregular, with areas of increased deposition. A 30% reduction in airway cross-sectional area may result in a greater than doubled deposition in the airways at the point of division (Kim and Hu, 1998). Regional deposition dose, or local tissue burdens, can be different between individuals, even if total lung deposition values are comparable. Therefore, it is possible that particle burdens could reach threshold limits in local lung regions under exposure conditions that are normally acceptable, particularly in individuals with compromised lungs.

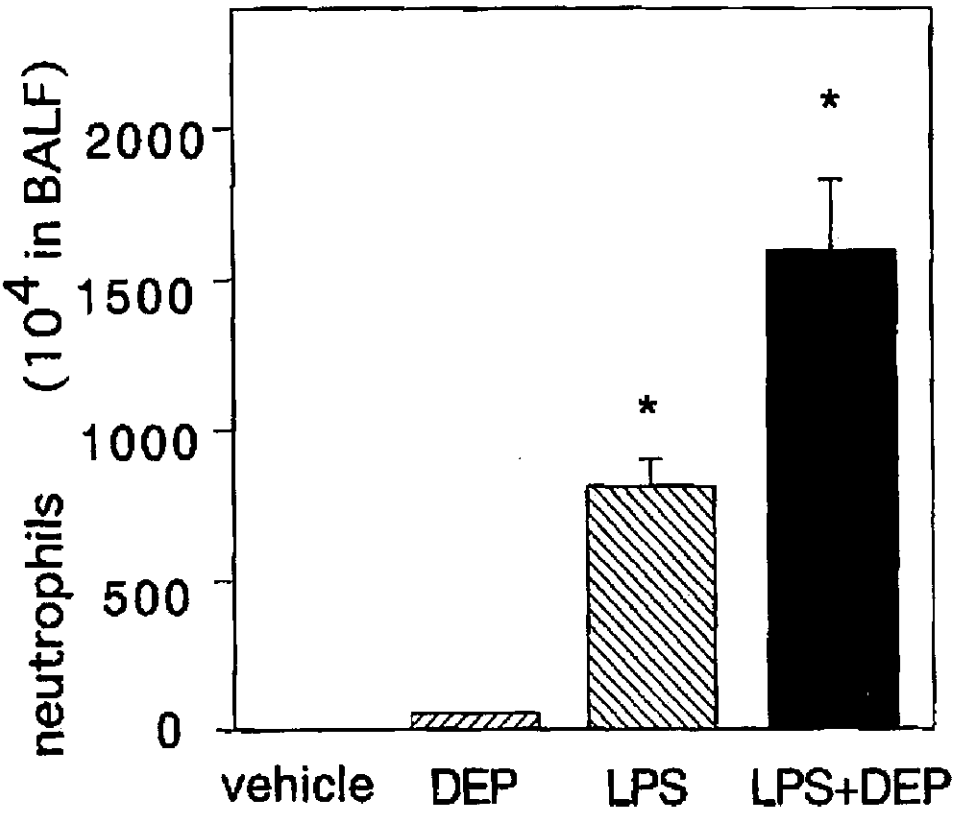
Chemical reactivity of the particles may cause an alteration in the function (activation) of macrophages leading to release of chemical mediators and to inflammation in the alveolus or in its pathway to the lymph nodes within the lung.

From the above arguments it will be clear that for particles in ambient air to be harmful

they must be both toxic and present in sufficient amount.

Figure 17

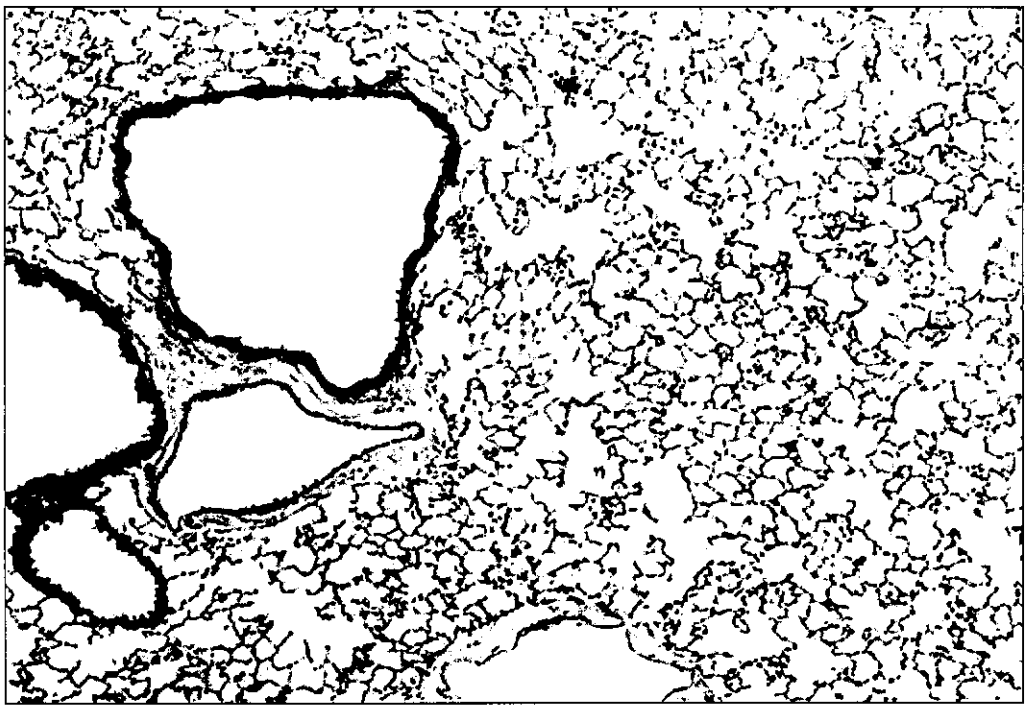
Additive Effect Diesel Exhaust Particles have on Inflammation (Neutrophils) in Lungs



H. Takano et. al. 2002

Figure 18

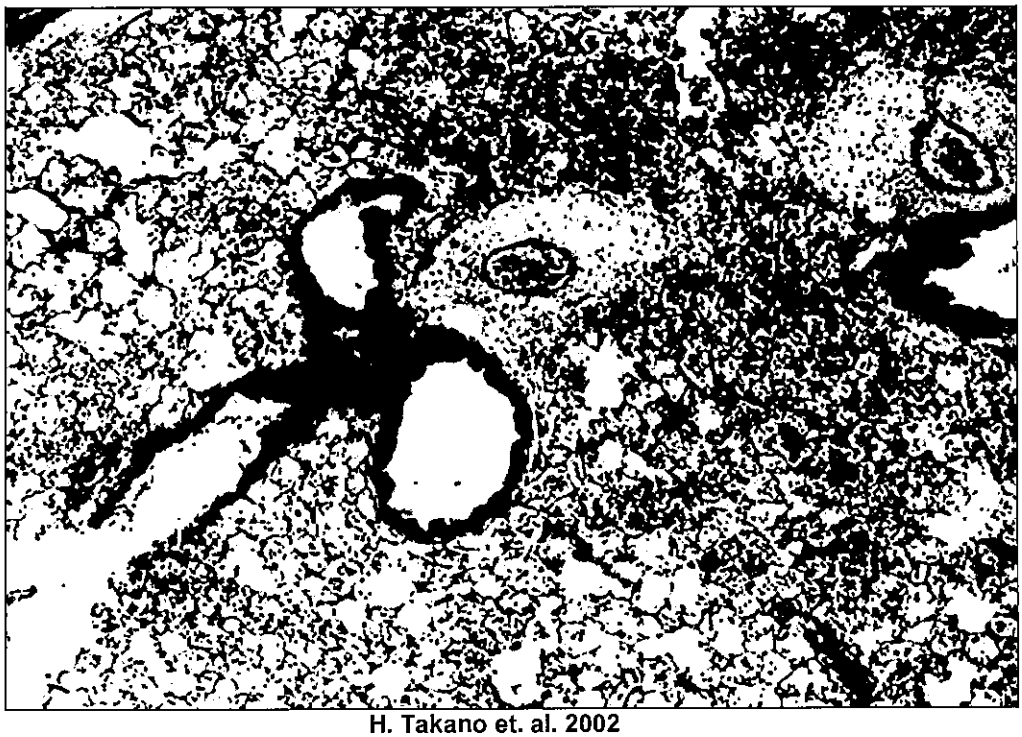
Normal Lung (mouse)



H. Takano et. al. 2002

Figure 19

Inflammation of mouse lung exposed to Diesel Exhaust Particles and Bacterial Cell Wall Lipopolysaccharide (LPS)



H. Takano et. al. 2002

Figure 20

Diesel Exhaust Particles (DEP) Injure Lung Similar to Endotoxin

DEP 90% <1μm produce:

- **serious adverse effects on respiratory health especially people who are susceptible to bacterial infection**
- **enhanced lung injury similar to endotoxin**
- **neutrophil sequestration, interstitial oedema, and alveolar haemorrhage**
- **synergistic effects with endotoxin, rather than additive**

H. Takano *et al*, *Am.J.Resp.& Crit.Care Med.* (2002); 165:1329-1335

Figure 21

Mechanism by which Diesel Exhaust Particles (DEP) augment inflammation is by increasing receptors for bacterial lipopolysaccharide (LPS), called 'Toll-like Receptor'. The effect of DEP is to make the lungs highly sensitive to the presence of normal levels of bacteria. This results in greatly heightened production of pro-inflammatory mediators from the cells.

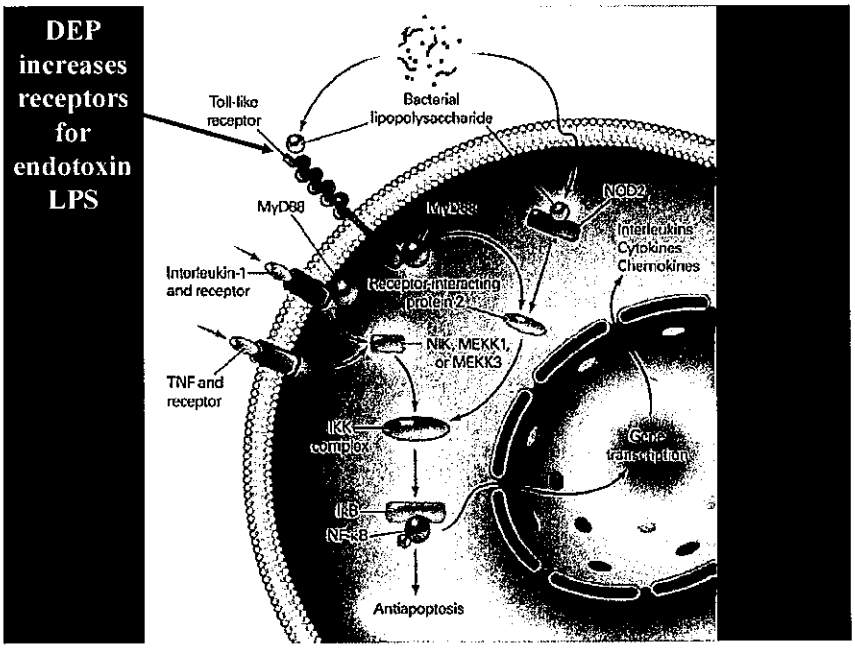


Figure 22

Effects of Diesel Particles on Lung

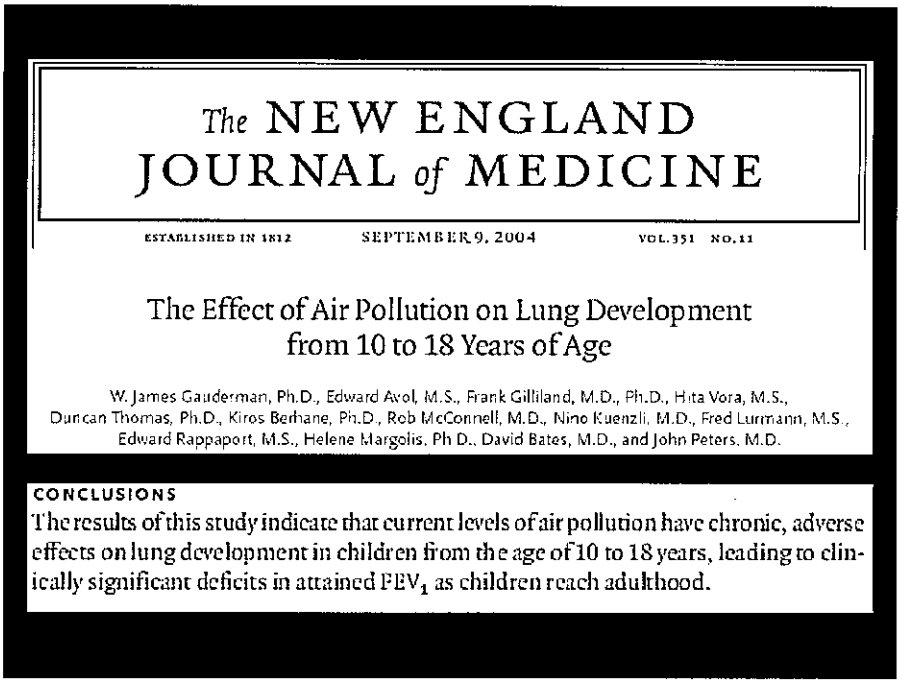
Diesel particles 90% < 1µm linked to:

- **lung cancer**
- **pulmonary fibrosis**
- **chronic alveolitis**
- **bronchitis**
- **oedematous changes**
- **airway inflammation with hyper-responsiveness**
- **enhancement of allergic asthma**

H. Takano *et al.*, *Am.J.Resp.& Crit. Care Med.* (2002); 165:1329-1335

These observations may partly be explained by the concept that in any large population there are always some vulnerable people, usually already suffering from a serious illness, in whom a relatively small influence is required to provoke worsening illness or even death. Moreover, much of the evidence of the health effects of air pollution has employed pollution data from single outdoor monitoring sites, but there is evidence that this may in some circumstances significantly under-estimate personal exposure to certain pollutants, since such exposure is determined also by activity patterns and proximity to local sources.

Some studies have demonstrated that the levels measured by personal PM10 monitors may be on average up to 2-5 times higher than those recorded at fixed site monitors (Lioy et al 1990, Watt et al, 1995).



The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 SEPTEMBER 9, 2004 VOL. 351 NO. 11

The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age

W. James Gauderman, Ph.D., Edward Avol, M.S., Frank Gilliland, M.D., Ph.D., Hita Vora, M.S., Duncan Thomas, Ph.D., Kiros Berhane, Ph.D., Rob McConnell, M.D., Nino Kuenzli, M.D., Fred Lurmann, M.S., Edward Rappaport, M.S., Helene Margolis, Ph D., David Bates, M.D., and John Peters, M.D.

CONCLUSIONS

The results of this study indicate that current levels of air pollution have chronic, adverse effects on lung development in children from the age of 10 to 18 years, leading to clinically significant deficits in attained FEV_1 as children reach adulthood.

The Relevance of Particle Size and Mass to Toxicity

The ambient particulate cloud comprises particles of different sizes, from a few nanometres up to several tens of micrometres, and of these all those below about 10 μm diameter have some potential to reach and be deposited in the airways and alveoli. This forms the basis of the use of the metric PM10, which represents the mass of such particles. However, in a given weight of ambient particles, the large majority of the mass will reside in the fraction greater than 1μm in diameter, but the greatest number of particles by far will be below this size. In other words, the smallest particles are most numerous but weigh the least.

Thus, when particles are collected and measured by weight, that weight may disproportionately represent the largest particles. PM10 is therefore dominated by the larger particles above 1 μm and below 10 μm and is thus appreciably affected by local sources of coarser particles, such as may be stirred up by the action of wind and abrasion of roads, soil, the sea surface and so on.
Data indicate a causal association between PM10 and ill-health. It seems likely on mechanistic grounds that the component of PM10 that is responsible is that fraction generated by combustion and photochemical reactions. These reside mainly, but not wholly, in the nucleation and accumulation mode particles, generally below about 2 μm in diameter.

The best indicator of toxicity may be the total surface area of particles inhaled.
(see Figure 5)

A key factor in promoting the toxicity of particles is their capacity to be transferred from the alveolar space into the interstitium of the lung. For this to occur, two barriers have to be penetrated. The first of these is a layer of waterproofing material, surfactant. Once through the surfactant layer, the particles have to pass through the second barrier, the alveolar epithelium.
This process would be assisted by the physical forces exerted by surfactant, but may also be effected by active transfer **through the cells** by a process known as **pinocytosis.**

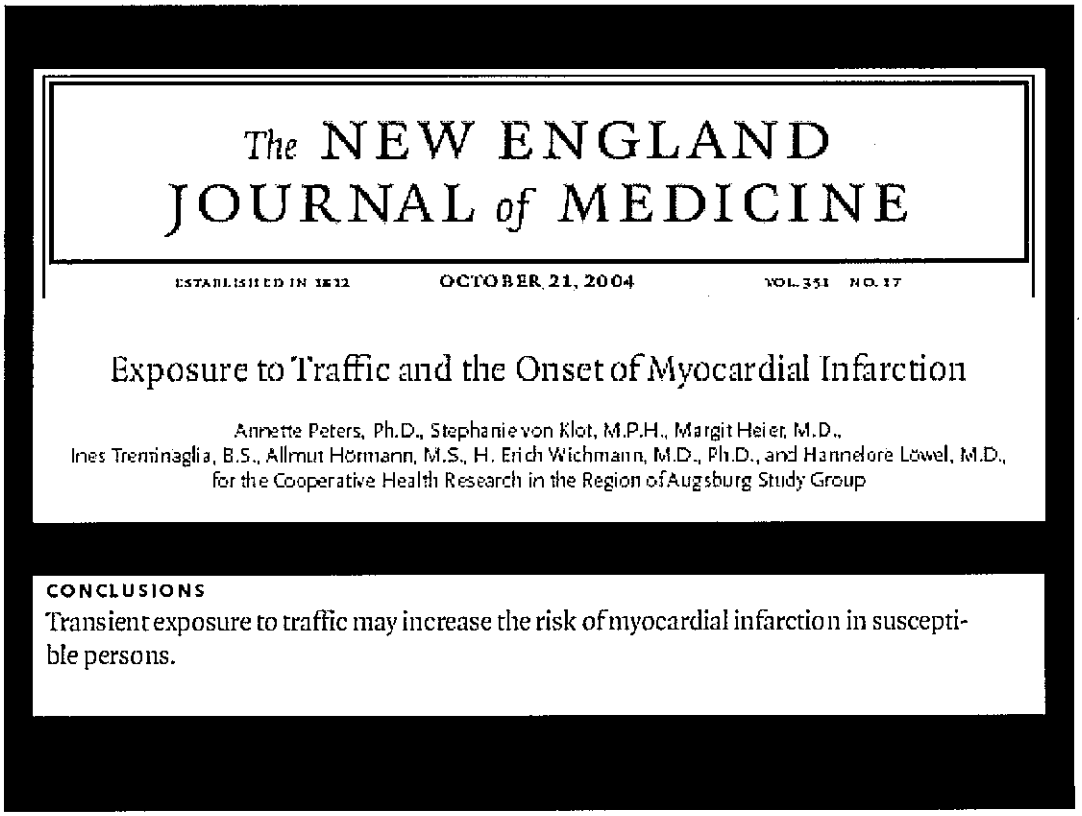
A plausible biological explanation of why pathological effects may differ, depending on the size of particles, is that large numbers of small particles cause inflammation even when their total mass is relatively low (Oberdorster et al 1994, 1995).

Exposure to PM_{10}: **Particulate Air Pollution and Blood**

- **reduced RBC count** **($p<0.001$)**
- **reduced platelet and factor VII** **($p<0.05$)**
- **elevated C-Reactive Protein** **($p<0.01$)**

NOTE: RBC probably sequested in lung by changes to endothelial cells

A. Seaton *et al.*, *Thorax* (1999); 54:1027-1032.



The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 OCTOBER 21, 2004 VOL. 351 NO. 17

Exposure to Traffic and the Onset of Myocardial Infarction

Annette Peters, Ph.D., Stephanie von Klot, M.P.H., Margit Heier, M.D., Ines Trentinaglia, B.S., Allmut Hörmann, M.S., H. Erich Wichmann, M.D., Ph.D., and Hannelore Löwel, M.D., for the Cooperative Health Research in the Region of Augsburg Study Group

CONCLUSIONS

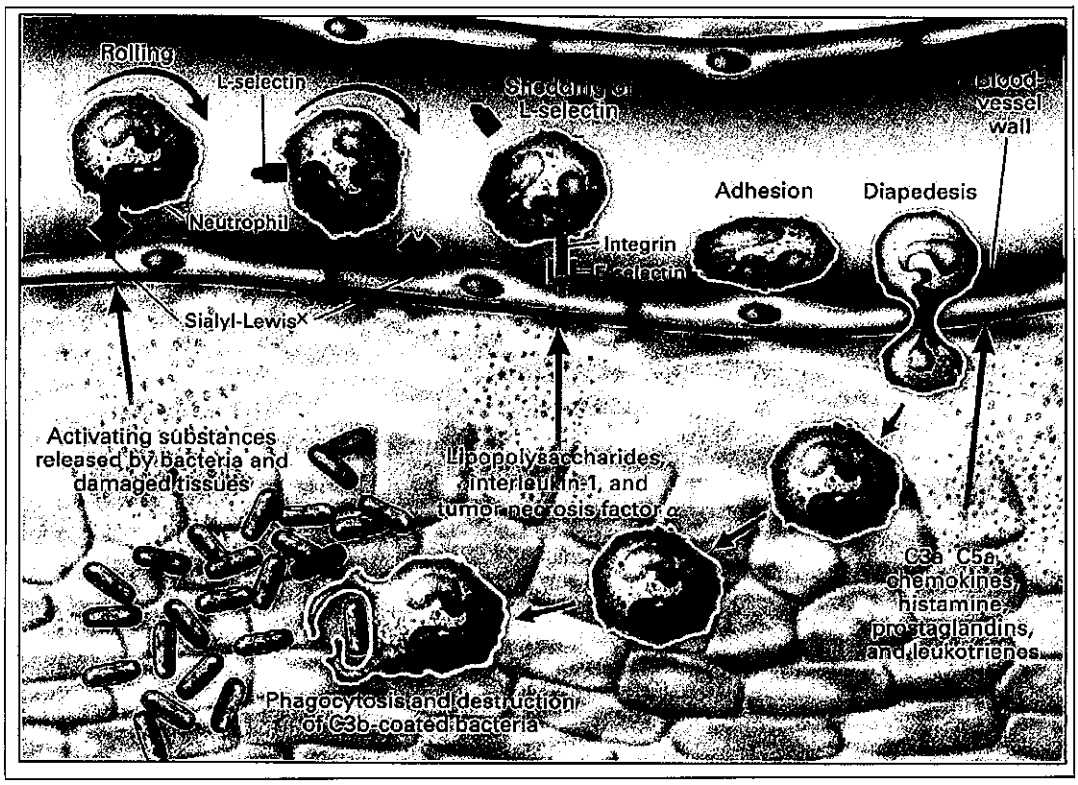
Transient exposure to traffic may increase the risk of myocardial infarction in susceptible persons.

Mechanisms of Toxicity of Particles to the Lung

Following deposition in the lung, particles are rapidly phagocytosed by alveolar macrophages which migrate towards the bronchoalveolar junction. Large numbers of particles, however, may overwhelm the macrophage phagocytic system and result in increased numbers of particles coming into contact with the respiratory epithelium. Impairment of clearance of particles by macrophages begins when the particles occupy 6% of the macrophage volume and is completely inhibited when 60% of macrophage volume is occupied. This inhibition occurs at a lower percentage if the particles are of very small size, suggesting that the effect is less due to the volume than to the surface area in contact with the inside of the cell. Oxidative damage mediated by particles then leads to activation of macrophages with the subsequent release of pro-inflammatory mediators.

Figure 23

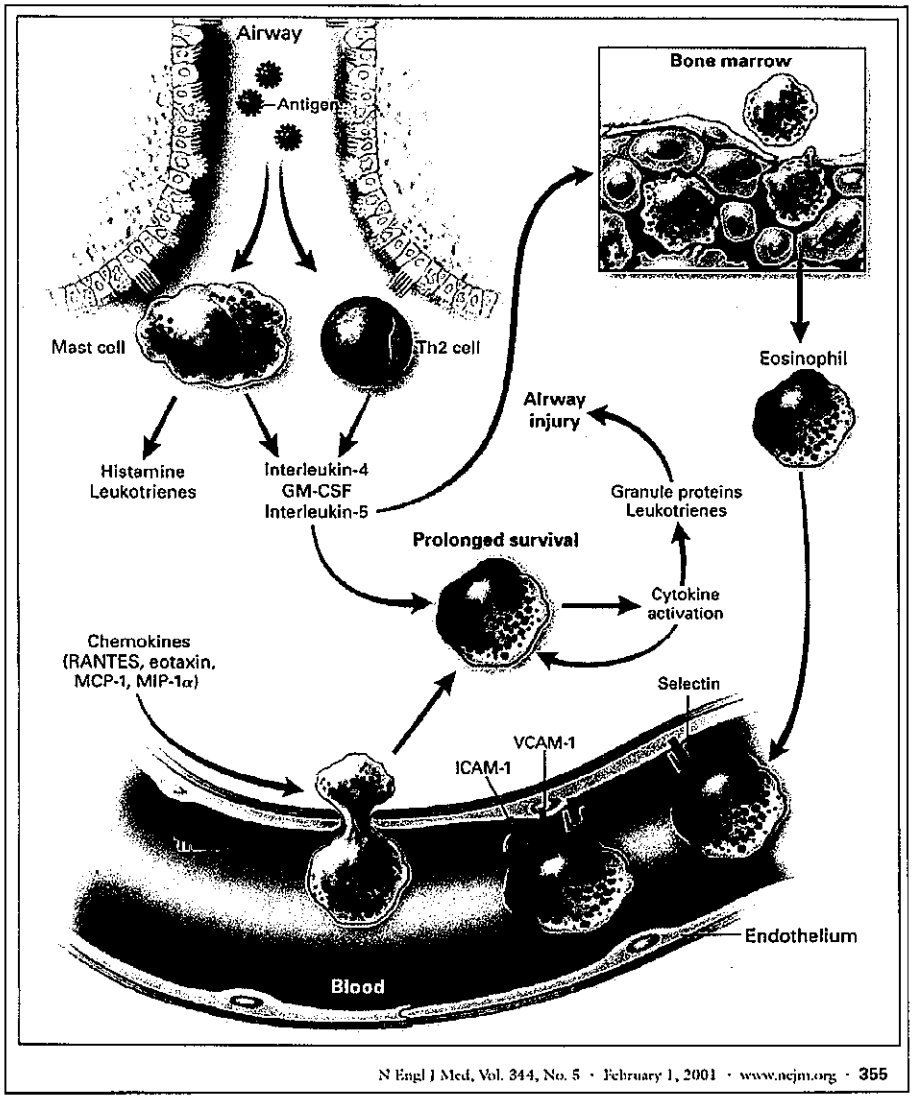
Acute inflammation (response to tissue injury) to bacterial invasion



Human airway epithelial cells in culture also phagocytise diesel exhaust particles and then release several pro-inflammatory cytokines in a dose-related manner. This suggests that these cells can perform phagocytic functions in the same manner as macrophages and neutrophils, and may also act as initiators of inflammatory responses. Similar responses have been seen on exposure to particles collected from ambient urban air.

