

2013 Federal Senate Standing Committee Inquiry

‘The impacts on health of air quality in Australia’

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 20 years involvement with air quality and health-impact issues of traffic tunnels (e.g., Lane Cove Tunnel, M5 East Tunnel and Cross City Tunnel) in Sydney from their earliest beginnings. I have appeared in no fewer than five NSW Parliamentary Inquiries and a contributor to the 2008 NH&MRC Report [http://www.nhmrc.gov.au/ files nhmrc/publications/attachments/eh42.pdf](http://www.nhmrc.gov.au/files/nhmrc/publications/attachments/eh42.pdf) - all related to the health impacts of air pollution e.g., arising from unfiltered vehicle tunnels.

Its presentation is true and does not, by its presentation of information or omission of information, materially mislead or intend to materially mislead.

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Terms of Reference:

http://www.aph.gov.au/Parliamentary_Business/Committees/Senate_Committees?url=clac_ctte/air_quality/tor.htm

The impacts on health of air quality in Australia, including:

- (a) **particulate matter, its sources and effects;**
- (b) **those populations most at risk and the causes that put those populations at risk;**
- (c) **the standards, monitoring and regulation of air quality at all levels of government;**
- (d) **any other related matters.**

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Summary

1. Diesel exhaust is a mixture containing over 450 different components, including vapours and fine particles coated with organic substances. Over 40 chemicals in diesel exhaust are considered toxic air contaminants overseas. Exposure to this mixture may result in cancer, respiratory effects, and other health problems. Weight-for-weight diesel particles are more than 40 times as carcinogenic as tobacco smoke.

2. In June, 2012, the WHO declared **diesel fumes** to be a **Level-1 Human Carcinogen** i.e. proven evidence of carcinogenicity in human beings. http://www.iarc.fr/en/media-centre/pr/2012/pdfs/pr213_E.pdf

3. The United Nations defined the right to health in Article 12 of the [International Covenant on Economic, Social and Cultural Rights](#) in 1966. The Covenant guarantees the “*right of everyone to the enjoyment of the highest attainable standard of health*”. Article 12.2 (b) comprises e.g., “*the prevention and reduction of the population's exposure to harmful substances such as radiation and harmful chemicals or other detrimental environmental conditions that directly or indirectly impact upon human health*”.

Through ratification of international human rights treaties, Governments undertake to put into place domestic measures and legislation compatible with their treaty obligations and duties. Australia is in breach of the ‘Declaration of Human Rights’!

4. Yet despite the volumes of evidence of health impacts of PM_{2.5} particles, the National Environment Protection Council (NEPC), has NOT implemented a STANDARD for carcinogenic Particulate Matter (PM) of 2.5µm or less (PM_{2.5}). That only a ‘guideline’ is set is indicative of the overt influence of the Oil/Petroleum/Automobile cartels and their representatives/lobbyists as well as alleged NEPC’s negligence – along with that of the various instruments of State and Federal Governments – Health, Environment, Energy, Roads & Transport, Planning etc! The reasoning it would appear is ‘*economic rationalism*’ i.e., if the taxes and excise income exceeds the cost of health impacts on society and there is left-over money in Treasury’s coffers then it is justified – but there is no morality!

Why is this so? As with the telecommunications industries:

“Oil and car industries have acted again and again to deceive regulators about the hazards of their products and have used their wealth to hamstring attempts by state and federal legislators to make laws that address such threats/hazards.”

Terry Tamminen ‘*Lives per Gallon*’ (2006)

5. 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 PM₁₀, PM_{2.5} and PM₁, are as follows:

*“PM₁₀ 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.”

“PM₁ 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 PM₁₀ and PM₁ standards would be most desirable from the legislation point of view.” (Morawska & Thomas, 2000).

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 - 0.03µm and 0.1µm – excluded from most air-quality monitoring!

6. California's Scientific Review Panel has unanimously endorsed the official listing of diesel exhaust as a toxic air contaminant, due to its cancer and non-cancer health effects. Australia is slow to follow!
 7. For the same load and engine conditions, diesel engines exhaust 100 times more sooty particles than petrol engines. As a result, diesel engines account for an estimated 26 percent of the total hazardous particulate pollution (PM10) from fuel combustion sources in our air, and 66 percent of the particulate pollution from on-road sources.
 8. Cleaner alternatives to diesel engines are readily available. Alternatives include electric, liquefied natural gas (LNG) or compressed natural gas (CNG) buses and trucks. For cars, in addition, alternatives include ethanol and ‘biodiesel’. E10 (10% ethanol in unleaded petrol) is reported to reduce fine particles by a qualified 50%. Most E10 is under-blended as E8.
- Diesel buses and trucks are important contributors to smog (ground-level ozone) and fine toxic soot, two pollutants that have recently come under increased scrutiny because of their important public health impacts. Purchasing alternative fuel vehicles will reduce smog and fine soot emissions considerably. For example, operating a natural gas bus instead of a new diesel bus is equivalent to eliminating the smog and soot from 17-55 passenger cars.
9. The health risk from diesel exposure is greater for children, the elderly, people who have respiratory problems or who smoke, people who regularly strenuously exercise in diesel-polluted areas, and people who work or live near diesel exhaust sources. Asthma is on the rise.
 10. According to an expert estimate, lifetime exposure to diesel exhaust at the outdoor average concentration (2.2 micrograms per cubic meter (µg/m³) may result in about one in every 2,000 people developing cancer due to this exposure. Some organisations e.g., NSW Cancer Council and ‘experts’ down-play the importance of diesel fumes as a cause of cancer – presumably because it undermines their campaigns and lobbying against “passive tobacco smoke” A moratorium on cancer research and inquiry into such publicly funded cancer societies are warranted.

Cancer is a multi-factorial process involving initiation by a chemical, virus or radiation followed by ‘promotion’ such as dietary omega-6 fatty acids, [reduction in melatonin](#) (e.g., beta-blocker medications, fluoride and by radiation from base phone stations). Cancer prevention, therefore, should be a multi-pronged strategy.

The effects of [long-term exposure](#) to wireless technologies including emissions from cell phones and other personal devices, and from whole-body exposure to RF transmissions from cell towers and antennas is simply not known yet with certainty. However, the body of evidence at hand suggests that bio-effects and health impacts can and do occur at exquisitely low exposure levels: [levels that can be thousands of times below public safety limits](#). [Exposure to diesel fumes and emissions from cell phones and other personal devices and from whole-body exposure to RF transmissions from cell towers and antennas reduce melatonin and are likely to be synergistic or additive in causing health impacts.](#)

11. The WHO has stated that for particle matter (PM), the PM₁₀ is a 'marker' and there is no threshold below which there is no health impact. Nor are PM₁₀ levels relevant for particles in tailpipe exhaust. The current 24-hour PM₁₀ standard of 50µg/M³ is set at limits for economic reasons and NOT for preventing health-risk. Further, professors Lidia Morawska and Michael Moore (2004) - of Queensland, have made 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."

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

12. 1000 particles of 1µm are equivalent to one 10µm particle and have 10-times the surface area

- 1million particles of 0.1µm are equivalent to one 10µm particle and have 100-times the surface area.
- 1billion particles of 0.01µm are equivalent to one10µm particle and have 1000-times the surface area.
- More than 85% of particles in diesel exhaust are less than 1µm, peaking at 0.1 µm.

13. Exhaust pollution including coarse, fine and ultra-fine particles, gaseous irritants, 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
- 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 nanogram 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
- Exacerbation of viral asthma

The effect is a major increase in sickness-care costs to the nation's health budget. In France, a study showed 2/3 of health care costs due to pollution resulted when levels of pollution were below the national standard for Particulate Matter (PM), less than 10 micrometre in diameter, i.e., PM₁₀ of 50µg/M³/24 hours. According to NSW Health, some 1,400 people die annually from air pollution i.e., air pollution causes more premature deaths in NSW each year than road accidents. 2.3% of all deaths in Australia are attributable to air pollution. The annual health cost of air pollution to NSW is estimated at \$4.7 billion.

14. 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. Whilst diesel fumes are a known cause of lung cancer, health bureaucrats state "we have yet to understand the underlying mechanism."

This is the classic risk-based approach. Use uncertainty as an excuse to delay. Remember tobacco and asbestosis? Precaution is not (yet) fashionable while risk-assessment is!

If a substance is known to be harmful, it should not be necessary to demonstrate 'scientifically' that it is actually causing harm before doing something about it.

15. The opening of the Lane Cove Tunnel resulted in a number of complaints from residents about air-pollution **odour**. The implication being that this is associated with ‘groundstrike’ by fumes of unfiltered tunnel pollution from the tunnel’s exhaust stacks. The same experience continues to be reported in ‘odour diaries’ by residents affected by toxic fumes from the M5 East stack. In the past, unpleasant environmental odours have been considered warning signs or indicators of potential risks to human health but not necessarily direct triggers of health effects. The latter assumption has now changed because certain environmental malodours have now been shown to trigger health symptoms.

Heightened symptomatic responses to odours are well documented in two medical conditions – pregnancy and asthma. The majority of women report nausea and vomiting in the first trimester of pregnancy. Odours (e.g., household, food and industrial chemicals) can act as triggers of nausea and vomiting during this period. For asthma, Shim and Williams (*Amer.J Med.* 1986; vol. 80, p18-22) found that 90% of a group of 60 asthmatics surveyed reported exacerbations of asthma in relation to odour exposures, and nearly 40% had visited emergency rooms after such incidents. On repeat testing with a placebo (saline), the results of abnormal lung function in the asthmatics were not affected. Several separate studies have shown that exposure to odours by asthmatics, whose skin prick-testing confirmed a lack of immunological reaction, could provoke asthmatic attacks. Therefore, individuals with odour-triggered respiratory symptoms or ‘sensory hyper reactivity’ should not be assumed to have asthma, but rather should be considered in terms of a broader differential diagnosis.

16. Regarding the question of filtration in stacks and tunnels there is clearly a need for Governments 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**. At the moment, Governments and the statutory authorities are dicker about **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 end result, to date, is a patronising ‘**do-nothing**’ **outcome!** The **totality of the evidence** is, beyond reasonable doubt, in favour of installing filtration and makes it **obligatory** for Governments, to **unanimously endorse** the installation of filtration technology in tunnels and/or in stacks as a **responsibility** and a ‘**duty of care**’ – as endorsed by the Australian Medical Association.

17. Recently, mycologists brought to the attention of Lane Cove Council an alarming observation referred to as ‘*Rosecomb*’ (abnormal gills – due to diesel toxicity) in protected *Hygrocybe reesiae* and similar malformations in other endangered fungi. The analogy is thalidomide teratology. Are these epi-genetic effects an additional ‘wake-up’ call to draw attention to the poisoning of human beings and our life-support systems?

18. Government/consultant Air Quality Reports fail to respond to the results of research that confirms there is **no safe threshold level of particulate exposure.**

The lack of an identifiable safe threshold level for particulate exposure points to the need to reduce ambient particulate concentrations to as low a level as practical.

The **24 hour averages** of PM₁₀ do not identify *short-term pollution sources* e.g., a.m. and p.m. peak periods.