Figure 24

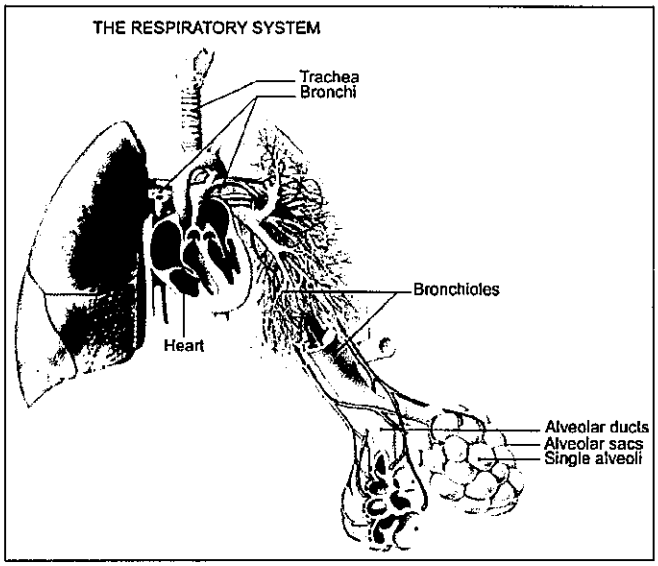
Response to Allergic Reaction in Alveolus



In mice with allergic airways disease, a condition with similarities to human asthma, large doses of fine particulate matter obtained from the ambient urban atmosphere have increased airway hyperreactivity (Takano et al 1997). The carbon core of diesel particles has also been shown to have a significant adjuvant effect on the local immune-mediated inflammatory response as well as on the systemic specific IgE response to allergen (Lovik et al 1997). Diesel exhaust particles have also been shown to adsorb allergens from grass pollen onto their surface, thus potentially increasing allergen deposition in the respiratory tract (Knox et al 1997). Such studies give support to the hypothesis that allergic individuals may be made more sensitive to allergens as a result of exposure to particulate pollution.

Figure 25

Lung and Heart Relationships in Gas Exchange



Healthy human volunteers, when exposed to diesel exhaust particles of uncharacterised size distribution and at high mass concentrations for 1 hour, show evidence of airway, alveolar and a more general inflammatory reaction (Rudell et al 1999, Salvi et al 1999). Nasal instillation of diesel particles, with or without allergen, in atopic human subjects has produced an increase in the allergic antibody, IgE, in nasal lavage fluid (Diaz-Sanchez et al 1994, 1997). These, and other studies of the mechanism of ragweed allergy (Fujieda et al 1998), suggest that diesel particles can contribute to the enhancement of mucosal allergy in humans.

Figure 26

Allergic Skin Tests to Illustrate Acute Response and Late Response

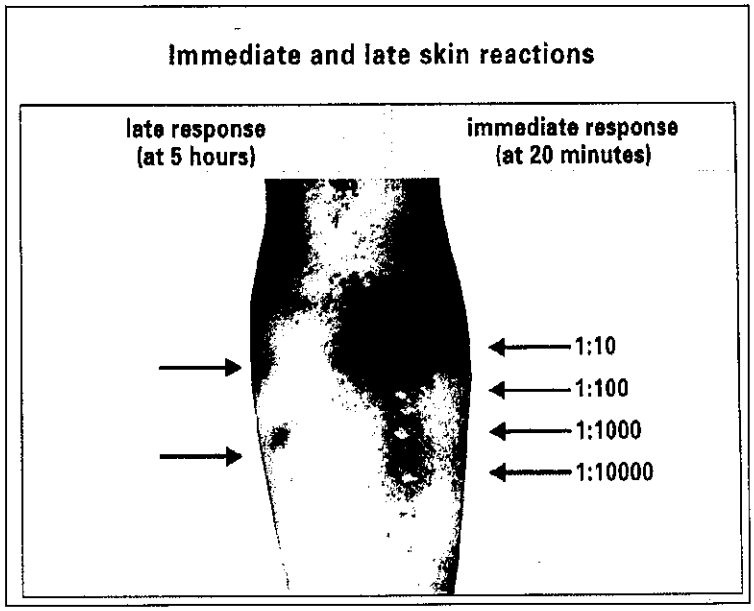


Figure 27

Graphic Illustration of Early or Immediate Response and Late Prolonged Response to Allergen. It is the late response that is cellular and augmented by diesel exhaust particles.

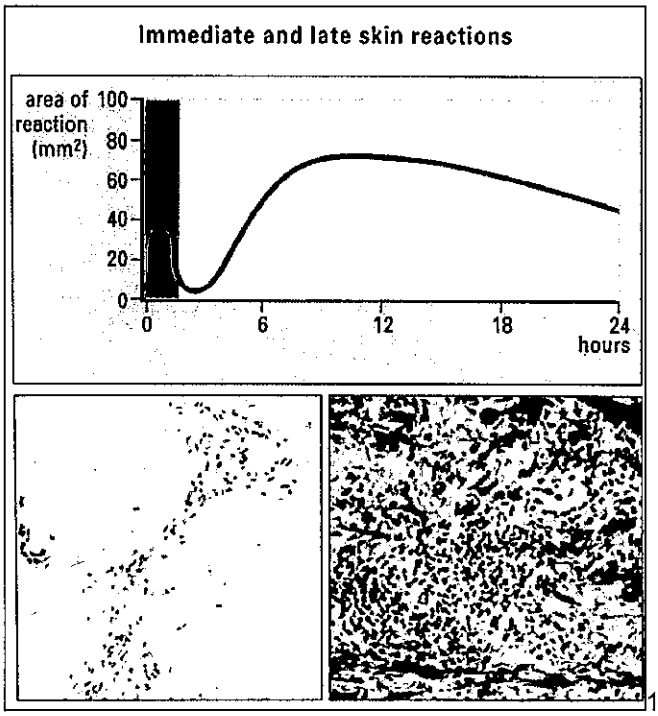


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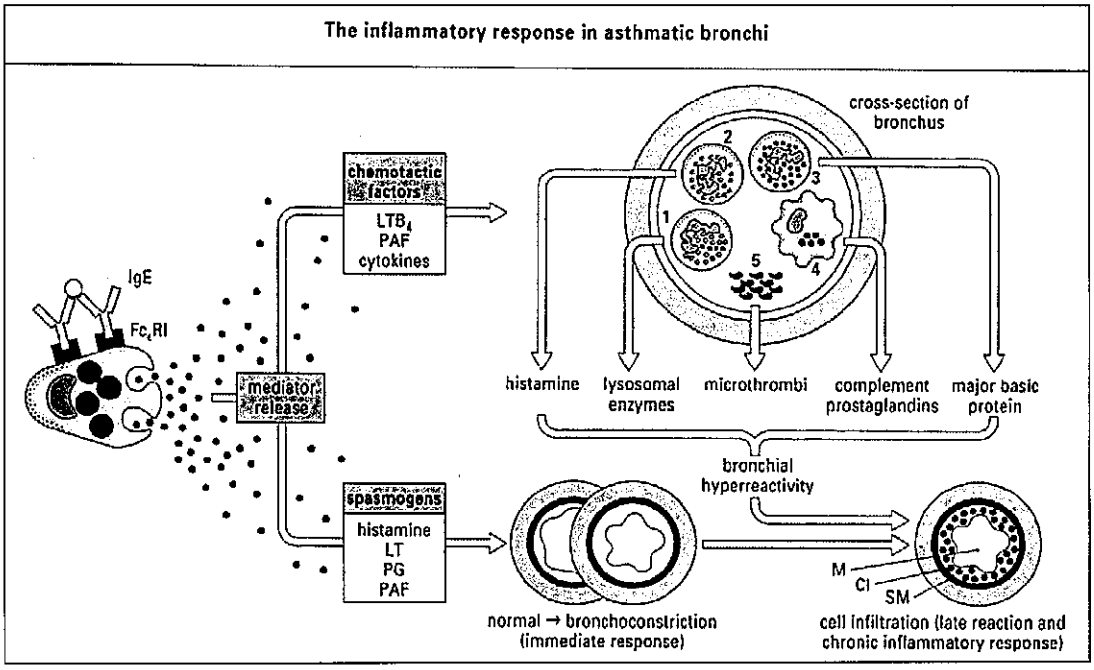
Figure 28

Pro-inflammatory Mediators Released from Allergic Hypersensitivity in which Diesel Particles Augment the Late Reaction in the Presence of Bacterial Lipopolysaccharide (LPS)



Most importantly with respect to the relevance of particle size (and numbers) to toxicity, experimental studies in rats have shown that nanometre-sized particles cause more lung injury than the same deposited mass of fine respirable particles of the same material (Oberdörster et al 1994, Donaldson et al 1998).

More free radical activity is generated by **ultrafine** particle samples than by coarser samples of the same substance (Donaldson et al 1998, Zhang et al 1998). Hence the differences in their ability to cause lung inflammation may be explained on the basis of different amounts of free radical activity, probably related to the greater surface area available. In vitro exposure of phagocytic cells to ambient particles collected from different urban settings causes oxidative stress which correlates with the iron content of the particles (Prahalad et al 1999).

PM2.5 collected from the ambient air induces the expression of genes for stress-related proteins, such as heat shock proteins and the magnitude of this has been correlated with the amount of soluble copper in the particle preparations (Vincent et al1997). It has thus been suggested that the lung dose of bio-available transition metal, rather than instilled particulate mass, may be the primary determinant of the acute inflammatory response (Costa and Dreher 1997). However, two notes of caution need to be sounded. First, Brown et al (2000) have shown that ultrafine particles themselves, without transition metals, may induce inflammation in rat lungs. Secondly, Monn and Becker (1999) have drawn attention to the important cytotoxicity of endotoxin derived from biological components of the summer air. (see Figures 17-22)

A large number of transition metals appear to be adsorbed onto the surface of particles and these are capable of generating reactive oxygen intermediate species from various airway cells. These in turn activate factors that lead to the release of a large number of pro-inflammatory molecules such as cytokines, cell adhesion molecules and inflammatory mediator receptors (Meyer et al 1994, Jimenez et al 2000). (see also Figures 14 and 15)

Overall, the experimental evidence points towards a central hypothesis explaining the initiation of the pathological effects associated with exposure to particles. This is that something associated with the particle surface, most likely adsorbed transition metals but also possibly some other physico-chemical property, is able to initiate oxidative stress when it comes into contact with lung cells. This then activates chemical messengers that switch on genes for inflammation. The inflammation may then have different consequences in individuals with differing susceptibilities.

Particles and the Cardiovascular System

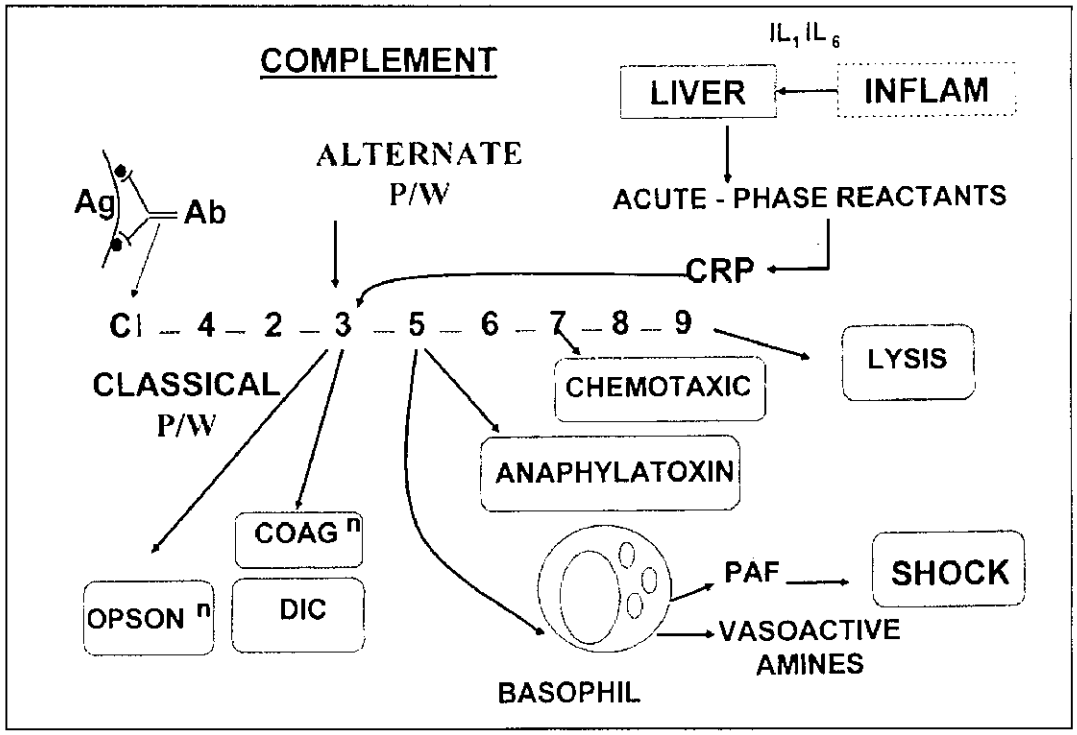
Ambient particles have been associated with cardiovascular mortality and morbidity, especially among the elderly population (Dockery et al 1993, Poloniecki et al 1997). While it is intuitively possible that inhalation of particles may lead to exacerbation of an underlying respiratory condition, why this should lead to an effect on the cardiovascular system might not seem immediately apparent.

Seaton et al (1995) suggested that low grade inflammation, caused by ultrafine particles deposited in the alveoli, might lead to increased **coagulability** and that the altered blood flow characteristics might therefore be a part of the pathological mechanism linking particulate pollution with cardiovascular mortality and morbidity.

Epidemiological studies have demonstrated a consistent increased risk for cardiovascular events in relation to both short- and long-term exposure to present-day concentrations of ambient particulate matter. (Ref: R.D. Brook et al :*Circulation,* 2004;109:2655-2671

Figure 29

Complement Cascade in Classical Antibody Pathway as well as Non-specific Alternative Pathway Leading to Pro-inflammatory Responses.



Several haematological factors, including plasma viscosity, fibrinogen, factor VII and plasminogen activator inhibitor occurring as a consequence of inflammatory reactions, are predictive of cardiovascular disease. Release of IL-6 from macrophages following particle phagocytosis, as has been demonstrated in vitro (Terashima et al 1997), could stimulate hepatocytes to secrete fibrinogen (Akira and Kishimoto 1992) and increase blood viscosity.

In support of this hypothesis, Peters et al (1997) have shown increased plasma viscosity, a measurement largely determined by fibrinogen concentrations, in a large population sample coinciding with an air pollution episode in Europe. was taken in this study concurrent falls in temperature.

In a direct test of the hypothesis, Seaton and colleagues (1999) showed that exposure to PM10 (measured both at a central point and as estimated personal exposures) correlated inversely with haemoglobin, red cell count, packed cell volume and platelet count, but not with coagulation factors. These relationships persisted after correction for plasma albumin concentration, suggesting that the blood changes were due to red cell and platelet sequestration. This study was carried out over two years in cities in which the ambient concentrations of PM10 never exceeded 100 µg/m3. The authors argued that alveolar inflammation may lead to alterations in **red cell and platelet adhesiveness**, and that consequential sludging in capillaries may be a factor in cardiovascular morbidity.

Other workers have observed changes in heart rate variability on exposure to sulphur dioxide and to the acid derived from sulphur dioxide (sulphuric acid) in human exposure studies.

A study of heart rate variability may be a predictor of acute cardiac episodes in elderly people (Liao et al 1999). These investigators showed rises in PM2.5 to be associated with reductions in heart rate variability, suggesting an effect on neural control of the heart. A pilot study of subjects with implanted cardiac defibrillators showed a tendency for increased episodes of arrhythmia to occur following rises in several indices of traffic-related pollution including black carbon and PM2.5 (Peters et al 2000). The delay between the initial rise in pollution and the onset of arrhythmias suggested that haematological rather than reflex stimuli were responsible. It should be noted that in general ischaemic stress is more likely initiator of an arrhythmia than is a neural reflex.

Cardiovascular Failure, Coagulopathy and Air Pollution

PM_{10} pollution episodes have been consistently associated with increased mortality, particularly resulting from cardiovascular causes (Dockery *et al.*, 1994; Schwartz, 1994). In cardiovascular deaths, evidence indicates that an increase risk of blood clotting is related (Seaton *et al,* 1995). Air pollution episodes clearly increase plasma viscosity in men and women (Peters *et al.*, 1997). The exact mechanism by which ultra-fines (<1μm) mediate adverse health effects is unknown. Donaldson et al., (2001) found significant rises in C-reactive protein (CRP), an index of inflammation, as a likely interaction between the ultra-fine particles, CRP and cardiovascular disease. It is also noteworthy that CRP is able to activate complement by the alternate pathway thereby linking into coagulation pathways via C_3b. Formation of platelet-activating factor (PAF) via basophils stimulated by C_5 complex in the complement activation cascade induces clotting via platelet aggregation. Other pathways leading to coagulation are also documented.

Seaton *et al* (1999) also found that there was an association between exposure to PM_{10} air pollution and reduced red-cell count ($p<0.001$) as well as lowered fibrinogen ($p<0.05$). Sequestration in the capillaries by red cells was believed to account for the reduced count. Evidence showed that ultra-fine particulates alter the pulmonary endothelium to cause expression of adhesion molecules (e.g., ICAM-1) to make passing red cells adhere, accompanied by early consumption coagulopathy. The potential for ischaemic damage in individuals with vulnerable coronary circulation is then possible.

Figure 30

Pollution can trigger heart attack

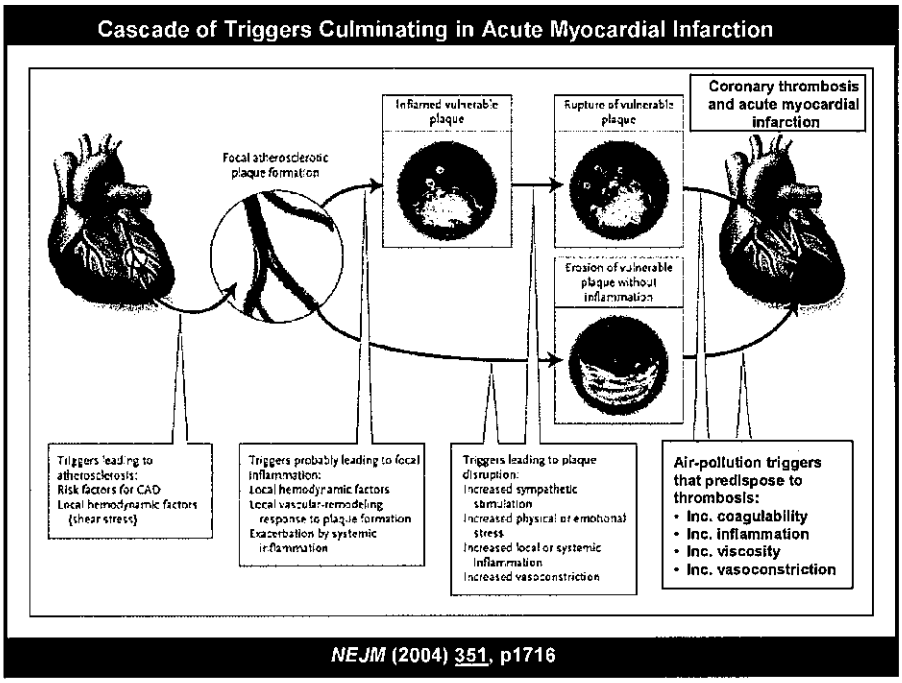
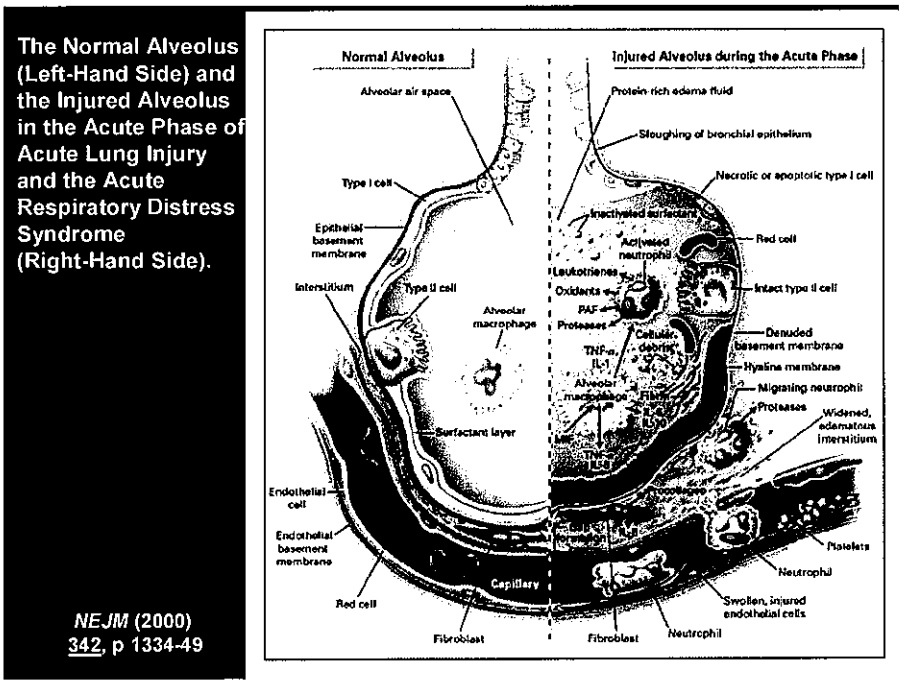


Figure 31

Pollutants trigger inflammation in the lungs and promote blot clots that can be carried to the coronary arteries to cause a heart attack



Asthma and Exposure to Air Pollution

The association between air pollution and the prevalence of asthma is supported e.g., by significantly higher episodes of coughing and wheezing among children who lived within 100m of a heavily trafficked freeway (Van Vliet *et al.*, 1997). For children, asthma events were strongly associated with NO_2, CO and SO_2 while with adults, the strongest associations for asthmatic episodes were with PM_{10} (Hajat *et al.*, 1999).

Figure 32

24-Hour Stagnation and Asthma
(Hospital Emergencies)

Seattle - increased 63%
Spokane - increased 28%

NOTE:

- **stagnation - surface wind speed <annual hourly mean value**
- **stagnation is not the trigger**

G. Norris *et al., Thorax* (2000); 55:466-470

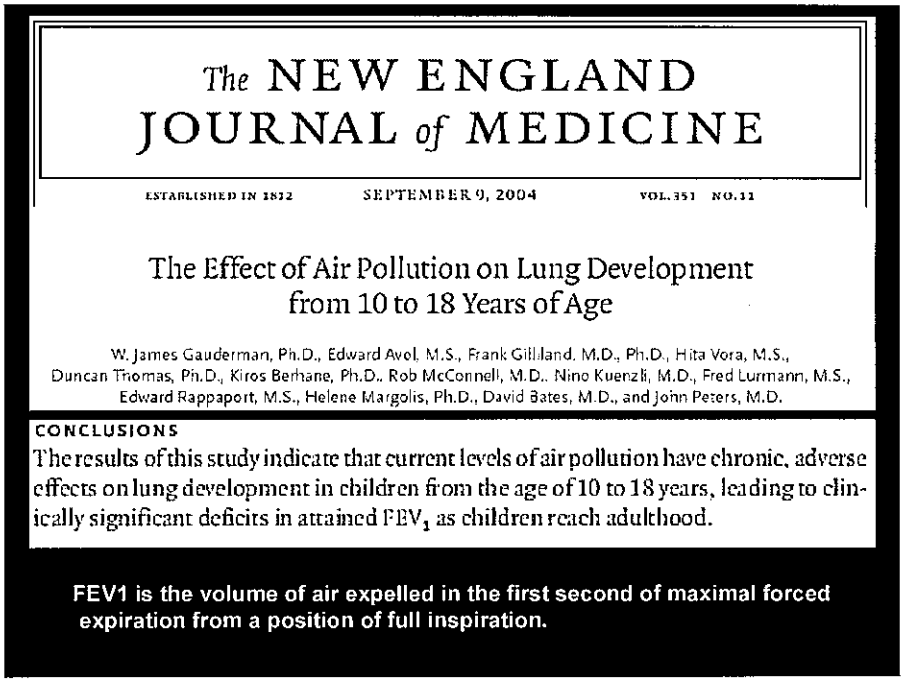
Exposure of the lung to PM_{10} and ultra-fines leads to oxidative stress within various lung cells, including alveolar macrophages, and an activation of gene expression resulting in the production of various pro-inflammatory mediators. Such inflammation, extending over a wide lung surface area including about three billion alveoli may exacerbate any pre-existing pulmonary or cardiovascular disease (Stone, 2000; Kennedy *et al.*, 1998; Rahman *et al*, 1998).

Despite a large amount of information on the biological activities of cytokine and on their potential involvement in various aspects of asthma inflammation, little is still known about cytokines in day-to-day asthma episodes.

Lung Growth Affected by Air Pollution

Results from investigations by Frank *et al* (2001) indicate that during the teen years of development, the rate of lung function growth can be altered by a change in exposure to air pollution. Such changes may be reversible during periods of rapid lung growth accompanying physical development during the teen years (Gauderman *et al*, 2000).

Figure 33



The NEW ENGLAND JOURNAL of MEDICINE

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The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age

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CONCLUSIONS

The results of this study indicate that current levels of air pollution have chronic, adverse effects on lung development in children from the age of 10 to 18 years, leading to clinically significant deficits in attained FEV_1 as children reach adulthood.

FEV1 is the volume of air expelled in the first second of maximal forced expiration from a position of full inspiration.

Particles and Long-Term Health Effects

No studies have directly addressed the explanation of the observed long-term increases in risk of death associated with exposure to particulate air pollution.This apparent excess of mortality applies to death from cardio-respiratory disease and lung cancer. From the discussion above, it is possible that pulmonary inflammation could increase risks of chronic obstructive lung disease in the same way that it does as an effect of smoking; **inflammation from one cause is likely to add to the effects of inflammation from another.**

With respect to lung cancer, it is known that urban particles carry polycyclic aromatic hydrocarbons, several of which are bronchial carcinogens (EPAQS 1999). They will therefore act as a vehicle for their transport into the lungs, again adding to the risks derived from other sources such as cigarette smoke.

If particles do indeed increase risks of death from heart disease, the effect is likely to be mediated through modulation of biochemical or cellular risk factors, such as by increasing levels of blood fibrinogen leading to increased atheroma formation in arteries.

Figure 33

Diagram illustrates dormancy of a tumour is when programmed cell death (apoptosis) balances cell multiplication (Mitosis).
Inflammation down-regulates apoptosis that results in acceleration of tumour growth. Inflammation associated with exhaust particles may act not only as an initiator of tumours but also the acceleration of their growth.

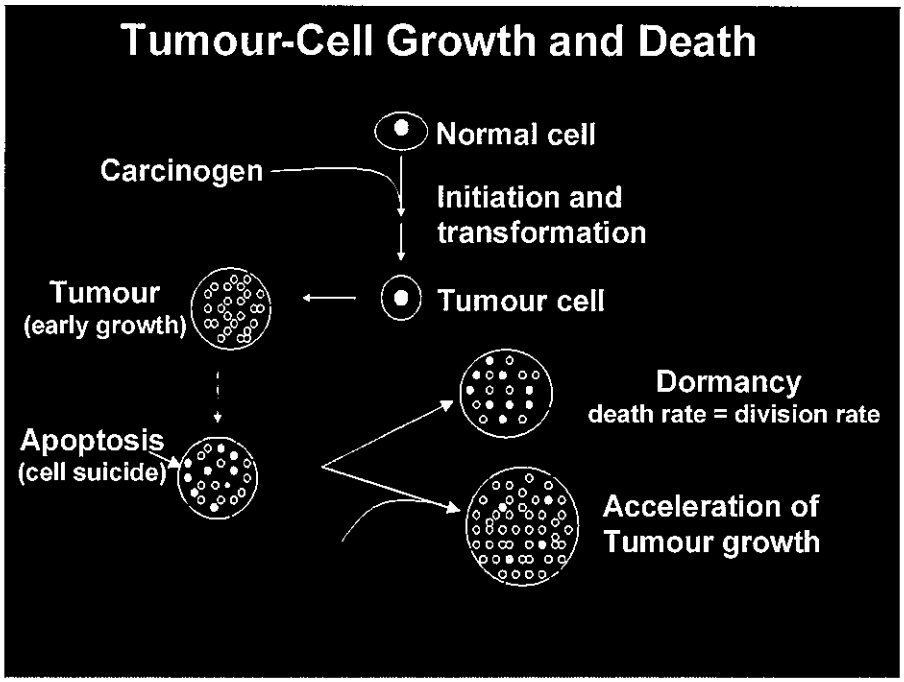

Figure 34

Photograph by Dr. R. Kearney of a tumour to show induction of blood vessels for continued growth. Dormancy of a tumour is not sleepiness but occurs when cell death equals cell multiplication. Inflammation accelerates tumour growth by 'switching off' cell suicide (apoptosis)

Figure 35

Demonstration by Dr. R. Kearney of acceleration of experimental tumour growth by inflammation induced by LPS. It would be expected diesel particles plus LPS would give greater acceleration of tumour growth.

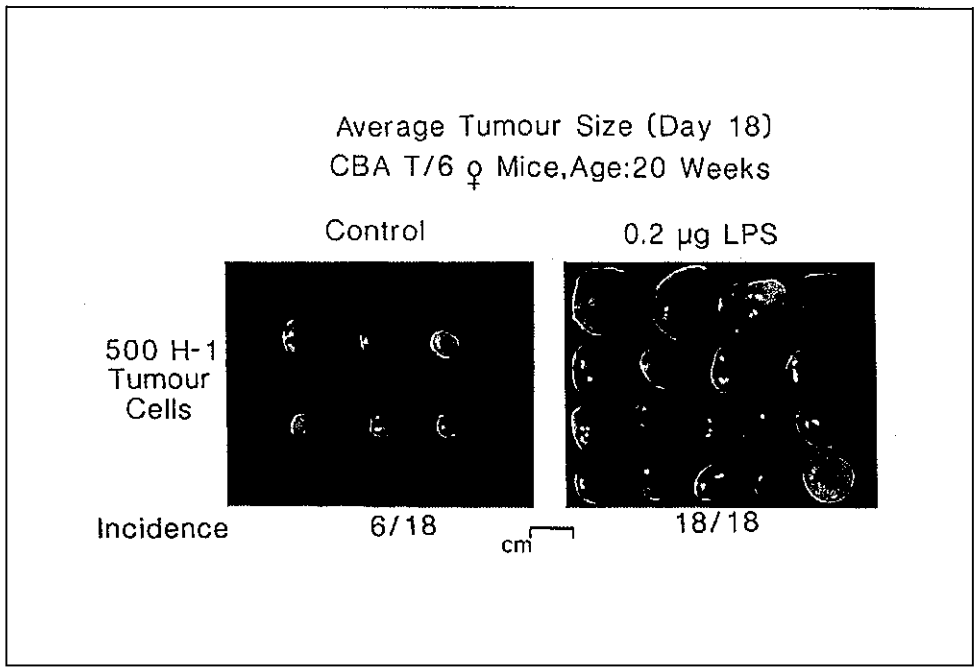

Figure 36

Common infection of the stomach due to *Helicobacter pylori,* accompanied by chronic inflammation that will accelerate tumour growth both locally and distal to the stomach

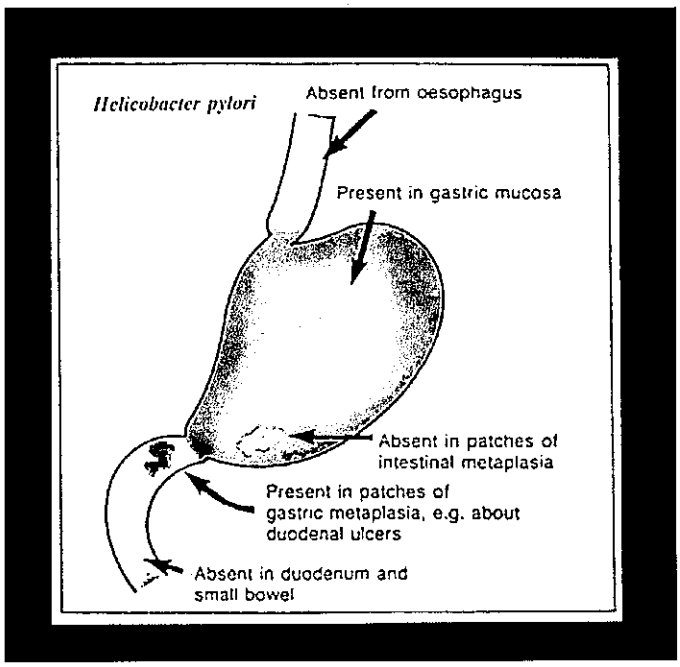

Figure 37

Shows the significantly higher prevalence of certain cancers in patients with *H. pylori* infection suggesting the chronic inflammation is associated with tumour growth.
The possibility exists that since lung cancer can be associated with pollution and cancer growth is accelerated by inflammation in the lungs. An even higher risk is carried when *H. pylori* gastritis is also present.

Prevalence (%) of Anti- *Helicobacter pylori* Antibodies.

Carcinoma	*n*	Pr ±pre
Lung	38	88.4 ± 4.9
Stomach	45	69.2 ± 5.7
Colon	55	78.6 ± 4.9

Pr ± pre= prevalence ± prevalence error
* Difference significant at the p=0.02 level
J. Muszynski *et al.*, *Scand J. Gastroenterology* 1995; 30: 647-651

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

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Michael J. Thun, MD
Eugenia E. Calle, PhD
Daniel Krewski, PhD
Kazuhiko Ito, PhD
George D. Thurston, ScD

Context Associations have been found between day-to-day particulate air pollution and increased risk of various adverse health outcomes, including cardiopulmonary mortality. However, studies of health effects of long-term particulate air pollution have been less conclusive.

Objective To assess the relationship between long-term exposure to fine particulate air pollution and all-cause, lung cancer, and cardiopulmonary mortality.

Design, Setting, and Participants Vital status and cause of death data were collected by the American Cancer Society as part of the Cancer Prevention II study, an ongoing prospective mortality study, which enrolled approximately 1.2 million adults in 1982. Participants completed a questionnaire detailing individual risk factor data (age, sex, race, weight, height, smoking history, education, marital status, diet, alcohol consumption, and occupational exposures). The risk factor data for approximately 500 000 adults were linked with air pollution data for metropolitan areas throughout the United States and combined with vital status and cause of death data through December 31, 1998.

Main Outcome Measure All-cause, lung cancer, and cardiopulmonary mortality.

Results Fine particulate and sulfur oxide–related pollution were associated with all-cause, lung cancer, and cardiopulmonary mortality. Each 10-$\mu g/m^3$ elevation in fine particulate air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively. Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality.

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

Based on several severe air pollution events,[1-3] a temporal correlation between extremely high concentrations of particulate and sulfur oxide air pollution and acute increases in mortality was well established by the 1970s. Subsequently, epidemiological studies published between 1989 and 1996 reported health effects at unexpectedly low concentrations of particulate air pollution.[4] The convergence of data from these studies, while controversial,[5] prompted serious reconsideration of standards and health guidelines[6-10] and led to a long-term research program designed to analyze health-related effects due to particulate pollution.[11-13] In 1997, the Environmental Protection Agency adopted new ambient air quality standards that would impose regulatory limits on fine particles measuring less than 2.5 μm in diameter ($PM_{2.5}$). These new standards were challenged by industry groups, blocked by a federal appeals court, but ultimately upheld by the US Supreme Court.[14]

Although most of the recent epidemiological research has focused on effects of short-term exposures, several studies suggest that long-term exposure may be more important in terms of overall public health.[4] The new standards for long-term exposure to $PM_{2.5}$ were originally based primarily on 2 prospective cohort studies,[15,16] which evaluated the effects of long-term pollution exposure on mortality. Both of these studies have been subjected to much scrutiny,[5] including an extensive independent audit and reanalysis of the original data.[17] The larger of these 2 studies linked individual risk factor and vital status data with national ambient air pollution data.[16] Our analysis uses data from the larger study and

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New Scientist - 9 March, 2002

Big city killer
If the cigarettes don't get you the traffic pollution will

in cities are caused by tiny particles of pollution, most of them from vehicle exhausts.

That's the conclusion of the biggest study into city pollution to date, which tracked half a million Americans for 16 years. It suggests the impact is far greater than feared.

The research focused on particles less than 2.5 micrometres in diameter, known as PM2.5s. These fine particles are thought to kill by lodging deep in the lungs.

The researchers found that the long-term death rate from lung cancer rose by 8 per cent for every 10-microgram increase in the average concentration of PM2.5s per cubic metre. The increased risk is comparable with the risks to long-term passive smokers.

C.A. Pope *et al*: *The Journal of the American Medical Association* (2002) 287, p 1132

Epidemiological Evidence for Health Effects of Particles

Few epidemiological studies have addressed directly the independent health effects of the fine as compared with the coarse fraction of particles. The limited evidence that is available comes from time-series studies that have related short-term fluctuations in mortality, hospital admissions and other measures of morbidity to temporal changes in the concentrations of air pollutants; and from cross-sectional and longitudinal nvestigations that have compared rates of disease or death in populations according to indices of their long-term exposure to pollutants.

The ability of these studies to discriminate between the effects of different particulate fractions is limited by the generally high correlation between different measures of particles, and especially between PM10 and PM2.5. Also, in the studies of long-term exposure, full adjustment for potential confounders has not always been possible. Overall, however, the variation in risk across the range of ambient levels has been similar for PM10 and PM2.5. PM10-2.5 has shown less consistent associations with health effects and appears less robust than PM2.5 in multi-pollutant models.

Sulphates, which form part of PM2.5, have also been associated with health effects, but probably do not account for all of the effects of PM2.5. In studies of summer acid haze in eastern North America, the effects of sulphates have been difficult to separate from those of acid aerosol and ozone. Black Smoke tends to show similar effects to those of PM10 and to be robust in multi-pollutant models. This suggests that material monitored by the Black Smoke method is responsible for some of the toxicity of PM2.5. The limited evidence that is so far available suggests that associations with PM2.5 are similar to those with counts of particle numbers in the sub-micrometre range.

Because fine particles are more uniformly distributed over a given area than coarse particles, it is likely that a central monitor will provide a less accurate reflection of population exposures to PM10 than to PM2.5. Such misclassification of exposures would normally be expected to bias estimates of risk towards the null. Thus, even where morbidity and mortality appear to be more strongly associated with PM2.5 than with coarser fractions of particles, it does not necessarily follow that the former is more toxic.

Current epidemiological evidence suggests that PM2.5 and PM10 are equally effective for detecting and monitoring adverse health effects from particulate pollution in the general population. There is some indication that the toxicity of PM10 resides largely in the PM2.5 fraction.

Issues in Particulate Matter Measurement and Monitoring

Unresolved issues related to vehicular PM emissions:

- The chemical composition of gas/diesel particulate emissions and how they vary by size;
- The way rates, chemical composition, and particulate size distribution change with operating conditions and malfunctions;
- The evolution and fate of ultra-fines in the atmosphere;
- Are emissions measurements relevant to exposure? Can we determine urban exposures with ambient measurements?

Polycyclic Aromatic Hydrocarbons and $PM_{2.5}$

Studies have identified that, for $PM_{2.5}$, motor vehicles contributed one-third of the total PM in ambient air, with petrol vehicles responsible for more than half of that. Diesel contributed approximately half the elemental carbon, and the organic carbon came mostly from petrol vehicles. With respect to PAH's (polycyclic aromatic hydrocarbons) in petrol and diesel emissions, diesel was found to have mostly lower levels of gas-phase PAH's as well as particle-phase.[20]
Studies by Dravitzki & Kvatch (2000) were undertaken to test the polycyclic aromatic hydrocarbon (PAH) content of $PM_{2.5}$ and PM_{10} combustion pollutants in the 625m long Mt. Victoria tunnel in Wellington, New Zealand. Studies found that the major amount of particulates (>75%) emitted had a size <$PM_{2.5}$. It is noteworthy that PAH compounds of which several are known carcinogens e.g., benz(a)anthracene, dibenz(ah)anthracene and benzo(a)pyrene were found to be mainly associated with respirable particles <2μm in diameter.

It is particularly noteworthy that UP TO a fifth of all lung cancer deaths in cities are caused by tiny particles of pollution, most of them from vehicle exhausts.That's the conclusion of the biggest study into city pollution to date, which tracked half a million Americans for 16 years. It suggests the impact is far greater than feared.

[20] Symposium Summary. The Future of Diesel: Scientific Issues 2000 Air Pollution Symposium. December 2000 Massachusetts Institute of Technology

The study is important because it followed individuals, says British expert Roy Harrison of the University of Birmingham, allowing the researchers to separate the effects of smoking and pollution. "In the past, we have often just compared urban areas and rural areas. But more people smoke in cities, and it is difficult to compensate for that."

The research focused on particles less than 2.5 micrometres in diameter, known as PM2.5s. These fine particles are thought to kill by lodging deep in the lungs. The researchers found that the long-term death rate from lung cancer rose by 8 per cent for every 10-microgram increase in the average concentration of PM2.5s per cubic metre. The increased risk is comparable with the risks to long-term passive smokers.Typical PM2.5 levels in the US are 20 micrograms in Los Angeles and 16 micrograms in New York-the limit set in 1997 by the Environmental Protection Agency is 15 micrograms. British levels are similar, though one PM2.5 monitor at Marylebone Road in London records an average of 32 micrograms. "I'd say London has a special problem because of the high proportion of diesel fumes," says George Thurston of New York University, co-leader of the study. (Ref: Moore et al: The Journal of the American Medical Association (2002, vol 287, p 1132).

The following Table illustrates the cancer-causing risk of carcinogens in diesel combustion:

Cancer-causing Pollutants in Diesel Exhaust

Pollutant	Diesel Emissions % of all Mobile 1996[52]	EPA Carcinogen Status	Cancer Risk (per million/microgram in 70-yr life)
Formaldehyde	52%	probable	1 in a million
Acetaldehyde	59%	probable	1 in a million
Butadiene	8%	probable	2 in a million
Acrolein	50%	possible	n/a
Benzene	5%	known	2-8 in a million
Diesel Particulate Matter	77%	probable[53]	EPA: 12 to 1210 in a million; CARB: 300 in a million[54]

http://www.catf.us/publications/reports/Diesel_Health_in_America.pdf

Table 3 PAH Concentration (ng/m^3). Distribution between the aerosol and gas phases (26/05/99)

Pollutants	Tunnel centre		Portal	
	Pat.	Gas	Pat.	Gas
Naphthalene	<	10669.3	<	2117.7
Acenaphthylene	<	614.3	<	156.8
Acenaphthene	<	42.0	<	15.7
Fluorene	<	216.6	<	52.3
Phenanthrene	<	223.1	<	49.5
Anthracene	1.6	64.7	0.2	11.6
Fluoranthene	6.8	29.1	1.4	9.9
Pyrene	10.3	38.8	2.9	12.9
Benz(a)anthracene	16.2	<	2.0	<
Chrysene	19.4	<	2.3	<
Benzo(b)fluoranthene	13.3	<	2.8	<
Benzo(k)fluoranthene	13.6	<	2.2	<
Benzo(a)pyrene	17.1	<	1.9	<
Indeno(123cd)pyrene	12.0	<	1.6	<
Dibenz(ah)anthracene	1.6	<	0.3	<
Benzo(ghi)perylene	30.1	<	3.6	<
Total of 16 Pollutants	141.9	11898	21.1	2426

< = Less than limit of detection

From V. K. Dravitzki and I. A. Kvatch (2000)

At least seven of the above PAH's are known carcinogens and as the authors have shown are associated with the <u>fine particles</u> less than PM 2.5.

The significance of this is found in studies by Seaton (1995) and Bates (1996) who estimated the "inhalable" particles less than PM_{10} deposited in the lung alveoli rises from about 10% at 5µm to 30% at 0.1µm and 50% at 0.05µm. Particles in cities are commonly at concentrations of 50,000 particles/cm^3 (Bates, 1996). An adult inhales about 18M^3 of air each day (Seaton, 1996). Assuming a concentration of 1000/cm^3 of 0.05µm, which would represent relatively clean air, the total number of particles deposited in the lung in a day would be 8×10^{10} i.e., about **270 particles in each of the 296 x 10^6 alveoli in the human lung.**

The lung's formidably large surface area would provide highly significant cumulative effects (exposure and absorption) of toxic substances to the alveolar membrane and therefore the potential harmful effects, including carcinogenesis, in local as well as in distal sites. Alveolar macrophages will hydrolize off chemicals adsorbed to the indigestible char particles to then be absorbed by the bronchocapillary and associated lymphatic vessels.

In the calculations of the public health impact of ambient air pollution, international experts maintain it is crucial to decide what level of exposure may be considered as the 'reference exposure'. In the study by Kunzli *et al* (2000), the lowest assessed level was set at 7.5µg/M^3 upwards. NSW Health seem not to define a *"lowest assessed level"* for health impacts in communities potentially affected by emissions from unfiltered tunnel stacks.

Particle composition in addition to size is important in determining the adverse effects of $PM_{2.5}$ (Saldiva *et al*, 2002). These authors found vanadium, bromine, lead and organic carbon from combustion sources to be powerful inducers of pulmonary inflammation.

It was confirmed by a N-Z Report that 75% of PAH's in traffic tunnels are confined to $PM_{2.5}$ or less.[21] Hence removal of ultra-fines (e.g., by E.P. filtration) also removes the bulk of the PAH's. Reports by Regulatory Authorities seem not to comprehend the applied relevance of these published facts and to acknowledge the value of filtration of particulates that also removes a proportion of highly toxic PAH's.

Tunnel studies have shown that particle size distribution in background averaged 100 nanometres (0.1 µm). At the tunnel inlet, ultra-fines (approx. 10nm or 0.01µm) mixed with the background distribution. At the tunnel outlet, they were virtually all ultra-fines [22] While ultra-fine particles can account for most of the particles by number (90%), they do not account for a large part of the total mass, only about 30%.

Particulate matter from on-road spark-ignition petrol vehicle emissions contains a higher fraction of particulate PAH's and these emissions are comparable to or exceed diesel vehicles on a per-km basis. However, these are **under-estimated** in current RTA/EPA inventories relative to diesel emissions.[23] A greater fraction of semi-volatile organic compounds exists in the particulate phase near the source. To date, the relative contributions of petrol and diesel vehicles to ultra-fine particle exposure have not yet been evaluated.[24] Therefore, LCTAG maintains that the failure to provide such information is a case for the Regulatory Authorities to adopt the Precautionary Principle and install filtration and gas-cleaning systems.

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[21] Dravitzki Vince K. and Kvatch Igor A. *Hazardous emissions from road transport.* 15th International Clean Air & Environment Conference. Sydney, November 2000 Vol. 1 pp 574-579.

[22] Ibid

[23] *Symposium Summary The Future of Diesel: Scientific Issues 2000 Air Pollution Symposium*, December 2000 Massachusetts Institute of Technology.

[24] Ibid

Fine Particulate Matter from Diesel Exhaust

In NSW, measuring ultra-fine particles presents major technical and bureaucratic problems as yet unresolved. Improved procedures are needed to properly characterize petrol and diesel particles in the environment. Studies have found that particle size distribution is strongly influenced by the formation of ultra-fine particles as exhaust gases are diluted by ambient air. Factors affecting ultra-fine particle formation include humidity, the dilution ratio, and residence time.[25]
Working Paper Nine fails to take account of the influence of dilution ratios on nanoparticle counts. Studies show the bigger the dilution ratio, the smaller the number of particles nucleated at the peak size. Nucleation of sulphuric acid followed by growth in the particle size due to sorption of volatile organic compounds is also strongly affected by ambient temperature. More particles at the peak size are nucleated in the plume at colder ambient temperature.[26] The failure to take account of the formation of ultra-fines and their significance in exhaust emissions and hence in the stack pollution reveals a high degree of incompetence in this area.

Studies of diesel nanoparticles[27] showed that the majority of 25 nm (0.025μm) particulate mass consisted of hydrocarbons (alkanes, alkenes and cyclic compounds), and that polycyclics were below detection level. These studies also found that these particles were enriched with heavy hydrocarbons relative to undifferentiated hydrocarbons in the range of a molecular mass of thirty or higher.

Current air-quality monitoring generally fails to acknowledge the PAH's are largely associated with particulates. Thus, the potential for removal of particulates and *concurrently* the adsorbed PAH's is rarely acknowledged by the regulatory bureaucrats in Australia, unlike overseas countries such as Japan and Norway.

Impact of Vehicle Emissions on Air Quality & Public Health

Studies[28] conclude that vehicle emissions (petrol and diesel) are unhealthy. Diesel exhaust is a major contributor to ambient NO_x (which is a precursor to ozone and to secondary fine particulates) and also contains diesel particulate matter and over 40 substances identified as toxic air contaminants.
Vehicle emissions carry a high cancer risk.[29] While health bureaucrats acknowledge health risks are associated with exposure to emissions, to date in Australia they have almost totally failed to comply with growing international recommendations that exposure to known identified toxic hazardous agents must be reduced. Community pressure to comply will continue, despite the propaganda that arises and demonstrable incompetence from many of the bureaucratic departments.

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[25] Ibid

[26] Ibid

[27] Ibid.

[28] Ibid

[29] Ibid

Diesel engines, irrespective of design and operating point, emit solid particulates in the range of 100nm (0.1μm), at concentrations *above 10 million particles per cm*^{3}. Regulatory Authorities generally fail to comprehend the published data that establishes a drastic curtailment of pulmonary intruding particulates is: *not feasible by further development of the engine combustion, nor by re-formulation of fuels, nor by deployment of oxidative catalytic converters.*[30] Filtration should be adopted as a health measure, coupled with alcohol-blended fuels.

Germany, Austria and Switzerland have already mandated the deployment of particulate traps to curtail the total solid particulate count, in the fine particulate range below 50nm. The curtailment of two orders of magnitude can be re-inforced through fuel additives.[31] Additional reductions can be achieved by incorporating alcohol in fuels as already recognised overseas.

In general, there is no clear public acknowledgement, by the Regulatory Authorities, that for studies of pulmonary intruding particulates from vehicle emissions (and the evaluation of particulate traps), the usual gravimetric evaluation of the total particulate mass PM (e.g., High Vol system) must be abandoned or at least enhanced. This is necessary as gravimetry is *non-specific* with respect to the chemical composition and the aerosol properties (such as size and surface) and, hence, *delivers no toxically relevant information*. The counted particulate concentration appears to be the significantly more specific and sensitive criterion.[32] Suitable equipment is available to conduct those measurements and its widespread use must be introduced urgently.