19. The multi-layered, utterly immoral, petroleum cartel has become institutionalized, having “got government off its back”. The Australian Government must be more aggressive in taking on the staggeringly profitable and tax-favoured big oil companies. As with Big Tobacco it is time for the community to initiate Class Actions against the oil cartels for the sickness and death they have afflicted!

20. Today, when ordinary citizens and others rise up in anger and hope, they have power to defy governments and focus debate by politicians. Such moments are upon us now to bring change for good. In the words of the '*Desiderata*' (1927) - Latin: "desired things" - written by Max Ehrmann: "*You are a child of the universe no less than the trees and the stars; you have a right to live ...*" The poem is a list of things desirable in life - technically, things considered necessary or highly desirable or something lacking and wanted. Sustainability! The poem's simplicity and affirmation of life resonate more than ever.

<http://www.fleurdelis.com/desiderata.htm>

Preface

Free access to clean fresh air and water of acceptable quality is a fundamental necessity and a *human right*.

The [Preamble](#) to the [World Health Organisation's](#) (WHO) constitution declares that it is one of the fundamental rights of every human being to enjoy "*the highest attainable standard of health*". The right to health is not to be understood as a right to be *healthy*. The right to health contains both freedoms and entitlements. The *entitlements* include the right to a system of health protection which provides equality of opportunity for people to enjoy the highest attainable level of health.

In June, 2012, the WHO declared **diesel fumes** to be a **Level-1 Human Carcinogen** i.e., proven evidence of carcinogenicity in human beings. http://www.iarc.fr/en/media-centre/pr/2012/pdfs/pr213_E.pdf

Australia continues to breach The Universal Declaration of Human Rights <http://www.un.org/en/documents/udhr/index.shtml> (Article 25) by allowing its citizens to be exposed to the carcinogenic fumes of diesel, despite the Government knowing the dangers. This is similar to deliberately exposing servicemen to the radiation of a nuclear bomb test at Maralinga 61-years ago – again, despite government knowing the dangers!

The United Nations further defined the right to health in Article 12 of the [International Covenant on Economic, Social and Cultural Rights](#) in 1966. The Covenant guarantees the "*right of everyone to the enjoyment of the highest attainable standard of health*". Article 12.2 (b) comprises e.g., "*the prevention and reduction of the population's exposure to harmful substances such as radiation and harmful chemicals or other detrimental environmental conditions that directly or indirectly impact upon human health*".

Through ratification of international human rights treaties, Governments undertake to put into place domestic measures and legislation compatible with their treaty obligations and duties.

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.*"

It is well known that levels of cancer, neurological disease (e.g., dementia), respiratory disease (e.g., asthma) and infertility have increased dramatically over the last century, in

the latter half especially. Is this just co-incidence or is there a correlation between disease, poor health and the prevalence of toxic chemical contamination of our life-support systems – air, water, food and land?

Already we witness this in action in Australia. For example, for decades, we have known that the combustion of fossil fuels e.g., **petrol and diesel** yields highly toxic particles that are predominately *ultra-fine* (about 0.03 micrometres - μm - in aerodynamic diameter), *soluble* and carry *toxic and carcinogenic chemicals* e.g., polycyclic aromatic hydrocarbons (PAHs).

It is emphasized that in June, 2012, the WHO declared **diesel fumes** to be a **Level-1 Human Carcinogen** i.e. proven evidence of carcinogenicity in human beings.
http://www.iarc.fr/en/media-centre/pr/2012/pdfs/pr213_E.pdf

Yet despite all this and the volumes of evidence of health impacts of PM2.5 particles, the dicker around for over 2 decades by the National Environment Protection Council (NEPC), has NOT led to the implementation of a STANDARD for carcinogenic Particulate Matter (PM) of 2.5 μm or less (PM2.5). That only a ‘guideline’ is set is indicative of the overt influence of the Oil/Petroleum/Automobile cartels and their representatives/lobbyists as well as alleged NEPC’s negligence – along with that of the various instruments of State and Federal Governments – Health, Environment, Energy, Roads & Transport, Planning etc! The reasoning it would appear is *‘economic rationalism’* i.e., if the taxes and excise income exceeds the cost of health impacts on society and there is left-over money in Treasury’s coffers then it is justified – but there is no morality!

Why is this so? As with the telecommunications industries,

“Oil and car industries have acted again and again to deceive regulators about the hazards of their products and have used their wealth to hamstring attempts by state and federal legislators to make laws that address such threats/hazards.”

Terry Tamminen *‘Lives per Gallon’* (2006)

A ‘guideline’, unlike a ‘standard’ is legally unenforceable! This favours the oil cartels and vehicle manufacturers. Consequently, the function of the transnational corporations (oil, pharmaceuticals, agribusiness, car manufacturers etc) is not to promote a healthy ecology, but to extract as much marketable value out of the natural world as possible, even if it means treating the environment like a septic tank. Profit is the name of the game! Indeed, the 2009 documentary *The Idiot Cycle* claims that certain chemical manufacturers, including oil cartels are profiting from the production of cancer-causing products and then some of the same companies are investing in profitable cancer treatments - *making pollution itself a cash-cow*.

Though ratifying international human rights treaties, the Australian Government has FAILED to undertake to put into place domestic measures and legislation compatible with their treaty obligations and duties e.g., “Article 25: *Everyone has the right to a standard of living adequate for the health and well-being of himself and of his family....*”

Public health experts fear global trade agreements and powerful transnational companies continue to create a boom industry in death and disease. Like the tactics of Big Tobacco, the Oil Cartels also seek to augment their profits at the expense of people’s lives – through aggressive marketing and lobbying. The problem is that people are not being represented ethically and honestly by their politicians and bureaucrats. People feel powerless, manipulated and betrayed!

It is no longer a level playing field when it comes to making healthy choices! Society is saturated by advertising, covert marketing and sponsorships! Researchers are being bought off! We are

dominated by blatant and sophisticated lies! The deceitful mantra “*Ethanol damages engines*” from the oil cartels helps account for the 30% increase in sales over the last three years of vehicles powered by *carcinogenic diesel*! Such companies should no longer be given a free place at the table when it comes to negotiating a public health response to the growing health impact of air pollution.

‘*Globalization*’ is too often confused with a beneficial notion of genuine mutual interdependence and cooperation between nation states – but in reality it clearly is not. In an effort to maintain profit margins, here and abroad public assets are plundered, human life trampled upon as well as the environment destroyed. In secret negotiations behind closed doors, the Trans-Pacific Partnership (TPP) Agreement involving Australia is a global economic coup that will redefine the terms of globalization in a way that completely resets the social contract in favour of corporate right rather than human rights. The TPP will allow corporations to sue nations if *laws such as those **protecting the environment** interfere with corporate profits*. How are we not to sit back and see our health and biodiversity hollowed out?

It is hoped that this Senate Committee Inquiry will not emulate all the numerous previous similar meetings, task forces, parliamentary inquiries, government reviews etc, etc that have come to essentially nothing to allow *health impacts of air pollution to continue as ‘business as usual’!*

Terms of Reference – 1. Particulate matter, its sources and effects

Table 1. Comparisons of ambient fine and coarse mode particles

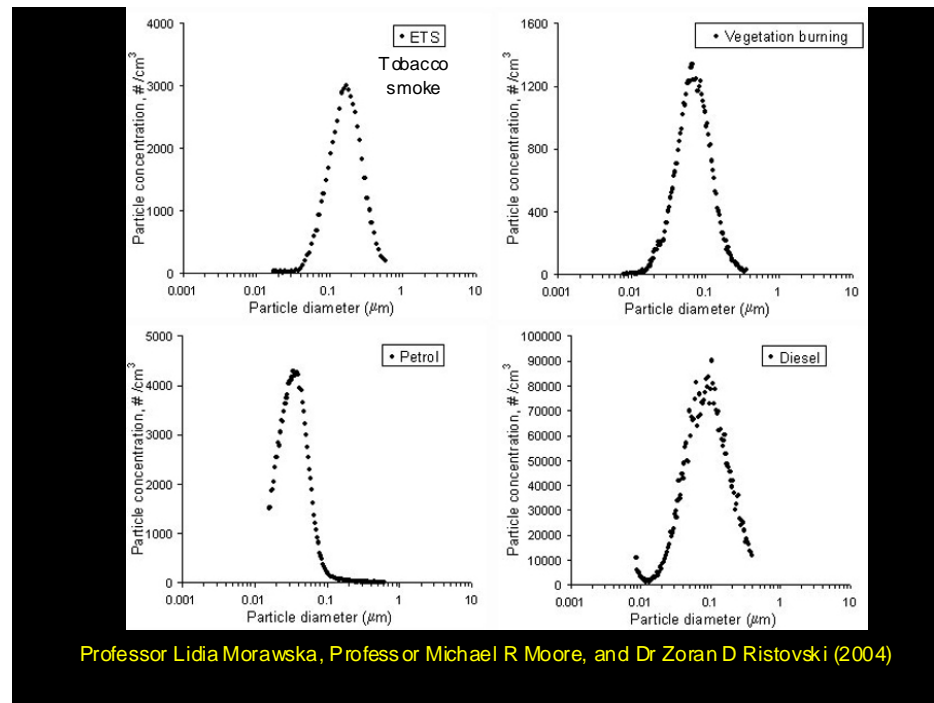
| | Fine Mode (<PM 2.5) | Coarse Mode PM10 |
|-----------------|--|---|
| Formed from: | Gases | Large solids/droplets |
| Formed by: | Chemical reaction ; nucleation; condensation; coagulation; evaporation of fog and cloud droplets in which gases have dissolved and reacted. | Mechanical disruption (e.g. crushing, grinding, abrasion of surfaces); evaporation of sprays; suspension of dusts. |
| Composed of: | Sulphate, SO_4^{2-} ; 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. | Re-suspension 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

1(a) 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 peaks¹ - 0.03 μm and 0.1 μm . Neither of these particle sizes (mass or number) is measured in any of the routine Air Quality surveys. Why? NEPC has not seen fit, in their alleged negligence, to set a *standard* for PM_{2.5} or less – only a ‘guideline’.

See also diagram below.



Morawska & Thomas² report that only 3% of combustion particles between 0.1 μm and 1 μm are present in PM₁₀ measurements. Despite this research conducted for the Queensland Environment Protection Agency, the RTA continues to claim that PM₁₀ measures all particles less than 10 μm in size. Monitoring by NSW EPA excludes most particles 1 μm and less. About 90% (by mass) of diesel particulates³ are less than 1 μm and are missing in the PM₁₀/M³ measurements in most EIS Reports. As a consequence, PM₁₀ measurements of air quality and those of air-streams vented from tunnel stacks are seriously under-estimated.

Morawska & Thomas⁴ concluded: "*PM₁₀ 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.*"

¹ 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.

² 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.

³ 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.

⁴ Morawska. Op cit.

Therefore, if the PM₁₀ 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 PM₁ measurement provides very good information about contributions from the combustion engine, and distinguishes it from suburban background.