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[30] *VERT: Curtailing Emissions of Diesel Engines in Tunnel Sites* by Andreas Mayer, DieselNet Technical Report, April 1998

[31] Ibid

[32] Ibid

Health Impacts in the USA

National Annual Diesel Fine Particle Health Impacts[7]

Annual Cases in the U.S., 2010	
Premature Deaths	21,000
Lung Cancer Deaths	3,000
Hospital Admissions	15,000
Emergency Room Visits for Asthma	15,000
Non-fatal Heart Attacks	27,000
Asthma Attacks	410,000
Chronic Bronchitis	12,000
Work Loss Days	2,400,000
Restricted Activity Days	14,000,000

http://www.catf.us/publications/reports/Diesel_Health_in_America.pdf

Population Benzene Overload

In June, 2001, the National Industrial Chemicals Notification and Assessment Scheme (NICNAS), Australia's regulator of toxic chemicals, recommended in its Report that the national exposure standard for **benzene**, a known carcinogen, **be cut by 90%.** The director of NICNAS, Dr. Margaret Hartley, stated: "*There is no known threshold for the carcinogenic effects of benzene, but since the risk for leukaemia increases with exposure, it can be reduced by controlling exposure to the highest practicable standard.*"
Again, the regulatory bureaucrats, in somewhat cavalier fashion, largely ignore the potential impact of benzene. No mention is made of the NICNAS recommendation nor of the fact that the UK Government's plan to replace the current 5ppb standard for benzene to 1ppb by 2003. Australia appears not to have an official standard for benzene in the atmosphere, only an occupational and health standard. Ofequally concern is that EIS predictions of ground-level benzene concentrations from stacks seem not modelled using either the Calpuff or the Cal3qhc systems.
In the NSW RTA's *International Workshop on Tunnel Ventilation, (Sydney, June, 2000),* it was disclosed, upon cross-examination, that the benzene levels in the M5 East stack plume were estimated to be **twenty-fold higher than background**.

International Experts Take Account of Pollutants Known to Enhance Response to Allergens

Airborne pollutants not only have the effect of sensitizing the respiratory tract directly but will augment the specific hypersensitivity responses to known allergens (e.g., Rusznak, 1997). As little as 0.25ppm NO_2 will lead to airway narrowing. If at the same time as the asthmatic is exposed to specific allergen and inhales irritants e.g., SO_2, SO_3, $PM_{2.5}$ and/or ozone, then the addition of the secondary irritation to the primary hypersensitivity response can quickly reach the threshold to precipitate acute asthma. Conversely, exposure to irritants such as NO_2, can precipitate asthma involving a response to a **lower dose** of the allergenic trigger e.g., grass pollen. Also relevant is that temperature inversions higher in the atmosphere, can contribute to increases in air pollution at ground level and therefore impact adversely on health (Atkinson, 1999), especially in winter months.

Composition and Concentration of PM_{10} in Stack Emissions are Not the Same in Ambient Atmosphere

The average annual concentration of Sydney's airshed pollution is 25-30µg/M^3 PM_{10}. This figure is derived from averages of the 24 hour averages of prescribed monitoring stations throughout the Sydney regional area. Such a figure of 25-30µg/M^3 PM_{10} does not reflect numerous 'hot spot' areas e.g., CBD where 10-15 minute excursions can exceed 450µg/M^3. Nor does this figure of 25-30µg/M^3 PM_{10} reflect exceedances (above 50µg/M^3 PM_{10} 24 hr) that occur daily during busy morning and afternoon peak periods. Thus quoting 25-30µg/M^3 PM_{10} can be misleading when assessing health risks to local communities. Janssen *et al* (2000) showed that **personal** $PM_{2.5}$ concentrations are highly correlated with ambient concentrations within susceptible subjects (indoors elderly persons with cardio-vascular disease) over time. Ramachandran *et al* (2000) concluded numerous short-term (15-minute average) excursions were 3-4 times the measured average concentrations. Health impacts of 15 minute exposure to $PM_{2.5}$ pollutants are not reflected by measuring only 24-hour average concentrations.

Such short-term variations are important in light of reports (e.g., R.A., Michaels, 1996; 1998) that short-term excursions can explain some of the excess mortality and morbidity attributed to ambient particulates.

A pollution level expressed as PM_{10} at 25µg/M^3 gives no indication of the relative health hazard. Thus 25µg/M^3 consisting predominantly 0.1µm diameter particles will contain 1 x 10^6 times more particles and have a surface area 100 times that of 25µg/M^3 of 10µm diameter particles. Thus weight for weight the fine emission particles in fuel combustion are potentially 100-fold more toxic than particles resuspended from the road surface. Furthermore, in the light of the studies of the Morawska's group (2000), 25µg/M^3 PM_{10} stack exhaust from a traffic tunnel where the particulates are resuspended road-dust mixed with tail-pipe emission, the equivalent ambient air could be up to 4-fold higher i.e., 100µg/M^3. Failure to take such differences into account may partly explain the serious under-estimation of the health risk to residents whose health and well-being have been adversely affected by exposure to unfiltered emissions from the M5 East stack. The failure to monitor fine airborne particulate matter on the basis of size and number below 1µm makes it virtually impossible for NSW health to evaluate health risk. Australia now has a 'guideline' for $PM_{2.5}$ but only a standard for PM_{10}. The PM2.5 guidelines are not enforceable until 2005.

Term of Reference (e)

The financial impacts of air pollution on the NSW Health system

The highest air pollution costs occur for vehicles driven in the central areas of the capital cities. The high values in these areas are due to the greater impact of particulate pollution (the main pollutant of concern for health impacts) when emitted in areas of high population density.

Air pollution costs vary for different vehicles within urban areas. Larger vehicles have higher air pollution costs. Modern vehicles have much lower air pollution costs than older vehicles. However, particles generated by modern vehicles are smaller and on a weight for weight basis with pollution from older vehicles, modern car emissions may be more toxic in terms of particle numbers and surface area. Watkiss (2002) claims the largest benefits (i.e. the greatest reduction in air pollution costs) occur when comparing Euro 1 vehicles (which have catalytic converters) against pre-Euro vehicles. The results indicate that Euro 1 vehicles have air pollution costs around half that of older vehicles. Euro 2, 3 and 4 vehicles have even lower air pollution costs, though the levels of reductions are not as dramatic as from Euro 1 technology. The age of vehicles and the overall fleet mix therefore has a very large impact on the air pollution costs in Australia. As older vehicles are removed from the fleet and newer vehicles introduced, the air pollution costs for the road sector will change.

Air pollution costs also vary with fuel type in urban areas. Petrol vehicles, which have lower particulate emissions, have much lower air pollution costs than diesel vehicles. The differences between petrol and diesel vehicles are most pronounced for older vehicles (pre-Euro). Petrol vehicles emit higher NO_x and hydrocarbon levels, which tend to impact on health mainly through the formation of secondary pollutants, i.e. ozone and (for NO_x) secondary particles. Because these pollutants form over time and distance, the importance of local population density is less than for particulates.

Vehicles running on low sulphur fuel also have lower air pollution costs, though these benefits are small in comparison to the benefits achieved from tighter vehicle emission standards. European data also indicates that moving from low sulphur diesel to ultra-low sulfur fuel leads to lower air pollution costs (though the benefits are not as great as moving from conventional to low sulphur fuels). However, Australian test data on the benefits of ultra-low sulphur fuel over low sulphur fuel shows mixed results Watkiss (2002).

Understanding Differences in Air Pollution Costs

Emissions from road transport, e.g. from a busy road, lead to high concentrations of pollutants in the immediate area around the road (out to a distance of a few hundred metres), but drop off fairly rapidly thereafter. However, these emissions also lead to small increases in pollution well beyond this distance (in fact up to many kilometres away). These latter effects are important for major urban areas, as it will increase the numbers of people exposed to pollution[33].

For conservative pollutants, such as particulates, the exact levels of pollution across any area will be determined by the local meteorological conditions (primarily wind speed and direction) and the deposition rate. However, the key factor in determining the size of the air pollution costs is the local population density.

Taking all this into account it is clear that different area types are needed when looking at Australia to take account of differing population densities, particularly for urban areas. Watkiss (2002), therefore proposed different unit pollution costs for conservative air pollutants for three different area types. These are:

- **Areas of very high population density.** These include the central areas of all the larger capital cities (the exceptions are Canberra, Hobart and Darwin). These areas have average population densities of > 2,500 people/km^2. The central areas of Sydney, Melbourne and Brisbane have average densities of > 5,000 people/km^2.
- **Low - Medium population density.** This includes the outer areas of the larger capital cities, other capital cities (Canberra, Hobart, Darwin) and other urban areas. All of these have broadly similar population densities.
- **Very low population density.** All non-urban areas (rural and semi-rural areas) with population densities below 100 people/km^2.

[33] Note there are issues here with potential threshold effects. If there is a specific threshold, below which no health effects occur, then in areas of low background concentrations, the small contribution of road emissions will have no effect (as they will not lead to exceedances of the threshold). However, for the main pollutant of concern, particules, the current consensus is there is no threshold and therefore these lower level exposures are added to high level concentrations near the source.

Typical population densities for each of these areas were used by Watkiss (2002) to derive specific unit pollution costs for conservative pollutants (based on the regression analysis in Europe). The values for particulates, CO, benzene, butadiene and SO_2 (direct effects) are calculated using linear regression based on unit costs and population densities in Europe. Average densities assumed are: Large capital cities = 2,750 people/km^2. Other urban areas = 750 people/km^2, non-urban areas = 10 people/km^2.

However, it is also apparent that certain areas of Sydney will have different levels of secondary pollution (especially ozone). This led to an additional separation for urban areas, to differentiate between those areas with and without ozone problems. For secondary pollutants, unit pollution costs were transferred, based on local assessments for similarly sized urban areas. The values for NOx include nitrate and ozone effects. The values for hydrocarbons include ozone effects only. It is assumed that ozone effects will only occur in the major capital cities.

Overall, this led to four different area categories, each of which has a different set of air pollution costs. The areas proposed by Watkiss (2002) are:

- ***Band 1. Inner areas of large capital cities*** (Melbourne, Sydney, Brisbane, Adelaide and Perth). These areas have very high local population density and are areas where ozone is an issue.
- ***Band 2. Outer areas of large capital cities***. These areas have lower population density than the central areas, but have ozone problems and therefore secondary pollution effects.
- ***Band 3. Other urban areas***. This includes smaller capital cities (Canberra, Hobart and Darwin), and other urban areas. These areas have similar population density to the outer areas of the capital cities, but in general do not have ozone problems.
- ***Band 4. Non-urban areas***. These areas have very low population densities and no ozone problems.

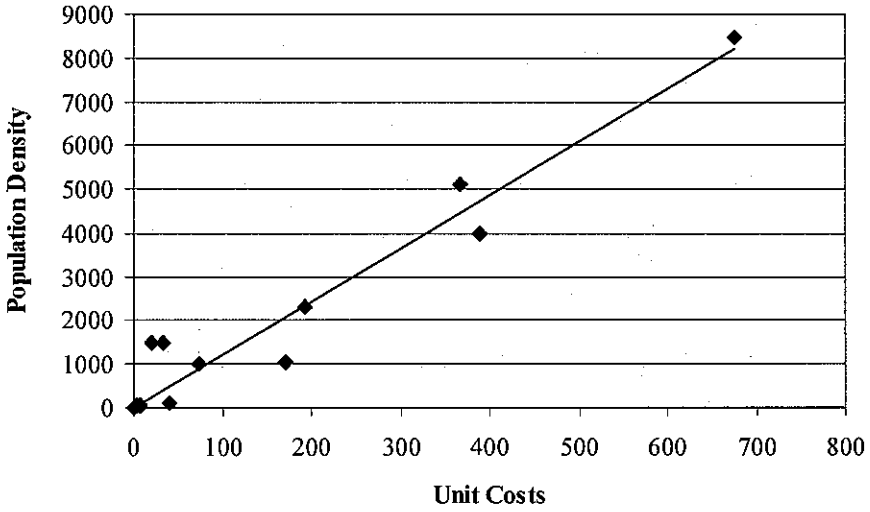
The relevant unit costs for the four area types are shown in the Table below. For comparison, the values from the NSW study are presented in the final column. It can be seen that the values proposed here have similar costs, with the exception of particulates.

Unit Pollution Values.

	Unit Values for this Study $A/tonne				Recent Australian
	Band 1	Band 2	Band 3	Band 4	values $A/tonne
Particles	341,650	93,180	93,180	1,240	17,600
CO	3.0	0.8	0.8	0	12
NOx	1,750	1,750	260	0	1,385
THC	875	875	175	0	1,440
SO_2	11,380	4,380	2,800	52.5	
Benzene	2,425	660	660	0	
1,3-butadiene	90,730	24,745	24,745	0	

Band 1. Inner areas of larger capital cities (Melbourne, Sydney, Brisbane, Adelaide and Perth).
Band 2. Outer areas of larger capital cities.
Band 3. Other urban areas. Includes other capital cities (Canberra, Hobart and Darwin), and other urban areas.
Band 4. Non-urban areas.

For conservative pollutants, the key factor in determining the relevant damage costs is the population density. Indeed, it is possible to show this by looking at the statistical relationship between damage costs and population density. A relatively good fit emerges[34]. For this study, Watkiss used a similar regression analysis to derive a function that directly relates population density to damage costs (i.e. cost per tonne) for each of the conservative pollutants (PM, CO, SO_2, benzene and butadiene). The regression has then been used to select relevant unit pollution costs for Australia based on population density .



The Relationship between Population Density (km^2) and Damage Costs. (Watkiss, 2002)

The author of this submission in 2005 contacted Chris Eiser of the NSW EPA (DEC) to provide the latest data on the levels of each of the pollutants for which Dr Watkiss had assigned the costs per unit of pollutant. The following tables were constructed:

[34] The outlying points arise as average population density for the entire area is presented in the graph. More specific population data for the exact area of emission would produce an even better fit.

Unit Pollution Health-Costs[1] for Sydney[2] - BAND[3] 1

Pollutant	Tonnes/year[6]	Unit cost[1] $A/tonne	Total cost annually
Particles (TSP)	24,370	341,640	8,325,766,800
CO	533,700	3.0	1,601,100
NOx	88,600	1,750	155,050,000
VOC[4]	135,870	875	118,886,250
SO_2	23,010	11,380	261,853,800
Benzene[5]	2,850	2,425	6,911,250
1,3-butadiene[5]	285	90,730	25,858,050
			8,895,927,250

1. Data from Table 23 in *Fuel Taxation Inquiry: The Air Pollution Costs of Transport in Australia* by Paul Watkiss (March, 2002)
2. Data (2002) provided by Chris Eiser
   Manager Atmospheric Science
   Department of Environment & Conservation (NSW)
   Note: Data is for all sources (Mobile, Industry, Domestic/Commercial)
3. Band 1: Inner areas of larger capital cities (Sydney, Melbourne, Brisbane, Adelaide and Perth) – P. Watkiss 2002.
4. Non-methane hydrocarbons
5. Data for 2000, Sydney (C. Eiser)
6. Data provided by C. Eiser

The costs are based on the assignment by Paul Watkiss of $6 million for a cost of a human life. It is noteworthy that the BTRE assigns a value of $1.3 million to a human life. Hence the lower cost estimate by BTRE for health impacts. See Table 9 below

Unit Pollution Health-Costs for Sydney - BAND 2

Pollutant	Tonnes/year	Unit cost $A/tonne	Total cost annually
Particles (TSP)	24,370	93,180	2,270,796,000
CO	533,700	0.8	426,960
NOx	88,600	1,750	155,050,000
VOC	135,870	875	118,886,250
SO_2	23,010	4,380	100,783,800
Benzene	2,850	660	1,881,000
1,3-butadiene	285	24,745	7,052325
			2,654,876,235

Legend: as above, except-
Band 2: Outer areas of larger capital cities (P. Watkiss, 2002)

Unit Pollution Health-Costs for Sydney - Average of BANDS 1 & 2

Pollutant	Tonnes/year	Ave. unit cost $A/tonne	Ave. total cost annually
Particles (TSP)	24,370	217,410	5,298,281,400
CO	533,700	1.9	1,014,030
NOx	88,600	1,750	155,050,000
VOC	135,870	875	118,886,250
SO_2	23,010	7880	181,318,800
Benzene	2,850	1542.5	4,396,125
1,3-butadiene	285	57,737.5	16,455,187
			5,775,401,792

Legend: as above, except data from Band 1 and Band 2 have been averaged.
Band 1: Inner areas of larger capital cities (Sydney, Melbourne, Brisbane, Adelaide and Perth) – P. Watkiss 2002.
Band 2: Outer areas of larger capital cities (P. Watkiss, 2002)

At the national level, these health effects are important. It is estimated some 2,400 people die each year in Australia from air pollution, and some 10-15% of the population display respiratory symptoms (NEPC, 1998). These health impacts have major economic costs, estimated at around $A18 billion/year[35]. Transport is a major source of these atmospheric pollutants and therefore can be assumed to be a major cause of these health effects.

[35] Note this estimate (NEPC, 1998) is considered high for the effects quantified, as it applies a full Value of Statistical Life (VoSL) of $A7million to value acute mortality (deaths). The literature now suggests that these deaths only reduce life expectancy by a relatively short period of time and more recent studies factor this into account with the use of a very much lower VoSL.

Air Pollution Costs for Australian Vehicles. Based on Emissions from the NEPM (2000) Project 2.2 Vehicle Testing (presented in Table 1). Urban Factors for Diesel Vehicles. (from Watkiss, 2002)

Vehicle Type	Year manufacture	Band 1 cents/km	Band 2 cents/km	Band 3 cents/km	Band 4 cents/km
Diesel passenger	1980-89	26.99	7.61	7.32	<1
car and	1990-95	14.55	4.11	3.94	<1
off-road	1996-99	9.46	2.75	2.55	<1
Diesel Light	1980-89	22.09	6.19	6.00	<1
Commercial	1990-95	12.83	3.64	3.48	<1
<3.5 t GVM	1996-99	11.27	3.31	3.03	<1
Medium	1980-89	24.89	7.25	6.72	<1
goods vehicle	1990-95	33.72	9.62	9.13	<1
3.5 – 12 t GVM	1996-99	9.31	3.10	2.45	<1
Heavy bus	1980-89	40.57	13.23	10.70	<1
	1990-95				
	1996-99	25.57	8.17	6.77	<1
Goods vehicle	1980-89	26.61	8.48	7.06	<1
12 – 25 t	1990-95	26.07	8.18	6.93	<1
GVM or GCM	1996-99	14.73	4.79	3.89	<1
Heavy Goods Vehicle	1980-89				
>25 t	1990-95	15.71	6.27	3.95	<1
GVM or GCM	1996-99	25.03	7.89	6.65	<1

Vehicle Type	Year manufacture	Band 1 cents/litre	Band 2 cents/litre	Band 3 cents/litre	Band 4 cents/litre
Diesel passenger	1980-89	138.41	39.02	37.54	<1
car and	1990-95	81.29	22.98	22.03	<1
off-road	1996-99	46.16	13.44	12.45	<1
Diesel Light	1980-89	129.20	36.21	35.08	<1
commercial	1990-95	78.73	22.33	21.33	<1
<3.5 t GVM	1996-99	64.01	18.83	17.23	<1
Medium	1980-89	110.14	32.08	29.72	<1
goods vehicle	1990-95	162.11	46.23	43.88	<1
3.5 – 12 t GVM	1996-99	43.73	14.56	11.49	<1
Heavy bus	1980-89	96.82	31.59	25.54	<1
	1990-95				
	1996-99	57.98	18.53	15.36	<1
Goods vehicle	1980-89	82.40	26.24	21.85	<1
12 – 25 t	1990-95	79.24	24.86	21.08	<1
GVM or GCM	1996-99	43.32	14.09	11.44	<1
Heavy Goods Vehicle	1980-89				
>25 t	1990-95	32.53	12.98	8.18	<1
GVM or GCM	1996-99	55.14	17.37	14.65	<1

Note, diesel vehicles manufactured 1996 or later are Euro 1.

Key:
Band 1. Inner areas of larger capital cities (Melbourne, Sydney, Brisbane, Adelaide and Perth).
Band 2. Outer areas of larger capital cities.
Band 3. Other urban areas. Includes other capital cities (Canberra, Hobart and Darwin), and other urban areas.
Band 4. Non-urban areas.

Air Pollution Costs for Australian Vehicles. Based on Emissions from NEPM 2001 (from Watkiss, 2002)

	Band 1 cent/km	Band 2 cent/km	Band 3 cent/km	Band 4 cent/km
Cars (diesel)	17.33	4.91	4.70	<1
LCVs (diesel)	22.45	6.30	6.09	<1
Rigid 1	23.54	6.90	6.34	<1
Rigid 2	22.20	7.04	5.89	<1
Artic	18.73	6.56	4.87	<1
Buses	27.84	8.95	7.36	<1

Key:
Band 1. Inner areas of larger capital cities. Band 2. Outer areas of larger capital cities.
Band 3. Other urban areas. Band 4. Non-urban areas.

The following Table and two Figures illustrate health benefits of reducing PM2.5, 24 hour and Annual Averages, in Sydney, are copied from the Final Report (September, 2002) titled 'Exposure Risk and Exposure Standard for the Development of a PM2.5 Standard by M. Burgers and S. Wals

Table S1.1 - $PM_{2.5}$ Scenario Assessment - Health Outcomes Avoided per year - SYDNEY

	Short Term Health Endpoint						Long Term Health Endpoint		
	S1	S2	S3	S4	S5	S6	L1	L2	L3
	Mortality	Mortality	Mortality	Hospital Admissions	Hospital Admissions	Hospital Admissions	Mortality	Mortality	Mortality
€	All cause	Respiratory	Cardiovascular	Asthma	Cardiovascular disease	COPD	All cause	Lung cancer	Cardio-pulmonary disease
Scenario									
Scenario: 24h PM2.5 <35	70	21	14	40	63	15			
Scenario: 24h PM2.5 <30	104	31	21	60	94	22			
Scenario: 24h PM2.5 <25	138	41	28	79	125	29			
Scenario: 24h PM2.5 <20	172	51	35	99	155	36			
Scenario: Annual PM2.5 <10							150	12	75
Scenario: Annual PM2.5 <8							329	39	247
Scenario: Annual PM2.5 <5							677	80	510

Figure S1.1 - $PM_{2.5}$ Scenario Assessment - Health Outcomes Avoided per year - SYDNEY

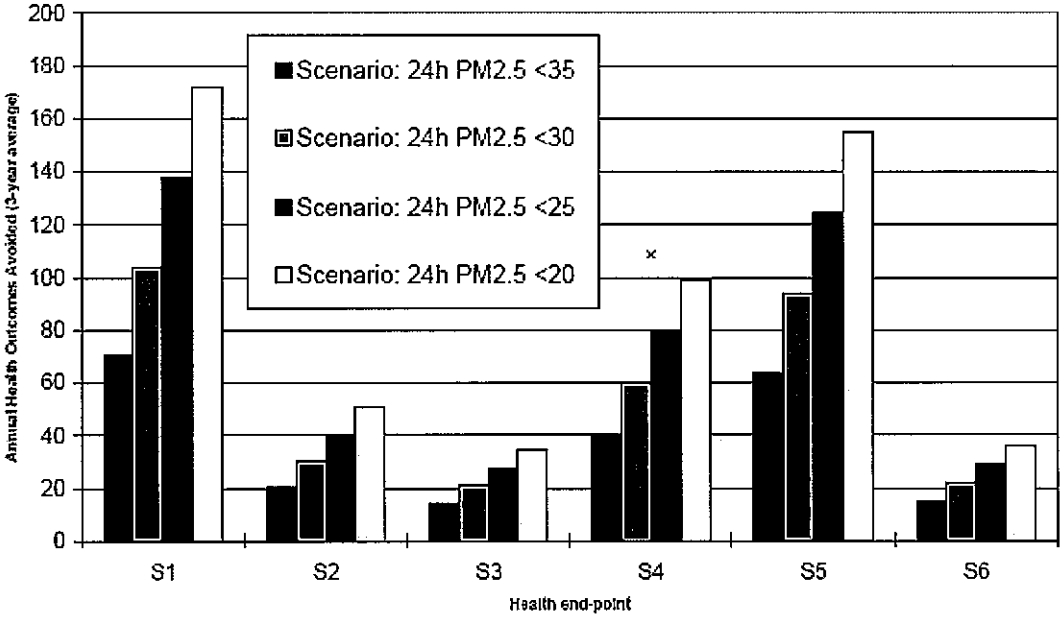
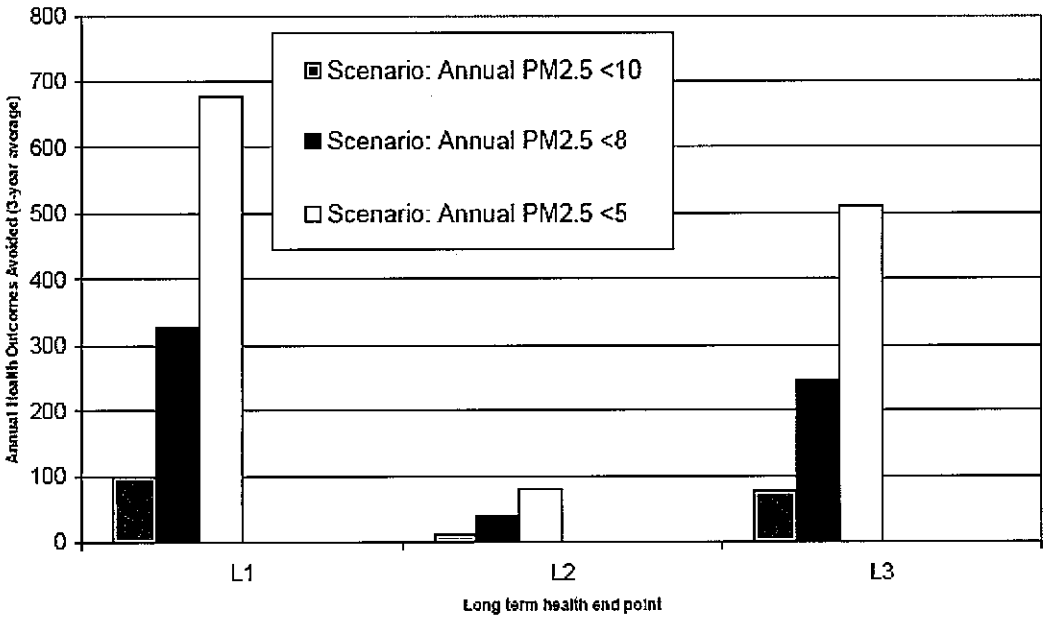


Figure S1.2 - $PM_{2.5}$ Scenario Assessment - Health Outcomes Avoided per year -SYDNEY



Two-Thirds Annual Health Costs are Incurred When Particulate Levels are Within Limits i.e., <50μg/M^3 24 Hour Average

A study by Zmirou et al (1999) showed that in an urban French population of one million inhabitants, **two–thirds** of the annual health costs in 1994 occurred during days when 24-hour average PM_{10} particulate levels were lower than 50μg/M^3. Economic loss or premature mortality due to pollution accounts for high health costs. Only 5% of health costs are hospital expenditures, very few cases require hospital care. Over-the-counter medication consumption costs are the most important expenditure because prevalence rates for respiratory symptoms followed by medicine intake are large in the general population. The next largest expenditure is absenteeism after respiratory conditions are diagnosed by G.P.'s

Overseas studies have estimated that to reduce PM_{10} concentrations from 30μg/M^3 to 10μg/M^3 will increase life expectancy by at least a year. In North America, modest reductions in pollution have been calculated to produce savings of the order of $40 billion p.a. by 2010.

Cost of Mortality and Morbidity Due to Vehicle Pollution in Australia

The information reproduced in the following Tables highlights the serious and costly impacts on health due to exposure to vehicle exhausts:

TABLE 10: CAPITAL CITY POPULATION VEHICLE POLLUTION INDUCED DEATHS AND ROAD FATALITIES (2000)

City	*Total population*	*Pollution induced deaths (range)*		*Road fatalities*
Sydney	3 502 301	339	762	267
Melbourne	3 160 171	213	478	230
Brisbane	1 508 161	94	210	44
Adelaide	1 002 127	54	120	70
Perth	1 176 542	49	111	91
Hobart	126 048	4	9	5
Darwin	71 347	2	5	19
Canberra	309 799	4	9	14
All Capital Cities	11 856 496	758	1 703	740

Sources: BTRE and ATSB

Amoako, L., Ockwell, A., Lodh, M., 'The Economic Consequences of the Health Effects of Transport Emissions in Australian Capital Cities' Bureau of Transport and Regional Economics, 2003

TABLE 8: HEALTH CASES OUTCOME –MORBIDITY (VEHICLE SHARE)

	Morbidity Cases			*Asthma Attacks (No of days)*		
City	Base case	Lower	Upper	Base case	Lower	Upper
Sydney	1 085	373	1 778	7 441	4 230	10 591
Melbourne	693	244	1 129	9 025	4 833	13 074
Brisbane	326	114	532	2 810	1 513	4 060
Adelaide	170	57	280	922	513	1 318
Perth	167	57	274	890	500	1 270
Hobart	10	4	16	3	2	5
Darwin	9	3	15	5	3	8
Canberra	na	na	na	na	na	na
All capital cities	2 460	851	4024	21 095	11 594	30 325

na= not available. ACT morbidity data was not available

Amoako, L., Ockwell, A., Lodh, M., 'The Economic Consequences of the Health Effects of Transport Emissions in Australian Capital Cities' Bureau of Transport and Regional Economics, 2003

Problems of Diesel

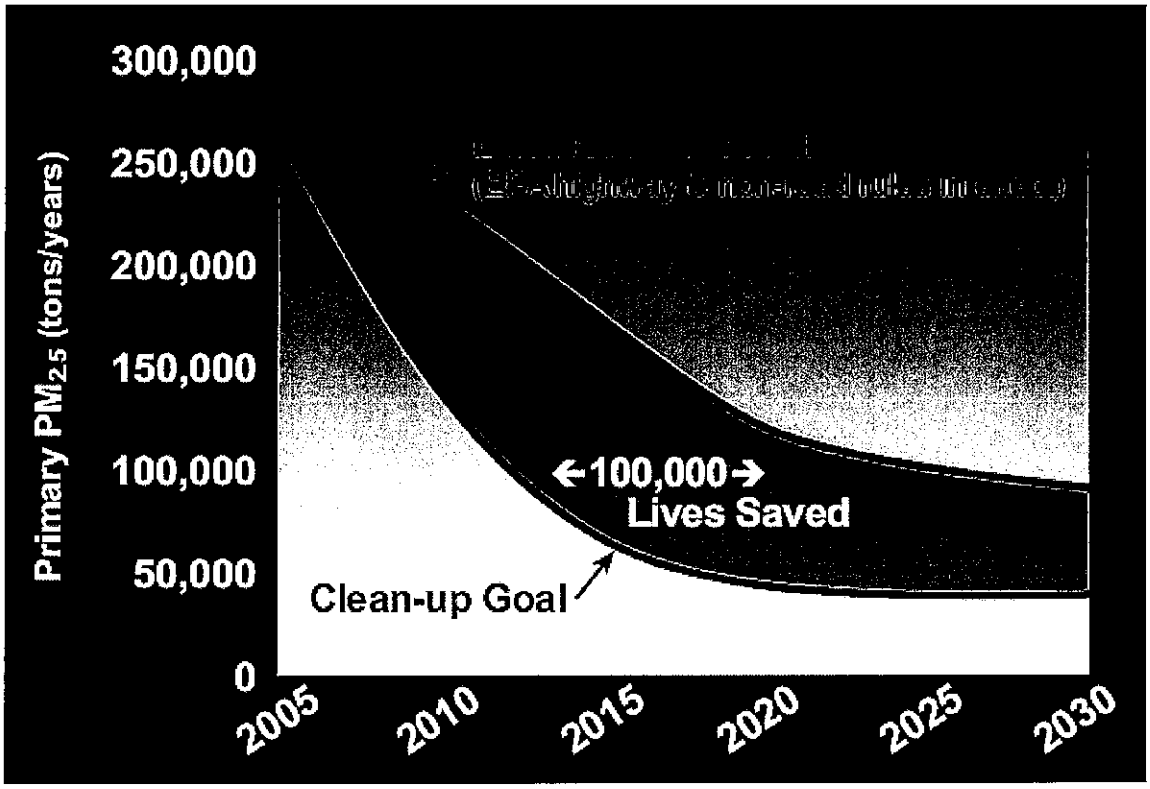
- **Complex engineering problem**
- **Environmental problem**
- **Medical / biological problem**
- **Legal problem**
- **Management problem**
- **Enormous public health problem**
- **Ethical problem**
- **Problem of public administration and good government**
- **Problem of fairness and justice - those most harmed are those least able to defend themselves - children of the urban poor.**

TABLE 9: TOTAL ECONOMIC COSTS –(MOTOR VEHICLES SHARE $A MILLION)

Capital Cities	*Mortality*			*Morbidity*		
	Base case	*Lower*	*Upper*	*Base case*	*Lower*	*Upper*
Sydney	713	441	990	785	782	788
Melbourne	448	276	621	466	465	468
Brisbane	197	122	273	223	222	223
Adelaide	113	70	156	109	109	110
Perth	104	64	144	110	110	111
Hobart	8	5	11	7	7	7
Darwin	5	3	7	10	10	10
Canberra	8	5	12	na	na	na
All capital cities	1 596	986	2 214	1 712	1 705	1 718

Amoako, L., Ockwell, A., Lodh, M., 'The Economic Consequences of the Health Effects of Transport Emissions in Australian Capital Cities' Bureau of Transport and Regional Economics, 2003

Cost Benefits in USA to Reduce Fine Particulates



http://www.catf.us/publications/reports/Diesel_Health_in_America.pdf

Term of Reference (f)
The effectiveness of current laws and programmes for mitigating air pollution

Limitations in the National Environmental Protection Measure (NEPM) and how air quality standards are abused by the Regulatory Authorities.

- Environmental Policy principles are embodied in the following:

  (a) Precautionary principle
  (b) Intergenerational equity
  (c) Conservation of biological diversity and ecological integrity
  (d) Improved valuation, pricing and incentive mechanisms

- Definitions were provided for 'standard', 'guideline', 'goal' and 'protocol' in the Inter-Governmental Agreement on the Environment (IGAE) in 1992.

The IGAE implied definition of the term 'standard' was '**standards are mandatory.**'

- The IGAC definition of 'standard' was intentional and was to make clear the full mandatory nature of any standard set in a NEPM. However, the IGAC definition has been **dropped** from the definition in the NEP Council Acts. Similarly for the IGAC definition of 'guideline' i.e., **"Guidelines are not mandatory."**

- Thus, the failure to carry these definitions through in their entirety into the various NEPC Acts has, it seems, allowed the NSW Regulatory Authorities e.g., RTA and DEC to **abuse** the "standards". Whilst the "standards" and the "goals" are numerically equal, goals and guidelines are non-mandatory. "Guidelines" or "goals" can be interpreted to suit political and bureaucratic agendas. The NSW Statutory Authorities have consistently ABUSED the spirit and intent of the NEPM Air Quality Standards and Guidelines. It is demonstrably proven that the NSW Government bureaucracies, led especially by the NSW RTA, have corrupted the NEPM science to favour the corporate stakeholder in Public Private Partnerships as well as accepting the trade-off in sickness and death.

The standards of air quality should not be used as a license to pollute up to a certain level.

Thus, community stakeholders now take the view that risk assessment is a "black box" process in which they are asked to trust outcomes derived by the bureaucrats (Health, RTA, EPA and Planning NSW) and their "experts." Not surprisingly community groups are reluctant to do so as demonstrated in recent EIS Reports (M5-East, Cross City and Lane Cove Tunnels) and NSW Parliamentary Inquiries. Risk levels regarded as negligible or quite low by the bureaucratic practitioners are **not** regarded as such by **educated and well-informed groups in the community and those who suffer.**

- The Air NEPM standards provide a nationally accepted process for estimating and reporting emissions of six most common indicators discharged to the air, **and that is all the process has done. There are key elements missing.**

- There is no national consistency in applying these standards or in determining what constitutes an **unacceptable level of emission in relation to these standards.**

- Present indications are that their nine jurisdictions (including NSW Regulatory Authorities) remain reluctant to yield their separate air (or environmental) management powers to a single national body. Thus a "national" air quality mind-set has not yet developed and is unlikely to do so. (P. Morgan, Clean Air & Environment Conference, Sydney 2000. Vol. 1 p. 252).

- Consequently, control of air quality is essentially a state-managed issue. Thus the NEPM process **does not address the question of how States are to apply the standards in decision-making.** Once a measure is made, NEPC has no role in advising or interpreting **how it is meant to be applied.** Consequently the NSW Regulatory Authorities have exercised this role and clearly have **abused the application of the standards.**

- In NSW, the NEPM standards are used as a basis for assessing proposals that would involve the emissions of the NEPM indicators e.g., through the M5–East exhaust stack. Compliance with the NEPM indicators is **not** being required, by the NSW Regulatory Authorities, even at performance monitoring stations, **let alone wherever people live.**

- In Section 14(1) of the NEPC Act, the NEPM standards apply only to regions with populations **above** 25,000. Thus, the NSW Regulatory Authorities, in determining any impact assessment on the community around the stack(s) of the M5–East stack, CCT or Lane Cove Tunnel are not necessarily bound by the NEPC Act to adopt performance monitoring. It is regrettable that the interpretation of the Act and the NEPM standards applied to the populations in the M5 precinct, CBD and Lane Cove is left to the NSW Regulatory Authorities who are generally beholden to and aligned politically/ financially (PPD) to the Corporate Stakeholders.

- Whilst it is therefore extremely likely that the emission e.g., from the M5–East exhaust stack, during busy pm and am peak periods will exceed 50$\mu g/M^3$ PM_{10}, ***daily***, the NSW Regulatory Authorities are given the role to determine its significance. The relevant PM2.5 levels, though commonly exceeding the annual average, is only for 'reporting purposes'.

- To date, the NSW Regulatory Authorities consistently refer to Sydney's Regional Air Basin (or Air Shed) as being 25-30$\mu g/M^3$ PM_{10} and is "good on world standards." The abuse of this reference is that it **does not** sufficiently characterize the true magnitude of the threat air-pollution poses in 'hot-spots' to public health. These references are not as accurate as down-wind exposure analysis from the M5- East Exhaust Stack on relatively **very small areas** where residents reside.

Arnold Dix, Facilitator of the Sydney International Tunnel Ventilation Workshop, records his following observation:
"During the course of the Workshop, professionals from Government Departments were unable, when asked, to fully explain the significance of the NEPM's in terms of outcomes for the community."
(Arnold Dix's paper presented at Clean Air & Environment Conference, November 2000 Sydney. Vol. 1, p. 444).

- A serious flaw in the Reports of the Air Quality Assessment (Holmes Air Science) is the consistent attempt, we believe, to patronise the RTA's objective and ignore or delete 'hot-spot' readings when exceedances of the NEPM standards give "cause for alarm."

- The role it would seem for certain consultants or clients, commissioned by the RTA, is not to provide **independent** and objective expert advice. They seem to act as **'technical advocates'** for the **agents** e.g., RTA, and their agenda.

- Thus, not surprising, the validity of the **air quality base data** and the **probity of modelling techniques** are seriously brought into question, by independent assessors eg, CSIRO.

Does NSW Health have a legislative role in approving major projects? No.

The following is an extract from the NSW Health's Submission (May, 2001) to the Parliamentary Inquiry into the M5 East Stack Ventilation:

"The NSW Department of Health has no legislative or regulatory requirement to participate
in the assessment or approval of major developments such as the M5 East tunnel. The Department provides advice to other departments or members of the public, when requested, in relation to such developments".

The problem that has arisen is when contractual decisions are made between the RTA and a successful tenderer, the NSW Health is NOT consulted about potential impacts of changes e.g., the deletion of the $60 million ventilation tunnel from the RTA's Lane Cove Tunnel Representation's Report.

The problem here, from recent experience, is that NSW Health is involved when it is too late. The catalyst for NSW Health to give advice comes from community concern, and not usually from the Statutory Authorities. The late arrival of NSW Health to the project places them in a distinctly compromising situation of endorsing approvals made by the other Authorities and to avoid a confrontation with them in public.
Therefore, the advice provided when only "requested" is clearly a major flaw in the current process that involves the RTA, EPA and finally Planing in giving approvals.
NSW Health should be a partner **right from the beginning** as its statutory responsibility and not have to wait to be asked.
The problem is with the legislation that should be changed so that there is a requirement of the Health Department to be actively involved in the **planning stages** of major projects of this kind.

ACTION: Recommend that the legislation be changed accordingly.

The Art of Perpetuating a Public Health Hazard

"Denial" of the hazard by an expert may not imply – *"the truth, the whole truth and nothing but the truth"*
M. Greenberg (*J. Occup. & Environ. Med.* 2005;Vol 47: 137-144)

The scandalous approval by the Federal Government for the new Hume Highway to pass through Albury rather than by-pass it was a DENIAL of the medical evidence already supporting a high cancer incidence in Albury likely to be due to higher exposures to carcinogenic exhaust fumes. It is clear that the ensuing mortality and morbidity is part of the "collateral damage" governments are prepared to accept for personal and political gain and expediency.

In April 2004, NSW Health released its findings from Phase 2 of its *'Investigation into the possible health impacts of the M5 East Tunnel Stack.'* The conclusion reached was there was *"no evidence of an association between the prevalence of reported symptoms and the modeled emissions* (annual averages of pollution levels in previous year) *from the M5 East stack."* The results of the study were subsequently used by the NSW RTA and the former Roads Minister Carl Scully to claim that the impacts of tunnel emissions are free of risks. The results have also been used by NSW Health in providing advice that a major development incorporating a primary school did not have health impacts from the M5 East stack, despite knowing that their assessment excluded children and long-term health impacts.

In the knowledge of glaring inadequacies in the NSW Health Report, Lane Cove Council (LCC) commissioned an independent review by three experts, outside of NSW, and coordinated by Dr Peter Best of Katestone Environmental in Queensland.

After very detailed examination of the NSW Health Report, the Katestone Review recommended that *"Council not accept the findings of the Phase 2 report"* noting that *"The Phase 2 findings of no association between the prevalence of reported symptoms and modeled emissions from the M5 East stack are readily criticized for potential flaws in study objectives and design."*

NSW Health is on notice in the face of documented scientific and methodological defects, that to proceed with their attempt to publish without correction would be tantamount to 'scientific misconduct.'

Why did Dr Michael Staff and his team at NSW Health not correct the record when their Report had been deliberately misused publicly and politically, knowing full-well that 'at-risk receptors' such as children were excluded, producing bias for a negative finding. Only acute effects, not long-term ones, were assessed using methodology that did not and could not determine the pollution exposure of the respondents to the NSW Health 'phone questionnaire conducted over four weeks.

Whilst it was common for complainants to report on odour issues, NSW Health was quite dismissive without explanation of the odour source. Recent scientific reports confirm that odours can be indicators of potential risks to health due to one or more co-pollutants.

To date, the NSW Health, RTA, EPA (now DEC) and Planning (now DIPNR) appear to adopt the **same strategies** used successfully to support the use of white asbestos (chrysotile) as a safe material (*J. Occup. Environ. Med.*, 2005; 47: 137-144). The same techniques are being used to subvert the community into thinking exposure to vehicle pollutants is without risk to health and well-being. It can be readily inferred from the highly critical Katestone Review of the NSW Health Report that, as with the asbestos scandal, a "denial" of the hazard of an agent by its protagonists, no matter how distinguished, may not correspond with *"the truth, the whole truth and nothing but the truth."*

The conclusion of NSW Health's findings seems consistent with a popular form of "denial" used by the advocates of asbestos and runs like*: "We did not find the evidence for a causal association between an agent and its alleged effects"* when the evidence is based on such factors as:

- Unsound "negative" results derived from flawed data, methodology and study-design.
- Concealment of data that effectively removes scientific rigour and renders a reviewer powerless.
- Sampling (or questionnaire) is not properly conducted in the true exposure and breathing zones.
- Subverting the thinking of people by the release of false information, rather than a disclosure of the true facts publicly.
- Deliberately avoiding definitive answers to a number of important questions by failing to establish and operate a long-term sampling strategy for determining the qualitative and quantitative measures of hazard exposure of subjects in the study.
- Keeping opinions to themselves, when confronted with the facts, allowing government or industry agents to effectively operate a policy of concealment by silence in the face of error while evidence of proven causal effects is kept confidential by agreement with management.
- Early denial is given authority when made by government or industry medical officers or by some medical consultants and others, often with 'conflicts of interest'. The significance of the hazard is down-played with a "so what?" attitude.
- Claiming to adopt "world's best practice" to imply, falsely, there are no risks to health.
- Omitting significant numbers of workers (receptors) and thereby introducing a 'negative' bias.
- Applying inappropriate standards or methods to effectively minimize the concentration of the hazardous agent in the exposure.
- By initiating an 'epidemiological survey', as a ploy, when faced with a health problem, or to simply ignore the problem. It buys time, similar to RTA's 'filtration trial.'
- Deliberately terminating studies at a stage when findings are suggestive.
- Failing to adopt Precautionary Principles to contain the toxic agent by not installing adequate environmental control technology.
- Suppressing highly critical 'audits of performance' for political expediency.

There have been too many studies world-wide which directly link vehicle emissions with mortality and morbidity for NSW Health to engage in a study where they would not be able to find the associations between stack emission and community health It is now evident that these strategies used to hide the public health hazards of asbestos for over a century also feature in the techniques adopted by NSW Health to perpetuate the **myth** to the NSW Carr Government and its bureaucrats that the exhausting of vehicle pollutants from tunnel stacks, in residential areas, poses no health risk, either short or long term, for anyone.

In a recent document headed, *'Fuel Taxation Inquiry: The Air Pollution Costs of Transport in Australia'*, submitted to the Federal Government, data shows that for the inner parts of Sydney (covering 2.5 million), the annual health 'pollution' cost is $342,000 per tonne of particles and $1750 per tonne for oxides of nitrogen (NOx). When this information is applied to current stack pollution from the **unfiltered** M5 East tunnel, the annual health-costs are about $6 million. For the Lane Cove Tunnel (LCT) and using under-reported LCT EIS stack-pollution data for 2006, the annual health-costs alone are about $5 million.

It is high time lessons from asbestos, tobacco, exposure to radiation and the like are learnt and as the Hon Ms Sandra Nori, a Government Minister in the Carr Government and Member for Port Jackson said recently that action *"must be taken to protect our communities from the impact of car emissions by using the latest and best tunnel filtration technology available".* Ms Nori should know the health impacts of vehicle emissions as she is Secretary of the ALP's Air Pollution Task Force.

A multifactorial approach to addressing the need to clean up our toxic atmosphere is suggested by adopting the following additive measures:

The Precautionary Approach

Principle is to search for and adopt alternatives.

- Other fuels (e.g., LPG, ethanol, biofuels)?
- Other kinds of engines?
- Filters for trapping the fumes and soot?
- Innovative modes of transport for moving goods and people?
- Other ways of planning city growth to reduce reliance on trucks and buses?
- Hydrogen?
- Steam?
- Compressed air?

Focus attention on eliminating the problem.

Why, It seems, is No One Taking a Precautionary Approach?

- Because the risk-based approach to public health is adopted i.e., wait until the dead bodies can be counted.

- Whilst diesel fumes are known to cause lung cancer, health bureaucrats state they are "not yet sure" how big the problem is and "we have not identified the extent of the problem."

This is the classic risk-based approach. Ignore the evidence so long as it is not 100% watertight. Use uncertainty as an excuse to delay. Wait for the dead bodies to pile, then slowly acknowledge the need for action.

Our Society Has Been 'Taken For A Ride'

- Influence of powerful corporate wealth on our daily lives by opposing a more humane, sustainable and less polluting nation.

- Scrapping of e.g., clean trams, for dirty diesel-powered buses is about how behind-the-scenes elements affect public policy. First implemented in USA, later adopted here. Some lobbyists testifying about the harmlessness of bus fumes.

- The dangers of breathing diesel fumes have been known for at least 20 years.

The NSW State and Federal Government should:

- Pass legislation for mandating the introduction of clean alternate fuels.

- Pass legislation to 'clean up' state fleet vehicles.

- Pass legislation to implement, retain and enforce tighter new engine and clean fuel standards for highway and non-road diesel.

Term of Reference (g)
Strategies to reduce the health impacts of air pollution

Technology

There are major reductions in air pollution costs from the introduction of modern vehicles. The greatest benefits arise from going from pre-Euro to Euro 1 vehicles (fitting of catalytic converters) for both petrol and diesel vehicles. Note that lead was removed from petrol not principally for health reasons but because lead 'poisoned' the catalytic converter. These benefits are shown across all emission test data, e.g. for the NEPM data.

The use of Euro 1 vehicles leads to very significant reduction in the costs per km, and the costs per litre of fuel used, especially in the major urban areas. The air pollution costs of Euro 1 vehicles are generally around half that of pre-Euro 1 vehicles (in some cases even greater benefits occur). Note, the benefits of introducing Euro 1 vehicles leads to much greater benefits (per km and per litre) than the introduction of lower sulfur fuels.

The European data indicate there are continued benefits in introducing Euro 2, 3 and 4 vehicles. In terms of the cents/litre, larger improvements occur for diesel vehicles. In general there are order of magnitude (i.e. factor of ten) differences in the air pollution costs of pre-Euro and Euro 4 vehicles.

Fuel Type (Petrol vs. Diesel)

Petrol vehicles, which have lower particulate emissions, have much lower air pollution costs than diesel vehicles. These benefits are most apparent for densely populated urban areas (capital cities). The differences between petrol and diesel vehicles are most pronounced for older vehicles (pre-Euro). The reason for these differences is that older diesel vehicles emit higher levels of particles, which is the primary pollutant of concern with respect to human health impacts.

Petrol vehicles emit higher NO_x and hydrocarbon emissions. These pollutants have less direct effects on health. Instead they lead to health impacts through the formation of secondary pollutants, notably, ozone and (for NO_x) secondary particles. Because these pollutants form over time and distance, the location of emissions has a smaller role in determining the size of impacts. Previous European studies (e.g. ExternE) have assigned higher air pollution costs to petrol cars than in the study by Watkiss (2002). This is because regional pollution is more of an issue in Europe due to the high background levels of other reactive species involved in secondary pollution formation and because population numbers in Europe are much higher (and therefore more important) for the regional formation of secondary pollutants.

Low Sulfur Fuels

Lower air pollution costs occur for all vehicles when running of lower sulfur fuels. The greatest benefits of low sulfur fuel arise from its use in older diesel vehicles. Note, the benefits of lower S fuels are nowhere near as beneficial as for the introduction of vehicle technology standards.

The Australian BIC test data and Parsons test data indicate particularly good benefits from moving from 1200 to 500 ppm S diesel. The European data confirms this – for larger diesel vehicles, there are important benefits from moving to low sulfur diesel (LSD >500 ppm S). The benefits of ultra-low S diesel (ULSD) are less apparent in the Australian data, though some benefits are seen for certain vehicles tested (Watkiss, 2002). The European data indicates benefits from the use of ULSD, though the spread of data across vehicle type indicates that the benefits of moving from LSD to ultra-low sulfur diesel are only around half that of moving from diesel to low-sulfur diesel. The European data indicates that the benefits of moving to 10 ppm S fuel are negligible.

The reduction in cents/litre with LSD and ULSD varies significantly with vehicle age and with the location of the vehicle (i.e. small or large urban areas). It is therefore difficult to generalise on appropriate pricing differentials, other than to say that for LSD the benefits over conventional fuels are at least a few cents/litre (and for older vehicles can be tens of cents/litre). The additional benefits of ULSD over LSD are much smaller, though the

European data indicates these could still be a few cents/litre for older vehicles in central urban areas (Watkiss, 2002).

Combustion Emissions from Alternative Fuel Trucks.