The fact that PM₁ contributions are about two-fold greater in traffic aerosol than in ambient air⁵ is a serious deficiency in pollution monitoring by the RTA. Use of PM₁₀ data serves to hide exceedances of air quality standards. By using PM₁₀ measurements, the bulk of tail-pipe particulates are excluded and what is analysed is mainly re-suspended dust from the road surface⁶. The exclusion of combustion particles suppresses data of the enormous surface area (> 100 fold) of toxic respirable particles missing from PM₁₀ measurements.

Several overseas countries have a standard for PM₁₀ and PM_{2.5}. Australia has only now introduced a 'guideline' for PM_{2.5} additional to a standard for PM₁₀. It is misleading to imply PM₁₀ is stringent and includes all particles as fine as PM_{2.5} and PM₁.

A major flaw in monitoring pollution from tunnel stacks is that the fine particulate matter of less than one micrometre (<PM₁), representing 90% of particulate pollution from vehicle exhaust is excluded by the measuring devices (Tapered Element Oscillating Measure –TEOM) used, to date, in the monitoring PM₁₀.

1 (b) CASE STUDY: Example of how the air-quality guidelines and standards *are abused* is found by CSIRO's Study Report (2012) commissioned by NSW Government to assess the efficiency of a 'trial filtration study' of the M5 East traffic tunnel in Sydney.

CSIRO REPORT

http://www.rta.nsw.gov.au/roadprojects/projects/building_sydney_motorways/tunnel_air_quality/m5_east/project_documents.html

CSIRO Executive Summary

1. Only 0.5 -2.5 µm particles were measured (**Excludes particles from tail-pipe**, see graphs above).
2. 'More research' – typical CSIRO recommendation for self-serving research funding when answers to tabulated questions are already known from overseas experience. Compare to CSIRO's bio-ethanol government-funded research to "Test ethanol fuel E10 under Australian conditions"! The benefits of E10 were already known from overseas research!

CSIRO Report:

1. On p11 CSIRO confirms particles in tunnel are dominated by "*finer particle fraction*" – but **did not** measure them!
2. On p12, CSIRO seems to say that the instrumentation used was expected to measure down to 0.02 µm (20nm).
3. On p13, CSIRO chose an instrument with a size range from 0.5 µm – 20 µm. CSIRO state: "*Particles with diameters of less than 0.5µm have **not been considered in this study** due to the diminishing contribution of particles less than 0.5 µm to PM10 and PM2.5.*" Thus CSIRO used

⁵ Morawska. Op cit.

⁶ Ibid

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

⁶ 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.

⁶ Kittleson D.B. *Engine & Nanoparticles: A Review*. Op cit

equipment they KNEW was **inadequate** for proper measurements of the TOXIC fine fraction. CSIRO's statement is grossly misleading!

4. Why CSIRO were allowed to use irrelevant measurements etc. needs an answer.
5. Electrostatic Precipitators (ESPs) operate with efficiencies > 95% overseas. Why the assertion by CSIRO of "poor" filtration performance here? Is this common political patronage? CSIRO's conduct seems questionable! As a consequence, the community suffers!
6. What is needed is the filtration equipment be designed and built by credible and experienced overseas manufacturers.
7. The cost analysis is misleading. The CSIRO testing FAILS to account for the particle removal by filtration as per stack monitors. The filtration is more efficient than CSIRO calculated from selective 'fiddled' data in its flawed study. Who suffers from this alleged incompetence? The community!

Approximately 90% (by mass) of diesel particulate emission has a size less than $1\mu\text{m}$ (Brown *et al.*, 2000). NSW 'EPA' disclosed that its monitoring of atmosphere pollutants does **not** include those particles $1\mu\text{m}$ or less. (International Workshop on Tunnel Ventilation, June 2000). [Therefore, the 90% of diesel emission is not included in the mass of \$\text{PM}_{10}/\text{M}^3\$ recorded in routine air-quality monitoring.](#)

1(c) Scientific misconduct

In conventional scientific/medical research, investigators who publish results and *knowingly*:

- Derive them from the inappropriate application of methods;
- Fail to incorporate proper factors for valid readings;
- Skew the results to a pre-determined outcome;
- Remove data that are inconveniently high;
- Adopt standards that are known to be flawed or inappropriate;
- Deliberately mislead in the interpretation and analysis as well as in their data presentation, and
- Ignore the numerous, clear and specific directives in international expert advice to amend the study design for scientific stringency and methodological validity -

are deemed **incompetent**, or worse, judged to be guilty of **scientific misconduct** and treated harshly.

The flawed air quality assessment as well as the health-risk analysis of the tunnel emissions have consistently ignored independent professional advice, (e.g., Prof. Lidia Morawska (Q'land), air quality expert and adviser to WHO), that PM_{10} measurements, unlike PM_1 , provide no information regarding vehicle emissions that constitute the tunnel haze. Thus, standards set for the emissions e.g., of the Lane Cove Tunnel western stack ($1600\mu\text{g}/\text{M}^3/30\text{min.}$) relate **only** to PM_{10} and **grossly under-estimate** the actual particulate pollution. *Planning NSW* has also ignored the fact that the National Standards of regional air quality **do not apply to point source emissions** (stacks) or to tunnels as the standards do not measure the most harmful fine particles in vehicle exhaust.

The invalid PM_{10} data for measurements of traffic/industrial emissions implies the health risk "assessment" commonly undertaken in Environmental Impact Statements/Studies (EIS) is also **grossly under-estimated and flawed**.

By using PM_{10} measurements, government/consultant Air Quality Reports exclude virtually the bulk of the constituent particulates arising from the tail pipe. This exclusion then totally fails to acknowledge the existence of the **enormous surface area** (> 1000 fold) of respirable particles that carry toxins.

Government/consultant Air Quality Reports fail to disclose the levels of pollution at 15 minute intervals i.e., that demonstrate major exceedances during a.m. and p.m. peak traffic periods. Such

exceedances are diluted by 24 hour averages and further diluted by annual averages on which health risk assessments are guessed.

Contemporary risk management is about trying to get the levels as low as possible. We should not work up to a standard, we work down to a risk – that is not what has happened in this process. **A solution is to install filtration in the vehicle tunnel stacks in residential areas.**

Government/consultant Air Quality Reports fail to respond to the results of research that confirms there is **no safe threshold level of particulate exposure.**

The lack of an identifiable safe threshold level for particulate exposure points to the need to reduce ambient particulate concentrations to as low a level as practical. This means that e.g., *NSW Planning* needs to acknowledge there are feasible, practicable and warranted measures to reduce toxic emissions in tunnels such as filtration of pollutants before leaving the stacks.

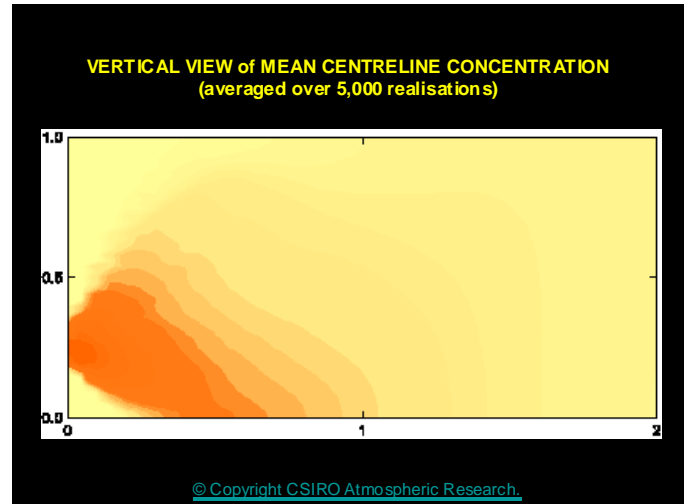
The 24 hour averages of PM₁₀ do not identify *short-term pollution sources* e.g., a.m. and p.m. peak periods.

Government/consultant Air Quality Reports and e.g., the NSW Regulatory Authorities including *NSW Planning* fail to acknowledge that it cannot be acceptable to increase health risks to one population (e.g., exposed to emissions from a single ‘unfiltered stack’) on the grounds that another might correspondingly benefit from vehicle tunnels when only the PM₁₀ mass/M³ measurements are made.

Exhaust particles in the ‘Fine Mode’ provide a formidable large surface area (wt/wt) for transport of adsorbed substances to the alveolar membrane

| | Golf ball | Rice | Sugar | Flour |
|---------------------|------------------|-----------------|-------------------|--------------------|
| Analogy | PM ₁₀ | PM ₁ | PM _{0.1} | PM _{0.01} |
| Number | 1 | 1000 | 1x10 ⁶ | 1x10 ⁹ |
| Surface Area | X | 10 X | 100 X | 1000 X |
| Solubility | — | + | + | + |
| Exhaust | + | ++ | ++++ | +++ |

Government/consultant Air Quality Reports fail to understand the *dynamics of pollution trajectories* from a stack. The following photo (LHS) has a cruise ship at Sydney's Darling Harbour discharging visible exhaust from a funnel. Superimposed is a photo of the Lane Cove Tunnel western stack (to scale). Note how the heavy particles 'fold downwards' from the point source.



The Top, RHS panel shows fallout of heavy particles in close proximity to a stack while light/fine particles remain in the air for days/weeks and travel 100's to 1000's of kms.

1 (d) Do PM₁₀ Measurements Apply to Emissions of Motor Vehicles? – No!

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

- Nuclei, N**, (<0.1µm). These originate from the condensation of hot, highly supersaturated vapours released during combustion.
- 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
- Coarse, C**, particle (>1.0µm). Almost all particles in the coarse particle mode originate from 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 PM₁₀, PM_{2.5} and PM₁, are as follows:

“PM₁₀ 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.”

“PM₁ 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 PM₁₀ and PM₁ standards would be most desirable from the legislation point of view.” (Morawska & Thomas, 2000).

Research shows that the 75% of the toxins, including carcinogens among the polycyclic aromatic hydrocarbons (PAH's), are carried on respirable particles less than 2.5µm i.e., particles not fully accounted for by air-quality reports/assessments based on PM₁₀ air quality data.

Despite the fact that the standards in the National Environmental Protection Measures (NEPM) *do not apply to a point source emission*, but to a regional airshed e.g., Sydney all NSW Air-Quality Report for traffic tunnels ignore the NEPM guidelines and apply an inappropriate PM₁₀ instrument for measuring emission pollution from the tunnel stacks.

Table 2 Fractional contribution of N+A and C modes to the volumes of PM₁, PM_{2.5} and PM₁₀

| 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 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 originate from natural and anthropogenic mechanical processes.

1 (e) Using Air Quality Goals as a License to Pollute

The national pollution guidelines do not apply PM₁₀ 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.

PM₁₀ 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 50µg/M³, 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.

Terms of Reference – 2. Those populations most at risk and the causes that put those populations at risk

2 (a) The relationship between air pollution, death and disease has been studied for decades, leading to the consistent 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>

See also NH&MRC Report (2008) on Air Quality Around Tunnels – link:

<http://www.nhmrc.gov.au/guidelines/publications/eh42>

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.

PAH Concentration (ng/m³). Distribution between the aerosol and gas phases (26/05/99)

| Pollutants | Tunnel centre | | Portal | |
|------------------------|----------------|---------|----------------|--------|
| | Pat. PM 2.5 | Gas | Pat. PM 2.5 | 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

Note: Several PAHs are known carcinogens and adsorb to FINE particles that are soluble.