	Combustion Emission (g/km)				
Fuel type	NOx	CO	NMVOC	PM_{10}	CO_2
LS diesel (Aus)	10.17	2.48	0.90	0.38	719
ULS diesel (Aus)	8.66	3.13	0.73	0.28	718
ULS diesel (100% hydroprocessing)	8.66	3.13	0.73	0.28	718
Fischer-Tropsch diesel	8.84	2.00	0.52	0.25	671
Biodiesel (canola)	11.51	1.36	0.04	0.27	0
Biodiesel (soybean)	11.51	1.36	0.04	0.27	0
Biodiesel (rape)	11.51	1.36	0.04	0.27	0
Biodiesel (tallow-expanded sys. boundary)	11.51	1.36	0.04	0.27	0
Biodiesel (tallow-eco.allocat.)	11.51	1.36	0.04	0.27	0
Biodiesel (waste oil)	11.51	1.36	0.04	0.27	0
Biodiesel (waste oil 10% original oil value)	11.51	1.36	0.04	0.27	0
CNG (Electric compression) 0.1%	1.25	0.04	0.02	0.07	599
CNG (NG compression) 0.1%	1.25	0.04	0.02	0.07	599
LNG (from existing transmission line)0.1%	1.98	0.03	0.02	0.07	620
LNG (Shipped from north west shelf)	1.98	0.03	0.02	0.07	620
LNG (perth)	1.98	0.03	0.02	0.07	620
LPG (Autogas)	0.53	0.19	0.01	0.04	649
LPG (HD5)	3.53	0.17	0.02	0.02	710
LSdiesohol	9.29	2.80	0.86	0.29	631
Ethanol azeotropic (molasses -Sarina exp.sys.boun)	8.69	3.14	0.73	0.28	0
Ethanol azeotropic (molasses - Sarina- Eco Alloc.)	8.69	3.14	0.73	0.28	0
Ethanol azeotropic (wheat starch waste -Bomaderry)	8.69	3.14	0.73	0.28	0
Ethanol azeotropic (wheat)	8.69	3.14	0.73	0.28	0
Ethanol azeotropic (wheat) fired with wheat straw	8.69	3.14	0.73	0.28	0
Ethanol azeotropic (woodwaste)	8.69	3.14	0.73	0.28	0
Ethanol azeotropic (ethylene)	8.69	3.14	0.73	0.28	439
Hydrogen (from natural gas)	0	0	0	0	0

Source: CSIRO (2001).

Combustion Emissions from Alternative Fuel Buses.

Fuel type	Combustion Emission (g/km)				
	NOx	**CO**	**NMVOC**	**PM10**	**CO_2**
LS diesel (Aus)	18.26	4.45	1.62	0.68	1291
ULS diesel (Aus)	15.55	5.62	1.31	0.51	1289
ULS diesel (100% hydroprocessing)	15.55	5.62	1.31	0.51	1289
Fischer-Tropsch diesel	15.88	3.59	0.93	0.44	1204
Biodiesel (canola)	20.66	2.43	0.07	0.49	0
Biodiesel (soybean)	20.66	2.43	0.07	0.49	0
Biodiesel (rape)	20.66	2.43	0.07	0.49	0
Biodiesel (tallow-expanded sys. boundary)	20.66	2.43	0.07	0.49	0
Biodiesel (tallow-eco.allocat.)	20.66	2.43	0.07	0.49	0
Biodiesel (waste oil)	20.66	2.43	0.07	0.49	0
Biodiesel (waste oil 10% original oil value)	20.66	2.43	0.07	0.49	0
CNG (Electric compression)	2.24	0.07	0.04	0.12	1076
CNG (NG compression)	2.24	0.07	0.04	0.12	1076
LNG (from existing transmission line)	3.56	0.06	0.03	0.12	1113
LNG (Shipped from north west shelf)	3.56	0.06	0.03	0.12	1113
LNG (perth)	3.56	0.06	0.03	0.12	1113
LPG (Autogas)	0.95	0.34	0.01	0.07	1166
LPG (HD5)	6.34	0.30	0.04	0.03	1275
LSdiesohol	16.68	5.03	1.54	0.52	1133
Ethanol azeotropic (molasses-expanded sys.bound.)	15.60	5.64	1.32	0.51	0
Ethanol azeotropic (molasses-economic allocation)	15.60	5.64	1.32	0.51	0
Ethanol azeotropic (wheat starch waste)	15.60	5.64	1.32	0.51	0
Ethanol azeotropic (wheat)	15.60	5.64	1.32	0.51	0
Ethanol azeotropic (wheat) fired with wheat straw	15.60	5.64	1.32	0.51	0
Ethanol azeotropic (woodwaste)	15.60	5.64	1.32	0.51	0
Ethanol azeotropic (ethylene)	15.60	5.64	1.32	0.51	789
Hydrogen (from natural gas)	0	0	0	0	0

Source: CSIRO (2001).

Air Pollution Costs for Alternative Fuels in Buses. Based on Emissions from CSIRO, 2001 Combustion and pre-combustion (urban). Cents per Litre. (from Watkiss, 2002)

Bus	Band 1	Band 2	Band 3	Band 4
	cents/litre	cents/litre	cents/litre	cents/litre
LS diesel (Aus)	54.99	21.16	15.19	<1
ULS diesel (Aus)	41.94	16.93	11.78	<1
ULS diesel (100% hydroprocessing)	41.81	16.80	11.73	<1
Fischer-Tropsch diesel	36.82	14.35	9.34	<1
Biodiesel (canola)	45.16	18.90	11.54	<1
Biodiesel (soybean)	45.39	19.13	11.59	<1
Biodiesel (rape)	45.18	18.92	11.55	<1
Biodiesel (tallow-expanded sys. boundary)	45.13	18.87	11.52	<1
Biodiesel (tallow-eco.allocat.)	44.38	18.12	11.16	<1
Biodiesel (waste oil)	44.36	18.09	11.15	<1
Biodiesel (waste oil 10% original oil value)	44.39	18.13	11.15	<1
CNG (Electric compression) 0.1%	9.14/m^3	3.19/m^3	2.45/m^3	<1
CNG (NG compression) 0.1%	9.24/m^3	3.30/m^3	2.49/m^3	<1
LNG (from existing transmission line)0.1%	6.12	2.31	1.60	<1
LNG (Shipped from north west shelf)	6.01	2.20	1.55	<1
LNG (perth)	6.07	2.26	1.60	<1
LPG (Autogas)	4.64	2.38	1.90	<1
LPG (HD5)	3.94	3.03	1.51	<1
LSdiesohol	37.23	15.06	10.30	<1
Ethanol azeotropic (molasses -Sarina exp.sys.boun)	23.23	8.97	5.95	<1
Ethanol azeotropic (molasses - Sarina- Eco Alloc.)	23.29	9.04	5.94	<1
Ethanol azeotropic (wheat starch waste - Bomaderry)	27.24	12.99	9.97	<1
Ethanol azeotropic (wheat)	27.52	13.27	10.14	<1
Ethanol azeotropic (wheat) fired with wheat straw	32.69	18.43	14.27	<1
Ethanol azeotropic (woodwaste)	29.12	14.86	11.18	<1
Ethanol azeotropic (ethylene)	25.65	11.39	7.06	<1
Hydrogen (from natural gas)	<1	<1	<1	<1

Note. Different values will result for urban pre-combustion emissions depending on the location of emissions. We assign all pre-combustion emissions a band 2 weighting.

Key:
Band 1. Inner areas of larger capital cities (Melbourne, Sydney, Brisbane, Adelaide and Perth).
Band 2. Outer areas of larger capital cities.
Band 3. Other urban areas. Includes other capital cities (Canberra, Hobart and Darwin), and other urban areas.
Band 4. Non-urban areas.

The use of the NEPM emission factors produces high air pollution costs for the diesel fleet. There are lower costs with more modern vehicles, but the high particulate emission from vehicles leads to high air pollution costs in urban areas, especially in central areas of capital cities.

Based on the NEPM data, the air pollution costs of the diesel fleet are estimated at 2 – 20 cents/km in urban areas. This is equivalent to a value of 10 cents/litre to A$1.4/litre. The highest values are associated with older vehicles, e.g. pre-1990 vehicles, travelling in the central areas of the major capital cities (where population density is very high). Interestingly most of the NEPM data does not indicate large differences in PM_{10} emissions between differently sized vehicles. This is reflected in the air pollution costs, which are very similar for all vehicles.

The lack of recent data for the Australian petrol car fleet makes it difficult to evaluate the air pollution costs of these vehicles. The data that is available does not include emissions of particulates, and does not include data on modern vehicles. It does appear that emissions from the Australian car fleet are much higher than the European fleet. It is not certain what the reason for this is, though it is likely to be a combination of the emission standards in place, the low vehicle turnover rate in the fleet, and the lack of a national vehicle testing and maintenance programme in Australia (Watkiss, 2002).

Higher environmental costs occur in capital cities because the population density is extremely high and the urban area is very large. This effect is particularly marked for the central areas of Sydney. Almost all the difference between the central areas of capital cities and other urban locations is due to the greater impacts of particulates.

Targeted measures to reduce vehicle emissions in these areas will have the greatest environmental benefits (and are likely to be most cost-effective). Very low emission vehicles including Euro 3 and 4 technology and alternative fuels should be targeted at these areas.

There is also another factor which exacerbates these urban effects. At low speeds, such as in heavily congested areas, emissions per km are higher. As congestion effects will be greatest in the central areas of Sydney and on urban highways, then the air pollution costs of vehicles in these areas may be even higher. It is also stressed that the values estimated by Watkiss (2002), for central areas of capital cities are at the lower end of damages and in the central areas of Sydney, air pollution costs are likely to be much higher due to the very high population densities in these areas.

Technology for Emission Curtailment

The notion that future technologies will lead to reduced levels of noxious particulations in vehicle emissions is speculative. Oxidative catalytic converters widely employed in diesel engines do not curtail solid particulate emission and also convert up to 40% of NO to more toxic NO_2. Attempts to curtail pollution through fuel re-formulation and new lubricants has decreased sulphur and aromatic components but have produced much higher numbers of ultra-fine respirable particulates at all point loads (Mayer, 1998). Similar outcomes have resulted with engine modifications. However, with the introduction of alcohol-petrol, a proportionate reduction in pollution will result.

Particulate Traps and Electrostatic Precipitators

Technology currently exists to remove particulates from vehicles by fitting particulate traps. Particulate removal can be more than 90% (Mayer, 1998). Filtration of particulates in traffic tunnels is best achieved by electrostatic precipitators (EP's). Such proven technology currently is in operation e.g., in Japan and Norway where EP's remove >90% on a numbers basis as well as on a mass basis for all classes of particles down to 0.3µm diameter. For particles greater than 1µm, the efficiency obtained is >95%. In-tunnel filtration clears the airstream within 15 minutes (e.g., Drammen tunnel, Norway) and helps keep the external local environment less polluted. Experience gained clearly shows a tunnel E.P. filtration/ventilation system can provide both economic advantage and cost benefits in reducing pollution from tunnels.

In NSW, two Parliamentary Inquiries into the M5 East tunnel recommended filtration. The current NSW Government's stand against filtration, coupled with invalid health-risk assessments based on flawed air quality monitoring seems an abrogation of a 'duty of care' that only a Royal Commission of Inquiry may unravel.

What is apparent in these tunnel projects is that such unacceptable political decisions not to install filtration arise and flourish in a political and bureaucratic climate when health professionals, it seems, with few exceptions, choose to remain silent. The appeal to such experts is, individually and collectively, to stand fearlessly as advocates for the health and well-being of local communities who are at major disbenefit.

Emission Curtailment through Oxidation Catalytic Converters

NSW Regulatory Authorities regularly assert that future technologies will lead to reduced levels of particulates in emissions. Such a position fails to acknowledge the fact that oxidation catalytic converters widely deployed in diesel engines **do not curtail** solid particulate emissions.
There are different effects for the oxygen–rich diesel exhaust gas in the oxidation catalytic converter.

The desired reactions are:	The undesired effects are:
$CO \Rightarrow CO_2$	$NO \Rightarrow NO_2$
$HC \Rightarrow CO_2 + H_2O$	$SO_2 \Rightarrow SO_3$

These reactions are very aggravating. NO_2 is more toxic than NO and SO_3 contributes to formation of sulphate particulates and to aerosols of sulphuric acid (battery acid).
The oxidative catalytic converter has the effect of converting up to 40% of the NO to NO_2 [36] The ratio of NO/NO_2 is usually 5-10% in diesel exhaust. Thus the catalytic converter substantially increases the NO_x toxicity. Emissions measurement in Swedish mines has substantially confirmed this effect.[37]
To date, EIS Reports fail to acknowledge that studies by gravimetric evaluation, of exhaust gas from utility vehicular engines, indicate no curtailment in the particulate's mass. Indeed a large increase is observed.[38]

[36] Ibid

[37] Ibid

[38] Andreas Mayer. Op cit

The bureaucratic expectation of improvements in air quality with better technologies e.g., with use of oxidative catalytic converters is **inaccurate** in light of the following facts:[39]

- The oxidation catalyst does not reduce the combustion of particulates (soot).
- The oxidation catalyst produces sulphate particulates.
- The oxidation catalyst has unfavourable gaseous phase reaction (increased toxicity).
- The positive effects of oxidation catalyst are irrelevant for construction site diesel engines.
- The oxidation catalyst does not curtail the actual solid combustion aerosols.

LCTAG contends that the negative effects far outweigh the benefits. In fact, there are indications that **additional solid particulates are created in the catalytic converter**. Thus, the installation of a filter or trap cannot be avoided for intermediate retention, coupled with a commitment to introduce alcohol-blended fuels.

Remember the catalytic converter was originally introduced to combat a smog problem caused by dirty fossil fuels. Lead in petrol 'poisons' the catalyst. The change to unleaded fuel was accompanied by the introduction of additional carcinogenic aromatic chemicals, as a substitute for lead, to combat pre-ignition of fuel under compression

Emission Curtailment through Fuel Reformulation

Studies have been undertaken to reformulate diesel fuel for curtailing emissions[40]. The main parameters are the diminished sulphur content, decrease in aromatic components and increasing the Cetane Index. The modest improvement i.e., to diminish total particulate emission by about 5 – 15% is insufficient to attain the joint European project VERT objectives i.e. only about 10%, which is disappointingly low.

In the same studies, the nano-particulate emissions were also investigated within the scope of this fuel comparison. Disappointingly, **not the slightest improvement in diminishing nano-particulates emissions was attained.**[41]

Thus, LCTAG believes the reformulation of diesel fuel cannot curtail the emission of respirable ultra-fine particulates. This route should therefore be abandoned and replaced by particulate traps and tunnel filtration. To this must be added the development of alcohol-petrol fuel which have proven benefits in reducing particulate emissions. Flexi-cars are now designed to be powered by 85% ethanol-petrol blends.

Potential Effect of Engine Modification

Engine development during the last decade has impressively succeeded in reducing the emission of particulate mass. Unfortunately, the **emissions now contain higher numbers of ultra-fine respirable particulates.**

Measures adopted collectively to suppress the combustion close to the cylinder wall include:

High supercharging, air intercooler, central nozzle position, increased number of nozzle perforations, very much higher injection pressure, lower air swirl and a shallower piston bowl. The mixture is thus prepared in the combustion air (air preparation) and not at the surface of the combustion space, as was previously the case. All large agglomerates are thus suppressed: the black smoke disappears and thus the particulate mass curtailed.[42]

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[39] Ibid

[40] Ibid

[41] Ibid

[42] Ibid

Andreas Mayer has shown that there is no improvement in the nano-particulate distribution, normalized with load. Indeed, **the new low-emission engine emits more ultra-fine particulates at all load points: at one load point six times more.**
The expectation that engine modification will solve the particulate problem, is inaccurate unless alcohol fuel is adopted with the engine modified accordingly as already available overseas. Engine development alone, at least at this time, without alcohol-blended fuels, is no strategy effectively (factor 100) to curtail the formation and emission of nano-particulates. Another solution must be sought. The installation of vehicular particulate traps coupled with in-tunnel filtration for treating the engine exhaust gas are viable proven measures to decrease particulates. The addition of alcohol fuels is imperative.

Emissions of Polycyclic Aromatic Hydrocarbons (PAH's)

Besides soot, PAH's always occur in the combustion of petrol and diesel. They are known to be at least partially carcinogenic and represent mostly unburnt fuel. These PAH's are in a gaseous state at exhaust gas temperatures that are expected at mean or even full load. The fact that the PAH's are found adhered to particulates less than 2.5μm diameter lends them to removal by particulate trap technology. The explanation for the association is that the PAH is adsorbed on the soot due to its large active surface. Removal can be more than 90%.[43]
The LCTAG contends that appropriate removal of particulates by the fitting of a trap to the exhaust and the installation of particulate filters such as electrostatic precipitators in tunnel can remove the PAH's along with toxic respirable particles.
We conclude, on the basis of published studies:

- The nano-particulate emissions of the combustion engine must be diminished at the in-tunnel source to 1/150 – 1/100 of the initial value. This is essential to attain the respiratory air-quality at the tunnel worksites and portals and can be achieved by fitting particulate traps and installation of EP's.

- With the exception of alcohol as a fuel, neither reformulated fuel, nor new lubricants, nor oxidation catalytic converters permit sufficient curtailment of particulate emissions.

- Further engine developments hold no promise to effectively curtail the ultra-fine particulate emissions through improved mixture preparation and combustion unless designed around alcohol petrol blends.

- Hot-gas filters and EP's are now able dependably to curtail the ultra-fine particulate concentration in exhaust gas by a factor 100 to 1000. This is valid for particulates of all sizes down to the range of 10 nm (0.01μm).

- Alcohol, iron and cerium-based fuel additives reinforce the particulate trap technology. They curtail the raw emissions and together with traps do not form secondary emissions such as ash emissions. They also do not form dioxins and furanes.

- The filter technology is therefore technically feasible, controllable in the field and is cost effective.

Wide scale deployment of these technologies to improve the respiratory air quality to help protect health is justified.

[43] Andreas Mayer, op cit.

Alcohol as a Fuel

The increasing cost of petrol in some countries and new laws requiring alternative fuels to reduce adverse impacts on health by fossil fuels have turned attention to car and truck designers to 'flexi-fuel'. Chief among alternative fuels is alcohol and 'biodiesel'.

'Alco-cars' have been used in Brazil and with modern design improvements is seeing a resurgence of sales.

Ethanol can be made from farm products such as sugar-cane and as a fuel has many advantages.

- Alcohol fuels burn cleaner than regular gasoline and produce less carbon monoxide and other pollutants.
- Vehicle manufacturers in some countries have designed engines with such fuel flexibility that they are capable of operating on 85% ethanol and 15% petrol, 100% petrol or any combination in between. A special sensor on the fuel line senses the petrol/ethanol mixture and computation automatically adjusts for air:fuel ratio and timing.
- Flexi-fuel alco-vehicles are manufactured at present by Chrysler, Ford and Mazda for the same price as petrol vehicles.
- For standard vehicles, alcohol can be blended in relatively small concentrations (10%) with petrol.
- Experimental tests have shown that alcohol-fueled spark ignition engines can produce as much or slightly higher power than petrol.
- Local State production of alcohol from sugar and will maintain viability of the sugar-cane farming as well as support agricultural jobs.
- Production of fuels in the State provides energy self-sufficiency.
- Federal Government must consider the introduction of tax incentives for alcohol fuels.

In the USA, these may include:
Exemption of tax per gasohol litre excise; cost pre-alcohol litre blender's tax credit; the cost per litre small ethanol producer's credit; the tax deduction of clean-fueled vehicles, and a tax credit for the production of unconventional fuels.
Such tax incentives, if enacted, would:

1. Encourage substitution of alternative renewable transportation fuels for petrol and diesel fuel, which would reduce petroleum consumption and importation.
2. Help support sugar-cane farmers for nearly all of Australia's ethanol production needs, and
3. Reduce green-house effects, adverse impacts on health as well as improve air quality.

Australia's capacity to produce alcohol from sugar-cane is immense. The best way to guarantee supply is the strong interest in revival of a new 'alco-car' with concomitant job creation and improved health.

Alternative Fuels

An analysis of alternative fuels show that CNG and in particular LPG have major benefits over other fuels when compared to current vehicles. However, the air pollution benefits from alternative fuels should be put in context. The air pollution costs of modern Euro 3 and Euro 4 vehicles are similar to the use of alternative fuels. The benefits of bio-diesel and bio-ethanol are modest compared to conventional vehicles and have similar air pollution costs to the use of low sulfur diesel (though they will have lower greenhouse gas emissions – their main reason for use). The benefits of alternative fuels, as with modern Euro technology, are greatest in central areas of capital cities. It is emphasized that the current 10% ethanol blended petrol, though able to reduce pollution levels significantly will in due course, as in overseas countries, be replaced with E85.

Health advantages to using ethanol-blends

- Ethanol is non-toxic, water soluble and highly biodegradable.
- The American Lung Association of Metropolitan Chicago credits ethanol-blended reformulated petrol with reducing smog-forming emission in the city by 25 % since 1990.
- Ethanol reduces tailpipe carbon monoxide (CO) emissions by as much as 25 %.
- Ethanol reduces particulate emissions, especially fine particulates that pose a health threat to children, senior citizens and individuals suffering from respiratory ailments.
- Ethanol is an 'oxygenate' that permits a cleaner burn much like an open gas ring on a Bunsen burner allows the gas to combust cleanly with a blue flame.

Federal Coalition 2001 Election Policy

- The Coalition undertook to promote the production, distribution and transport use of ethanol and biodiesel, in the knowledge that it will provide new industries and jobs for rural Australia and cleaner air for our cities.
- The Federal Coalition is committed to maintain the fuel excise exemption for ethanol and biodiesel.
- The Federal Coalition also undertook to provide from 2002/03 a capital subsidy for new or expanded production infrastructure for biodiesel until total production is reached by end 2006/07.

Current biofuels in Australia and overseas

- In Australia, biofuels with commercial prospects are ethanol and biodiesel.
- Ethanol is mainly derived here from two renewable sources – fermentation from sugars in grains such as wheat and corn and from 'C' molasses.
- Fuel ethanol in Australia is used as a fuel blend comprising 10 % ethanol and 90 % petrol (E-10).
- Brazil is currently the largest producer of ethanol at 12-15 billion litres per annum. Additional 30 sugar and ethanol plants should be in operation with ethanol production increasing by 50% over 2004/05 levels to 2.7 bin litres. In 2005/06 ethanol production will be 16.8 billion litres.
- The U.S. produces 10 billion litres per year, with production expected to rise to 19 billion by 2012.

- Overseas ethanol-blended fuels may contain as much as a 85% ethanol. Car manufacturers Chrysler, Mazda and Ford are now marketing cars to compute automatically to any alcohol-blended fuel.
- Biodiesel is derived from treatment of vegetable oil or animal fats.
- Canola oil is our principal oil seed and is harvested in November and December.
- Biofuels, unlike fossil fuels, are climate and rainfall dependent.
- The European Union in 2001 introduced a proposal to promote biofuels such as biodiesel, bioethanol or hydrogen fuels. The Commission's goal is to increase biofuel use from 2% in 2005 to 5.75% in 2010 and 20% by 2020.
- Approximately 8% of diesel fuel sold in Germany is biodiesel.
- Australia's Federal Coalition in October 2001 made a commitment to promote the use of biofuels such as ethanol and biodiesel to ensure biofuels provide 2% of our transport consumption by 2010.
- Fuel ethanol production and use is also being promoted in China, India, Thailand and Japan.

Issues

Problems of Diesel

- Complex engineering
- Environmental
- Medical / biological
- Legal
- Management
- Public health
- Ethical
- Public administration and good government
- Fairness and justice - those most harmed are those least able to defend themselves - children of the urban poor.

The Risk-Based Approach to Diesel and Petrol

Principle is to adopt "business as usual."

- Has backing of powerful special-interest groups harnessing governments to deflect and stymie the search for least harmful alternatives. This includes not only the Oil cartels but also to some extent self-interested groups such as the CSIRO who appear to patronise government with flawed reports. Patronage is the life-blood of politics!

- So long as the exact size of the problem is uncertain, risk assessors call for delay and more study. Research funding for some is a survival strategy where the aim in some cases is not to solve a problem but to create others. It all helps to maintain viability but without a social conscience.

- Because consultants can be 'bought' or 'hired' to reinterpret old data to cast doubt on the nature of the problem, action can be stalled for decades.

- Doubt is a powerful helpmate when your goal is to maintain "business as usual."

- The risk-based approach waits for the holy grail of scientific certainty to emerge from the data, meanwhile do nothing.

Why is the 'Precautionary Approach' not taken ?

- Because the risk-based approach to public health continues to be adopted i.e., wait until the dead bodies can be counted.
- Whilst petrol and diesel fumes are known to cause lung cancer, health bureaucrats state they are "not yet sure" how big the problem is and "we have not identified the extent of the problem."

This is the classic risk-based approach. Ignore the evidence so long as it is not 100% watertight. Use uncertainty as an excuse to delay. Wait for the dead bodies to pile, then slowly acknowledge the need for action. Precaution is not (yet) fashionable – Risk-assessment is !

Benefits of ethanol-blended fuels

- Many countries are adopting ethanol production to reduce harmful emissions from vehicles and enhance economic development.
- Ethanol contains 35% oxygen. Adding oxygen to fuel results in more complete fuel combustion, reducing harmful tailpipe emissions.
- Ethanol also displaces the use of toxic petrol components such as benzene - a carcinogen known to cause leukemia.
- Ethanol is a renewable fuel, typically produced from plant matter.
- Ethanol-blended fuels account for 18% (and growing) of all automotive fuels sold in the United States.
- Now is the time to promote ethanol in blended fuel with a banning of MTBE in USA.
- Ethanol-blended fuels reduced the $C0_2$-equivalent greenhouse gas emissions by approx. 3.6 million tons in the USA in 2001. i.e., equivalent to removing 520,000 cars from roads.
- Tripling the use of ethanol in USA would triple the greenhouse gas benefit.
- Ethanol fuels not only enhance energy security and boost rural economies, but can reduce harmful air pollution and greenhouse gas emissions.
- Biofuels can cause a renewal in agriculture and rural Australia and benefit the entire national economy.

What are the advantages to using ethanol-blends?

- Less dependence on imported crude oil
- Extends Australia's dwindling domestic supply of light crude petroleum used to produce transportation fuels.
- Expanded market opportunity for Australian farmers
- Rural economic development
- Displaces dangerous components in petrol, such as benzene
- Ethanol is made from renewable resources, whereas petroleum comes from limited fossil energy sources.
- Cleaner environment (lower carbon monoxide and smog-causing emissions)
- Cleaner burning engines
- Improved vehicle performance
- Bioethanol fuel shows better performance in reducing volatile organic chemicals (VOC), PAH, benzene and butadiene, relative to petrol.
- 10% ethanol blended petrol reduces fine particulates by up to a qualified 50%

Experience with biodiesel

- Emission reduction for particles, CO and gaseous hydrocarbons but increases in oxides of nitrogen (NO_x).
- In particulate emissions, the insoluble fraction (coarse mode) decreases while the soluble fraction (fine mode) increases with a net reduction in total PM. The soluble fraction can be reduced by using oxidation catalysts.
- Biodegradation of biodiesel is much faster than for diesel fuel.
- Studies show that, for greenhouse emissions, biofuels substantially out perform fossil fuels (but to a lesser extent) gas fuels.
- Biodiesel has significantly less ecotoxicity than diesel and ideal for sensitive rural areas.
- Biodiesel fuel has a biomodal distribution of fine particles with a 30% reduction in the 0.05 and 0.1µm diameter particles, but remained the same for larger and smaller particles.
- Recent studies showed biodiesel can reduce emissions of particulate matter by 47% when compared with petroleum in unmodified diesel engines.
- USA EPA report verified a 67% reduction in unburned hydrocarbons and a 48% reduction in CO_2 levels with pure biodiesel (B100). Smaller reductions (12%) were obtained with 20% biodiesel and 80% petro-diesel.

Assistance needed

- Assistance is needed in the general development of the biofuel industry. This includes new storages, crushers, refineries and associated infrastructure.
- Need to legislate fuel standards to include renewable biofuels such as biodiesel and ethanol-blended petrol.
- Address the escalating costs of growing crops e.g., canola, grains and sugar-cane, because of enormous amounts of inputs, disease levels and diminished yields giving unprofitable returns.
- There is an urgent need to increase farmer confidence for canola, grains and sugar cane.
- Long-term excise relief (or domestic producers credit) is required to engender confidence that lenders will have debts/loans re-paid.
- Capital subsidy be provided for enhanced ethanol production to attract investment capital from prospective owners.

Support from the Australian Medical Association (AMA) – Federal and NSW Branch

The Australian Medical Association, endorsed by the Australasian Lung Foundation, reported recently that the following interventions would reduce the negative health impacts of fossil fuel pollution:

- introduction of mandatory biofuel blends (petrol with 10% ethanol and diesel with 20% biodiesel)
- reduction of highly toxic aromatics such as benzene in petrol
- replacement of petrol/diesel vehicles with those that use liquid petroleum gas (LPG) or compressed natural gas (CNG);
- installation of in-tunnel filters and gas-detoxification systems in vehicular tunnels in heavily populated cities.

Recommendations

- The author advocates the expansion of the market for domestically produced renewable biofuels to reduce Australia's dependence on imported petroleum, spur rural economic development creating new jobs and tax revenue, and improve environmental quality by reducing emissions of harmful pollutants and greenhouse gases.

- The Federal Government is urged to enact a more aggressive Renewable Fuels Standard than is currently in the legislation, noting biofuels offer an immediate alternative to imported fossil fuels, are completely compatible with current transportation infrastructure as petroleum blending components of stand-alone fuels and in the longer term, are an ideal hydrogen source for fuel cells.

- Enact legislation to use ethanol as an oxygenate in petrol and to reduce levels of carcinogenic benzene.

- Enact legislation that allows durable excise rebates for the greenhouse credits, urban quality and health gains from ethanol and biodiesel in proportion to their proven environmental and health benefits.

- The technology exists right now to clean up these emissions from these engines so that most of the adverse health impacts can be prevented.

- The only thing that stands between us and dramatically healthy air is the political will at Federal and State levels to require these reductions and the funding to make them a reality.

Conclusions

In summary, these mechanistic possibilities can be divided into two broad areas, the physical
journey of particles into the lung and the biochemical/cellular mechanisms. The physical journey is clearly influenced by particle size. In general, smaller particles are more efficiently deposited in alveoli and small airways, especially in subjects with lung disease. Removal by macrophages is hindered if the cells become overloaded, and this is more likely with large numbers of very small particles than with small numbers of much larger ones.

The precise mechanisms of lung toxicity have yet to be fully clarified but most likely invoke the uptake of particles by phagocytic and epithelial cells, generation of extra- and intra-cellular reactive oxygen species, and subsequent release of pro-inflammatory and tissue damaging mediators. Once released, these attract other cells to the site of the response and, in the case of cardiovascular disease, may alter the coagulability and flow characteristics of the blood. In the case of allergic disease, such as asthma and hay fever, a synergistic interaction between tissue responses to inhaled particles and allergic responses to inhaled allergens may amplify the allergic tissue response. It should not be forgotten that such allergens are themselves included mainly in the larger size fraction of PM10. Thus pulmonary inflammation provides at least a theoretical explanation for most of the observed associations between exposure to particulate pollution and ill health, either by direct effects on the lung, by synergistic reactions with allergens or by secondary effects on the blood.

It is possible that movement of particles into the lung interstitium, where the inflammatory process may be initiated, is an important factor in the lung's reaction. This appears to occur predominantly by uptake of very small particles by epithelial cells. There is evidence that the toxic effects demonstrated experimentally are dependent not only on the dose in mass terms, but also on the size of the particles, the effects being greater (on a mass-for-mass basis), the greater the number of particles. Toxic fine and ultra-fine particles are expected to increase in the exhaust emissions of vehicles in the future.

This effect is likely to be related to surface area and probably due to substances on that surface. Current evidence suggests that transition metals initiating the release of free radicals are the most likely candidates for this toxic component.

For future epidemiological studies and for monitoring purposes it would be desirable to identify the components of the ambient aerosol most closely related to its toxicity, and to use this as a metric rather than PM10. Experimental studies indicate that these components would be likely to be included in the mass metrics, PM1.0 and PM2.5. However, particle number, particle surface area, or even content of certain transition metals that connect the measurement with cellular mechanisms linked to health outcomes may in the future prove more useful.

In addition to proven filtration systems both for exhaust and in-tunnel cleaning of air streams it is imperative that alcohol-fuel be introduced. Cars are already marketed by Ford, Chrysler and Mazda, without increased costs, for variable alcohol / petrol blends with as much as 85% alcohol content. More commonly up to 20% is becoming widely used world wide. In Australia, alcohol as an energy fuel will not only help reduce the green-house effect and adverse health impacts but will also provide economic viability to the grain and sugar cane industry. Our Government's support for such a venture is clearly warranted.

Term of Reference (h)
Any other relevant matter

1. Breaches of National Guidelines by NSW Regulatory Authorities

Are the standards prescribed by National Environmental Pollution Measures (NEPM) and adopted by NSW RTA and DEC (EPA) designed for 'point source' emissions e.g., from a stack?

(a) NSW Health states:

Department of Health acknowledges however that both the NEPM and US EPA goals were not designed to be applied to point sources, but to ambient air. Predicted dust deposition rates also exceed the NSW EPA goal of 2g/M^2/month."
(p. 8, NSW Health, Dr Greg Stewart to J. Betts (RTA), re: Lane Cove Tunnel EIS – 8.1.02 under Parliamentary Orders).

(b) NEPM states:

"*Conversely the air quality of some localised areas within major air sheds are dominated by local activities such as that experienced in a road tunnel or a heavily trafficked canyon street. Air quality management in these areas is complex and needs a different approach to that directed at meeting ambient standards intended to reflect the general air quality in the air shed".*
(NEPM (1998) p.13).

2. Over a Barrel: Corporate Corruption of Science

Whilst we may view occupational and environmental diseases as isolated and unique failures of the government, industry and science, the fact is that they are a sad outcome of a pervasive system of priority setting, decision making, and influence.

We were all appalled to be told that eating seafood from Sydney Harbour is a risk to our health because of toxic dioxins discharged into the harbour by a corporate environmental vandal who has now moved on. Why did it occur, despite the documented scientific evidence of the hazard? Where were the NSW bureaucrats whose role, under the NSW Environment Protection Act, is to enforce the law to protect the environment and the public?

Workers exposed to carcinogenic beta-naphthylamine in coal fumes at iron and steel works have a high risk of bladder cancer. Why is the risk, known 60 years ago, still present? Clusters of breast cancer and colon cancer are known to exist in certain other industries but the known hazards continue to be ignored. Why?

Research confirms that up to a fifth of lung cancer deaths are attributed to exposure to carcinogen-laden fine particles in the exhaust of vehicles powered by petrol or diesel. Why do such scientific claims inspire a flush of indignation among the anti-tobacco lobbyists? Is it a threat to passive-smoking litigation? Why is there corporate resistance to replace dwindling toxic fossil fuels with cleaner alternatives? Why are Sydney's traffic tunnels not installed with proven in-tunnel filtration systems to minimise known risks to human health?

Children living near petrol service stations have a proven, greater risk of certain leukaemias because of exposure to higher levels of benzene - a known cause of certain leukaemias. Where is the voice of the NSW Cancer Council and others in the cancer industry? Why, only recently, did Australia introduce an air-quality standard for benzene that is three times less stringent than that set for years in the UK? Why is there already a standard in the UK for 1,3-butadiene, a known carcinogen in petrol-powered vehicles, but Australia has no such standard yet? Over 285 tonnes of 1,3-butadiene and 2,850 tonnes of benzene are blown into Sydney's air-shed per year. Cost to taxpayers of health impacts due to exposure to vehicle exhaust in Sydney alone exceeds $2-3 billion annually. Yet, NSW Government switched its new bus fleet from clean LPG to dirty diesel.

The prevailing system is allowed to produce disease because political, economic, regulatory and ideological norms prioritize values of wealth and profit over human health and environmental well-being. Science is an integral part of this system but there continues a substantial tradition of manipulation of evidence, data, and analysis, ultimately designed to maintain favourable conditions for industry at both material and political levels.

Such is exemplified by the flawed NSW Health's study of health impacts on residents exposed to the toxic plumes from the M5 East stack and the illegal discharge from the tunnel's entries/exits. NSW Health's conclusion of "no health impacts" has been condemned by independent medical and scientific experts unwilling to compromise scientific probity. Why has NSW Health still not yet withdrawn its flawed report? Because it is politically damaging to do so! Rather than admit error, their lies continue to hoodwink the unsuspecting public. Worse, it helps protect the RTA, DEC, Planning, Health and the corporate stakeholders from litigation.

Such scandals are aided by the RTA Public Relations (PR) Unit out of which fictionalised reports have corrupted science about proven overseas tunnel filtration. Their cover-ups of the ongoing ventilation debacles of the M5 East tunnel are disgraceful. The role of the RTA PR Unit seems to be about ministerial patronage and 'keeping the lid' on scandals by denial and by corrupting damning evidence for corporate and political expediency. A system that changes the thinking of the public through media 'spin' and propaganda is about autocracy, not democracy!

Tabled internal documents disclose commissioned reports were narrowed by the RTA and its PR Unit to ensure the authors did not look at issues with potential to embarrass the RTA. This PR Unit has done immense damage to the credibility of the RTA and the NSW Government. However, truth does ultimately prevail, helped by privilege being denied on tabled internal documents. Bureaucratic corruption of the truth and a failure to admit error are costing taxpayers multiple millions of dollars.

The problem of corporate corruption of science is systemic at all levels. Startling published evidence confirms this 'underlying structure of harm'. Several features of the system include an unsustainable emphasis on growth, the failure of business competition, and the social and political power of corporations. The current economic and political system privileges corporate players and actually provides incentives for production of injury and disease rather than prevention i.e., the trade-off for 'economic growth' where profit is generated by shifting costs of health impacts to the taxpayer. Thus a corporation tends to be more profitable to the extent it can make other people pay for the bills for its impact on society.

Experts now concede the National Standards of Air Quality weight measures for particulates are inappropriate for assessing health risk. The NSW Regulatory Authorities have knowingly abused these inappropriate standards by applying them, contrary to the Guidelines, to a point source (tunnel stack) of pollution, thereby further corrupting the already flawed science. Why?

We have consistently observed, as members of the Lane Cove Tunnel Action Group Inc (LCTAG), that when favoured consultants are commissioned by either of these NSW regulatory/statutory authorities, it is commonplace that the extent science is carried out by and for the bureaucrats and for corporations, it becomes subject to the corporate logic of profit maximization. Thus, the imperative to reduce costs means keeping wages low, minimizing investment in environmental-friendly technologies, resisting regulation by the State and failing to implement "voluntary" safety and health standards e.g., not installing proven filtration technology in the tunnels.

Science is important to corporations, public health professionals and the public. Corporations have much at stake when their 'product' such as the ventilation system of a tunnel is put to scientific test. Numerous examples document how corporations and the NSW bureaucracies influence science and the effects of that influence on environmental and occupational health.

Released 'privileged' documents disclosed that in correspondence dated 10 May, 2004, the Operators or 'Joint Venture' of the M5 East lodged a Claim for $14 million (taxpayer's money) against the RTA. Documents also revealed the Operators sought a 'Change' as a consequence of the actual traffic being greater than traffic projections by the RTA. The Supplementary M5 EIS predicted 58,200 vehicles per day (vpd) would use the M5 tunnel in 2001 and 69,000 vpd in 2011. The traffic volumes in the M5 now exceed 110,000 vpd. The ventilation system was designed for 50% fewer traffic and so cannot cope with the pollution generated.

Health Science is now being corrupted by the NSW RTA and NSW Planning (DIPNR) by changing the Ministers M5 East's Conditions of Approval (MCoA) that forbids tunnel portal (entry/exit) emissions, except in an emergency. This shameful and unconscionable decision by Roads Minister Eric Roozendaal and Planning Minister Frank Sartor is about appeasing the Operator. Thus, to avert further Claims of compensation, proposed changes to the MCoA will allow the discharge of toxic pollution through the portals (as well as from the stack) into the residential precinct. The fault falls squarely on the RTA for not admitting error! The same is predicted for the Lane Cove Tunnel where RTA traffic projections are 50% fewer than in the LCT Company's Base Case Financial Model.

Former Minister for 'Cancer and Medical Science,' the Hon Frank Sartor MP (now Planning Minister) has the answer to cancer – blow it in the wind!
LCTAG condemns the NSW Government and its bureaucracy for continuing to persistently trash health science, community rights and entitlements.

The corporate corruption of science is not only widespread, with a long history, but is a real threat to the health and well-being of people and the environment. Such a problem deserves a concerted response from the electorate and also on the part of conscientious public health researchers.

Dr Ray Kearney
Chairman,
Lane Cove Tunnel Action Group Inc August, 2006

3. Urgent – Begin Replacing Fossil Fuels with Cleaner Alternatives!

Results published in the March edition of the *Journal of the American Medical Association* (2006) volume 295, p 1127-34 again confirmed the proven health impacts associated with exposure to fine particulate matter (PM) of which most is derived from the tailpipe of vehicles powered mainly by petrol and diesel. The *JAMA* article concluded that short-term exposure to PM of less than or equal to 2.5 μm in aerodynamic diameter ($PM_{2.5}$) increases the risk for hospital admission for cardiovascular and respiratory diseases.

While the underlying mechanisms are not yet fully understood, compelling evidence indicates that these health impacts are related to $PM_{2.5}$ - induced injury to the lung tissue. The associated inflammatory response that generates mediators of blood-clotting can cause blockage of coronary arteries already narrowed by atherosclerosis in at-risk elderly persons. The section of heart muscle starved of oxygen becomes non-functional precipitating 'heart failure'.

In March, 2002, the *JAMA* reported a 16-year study involving half-a-million Americans showed up to a fifth of lung cancer deaths was largely attributed to exposure to $PM_{2.5}$ vehicle pollution.

Unlike coarse PM_{10} particles, the fine $PM_{2.5}$ have on their surface condensates of carcinogenic products that dissolve in the airways, especially in the lung gas-exchange units called alveoli. By weight, a billion 0.01μm particles are equivalent to ONE coarse PM_{10} particle but have 1000x times the surface area of a PM_{10} and hence are far more toxic.

In Sydney, TWICE as many people die from air pollution than from road accidents. The total cost of morbidity and mortality due to vehicle pollution in Sydney alone is between $2-3 billion annually.

However, Australia only has a 'reporting' National Standard for $PM_{2.5}$ that is meant not to exceed 25μg/m^3 over 24 hours or an annual average of 8μg/m^3. Like most other Australian capital cities, Sydney's annual average of $PM_{2.5}$ is already exceeding the National Standard and is increasing. To date, this air-quality standard is not a 'compliant' standard and so there is no attempt of enforcement by the NSW DEC and RTA who know full well that the two stacks of the unfiltered Lane Cove Tunnel (LCT) will exhaust large amounts of toxic fine $PM_{2.5}$ pollution into an atmosphere already exceeding a prescribed safe limit. The levels of $PM_{2.5}$ in the M5 East tunnel are over five-times the national standard for background air quality.

At both Federal and State levels, under the respective 'Clean Air Act', the Environment 'Protection' Authority is required to set a particulate matter Ambient Air Quality Standard that protects public health with an "adequate margin of safety." Published findings now confirm an ongoing threat to the health of our community at all ages from the fetus to the at-risk elderly. Yet, politicians and their bureaucrats on all sides, at all levels, seem utterly seduced by the oil cartels to do anything to address this unconscionable situation. It is yet another example of certain corporate stakeholders augmenting their wealth and profitability by shifting health costs to the community and the environment. Treasury adopts principles of 'economic rationalism' where excise income exceeds sickness costs incurred, as for tobacco. But where is the morality?

Communities are further disadvantaged by e.g., the assertions in Parliament by Premier Morris Iemma who has allegedly mislead the NSW Legislative Assembly with his outrageous claim that filtration would cost $1 billion and he further hoodwinked the Assembly by a fictitious claim that "such filtration has no impact on the air quality inside the tunnel" (Hansard, March '06).

On the broader issue, Australia is a country which *Geosciences Australia* noted has a consumption of crude oil and condensate in 2004 that could be sustained by the remaining economic reserves for only 9.3 years. Global production of crude oil in 2005 was approx. 29 billion annually and the continued production at this level, based on total global reserves of one trillion barrels equate to about 35 years supply. Thus crude petroleum is a finite resource, and global reserves are limited. Already demand is outstripping supply.

Coupled with this is the problem of spare capacity. In crude oil production terms, spare capacity is pumping capacity that is currently unused but can be turned on immediately if needed in a crisis. The days of spare capacity in the global crude petroleum industry are gone. What this means is that should global crises arise (natural disasters e.g., Katrina hurricane, or wars in oil-producing countries such as Iraq and Iran) supply will barely if at all keep up with rising demand.

It is highly significant that the USA war on Iraq was launched on the allegation that Iraq had 'weapons of mass destruction', yet none was found. Similarly, the current posturing by USA toward Iran is based on a pretext that Iran has or is planning to develop 'nuclear weapons'. But no war is ever prompted by one factor alone. Oil played a role in USA's decision to invade Iraq. Iran has immense oil and natural gas reserves and will have a critical role in the world's future energy equation. However, major USA energy companies are prohibited from working with Iran by Executive Order, signed by President Clinton in 1995 and renewed by President Bush in 2004.

The truth is the current government of Iran has plans (as did former President Saddam Hussein of Iraq) to set up an OIL BOURSE i.e., an oil trading market based on the petroeuro rather than the current petrodollar. This poses a threat to USA's economic supremacy and to London's International Petroleum Exchange and Nymex in New York. Current membership of the proposed Iran Oil Bourse includes Russia, China and India. Iran has plans to open the Oil Bourse in 2006.

Tehran has the only military in the region that can threaten its neighbours (including Israel) and Gulf security. Thus, while publicly focusing on Iran's 'weapons of mass destruction', powerful western governments, led by USA, are thinking in geopolitical terms about Iran's role in the global energy equation and its capacity to obstruct the global flow of petroleum e.g., through the Strait of Hormuz. However, the economic threat of an Oil Bourse to the global economic supremacy of the USA is clearly the principal reason the USA is now likely to attack Iran, presumably via an 'anti-nuclear' strike by Israel.

The implications for Australia are simple, and alarming. As a country very highly dependent on road transport for the movement of both goods and people, and as a country currently committed almost entirely to the use of products derived from crude oil to fuel that transport, Australia is in a position of great vulnerability. This is exacerbated by the fact that Australia's indigenous reserves of crude petroleum are lower than the global average, and the rundown of Australia's oil reserves is occurring at a faster rate than the global average.

Clearly, in terms of escalating fuel costs, unmanageable constraints in fuel supply and unaffordable sickness-care costs attributed to toxic fossil fuel pollutants, Australia needs an urgent shift in demand from conventional fuels such as petrol and diesel to more sustainable cleaner alternatives.

The Australian government is urged to enhance incentives for increased alternative fuels, perhaps at least 20% by 2020, including natural gas and biofuels such as ethanol blends and biodiesel. Also needed is a policy requiring that a nominated percentage of Australian transport be powered by fuels other than petrol and diesel. It is economic folly to export our clean natural gas to China at 5c a cubic metre (equivalent in energy to one litre of diesel) and import overseas crude oil in excess of $A80 a barrel.

Australia has enormous capacity and now the perfect conditions to produce a range of other viable cleaner alternatives to petrol and diesel, not only to address our vulnerability but also to respond to an increasingly stressed global air quality, its associated impacts on climate and our national health budget.

Dr Ray Kearney May, 2006

Chairman, LCTAG Inc

4. Fossil Fuel Exhaust - The New 'Asbestos'

The major political parties at Federal and State levels, the oil companies and car manufacturers have known that whilst 'leaded' petrol is injurious to health, 'unleaded' petrol has even greater toxic properties.

The first internal combustion engines ran on 'motor spirit' or alcohol - an exceptionally clean fuel. As vehicle manufacturers strove to produce faster, more powerful cars, dirty fuels replaced clean fuels and later became even more toxic. The oil-companies' solution to stop the fuel pre-igniting and reduce the 'ping' under compression in engines designed for more power was to add cheap lead.

Very dirty fuels, however, produced a 'smog' problem that worsened due to the vastly increased toxins including sulphur/nitrogen oxides and poisonous particles. Rather than replace fossil fuel with a cleaner product, oil companies introduced the 'catalytic converter' that allowed exhaust gases to pass over a catalyst e.g., platinum, in the 'filter'. Lead in fuel, however, rendered the 'converter' useless. Hence the change from 'leaded' to 'unleaded' fuel. So, what about the 'pinging' problem? The alternatives to lead were **even more deadly**! Catalytic converters became law – thus allowing the oil companies to continue with production of 'dirty' fossil fuels.

Twenty years ago, the NSW Health Department knew that lead was being replaced with more toxic chemicals such as benzene, di-methyl benzene, toluene and xylene. Benzene is known to be associated with many cancers, particularly leukaemia. A decision was made to cut lead and **knowingly introduce substances that would increase cancer rates.** Sound familiar? The cover-up continues to this day, at all levels.

Modern car engines generate smaller exhaust particles in vastly greater numbers with immense surface areas, on a weight for weight basis. The technology created a highly efficient system that delivers toxins deep into our lungs where the very fine particles can dissolve to unload their cancer-causing cargo. Not surprising, a 16-year study, involving 500,000 Americans (*The Journal of the American Medical Association* 2002, vol 287, p 1132), found that up to a fifth of lung cancer deaths was attributed to exposure to fine particles, mainly from vehicle exhaust. See also:

http://www.newscientist.com/hottopics/pollution/pollution.jsp?id=23331100

In Australia, regular and premium unleaded petrol have high benzene levels of 2-3.3%, respectively, with increased tail pipe emissions of additional carcinogens, in gas and particle fractions. Britain has adopted a national standard for benzene of 1 part per billion (ppb). Australia has no such standard.

In NSW we have been subjected to a disinformation campaign by Government and the bureaucrats in the form of a seductively simple – no lead, no worries.

A UK study (*J Epidemiol Community Health* 1997, vol 51, p151) looked at 24,458 children dying of leukaemia and cancer in the UK over a 25 year period. It found that these children were **35% more likely than chance** to have lived **within 4 km of a major motorway**.

More than 25 ppb of benzene are emitted from the M5 East tunnel stack. Research has recommended that if the life-time risk of leukaemia is to be reduced to one in a million, compared to 10,000 at present (and increasing) then benzene levels have to be cut by 50-100 fold.

In a tabled document, released under Parliamentary Orders, the NSW EPA states, with respect to the Lane Cove Tunnel (LCT) Air Quality Impact Assessment of stack emissions:

" *While the predicted carcinogenic health risks associated with exposure to all pollutants are between **2 and 4** times higher than 1 x 10^6 at ground level, they are up to approximately **10 times higher** at elevated receptors* (individuals) *located at 40 metres above ground-level*

The incremental increase in carcinogenic health risks associated with exposure to various air toxic compounds has been predicted to be above 1 x 10^6. This level is commonly used within the EPA as a trigger for consideration about possible options for reducing the levels of pollutants predicted through the use of health risk assessment." (NSW EPA 26.6.2002)

This startling information, hidden from the community, is confirmed in another internal paper from the NSW EPA:
"The incremental increase in health risk due to tunnel vent emission exceeds the EPA's benchmark values for ALL the stack parameters assessed for the recommended locations at Marden St. and Sirius Road.

If the predicted increase in cancer risk is greater than 1 per million, the EPA has the installation of abatement measures to reduce the predicted cancer risk to below 1 per million. The risk assessment presented in the additional information predicts an increase in cancer risk to greater than 1 per million.

The argument presented by Holmes (Holmes Air Sciences) *that the risk is a small percentage of background levels is not relevant. The EPA requires all new point sources of air toxics to achieve the same benchmark level..."* (NSW EPA 24.7.02)

Typically, the RTA engaged their regular client who then incorporated a 'factor' that effectively reduced the cancer risk to 'insignificance' (NSW RTA 9.9.02). The RTA no doubt found this untested 'factor' most convenient.

The RTA and the LCT Company (LCTC) have proceeded to make major modifications to the tunnel ventilation that predicts greater levels (acknowledged by the RTA) of stack pollution, but without a proper and independent health-risk assessment, previously highly critical.

An obligation of due diligence applies to the Regulatory Authorities (RTA, EPA, Health and DIPNR) and to the LCTC. This obligation is to implement proven measures to clean and detoxify the polluted tunnel airstream consistent with the Precautionary Principles. The in-tunnel installations of electrostatic precipitators and denitrification systems are now confirmed effective by the RTA and Roads Minister Carl Scully MP.

The poisonous effects of vehicle emissions are no longer a matter of debate in the scientific and medical communities. It is incumbent on the LCTC, its constituent groups and its officers to understand that these emissions have the potential to harm.

LCTAG believes that Leighton Holdings Ltd, Thiess Pty Ltd and John Holland Pty Ltd have, to date, not exercised 'due diligence'. They intend, knowingly and wilfully to discharge toxic waste from the Lane Cove tunnel stacks in a manner likely to cause harm to the environment, motorists, residents, workers and in particular, those people most at risk because of their proximity to the stacks and/or their existing health status.