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

The Table above documents the poly-cyclic aromatic hydrocarbons (PAHs) in the PM_{2.5} particulate phase and the gas phase of a NZ vehicle tunnel. The majority of PAHs e.g., di-methyl benzanthracene (DMBA), on particles, are proven carcinogens. DMBA is commonly used to induce breast cancers in experimental animals e.g., rats. PM_{2.5} particles are **soluble** in the lungs, off-loading their cancer-causing cargo.

The bulk of the PM_{2.5} particles have a mean diameter of about 0.1 µm of which at least 30% are deposited in the alveoli.

The scientific evidence is clear: diesel exhaust is a complex mixture comprised of hazardous particles and vapours, some of which are known carcinogens and others probable carcinogens. Diesel exposure poses a significant and avoidable increase in human health risks. Compelling evidence from dozens of well-designed studies supports the conclusion that diesel exhaust causes cancer. In addition, fine particles from diesel exhaust aggravate respiratory illnesses such as bronchitis, emphysema and asthma and are associated with premature deaths from cardio-pulmonary disorders.¹ The evidence of health effects is derived from extensive studies of human workers as well as some studies in animals, and observations of various kinds of mutagenic activity in culture systems.

1. Shprentz D, "Breathtaking: Premature Mortality Due to Particulate Air Pollution in 239 American Cities", NRDC, New York, May 1996, pp. 13-32.

Table 2: Substances in Diesel Exhaust Listed by Cal EPA as Toxic Air Contaminants

| | |
|--|---|
| acetaldehyde | inorganic lead |
| acrolein | manganese compounds |
| aniline | mercury compounds |
| antimony compounds | methanol |
| arsenic | methyl ethyl ketone |
| benzene | naphthalene |
| beryllium compounds | nickel |
| biphenyl | 4-nitrobiphenyl |
| bis[2-ethylhexyl]phthalate | phenol |
| 1,3-butadiene | phosphorus |
| cadmium | polycyclic organic matter, including |
| chlorine | polycyclic aromatic hydrocarbons (PAHs) |
| chlorobenzene | and their derivatives |
| chromium compounds | propionaldehyde |
| cobalt compounds | selenium compounds |
| creosol isomers | styrene |
| cyanide compounds | toluene |
| dibutylphthalate | xylene isomers and mixtures |
| dioxins and dibenzofurans | o-xylenes |
| ethyl benzene | m-xylenes |
| formaldehyde | p-xylenes |
| <p>Note: California Health and Safety Code section 39655 defines a "toxic air contaminant" as "an air pollutant which may cause or contribute to an increase in mortality or in serious illness, or which may pose a present or potential hazard to human health."</p> | |

<http://www.nrdc.org/air/transportation/ebd/chap2.asp#table1#table1>

2 (b) Particles - size, numbers and surface area

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 (PM10), 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 *by filtration*.

2 (c) Who is most at risk?

Most of the human studies on the health risks of diesel exhaust looked exclusively at healthy, adult men. To extrapolate from male worker studies to the general population may not adequately protect women, children, and the elderly. Furthermore, worker studies provide little information about health effects in people with chronic illnesses or depressed immune systems. We do know something about the susceptibility of some of these groups from research on the health effects of fine particle pollution.

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).

2 (d) PM weight measures is inappropriate in evaluation of the likely biological effects of ultrafine particles

Queenslanders - Professor Lidia Morawska and Professor Michael Moore – writing in their excellent review found at:

<http://www.deh.gov.au/atmosphere/airquality/publications/health-impacts/index.html>
state under 'Toxicology':

*“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**”.*

2 (e) Pollutants interact with allergens

Studies have shown that pollutants such as exhaust particles can interact with allergens to amplify allergic reactions. Acutely high concentrations (e.g., a.m. and p.m. exceedances) of different pollutants can induce transient respiratory symptoms in asthmatic subjects.

Diesel emission of particulates (90% are not measured by current PM₁₀ monitors) enhance allergic inflammation.

2 (f) Environmental Impact Statements (EIS) are deficient

Government/consultant Air Quality Reports and EIS's do not provide evidence of the size of the 'at risk' population (e.g., asthmatics, bronchitics, cardiac patients, children < 5 years). The extent to which a 'risk' population is not quantified e.g., in the stack precincts, it is impossible to undertake specific risk assessments, only guesses are offered to hoodwink a concerned community.

2 (g) EIS reports do not take account of cumulative effects

The failure to take account of *cumulative effects* on health is bewildering. At least 43% of inhaled particulates are retained in the lung and is associated with serious cumulative effects. An adult breathes 18 M³ of air daily. Government/consultant Air Quality Reports and EIS's fail to provide a thorough and objective assessment of this matter. In the absence of certainty, the Precautionary Principle should be adopted.

2 (h) Most health impacts occur below the standard

Government/consultant Air Quality Reports fail to acknowledge that two-thirds of the health costs occur during days when particulate levels are less than $50\mu\text{g}/\text{M}^3$.

WHO challenges world to improve air quality

Dr Maria Neira, WHO Director of Public Health and the Environment stated:

*“By reducing particulate matter PM_{10} pollution to **$20\mu\text{g}/\text{M}^3$** as set out in the new guidelines, we estimate that we can cut deaths from air pollution by around 15% or 300,000 (of 2 million) deaths annually.”*

October, 2006

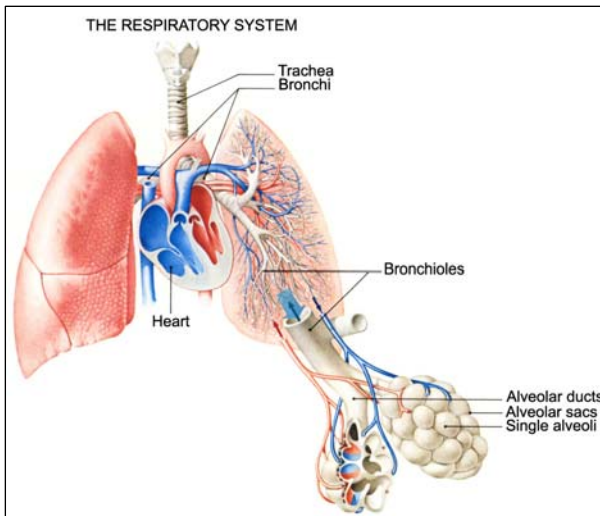
2 (i) Health impacts from short-term exposure during peak-hour are hidden

The 15-minute excursions in pollution levels during peak traffic periods that carry a 3-4 fold increase in health risk are hidden by PM_{10} averages. This is compounded by the fact that a.m. and p.m. peaks coincide with maximum pedestrian and residential exposure. Government/consultant Air Quality Reports often fail to acknowledge these facts in their analysis. Such short-term variations are important in light of reports (e.g., R.A. Michaels, 1996; 1998) that show **short-term excursions can explain some of the excess mortality and morbidity attributed to ambient particulates.**

2 (j) Adverse impacts on health and well-being

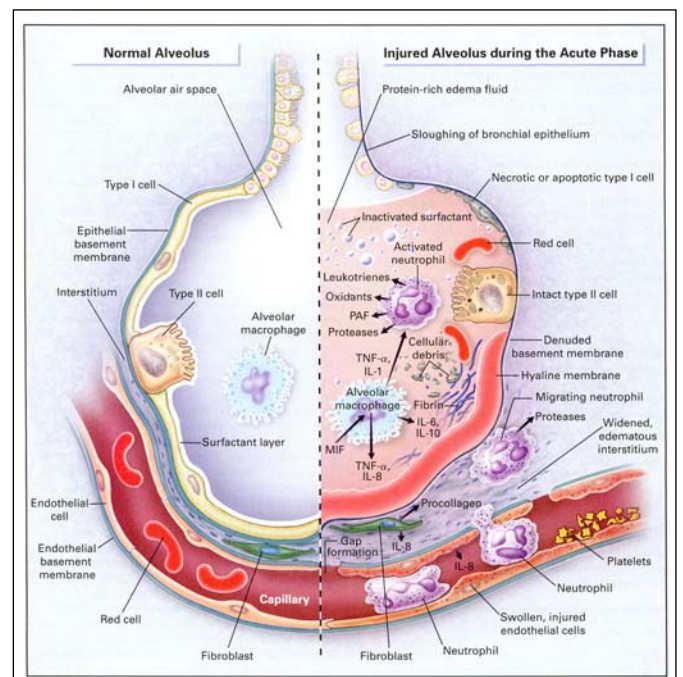
Exhaust pollution including coarse, fine and ultra-fine particles, gaseous irritants (e.g., O_3 and NO_2), and PAH's either alone or in combination, are known to be associated with, for example:

- a. inflammatory lung diseases e.g., asthma, bronchitis and alveolitis
- b. increased cardio-vascular disease
- c. increased risk of myocardial infarction in susceptible persons
- d. risk for exercise-induced heart damage
- e. limited blood flow and increased blood clotting
- f. increased mucous production and airway hyper-responsiveness
- g. 1 in 5 lung cancer *deaths* (USA) and accelerated tumour growth
- h. premature death
- i. symptoms of anaemia e.g., tiredness, headaches, fatigue and shortness of breath.
- j. low birth weight and small head circumference of neonate.
- k. intra-uterine growth retardation (for each 10 nanograms PAH's / M^3 increase)
- l. certain leukaemias e.g., from exposure to benzene.
- m. loss in productivity, absenteeism from work and school.
- n. increased sensitivity to bacterial products in airways
- o. more severe common viral asthma
- p. reduced male fertility
- q. significant risk of ovarian cancer from exposure to vehicle pollution
- r. adverse effects on lung development from the age of 10 to 18 years



Lung and Heart Relationships in Gas Exchange

Normal Alveolus and Effect of Acute Inflammation



Particulate Air Pollution and Blood

Exposure to PM₁₀:

- reduces RBC count (p<0.001)
- reduces platelet and factor VII (p<0.05)
- elevates C-Reactive Protein (p<0.01)

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

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

Heart Attacks and Short-Term Exposure to Fine Particles

- Within hours of exposure to PM_{2.5} fine particles, at levels of 10 µg/M³, can trigger a myocardial infarct in elderly persons at-risk with atherosclerosis.

C. A. Pope *et al.*, *Circulation*. (2006); 114: 2443-8

- A further increase in risk is gene-related

S. Y. Park *et al.*, *Circulation* (2006); 114: 2798-2805

Pollution 'increases risk of heart disease for women'

- The risk of post-menopausal women dying from a heart attack or stroke increases dramatically with higher concentrations of fine sooty particles from vehicles and factories.
- Researchers found that average particulate levels in and around 36 US cities ranged from about four to almost 20 µg/M³ of air. Each 10 µg/M³ rise was matched by a 76% increase in the chances of dying from any cardiovascular cause.
- For women living within, rather than between cities, the risk more than doubled, increasing by 128%, with each step up in pollution levels.

New Engl. J. of Med (Feb, 2007) v 356; p 447-58

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2 (k) Older adults are at increased risk of being hospitalized from exposure to fine-particle air pollution.