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The New South Wales Court of Appeal recently dismissed the appeal of a particular employer and relied on the High Court's statement in Southern Shire Council v. Heyman (1985) that: *"when there is a duty to take a precaution against damage occurring to others... breach of the duty may be regarded as materially causing or materially contributing to that damage, should it occur, subject of course to the question of whether performance of the duty would have averted the harm."*

The difficulty LCTAG has with the RTA, NSW Health and the other bureaucrats and certain of their clients is that their conduct sometimes obscures rather than elucidates the truth. The end result is a public denial of liability and the cancer, my friend, is blowing in the wind!

LCTAG appeals to the Board of the LCTC to demonstrate a duty of care to our community. YOU should seize the opportunity to demand Roads Minister Scully and the RTA permit a re-design of the ventilation systems in the LCT to incorporate world's best filtration technology so that this project becomes another Australian landmark, along with the M5 East and Cross City Tunnels that should be similarly filtered, as a gold standard in the application of such proven technologies.

Dr Ray Kearney
Chairman,
Lane Cove Tunnel Action Group Inc

Sept 2004

5. Will NSW Health Withdraw Sham M5 East Study?

Following numerous complaints, NSWHealth initiated Phase1 of a 3 phase investigation in 2003 into illnesses of residents near the M5 East tunnel stack at Turella. Under a Parliamentary Order, a declassified 'privileged' internal NSWHealth report disclosed: *"Experienced allergy, respiratory medicine and occupational medicine physicians, identified a substantial number of subjects who complained of eye, nose and throat discomfort. In some cases these symptoms were accompanied by headache. The time course of the symptoms, and consistency among subjects, convinced the attending physicians that there was prima facie evidence of adverse health effects related to the vent stack".* NSWHealth meanwhile released a different report claiming the findings were inconclusive.

The internally-managed NSWHealth Phase 2 study involved a random telephone survey asking respondents to comment on their health for the previous 4 weeks. The report was released on the 1st April, 2004. Without any form of actual pollution-exposure measurement, NSWHealth 'found' **no evidence of health effects due to the stack, and concluded there was no need to do any further investigation of any kind.** Confirmation of a *prima facia* cause and effect relationship between the reported illnesses and exposure to stack pollution would have profound implications, legally, politically and in terms of NSWHealth's own credibility.

The RTA's propaganda machine was energised by the NSWHealth's 'findings' and by Roads Minister Carl Scully's claim of 'a clean bill of health' for the M5 East stack. Quick to exploit the 'findings' and to justify, it seemed, the secret $40 million, cost-cutting ventilation changes to its tunnel contract, the Lane Cove Tunnel Company boasted: ***"There will be no health impacts from the unfiltered stack emissions."*** But how valid are NSWHealth's 'findings' and do they apply to other tunnels? Did the health study have scientific rigour, or was it rigged?

To test the validity of the NSWHealth Phase 2 findings, Lane Cove Council commissioned Dr Peter Best of Katestone Environmental to coordinate reputable experts to independently review the NSWHealth Study. The Reviewers - Professor Michael Moore (Q'ld) - a Medical Environmental Toxicologist, Professor David Fox (Vic) - a Chartered Statistician in Environmetrics, and Dr Peter Best (Q'ld) - Air Quality Science Consultant, all concluded that as the whole Phase 2 Study was so fundamentally flawed, its findings into the M5 East Health Effects **should not be accepted and recommended that the Phase 2 Study be withdrawn or substantially revised.**

The Reviewers found the internally designed and managed study was scientifically and methodologically deficient, being based upon numerous unproven assumptions that are either largely unfounded or misleading. Impartiality seemed compromised by Steering Committee members being also among the principal investigators.

NSWHealth engaged an overseas expert, Professor Bert Brunekreef, for comments on their proposal, submitted without full details. The study began before resolving issues raised by Prof Brunekreef. The Reviewers found it "odd" that only three of his 18 recommendations were adopted, and he was not asked to carry out any further independent analysis of the findings. The four weeks of self-reported symptoms, **without any form of actual measurement of pollution exposure**, were found highly restrictive and where no causal relationship was or could be established because of the study design. Control groups of residents, unexposed to stack emissions, were absent.

All the experts pointed out those self-reported symptoms are more related to acute exposure, while annual averages are more relevant to chronic health impacts. Yet, NSWHealth used annual averages in modeled pollution, from the previous year, rather than the actual exposures of the people studied. The experts asserted that these cannot be a substitute of acute exposure to temporary high levels of peak-hour stack pollution. Professor Brunekreef stated *"...it seems mandatory to estimate the contribution of the stack emissions to air pollution exposures for each individual for the four weeks before administration of the questionnaire."* Such advice was ignored by the NSWHealth and is a major and serious flaw.

The sample size was too low, by at least 5-fold, to obtain prescribed statistical significance. The experts could not establish or assess whether the 'weightings' applied by the NSWHealth to the unsatisfactory questionnaire were appropriate to adjust for bias.

The Phase 2 study depended on a comparison of symptoms between 3 zones of impacts from low to high levels of exposure to stack pollution, assuming no portal emissions. Portal emissions (ignored by NSWHealth) occurred almost daily, in breach of the Minister's Conditions of Approval, exposed some residents in the 'low' impact zone to high levels of pollution, thus invalidating any comparison with 'high' pollution residential zones.

The experts considered it "bizarre" that the NSWHealth excluded children and infants i.e., a high-risk group, from the study and believed the omission together with a strange predominance of older women may have biased the research to a 'no effect' outcome.

Odour diaries from a number of residents taken after the M5 East opened were under-used. The exclusion of a respiratory objective from the Phase 2 study was judged also to be "weird" and not "compliant" with the intent and recommendations of the Phase 1 Study. That asthma was conspicuously and oddly "de-emphasised" was found "extraordinary" when such expertise existed in the NSWHealth research team and in the Steering Committee. The NSWHealth study, however, did find a high proportion of health impacts e.g., 64% of all participants reported sore eyes, 66% reported nasal problems and 33% throat problems, but the NSWHealth failed to offer any explanation. Why? The specialist physicians who examined the affected residents in the Phase 1 study were convinced *"...that there was prima facie evidence of adverse health effects related to the vent stack."*

The experts determined the self-congratulatory quality statement by the NSWHealth of *"... the best feasible epidemiological approach"* was not justified. Rather, they found the objectives of Phase 2 were unrelated to or "disconnected" from the recommendations of the Phase 1 study to test causation between reported illness and stack emissions. They also strongly contested the relevance of the NSWHealth's findings to other tunnel situations. The Review recommended that an independently designed and managed study as well as a reanalysis of the collected data is justified.

In conventional scientific/medical research, flawed publications are normally withdrawn, amended or rejected. To date, NSWHealth has continued to justify the M5 Health Study, ignoring the fundamental flaws with a 'let it pass' mindset. It is hoped that the Katestone Critique will be heeded by the NSWHealth and the respective Ministers and take seriously community health impacts from the concentrated stack pollutants. The resultant faulty thinking and careless practices cannot continue to be perpetuated, backed by delegated legislative authority. This is fundamentally wrong, ethically and scientifically.

Put squarely, it can be inferred that the NSWHealth's Phase 2 Study was specifically designed to be undertaken with major political imperatives and self-interest in mind in an attempt to disassociate health impacts from the Government's own polluting of the environment and produce a pre-determined outcome to support a prevailing political agenda. Pseudo-science and patronage appear its key elements. Yes, whilst the statistical formula applied may have been appropriate, if the data is itself in error, then fundamentally so is the analysis. NSWHealth and its Minister were previously made aware of such failures but made no attempt to correct them.

Despite acknowledging that Phase 3 of the study was necessary, NSWHealth deliberately used its internal unscientific findings to disband any suggestion of completing Phase 3. The Reviewers strongly contend that not only should Phase 2 be redone but that Phase 3 is completed as a matter of urgency.

The NSWHealth's Phase 2 study is indeed a sham and is another example of a Statutory Authority acting as a servant of this Government rather than as a servant of the public. The critical omissions, coupled with numerous unfounded and misleading assumptions, raise questions about the competence of NSWHealth. In reality they are saying *'because we, through a flawed study have not found an effect, none exists.'* This is inconsistent with the NSWHealth's stated priority of ensuring that *'risks to health of the community are reduced through effective vigilance, surveillance, and responsiveness'* (Corporate Plan 2003-2005). Further, the Precautionary Principle has not been applied in this case, despite *prima facie* evidence of cause and effect with a documented high proportion of adverse health impacts. No attempt was made by the NSWHealth to explain the latter.

The conclusions by the NSWHealth of its M5 East Health Study cannot be accepted. The proper action is for NSWHealth to withdraw its findings publicly, forthwith. The Reviewers asserted that properly conducted Phase 2 and 3 studies should be implemented immediately and independently managed to obtain credible evidence to determine any causal associations of documented health impacts from the M5 East road tunnel stack emissions.

By the time this new study is done, Sydney will be circled by a toxic ring of tunnels, with the onus on victims to prove they are being poisoned. However, these are real people suffering daily. We are not talking about a lab experiment. The Lane Cove Tunnel Action Group Inc believes that in the light of all the overwhelming evidence about ill effects of unfiltered tunnels, it is time to act NOW. Fix the M5 East debacle and ensure Lane Cove does not suffer the same deal.

In view of the rejection by the Audit Office to extend an M5-East Performance Audit, an ICAC inquiry seems warranted to test the probity of the process that was adopted in the NSWHealth Study, and more generally by the government agencies to assess and manage the health risks of unfiltered tunnels to residents and tunnel users.

A copy of the Katestone Critique can be found at: *http://www.lanecove.nsw.gov.au/*

Dr Ray Kearney
Chairman, Lane Cove Tunnel Action Group Inc.
December, 2004

6. Ethics and Skeptics: The Lingering Threat of Fossil Fuel

The relationship between air pollution, death and disease has been studied for decades, leading to the recent conclusion that combustion of diesel and petrol is among the most toxic sources of emissions today (USA Clean Air Task Force Report, Feb. '05) viewed at the link: http://www.catf.us/publications/view.php?id=83

These exhausts contain numerous dangerous compounds, ranging from respiratory irritants to carcinogens including a host of air toxics, particulate matter, carbon monoxide and nitrogen oxides.

The very fine particles adsorb toxic gases and liquids onto their surfaces. On a weight basis, a billion ultra-fine particles are about equivalent to one coarse particle 10 micrometres in diameter, but have 1000 times the surface area. The fine particles are mainly soluble and penetrate deep into the lungs. Health research indicates that the invisible exhaust may be the most dangerous of all. Technology exists right now to clean up emissions from these engines and to remove such toxics from road tunnels. The only thing that stands between us and dramatically healthy air is the political will to require these reductions.

NSW Health Minister Morris Iemma knows that in Sydney, twice as many people die from exposure to vehicle pollution than from road accidents. Yet, despite NSW Health's protestations and legal advice, as disclosed in tabled internal documents, RTA deliberately refuses to install signs warning motorists to close their windows on entering the toxic M5 East tunnel.

In the USA, fine particle pollution kills 21,000 people each year. Diesel exhaust poses a cancer risk that is 7.5 times higher than the combined risk from all other air toxics. The risk of lung cancer for people living in urban areas is three times that for those living in rural areas (CATF Report, Feb. '05). Air pollution was not on the agenda of the Carr Government in deciding to power its new bus fleet with toxic diesel that replaced clean LPG.

Children are more susceptible than adults (except the elderly) to the adverse effects of air pollution because:

- Children are more active and breathe more rapidly.
- They have more lung surface area compared to their body weight and inhale more air kgm-for-kgm than adults.
- They have higher lung volume to body size, higher respiration rates and spend more active time in the polluted outdoor environment.
- When exposed to fine particles, children have slowed lung function growth, increased emergency room visits, increased incidence of asthma, bronchitis and crib death (CATF Report, Feb. '05).

Lane Cove Tunnel Action Group Inc. (LCTAG) is justifiably concerned about health effects from exposure to toxic unfiltered stack exhausts of the 3.7 km Lane Cove Tunnel (LCT). These concerns are heightened by:

- Failure of NSW Health to apply the recommendations of an overseas expert who identified numerous serious flaws, confirmed by three independent experts, in the design and methodology of the study into the health impacts of residents exposed to M5 East stack emissions. Tabled documents reveal NSW Health already knew in September, 2004 that their study required re-analysis, but continued to mislead both the Minister and the community. LCTAG believes NSW Health's insistence that their study was scientifically robust especially when frequent portal emissions corrupted the data and a failure to heed such criticisms are tantamount to scientific misconduct.
- Failure by the builders of the LCT, Thiess John Holland (TJH), to disclose the actual traffic numbers on which the modeling of stack pollution is said to be "compliant." Experience with the Gore Hill Freeway, LCT and M5 East projects, as documented by LC Council, confirms RTA's projected traffic volumes are greatly underestimated. A skeptic might think that claims of "compliance" are based on secretly 'chosen' traffic numbers to ensure compliance would be met fictitiously by 'filtering the facts'. Such would weaken the case for tunnel filtration. TJH's claim to 'commercial in-confidence' is unsustainable. On expert advice, LCTAG had agitated successfully to change RTA's original design of the LCT, from a twin two-lane to a three-lane tunnel.
- Failure of TJH to extend the pollution modeling of the stacks to include accurate background levels of pollution provides misleading health risks. Documents obtained by a Parliamentary Order disclose that the levels of pollution arising from traffic volumes on the Gore Hill Freeway alone, in the vicinity of the proposed LCT, will be at levels exceeding the National Standard. However, TJH continues to exclude relevant background levels in modeling pollution discharged from the stacks including benzene from the large Shell Service Complex adjacent to the western stack.
- In all formal community liaison/consultative meetings, to date, under the auspices of TJH and the RTA, there has been no representation by NSW Health. An embargo has been imposed on the Air Quality Community Consultative Committee not to discuss health impacts officially.
- LCTAG believes the proportional increase in privileged documents in a recent parliamentary call for internal papers is a reflection of the desire for the bureaucrats to keep their 'business' secret. It was a declassified report that disclosed in 2004 that medical specialists concluded from a Phase 1 Study there was *prima facie* evidence the illnesses reported by M5 East residents were causally related to stack emissions. Shocked by this unplanned public disclosure, NSW Health designed a Phase 2 study, proven so defective and methodologically flawed, that the predictable 'findings' would show no causal relationship.
- Tabled internal papers disclose NSW DEC (formerly EPA) protested strongly against the RTA for not notifying the DEC of a secret arrangement with the LCT company to eliminate the $40 million 1600 metre ventilation shaft in the design of the LCT approved by the Minister. DEC expressed dissatisfaction with the RTA's explanation that such changes were minor.

Community residents have a number of expectations of NSW Health, RTA, DEC, DIPNR and the LCT Consortium. We expect these agents will:

- Understand the public's point of view and that our concerns will be the agency's top priority.
- Ensure these concerns will be scientifically investigated, researched, studied, documented and addressed.
- Explain or find the reason for the illnesses of M5 East community residents and prevent further exposures as well as to learn from the agency's own mistakes.
- Adopt proper and effective enforcement of the Minister's Conditions of Approval and not to corrupt enforcement by delegating that responsibility to the RTA.
- Maintain a permanent documentation database of records and information.
- Validate the concerns of the residents.
- Implement all these duties in a timely manner.

LCTAG's expectations, however, have been dashed by bureaucratic dishonesty, incompetence, indifference and tardiness in each of the respective departmental portfolios. Concealment of data, overlooking published evidence, or the deliberate termination of studies at a stage where findings were suggestive have caused LCTAG to be mistrustful of government officials and suspicious of the activities they conduct with corporate clients.

Why then is the 'Precautionary Approach' not taken? Because the risk-based approach to public health is adopted instead i.e., wait until the dead bodies can be counted. Whilst diesel fumes are a known cause of lung cancer, health bureaucrats state they are "not yet sure" how big the problem is and "we have not identified the extent of the problem".

This is the classic risk-based approach. Ignore the evidence so long as it is not 100% watertight. Use uncertainty as an excuse to delay. Wait for the dead bodies to pile and then slowly acknowledge the need for action. Remember asbestosis? Precaution is not (yet) fashionable while risk-assessment is!

The risk-based approach to unfiltered tunnels, as it also is for diesel and petrol, is to adopt the principle 'business as usual'. This has the backing of powerful special-interest corporate groups harnessing governments to deflect and stymie the search for least harmful alternatives. So long as the exact size of the problem is uncertain, risk-assessors call for delay and more study. It is now clearly evident that RTA's insidious delaying tactic of implementing a 'filtration trial', as endorsed by former Roads Minister Scully, is to buy time politically as the tunnel projects continue. Tabled internal documents show because consultants can be 'bought' or 'hired' to reinterpret old data to cast doubt on the nature of a problem, action can be stalled for decades. Maybe DEC will be more forceful in going public with its concerns about the trial estimated to cost tens of millions of dollars and their reluctance to be part of the review panel. They know the trial is a waste of time and is under-rated to about 10% of the airflows in the LCT.

Doubt is a powerful helpmate when your goal is to maintain 'business as usual' and typifies the current mindset especially of the Carr Government, the NSW RTA and NSW Health regarding air toxics and tunnel filtration. The risk-based approach waits for the holy grail of scientific certainty to emerge from the data. Then, alas, the NSW Government is likely to enact legislation to take away yet another of your 'rights' i.e., to litigate against sheer bureaucratic negligence.

Finally, Minister Costa, 5 months is just too long not to pick up a phone and talk to the Federal Government about its $10 million offer!

Dr Ray Kearney
Chairman, Lane Cove Tunnel Action Group Inc.,
April, 2005

7. Poisonous Plumes, Politics and Patronage

Unlimited and free access to clean air of acceptable quality is a fundamental human necessity and right.

The lung is a critical interface between the environment and the human body. An average person takes about 10 million breaths a year and about 16 cubic metres of air every 24 hours. The internal surface area of the airways in the five lobes of the human lung is about equivalent to that of a tennis court. Hence toxic substances in air can easily reach the lung and produce harmful effects locally and in other organs.

Adverse effects of exhaust pollutants now include increased infant mortality (*New Scientist* 3 July, 2004); chronic deficits in lung development of children aged 10-18years (*New England Journal of Medicine*, 9 September, 2004); acute heart attacks (*New England Journal of Medicine*, 20 October, 2004); and an association between ovarian cancer and exposure to diesel exhaust fumes (*International Journal of Cancer*, 20 August, 2004).

The World Health Organisation recently reported serious concern about the health effects of vehicle pollutants and of the polycyclic aromatic hydrocarbons (PAH's) which are cancer-causing and can coat fine exhaust particles or exist as vapours. (http://www.euro.who.int/document/E83080.pdf). Diesel exhaust is around 40 times more carcinogenic than cigarette smoke on a weight/volume basis (Gong and Waring, 1998). Up to a fifth of lung cancer deaths have been attributed to exposure to fine particles of vehicle exhausts. (http://www.newscientist.com/hottopics/pollution/pollution.jsp?id=23331100).

Researchers reported a compound, 3-nitrobenzathrone, found in diesel exhaust fumes may be the strongest carcinogen ever analysed and warn that it could be partly responsible for the large number of lung cancers in cities. It produced the highest score ever reported in an Ames test, a standard measure of the cancer-causing potential of toxic chemicals. (*New Scientist*, 25 October 1997). (NewScientist971025-p4.pdf)

A UK study (*J Epidemiol Community Health* 1997; 51:151-159) looked at 24,458 children dying of leukaemia and cancer in the UK over a 25 year period. It found that these children were **35% more likely than chance** to have lived **within 4 km of a major motorway**.

Twice as many people died in Sydney in 2000 from air pollution than from road accidents (*Australian Bureau of Regional Economics Report,* September, 2003). Yet, despite the irrefutable evidence of a worsening situation, the Minister for 'Cancer and Medical Science,' the Hon Frank Sartor MP has, to date, not ensured vehicle-exhaust pollution is incorporated in the cancer-prevention program of his much vaunted multi-million dollar tax-payer-funded 'NSW Cancer Institute'. As a former Lord Mayor and involved with the Cross City Tunnel project, he also seemed disempowered to take a positive stand against the discredited "filtration is a placebo" ranting of Roads Minister Scully.

Where is the evidence of former Road's Minister Scully's 'inner purity' when exhaust emissions of 100,000 vehicles per day are discharged from a stack(s) into precincts where residents, young and old, are wilfully exposed to pre-determined cancer-risks several-fold higher in poisonous plumes from an un-filtered tunnel?

Fine particles, unlike coarse ones, are **mainly soluble** in the lung and represent more than 85% of the particle content of exhaust emissions. In NSW, continuous monitoring of atmospheric particles is underestimated by up to 40% (Katestone Environmental Report, Lane Cove Council, April, 2003) because the NSW Department of Environment and Conservation neglects to incorporate correction factors for accuracy. In conventional research, data manipulation is deemed 'scientific misconduct'. Without accurate measurements of the pollution levels it is not possible to determine the real health risks or to detect exceedances of the air-quality standards. Such tolerated abuses are well documented and seem exploited by the NSW RTA in managing the M5 East and Lane Cove Tunnel air-quality studies. It appears so much easier to establish 'compliance' when monitoring data are underestimated and skewed. This outrage is compounded when NSW Health incorporates such data into its 'internally managed' determinations of "no health-impact" studies based mainly on a pre-fabricated patchwork of guesses.

The *unfiltered* M5 East tunnel is a proven ventilation debacle. Evidence has emerged of a litany of breaches of the Ministers Conditions of Approval (MCoA), blatant cover-ups of illegal venting of emissions from the tunnel entrances (portals), allegedly known to the RTA and the tunnel operators. Such was disclosed in internal papers recently tabled by Parliamentary Order. Analysis of the tabled data revealed illegal venting occurred daily, mostly during peak hours stealthily, for almost a year in breach of the MCoA. The ventilation system is so defective and inefficient <u>without in-tunnel filtration</u> that the operators now close the tunnel for health and safety as exhausting from the tunnel portals is not an option, except in emergencies. Minister Scully, where is your 'duty of care' - or are YOU part of the problem?

The Report, by Child and Associates, into international developments in tunnel emission treatment systems has now been released by the RTA. Claims by the RTA that the Child Report supports RTA's 'Filtration Trial' are spurious because the first version of the 'independent' Report was completed before Minister Scully announced a 'Filtration Trial' in March, 2004. LCTAG is concerned that the subsequent April and September versions of the Child Report appear to have been subjected to interference by the RTA with major amendments imprinted. Why was the author prevented by the RTA from addressing issues such as the applicability of filtration systems to the M5 East, cost effectiveness and making recommendations, despite the MCoA of the M5 East requiring him to do so? Papers tabled by Parliamentary Order show the scope of the Child Report was narrowed by the RTA to ensure the author did not look at such issues with potential to embarrass the RTA. Detailed information in the April-version that contradicted RTA's misleading report about their visit to Japan was expunged in the final September version. As the master of his RTA servants, Minister Scully must take full responsibility for such anomalous conduct.

RTA's General Manager of Motorways, Gary Humphrey recently had the temerity to assert: *"Filtration will not be installed in the Lane Cove tunnel because air quality standards will be met".* Mr Humphrey's typically overblown comment engenders absolutely no confidence against a background of RTA's appalling track record of misleading information and tardy reporting, subject to critical attack recently by the Parliamentary Staysafe Committee. Yet, Roads Minister Scully could announce publicly in March, 2004, to his colleague - the Hon Angela D'Amori MP, Member for Drummoyne - "If the M4 tunnel is built, **it will have filtration**." Such a proclamation, before he announced RTA's 'Filtration Trial' appears to carry a strong whiff of political patronage coupled with a glint of ministerial 'inner purity', or is it just more RTA propaganda at play?

The Lane Cove Tunnel Action Group Inc (LCTAG) believes that an obligation of due diligence applies both to the Regulatory Authorities and also to the Lane Cove Tunnel Company (LCTC). This obligation is to implement proven measures to clean and detoxify the highly polluted tunnel airstream. Such measures would be consistent with the Precautionary Principles and include the in-tunnel installation of electrostatic precipitators and denitrification systems. Failure to remedy the known toxic emissions, LCTAG believes, may contravene the Protection of the Environment Operations Act.

LCTAG will persist and agitate to bring our concerns to the attention of the LCTC Board, its constituent companies and shareholders as well as to the NSW Government and its bureaucracies. We contend there is potential for all parties to breach the Act and that they have foreseen the potential for serious health impacts and threat of litigation.

Dr Ray Kearney,
Chairman, Lane Cove Tunnel Action Group Inc
November, 2004

8. RTA Persistently Trashes Community Rights!

A NSW Parliamentary Notice of Motion, on 21 June 2005, records that the Hon. Sylvia Hale MLC (Greens) will move (in relation to former Roads Minister Costa):
That this House condemns the Minister for Roads for allowing the Roads and Traffic Authority (RTA) to persistently trash community rights and entitlements, for its callous disregard for community amenity and public health, and its general mismanagement of the road system as evidenced by the RTA.

The Notice of Motion then continues and tabulates a 12-point litany of serious allegations about the RTA's arrogance and includes:

- *repeatedly misleading people regarding the full market value of compulsorily acquired land (as demonstrated by Land and Environment Court decisions over the last 12months),*

- *grossly miscalculating the traffic volumes and induced traffic growth associated with the M5East, and Lane Cove tunnel, and basing air quality measures inappropriately on grossly underestimated figures,*

- *neglecting to initiate modifications to Approval Conditions and introduce better pollution control measures for the M5 East Tunnel, the Cross City Tunnel and the Lane Cove Tunnel including in-tunnel filtration and more comprehensive air quality monitoring,*
- *neglecting to initiate modifications to Approval Conditions for the M5 East Tunnel and the Lane Cove Tunnel to take account of the gross underestimates of traffic volumes on which these tunnels were originally approved.*

Former Roads' Ministers Scully, Costa, Tripodi and now Roads Minister Eric Roosendaal respectively have utterly failed to quell the downright lying and deceit that have become the characteristic canker of the RTA bureaucracy. Those parts of the community who are aware of the facts are now in revolt against the betrayal by the current NSW Government and certain of its bureaucracies of basic human and democratic rights.

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(Includes additional references to those in the text of the Submission)

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