<http://www.sciencedaily.com/releases/2012/04/120417221835.htm>

The results showed an association between long-term exposure to fine air particles for all hospital admissions examined. For example, for every 10-µg/m³ increase in long-term PM_{2.5} exposure, the researchers found a 4.22% increase in respiratory admissions, a 3.12% increase in cardiovascular disease admissions, a 3.49% increase in stroke admissions, and a 6.33% increase in diabetes admissions.

Particulate air pollution is one of the largest avoidable causes of death and illness and unlike diet and exercise, does not require behavioural change. Off-the-shelf technology can be retrofitted onto sources of pollution at modest cost, with a large health benefit. This study shows that in addition to avoiding deaths, such measures will reduce chronic disease and medical care costs.

2(l) Exposure to air pollution linked to heart disease

The largest study (published Feb., 2013) yet to investigate the links between fine airborne particulate matter (PM) and patient survival after hospital admission for acute coronary syndrome (ACS) found death rates increased with increased exposure to PM_{2.5}.

<http://www.sciencedaily.com/releases/2013/02/130219201519.htm>

The study found that for every $10\mu\text{g}/\text{m}^3$ increase in PM2.5 there was a 20% increase in the death rate. For example, over one year of follow-up after patients had been admitted to hospital with ACS, there would be 20% more deaths among patients exposed to PM2.5 levels of $20\mu\text{g}/\text{m}^3$, compared to patients exposed to PM2.5 levels of $10\mu\text{g}/\text{m}^3$.

Dr Tonne and her colleague Paul Wilkinson, professor of environmental epidemiology at the London School of Hygiene & Tropical Medicine, estimate that death rates would be reduced by 12% among ACS patients if they were exposed to naturally occurring PM2.5 rather than the higher levels to which they were actually exposed. This translates to 4,783 deaths occurring earlier than they should do, due to exposure to PM2.5 from human-made sources. The implication is that reducing levels of PM2.5 will lead to increased life-expectancy.

2 (m) Causes that put people at risk - Most diseases including cancer and heart attacks are *multi-factorial* in induction and development

2 (m)-1 NSW Cancer Council's embarrassment: That WHO in June, 2012 declared diesel fumes to be a proven Level-1 carcinogen for human beings poses an immense embarrassment to the NSW Cancer Council whose former CEO was quick to publicly criticize anyone who stated that lung cancer was caused by anything other than tobacco.

<http://www.cancercouncil.com.au/lung-cancer/lung-cancer-risk-and-prevention/Minimal> and obscure mention is made by NSW Cancer Council of diesel fumes:

<http://www.cancercouncil.com.au/15559/cc-publications/understanding-cancer-series/understanding-lung-cancer/understanding-lung-cancer-qa/?pp=33312>

How utterly spurious and uninformed such comments are! In practical terms they ignore PM2.5 diesel fumes as a proven cause of lung cancer confirmed in e.g., a study (that excluded tobacco) in 2004 where one-fifth of lung cancer deaths was attributed to exposure to fine atmospheric PM2.5 particles derived from the exhaust pipes of motor vehicles. See links:

<http://www.reuters.com/article/2011/10/28/us-air-pollution-idUSTRE79R5NM20111028>

http://epw.senate.gov/107th/Levy_1.pdf

http://hydra.usc.edu/scehsc/web/Resources/Speaker%27s_Kit/AirPollutionImpacts.pdf

<http://www.telegraph.co.uk/science/science-news/3302094/Pollution-is-linked-to-deaths-from-lung-cancer.html>

2 (m)-2 Celebrity 'experts': Equally disturbing is when 'experts' seemingly fail to understand the importance of the *multi-factorial nature* of certain diseases such as cancer. For example, one 'expert' is reported to profess single cancer causations but excludes the documented importance of 'co-factors' such as inflammation and other factors e.g., reduced levels of the anti-cancer hormone – melatonin – in carcinogenesis/tumorigenesis. These 'co-factors' are misleadingly relegated to a category of "unproven risks" thereby obscuring the proven requirement of 'initiators' and 'promoters/co-factors' in cancer development.

<http://theconversation.edu.au/unproven-cancer-risks-diverting-focus-from-real-cause-lifestyle-5629>

Government departments (e.g., Energy, Health, Environment etc) 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 above articles that 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.”

Reports and studies from these sources often show findings which seem 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 M5 East ‘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 government to engage in a study where they would not be able to find the associations between vehicle emission and community health. These strategies were used to hide the public health hazards of asbestos for over a century also feature in the techniques adopted by bureaucracies to perpetuate the **myth** that the exhausting of vehicle pollutants from tunnel stacks, in residential areas, poses no health risk, either short or long term, for anyone.

The significance of ‘co-factors’ in cancer initiation by air pollution is commented as follows:

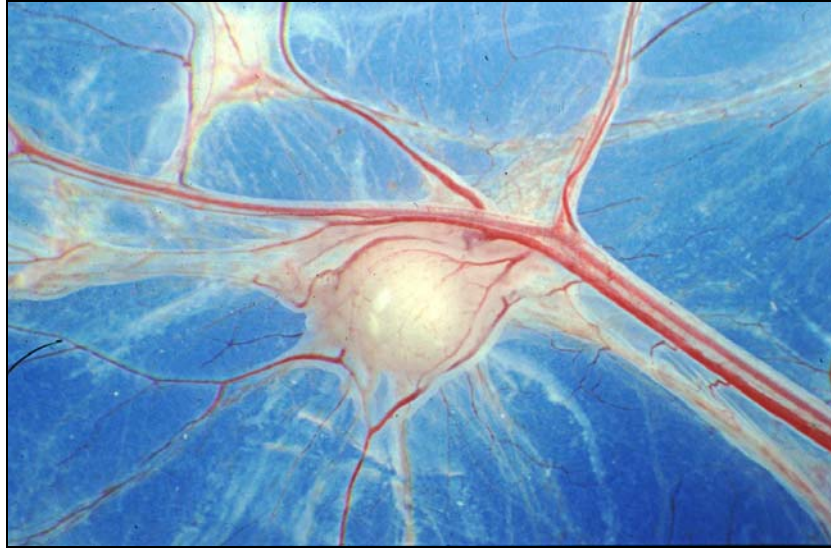
2 (n) Examples of the multi-factorial nature of cancer development.

2 (n) - 1 Inflammation: (i.e., body’s response to tissue injury e.g., sunburn, infection or a wound) is a *potent accelerator* of cancer growth *initiated* by a carcinogen e.g., diesel fumes,

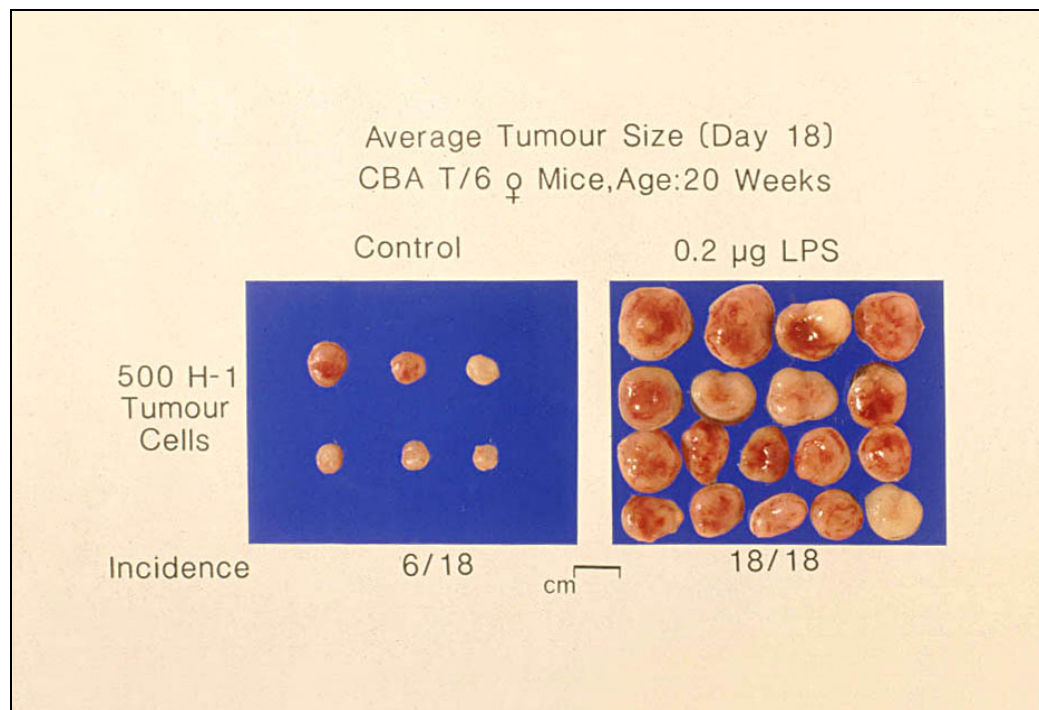
UV-radiation, asbestos or tobacco smoke. Surgery (tissue injury induced-inflammation) to remove a primary tumour is long documented to be a potent accelerator of any residual secondary tumour deposits.

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3365481/>

<http://jcb.rupress.org/content/192/4/547.full>



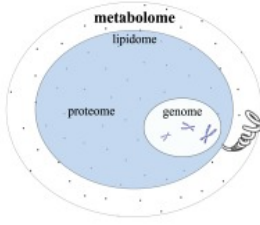
Photograph by Dr. R. Kearney of a tumour to show induction of blood vessels for continued growth. Dormancy of a tumour is not sleepiness but occurs when cell death equals cell multiplication. Inflammation accelerates tumour growth by 'switching off' cell suicide (apoptosis)



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.

Pro-Inflammatory Molecular Imbalances:

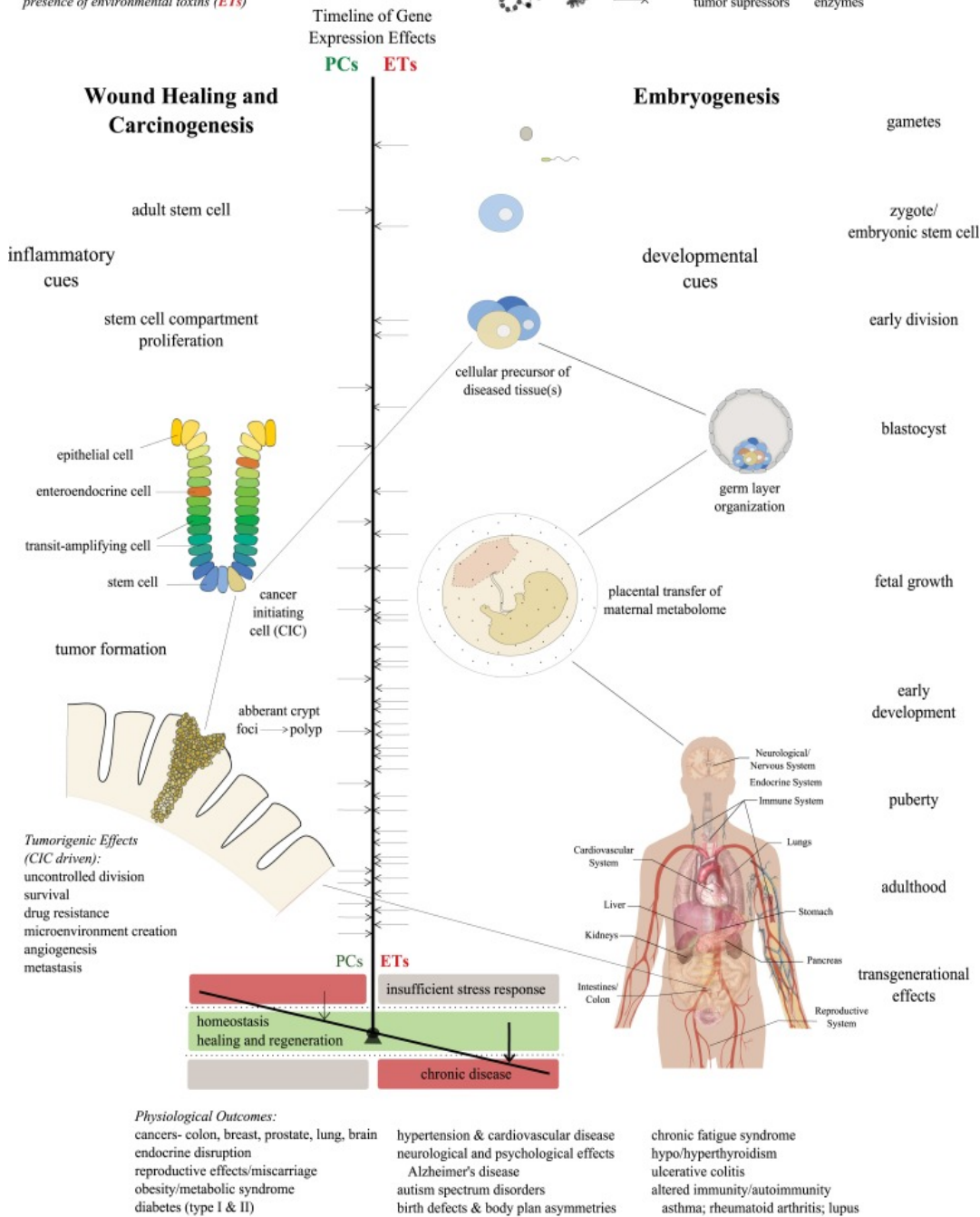
- free radical excess
- pH (acidosis)
- hypoxia
- osmolarity
- sugar levels
- electrolyte levels
- aberrant cell signaling
- stress (cortisol) levels
- circulating secreted factors
- caloric excess/energy usage
- lipid levels/omega 3:6 ratio
- heat shock/misfolded proteins
- lack of phytochemicals (PCs)
- presence of environmental toxins (ETs)



Influences on Gene Expression:

- Epigenetic Modifications:**
- DNA (CpG sites):** methylation
 - Histones:** acetylation, ubiquitylation, methylation
 - phosphorylation**
 - sumoylation**
 - biotinylation**
 - ADP-ribosylation**
- Others:**
- genetics- inheritance and mutations
 - non-coding RNA expression
 - transcription factor/machinery regulation
 - post-translational protein modifications
- Genes Affected:**
- oncogenes
 - tumor suppressors
 - receptors
 - enzymes

The



Dynamic epigenetic interplay of inflammation, environmental toxins, and phytochemicals theorized to lead to homeostatic imbalance and chronic disease in modernized societies; the manifestation and extent of which depends upon the hypothetical timing of critical gene expression changes in stem cells during embryogenesis, wound healing, and colon carcinogenesis (in this scenario), with other outcomes plausible.

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3365481/>

Terms of Reference - 3 The standards, monitoring and regulation of air quality at all levels of government

3 (a) Flaws in Monitoring of Air Pollution

Ayers *et al.*, (CSIRO, 2001) confirmed that a routinely used method of continuous sampling and monitoring PM₁₀ known as the Tapered Element Oscillating Microbalance (TEOM) *under-estimated PM₁₀ 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 are commonly 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 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.'

*Background levels of PM₁₀ have been **underestimated by 11–40%**, dependent on meteorological and air quality conditions (Katestone Environmental Report 25.1.2002)*

Why? Because NSW **refuses to incorporate a correction factor** into its continuous measurements of particles by the TEOM method. The correction factor is needed when ambient temperatures are below 17C. This omission enables the government to report on lower levels of pollution than exists and to avoid exceedances that require filtration in the tunnel to be installed.

Ventilation Stack Limits

West Stack: conc limit - 1.6 mg/ M³/ 30 min.

East Stack: conc limit - 1.2 mg/ M³/ 30 min.

Combined Annual load limit - 14 tonnes

Note:

- LCC analysis established 14 tonnes will be exceeded
- Weight measure is not appropriate for health risk

Noteworthy is the NSW Planning Minister's Conditions of Approval (MCoA) for the Lane Cove Tunnel:

Note that the MCoA allows 14 tonnes of PM10 each year to be discharged from the 2 stacks, but the existing monitoring stations were too far away to detect any of this PM10.

Ventilation Stack Limits

- Benzene
- Toluene
- Xylenes
- 1,3-Butadiene
- Formaldehyde
- Acetaldehyde

Combined stacks load limit:
154 tonnes / year

Note that the MCoA allows 154 tonnes of highly toxic volatile chemicals to be discharged each year from the 2 stacks. However, the monitoring stations that existed did NOT have the instruments to detect any.

Terms of Reference – 4 Any other related matters

4 (a) ‘Feelings’ are treated as evidence of fact

Throughout Government/consultant Air Quality Reports there seems to be a selection only of facts and arguments that tell in their favour. Coloured words and phrases appeal to the emotions rather than to reason, and sometimes mean nothing at all. Often their language lacks clarity and precision and is **without freedom of over-statements**. Such Reports usually do **not** give the community **all the available facts** to enable them to form an **independent opinion based on cold fact, rather than coloured assertions and feelings**. Repeatedly, ‘feelings’ are then allowed to judge facts. Even worse is when ‘feelings’ are treated as evidence of fact. Then, it appears, begins the transition, by an insidious artform, from scientific stringency to political expediency.

Health bureaucrats and telecommunication ‘experts’ state they are “*not sure yet*” how big the problem is and “*we have not identified the extent of the problem*” or “*we have yet to understand the underlying mechanism*”. Meanwhile it is business as usual and ***willful blindness to the truth!***

4 (b) Citizen activists

In the 2009 documentary *The Idiot Cycle*, it is claimed that certain chemical manufacturers are profiting from the production of cancer-causing products and then some of the same companies are investing in profitable cancer treatments. The function of the transnational corporation is not to promote a healthy ecology, but to extract as much marketable value out of the natural world as possible, even if it means treating the environment like a septic tank.

Profit is the name of the game! The support systems of the entire ecosphere – the Earth’s thin skin of fresh air, water and topsoil – are at risk. National and international regulatory mechanisms, *based on evidence*, must be put in place to ensure that basic resources such as land, water and the means for accessing fresh water do not become merely the means for profit accumulation for the wealthy. Enter the Citizen Activist!

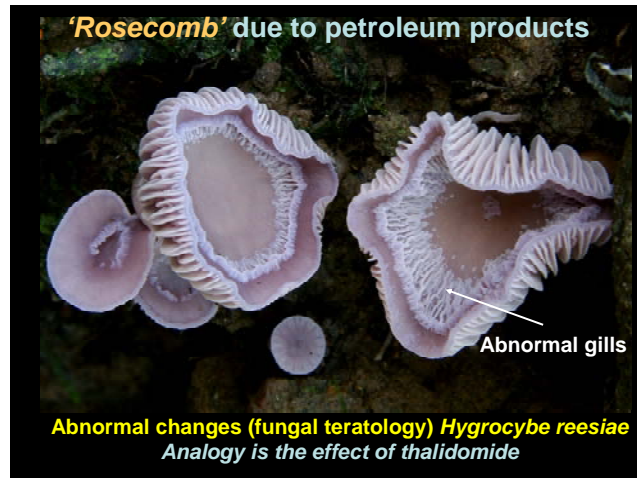
According to new information by teams of citizen scientists and experts, the Earth is now entering the sixth mass extinction event in its four-billion-year history. This die-off is the only

such event precipitated by human beings. Over 10,000 amateur scientists and professionals in the World Conservation Union have compiled data showing that currently 51 per cent of known reptiles, 52 per cent of known insects and 73 per cent of known flowering plants are in danger along with many mammals, birds and amphibians.

4 (c) Endangered fungi threatened by diesel fumes

However, plants and animal life along with amphibians are not the only species to face extinction. So too are species of fungi already listed as protected under NSW State legislation.

Mycology enthusiasts, Elma and Ray Kearney, recently brought to the attention of Lane Cove Council an alarming observation referred to as 'Rosecomb' (abnormal gills – due to diesel toxicity) in protected *Hygrocybe reesiae* and similar malformations in other endangered fungi.



The holotype (new), mushroom *Hygrocybe reesiae*, originally found in LCBP by the Kearney's, was listed (2000) under the *NSW Threatened Species Conservation Act, 1995*. For the *first time*, abnormal changes in protected waxcaps (*Hygrocybe* fungi) in Lane Cove Bushland Park are now recorded. 'Rosecomb' refers to the gross malformations when mushrooms are exposed to *petroleum products*. *H. reesiae* rosecomb is *not* a genetic mutation but rather genetic *instability* coupled with an *exogenous factor* e.g., diesel fumes to induce changes in *morphogenesis* of gills, late in development of the fruiting structure. The analogy is *thalidomide teratology*. Extinction may occur if threats remain, especially at 16°C and higher toxic levels. In response to these concerns, LCC has implemented a program to monitor diesel pollution in the tributary of Gore Creek that flows through LCBP. What is also relevant is that the World Health Organization declared in June, 2012 that diesel fumes are a *Level-1 human carcinogen*. Is *H. reesiae* rosecomb a wake-up call as another marker of an environmental genetic and epigenetic toxin (diesel) harmful to people?

At the time of *Rachel Carson's seminal work 'Silent Spring', published in 1962*, there was virtually no environmental movement. Her impassioned plea arose from her understanding of biological science and the fragile relationship of the ecosystem that we and all other living things rely upon.

In the words of the 'Desiderata' (1927): "You are a child of the universe no less than the trees and the stars; you have a right to live ..." Collective defiance and its subsequent disruption have always been essential to the preservation of democracy and our life-support systems!

4 (d) War Against Cancer - Have We Lost Our Way? Cancers on the increase!

Despite repeated assertions by the National Cancer Institute (NCI) that America is winning the war against cancer, the incidence of cancer has escalated to epidemic proportions, striking most families. Cancer now impacts about 1.3 million Americans annually and kills 550,000; 44% of men and 39% of women develop cancer in their lifetimes. While smoking-related cancers have decreased in men, there have been **major increases in non-smoking cancers in adults as well as childhood cancers**. Why are we not making more progress preventing cancer?

The NSW Cancer Institute predicts approximately a doubling of new cancer cases in the next 20 years from the present 35,000 per year. Total cancer costs in NSW are estimated to be \$1.627 billion for 2006. See link:

http://www.cancerinstitute.org.au/cancer_inst/publications/pdfs/abstracts/2007-11-16_cosa2007_cost-of-cancer.pdf

In 2005, R. Clapp, G. Howe, and M. Lefevre published a detailed report on the causes of cancer titled '*Environmental and Occupational Causes of Cancer; A Review of Recent Scientific Literature*'. They tell us that between 1950 and 2001 the incidence rate for all types of cancer increased 85%, using age-adjusted data, which means cancer is not increasing because people are living longer. People are getting more cancer because they are exposed to more cancer-causing agents!

Contrary to well-funded rumours, the *culprit is not just tobacco* or its numerous toxic chemical additives. Tobacco products remain the single most significant preventable cause of cancer, but ***they have not been linked to the majority of cancers*** nor to many of the cancers that have increased most rapidly in recent decades e.g., lymphomas, brain, and bone marrow cancers.

The bulk of the Clapp-Howe-Lefevre report is a cancer-by-cancer compendium of what recent human studies tell us about environmental and occupational exposures that contribute to all cancers. Many of the carcinogens are well-known: e.g., benzene, aromatic amines, petrochemicals and combustion byproducts (polycyclic aromatic hydrocarbons - PAHs), diesel exhaust, petroleum products, dioxins and outdoor air pollution.

The growing burden of cancer on children provides some of the most convincing evidence of the role of environmental and occupational exposures in causing cancers. Most children do not smoke, drink alcohol, or hold stressful jobs.

Clapp-Howe-Lefevre put it this way:

"We have learned how to save more lives, thankfully, but more children are still diagnosed with cancer every year. The incidence of cancer in all sites combined among children ages 0-19 increased by 22% from 13.8/100,000 in 1973 to 16.8 in 2000 and most of this increase occurred in the 1970s and 1980s. Epidemiologic studies have consistently linked higher risks of childhood leukaemia and childhood brain and central nervous system cancers with parental and childhood exposure to particular toxic chemicals including solvents, pesticides, petrochemicals, and certain industrial by-products (namely dioxins and PAHs)." So it is more complicated than just tobacco!

Their report makes a compelling case that numerous industrial chemicals contribute to many kinds of cancers. But where this report really sparkles is in its clear call for **prevention**.

The truth is, however, an epic struggle has been going on for 50 years between the "slash and burn" (surgery, radiotherapy and chemotherapy) camp, versus those who think the only real victory is prevention of disease. The struggle occurs across a fault line defined by money. To be blunt, there is no money in prevention. Whilst cancer treatment saves some lives, it is a booming business while cancer *primary* prevention has generally lost its way.

Apart from basic research, the cancer establishment's mindset remains fixated on 'secondary' prevention or damage control - screening, diagnosis, and chemoprevention (the use of drugs or nutrients to reduce risks from prior avoidable carcinogenic exposures) - and treatment. This is coupled with indifference to primary prevention, other than lifestyle factors - smoking, inactivity, and fatty diet. This exclusionary claim remains based on a scientifically discredited 1981 report by British epidemiologists, Sir Richard Doll and Sir Richard Peto. They **guesstimated** that lifestyle factors (e.g., smoking) are responsible for up to 90% of all cancers, with the balance arbitrarily assigned to environmental and occupational causes. The prevention-is-pointless crowd

latched onto the Doll and Peto study and spread it everywhere, contributing to numerous, preventable cancer deaths.

The USA federal NCI and the American Cancer Society (ACS) which, together are called the 'cancer establishment' adopted the Doll-Peto perspective, that cancer is a lifestyle disease - the victims themselves are responsible! The 'cancer establishment' ignored the fundamental and widely accepted Precautionary Principle and poured billions of dollars into new cancer treatments. Meanwhile, cancer incidence rates climbed relentlessly - making the cancer-treatment industry healthier and wealthier. Australia followed the ruse of the USA 'cancer establishment' and continues to refuse to shake off the stupor induced by the misleading Doll-Peto arithmetic, which pretended to prove that environment and occupational exposures are of no consequence.

Doll's fame and knighthood arose from his work in 1954 when he reported on the cancer association with e.g., smoking, exposure to asbestos and certain coal-tar chemicals. However, over subsequent decades, Doll drastically changed his views and gradually emerged as a major defender of corporate industry interests in the mistaken belief that greed helps the world go round. Doll insisted that anti-cancer campaigns should focus exclusively on scientific research, freely accessed by the pharmaceutical cartels, and not become involved in prevention research and education.

Since 1976, Sir Richard Doll's appalling track record of pro-industry bias allegedly helped to peddle their deadly wares by claiming otherwise by his subjective armchair science. Doll also allegedly accepted consulting fees from Dow Chemical Corporation, the Chemical Manufacturers Association and ICI for his work that exonerated vinyl chloride as a carcinogen - a conclusion the World Health Organisation rejected. See link: http://www.whale.to:80/v/walker_doll.html Among his numerous, outrageous scientific blunders, in 1985, Doll wrote to the judge of an Australian Royal Commission, investigating claims of veterans who had developed cancer following exposure to the herbicide Agent Orange in Vietnam, in strong support of the defense claims of its major manufacturer, Monsanto. He stated that, "*TCDD (dioxin), is at the most, only weakly and inconsistently carcinogenic in animal experiments*". In fact, dioxin is among the most potent known tested carcinogens, apart from confirmatory epidemiological evidence. Doll's defense, resulted in denial of the veterans' claims. What is so scandalous is that Doll received a daily consultant's fee of \$1,500 from Monsanto Corporation during the 1980s when he was investigating its product, Agent Orange, as a carcinogen. Separately, in 1985, the U.K. Society for the Prevention of Asbestos and Industrial Disease (SPAID) **criticized Doll for manipulating scientific information.**

The war against cancer must be fought by strategies based on primary prevention, rather than reactively on 'secondary' prevention or damage control. This war must be waged by leadership accountable to the public and not special interests. Comprehensive cancer prevention programs need to reduce exposures from all avoidable sources including cancer-causing vehicle emissions. Cancer prevention programs focused on tobacco use, diet, and other individual behaviours disregard the lessons of science - according to Clapp-Howe-Lefevre (2005) who offer some guidelines for preventive action:

1. The least toxic alternatives should always be used.
2. Partial, but reliable, evidence of harm should compel us to act on the side of caution to prevent needless sickness and death.
3. The right of people to know to what they are being exposed be protected.

While a handful of contemporary public health epidemiologists still work in this way, those linked to industry or to government bureaucracies invariably work not for the people, but to defend the profitability of corporations, and to legitimize the authority of the State with dependent patronage. Their research is constantly updating not 'what is best for the people' but what risk to the people that 'industry' can profitably get away with and how best to fund their

armchair science. Whilst some have argued that Sir Richard Doll should be remembered with fondness, respect and gratitude, there are poignant lessons for all to learn.

Prior to his death, Doll finally admitted: *"It does look as if it is the cancers that are principally caused by hormones that are not affected by smoking. Most of the other cancers throughout the body are induced by exposure to chemicals, often environmental ones."*

For decades, powerful groups of interlocking corporate interests, with the highly profitable cancer drug industry at their hub, have dominated the losing war against cancer. Research has found e.g., that up to one-fifth of lung cancer deaths are attributed to exposure to fine particles from fossil fuel combustion. See link:

<http://www.newscientist.com/article/mg17323331.100-big-city-killer.html>

This is unwelcome news for the campaigners against passive smoking! A cancer-research moratorium in Australia is long overdue.

In Australia, our 'regulators' need to confront the cancer-causing polluters directly. **The public should be concerned that uncertainty is too often parlayed into an excuse to do nothing until more research can be conducted.** Why the need for a 'trial' of filtration in the M5 East tunnel when it is already proven technology overseas? Government's answer to cancer is to **'blow it in the wind!'** Similarly, CSIRO conducts flawed studies of 'health benefits' of E5 and E10 fuels 'under Australian conditions' but excludes E85 as a clean alternative fuel. So, the winnable war against cancer continues to be lost! See link: http://www.preventcancer.com/press/pdfs/Stop_Cancer_Book.pdf

4 (e) Air-Pollution Odours and Health Effects

The opening of the Lane Cove Tunnel resulted in a number of complaints from residents about air-pollution odour. The implication being that this is associated with 'groundstrike' by fumes of unfiltered tunnel pollution from the tunnel's exhaust stacks. The same experience continues to be reported in 'odour diaries' by residents affected by toxic fumes from the M5 East stack. In the past, unpleasant environmental odours have been considered warning signs or indicators of potential risks to human health but not necessarily direct triggers of health effects. The latter assumption has now changed because certain environmental malodours have now been shown to trigger health symptoms.

Overseas, community-wide exposure to toxic substances is an increasing source of large-scale civil litigation, and claims for psychological damage are commonly included. However, in such a setting, the possibility of malingering can lead to an exaggerated symptom report and invalidate data.

During the last decade, great progress has been made in understanding the smell or olfactory system and how environmental odours can influence behavioural and cognitive function. From a regulatory perspective, 'odour pollution' is most frequently handled as a nuisance abatement issue pertaining to specific sources. Nuisances can be defined on both an attitudinal and behavioural basis where the latter might include such adaptations as staying indoors and keeping windows closed. Unfortunately, with the ongoing debacle of the M5 East tunnel ventilation, NSW public health officials have great difficulty in distinguishing odour effects as 'annoyance' or a 'health effect'.

The health impact of environmental odour pollution varies with both the source and the exposed population. Whilst some environmental odours herald toxicologically important exposures, some potent odourants, such as hydrogen sulphide (rotten-egg gas), have odour thresholds several orders of magnitude lower than their thresholds for irritant (or other toxic) effects.

Notwithstanding this margin of safety, residents impacted by such emissions often report not only odour-related annoyance but also symptoms of illness.

The community is well aware that some odour chemicals may be accompanied by other chemicals that are known to be toxic. Hence the perception of odour on the part of a community member may very well signal impending danger. For certain odours, especially sulphurous gases, this can give rise to a major paradox. The same concentration of hydrogen sulphide detected from a boiled egg may elicit tasting enjoyment but the same concentration perceived to be coming from an environmental source such as a tunnel exhaust stack can elicit a complaint and the report be accompanied by nausea, headache and shortness of breath.

Heightened symptomatic responses to odours are well documented in two medical conditions – pregnancy and asthma. The majority of women report nausea and vomiting in the first trimester of pregnancy. Odours (e.g., household, food and industrial chemicals) can act as triggers of nausea and vomiting during this period. For asthma, Shim and Williams (*Amer.J Med.* 1986; vol. 80, p18-22) found that 90% of a group of 60 asthmatics surveyed reported exacerbations of asthma in relation to odour exposures, and nearly 40% had visited emergency rooms after such incidents. On repeat testing with a placebo (saline), the results of abnormal lung function in the asthmatics were not affected. Several separate studies have shown that exposure to odours by asthmatics, whose skin prick-testing confirmed a lack of immunological reaction, could provoke asthmatic attacks. Therefore, individuals with odour-triggered respiratory symptoms or ‘sensory hyper reactivity’ should not be assumed to have asthma, but rather should be considered in terms of a broader differential diagnosis.

Reports have also shown that odours can serve as a cue for an acute ‘stress’ state in an individual who is pre-disposed to view the odour source as posing a toxicological threat. This stress could either precipitate symptoms directly through mechanisms (e.g., nausea and headaches) of the central nervous system (CNS), or lead to heightened awareness and reporting (e.g., irritation of the respiratory-tract mucosa).

Although somewhat controversial, odours are thought to play a part in some episodes of ‘mass psychogenic illness.’ Such episodes typically occur among closed populations under some degree of psychosocial stress. In recent studies, physiological monitoring and functional CNS imaging have, in real time, confirmed that the CNS system reacts differently to pleasant and unpleasant odours.

There are at least three mechanisms by which ambient odours may produce health symptoms. The first mechanism is where the symptoms can be induced by odourants at levels that also cause irritation or other toxicological effects. Irritation - rather than the odour - is the cause of the health symptoms. Examples include ammonia, chlorine and formaldehyde. Sensory irritation can be induced by a single odourous chemical or by the aggregate effect of low concentrations of compounds as in exhaust-pipe emissions containing multiple volatile organic chemicals. When irritant mixtures or compounds come into contact with the upper and/or lower respiratory tract many systemic effects can occur including 1) altered respiratory rate; 2) reduced respiratory volume; 3) increased nasal secretion; 4) lacrimation (watery eyes) and tearing; 5) sneezing; and 6) increased blood pressure.

The second mechanism is where health symptoms occur when the odourant concentrations are not irritating. The way this happens is not well understood. Brain structures that are stimulated by unpleasant experiences are preferentially stimulated when smelling e.g., hydrogen sulphide. Many studies have shown that unpleasant odours can impair mood. When malodours are present as a result of M5 East stack pollution, the residents have reported symptoms of increased levels of tension, depression, anger and fatigue. Some researchers have suggested that conditioned or learned associations may play a role in perceptions and health symptoms induced by malodours.

The third mechanism is where a co-pollutant is present in the pollution mixture and is responsible for the reported health symptoms. Odours can arise from the incomplete combustion of fossil fuels. Harmful effects may arise from certain odourless gases. Nitrogen dioxide, a gas produced in the combustion of fossil fuels, is odourless but is an irritant and can trigger asthma attacks. Fine exhaust particles that carry carcinogenic chemicals can dissolve in the respiratory tract and initiate tissue injury that induces the inflammatory response. The associated acute respiratory effects can be augmented in the presence of odourous compounds.

4 (f) 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. [Dr. Gary Whitten reports that E10 achieves a qualified 50% reduction in PM2.5 particle levels in test vehicles.](#)
- 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.

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 adverse impacts on health as well as improve air quality.

A problem in NSW is not only the lack of commitment by the 'Energy Minister' but a total absence of enforcement of the existing legislation. This is also illustrated by the Oil Cartels FAILING to provide genuine E10 but rather under-blended E8.

Australia's capacity to produce alcohol from sugar-cane and wheat is immense.

Recently the British Heart Foundation reported that even cyclists riding to the office along busy roads in cycleways adjacent to bus lanes do themselves more harm than good by inhaling the toxic diesel fumes from buses and urged that cycleways be located away from major roads.

Costs of morbidity and mortality due to exposure to fossil fuel-combustion in Sydney alone exceed \$2-3 billion annually and more people die each year from motor vehicle emissions than in road accidents.

A Case Study in Tokyo showed that by reducing particle pollution by 50% was accompanied by a health-cost benefit in one year alone of at least \$A47 billion.

The Australian Medical Association, (See below) endorsed by the Australasian Lung Foundation, who reported 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.

AMA BACKS MANDATORY USE OF ETHANOL IN PETROL ON HEALTH GROUNDS

AMA President, Dr Mukesh Haikerwal, has today written to the Government's Biofuels Taskforce detailing the AMA's support for the mandatory use of ethanol in petrol in the interests of protecting and improving human health. Dr Haikerwal said the AMA wants to see the biofuels debate in Australia shift from economic issues to human health issues.

"The AMA is a strong advocate on initiatives related to environmental impacts on human health such as global warming," Dr Haikerwal said.

"We are equally passionate about the impact of vehicle emissions on human health and we would encourage governments to pursue responsible measures to reduce emissions.

"The AMA considers the use of biofuels such as ethanol in petrol as a positive move.

"In our opinion, there is incontrovertible evidence that the addition of ethanol to petrol and biodiesel to diesel will reduce the deaths and ill-health associated with the emissions produced by burning those fuels," Dr Haikerwal said.

According to the AMA's submission to the Biofuels Taskforce, there are three components of present vehicle emissions that have been shown to damage human health: the particulates (particularly PM 2.5); the aromatic component (polycyclic aromatic hydrocarbons); and the gaseous irritants such as ozone (O3) and nitrous oxide (NO2).

The AMA supports any interventions that will reduce the above three emissions and their impacts.

The AMA believes that the following interventions would reduce the negative health impacts:

- 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.**

27 July 2005

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In general, the current State and Federal Governments and their respective bureaucracies have demonstrably failed to provide good governance and to constitutionally serve the electorate with a sense of common decency, justice and a duty of care.

Clean alternatives to fossil fuels

The Federal Government should establish a clear policy and maintain its commitment in support of expansion of alternative biofuels – especially ethanol-blended petrol, biodiesel and liquefied natural gas.

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 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

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 leukaemia.
- Ethanol is a renewable fuel, typically produced from plant matter.
- Ethanol-blended fuels account for 12 % (and growing) of all automotive fuels sold in the United States.
- Now is the time to further promote ethanol in blended fuel with a banning of MTBE in USA.
- Ethanol-blended fuels reduced the CO₂-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.

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 (and enforce) fuel standards to include renewable biofuels such as biodiesel and ethanol-blended petrol.
- Address the escalating costs of growing canola crops 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 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.

Our nation's dependence on diesel must be reassessed in light of growing scientific evidence that diesel exhaust poses a major health hazard, particularly to children and the elderly. Diesel emissions are comprised of a witch's brew of potent carcinogens, reproductive toxicants, irritants, and other hazardous chemicals. This complex mixture of fine particles and toxic chemicals is linked to cancer, asthma and other respiratory exacerbations, and premature death. Fortunately, alternatives such as electric, natural gas, ethanol and biodiesel and other less polluting fuels are technologically feasible and, in some cases, economically advantageous. The move away from diesel to clean alternatives will take a concerted effort by public and private operators and continued pressure from the people most affected by the health impacts of diesel exhaust.

The following is strongly recommended as an antidote to our nation's dependence on diesel:

- Federal and State governments must end their "hands off" diesel policy immediately and act quickly to set new stringent standards for heavy duty trucks, buses, trains, marine vessels, construction and agricultural equipment and other diesel vehicles and equipment to expedite the transition toward cleaner alternative fuel vehicles;
- Legislative and municipal bodies should provide financial incentives to operators of diesel vehicles and equipment to encourage them to purchase new alternative fuel vehicles or to retrofit their existing diesel vehicles;
- Public transit agencies and governmental entities should lead the way toward clean fuel vehicles by committing to purchase only alternative fuel buses and other vehicles for their fleets; and
- Private fleet operators should take a major step to protect their workers and local communities by immediately beginning to purchase alternative fuel heavy duty trucks, buses and equipment as part of their fleet.

HIGHLIGHTS

<http://ntp-server.niehs.nih.gov/files/DieselExhaust.pdf>

- Diesel exhaust is a mixture containing over 450 different components, including vapours and fine particles coated with organic substances. Over 40 chemicals in diesel exhaust are considered toxic air contaminants overseas. Australia does not list diesel as an air toxic. Exposure to this mixture may result in cancer, respiratory effects, and other health problems.
- California's Scientific Review Panel has unanimously endorsed the official listing of diesel exhaust as a toxic air contaminant, due to its cancer and non-cancer health effects. Australia has not!
- Diesel exhaust has been listed as a known carcinogen under California's Safe Drinking Water and Toxic Enforcement Act (Prop. 65) since 1990. Australia has not! Many components of diesel exhaust, such as benzene, arsenic, dioxins, and formaldehyde, are also known carcinogens. Other components, such as toluene and dioxins, are known reproductive toxicants.

- For the same load and engine conditions, diesel engines exhaust 100 times more sooty particles than petrol engines. As a result, diesel engines account for an estimated 26
- percent of the total hazardous particulate pollution (PM10) from fuel combustion sources in our air, and 66 percent of the particulate pollution from on-road sources.
- Diesel engines also produce nearly 20 percent of the total nitrogen oxides (NOx) in outdoor air and 26 percent of the total NOx from on-road sources. Nitrogen oxides are a major contributor to ozone production and smog.
- The health risk from diesel exposure is greater for children, the elderly, people who have respiratory problems or who smoke, people who regularly strenuously exercise in diesel-polluted areas, and people who work or live near diesel exhaust sources.
- According to an expert estimate, lifetime exposure to diesel exhaust at the outdoor average concentration (2.2 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$)) may result in about one in every 2,000 people developing cancer due to this exposure. This estimate increases to as many as one in every 1,200 at levels found in the South Coast Air Basin in Southern California ($3.6 \mu\text{g}/\text{m}^3$), and to even higher risks for those living near freeways or in highly polluted urban communities.
- Dozens of studies link airborne fine particle concentrations to increased hospital admissions for respiratory diseases, chronic obstructive lung disease, pneumonia, heart disease and death. Recent evidence indicates that diesel exhaust exposure may contribute to asthma.
- About 127 million Americans -- half of the nation's population -- live in regions with air quality that does not meet federal standards for certain air pollutants. More than 60 percent of preadolescent children, including children with asthma, live in "nonattainment" areas. In the United States, there are an estimated 10.3 million people living with asthma.
- In California, there are six million children under the age of fourteen, 90 percent of whom live in areas that fail to meet state and federal air quality standards. According to the American Lung Association, there are over a half million children with asthma in California.
- Asthma is on the rise. In the United States, age-specific death rates from the disease increased 118 percent between 1980 and 1993. African-American and Latino children have a higher risk of asthma than white children. Moreover, African-American children are four times more likely to die from asthma compared to Caucasian children.
- Cleaner alternatives to diesel engines are readily available. Alternatives include electric, liquefied natural gas (LNG) or compressed natural gas (CNG) buses and trucks. For cars, ethanol, CNG and LPG.
- Although initial purchase prices may be higher for alternative fuel buses and trucks, federal, state, and local funds are available to offset these higher costs. These funds are specifically earmarked for clean technologies and would not otherwise be available for these purchases.
- For transit authorities, use of alternative fuel buses can generate operational cost savings. Sacramento RTD's CNG bus fleet is currently demonstrating cost savings of 20-40% per mile when compared to diesel counterparts. Over its lifetime, a CNG bus will save

- 190,000 gallons of diesel fuel compared to a new diesel bus, decreasing our dependency on petroleum.
- Diesel buses and trucks are important contributors to smog (ground-level ozone) and fine toxic soot, two pollutants that have recently come under increased scrutiny because of their important public health impacts. Purchasing alternative fuel vehicles will reduce smog and fine soot emissions considerably. For example, operating a natural gas bus instead of a new diesel bus is equivalent to eliminating the smog and soot from 17-55 passenger cars.