28 Warne Street
Pennant Hills NSW 2120
9 September 2014

Development Assessment Systems & Approvals
Department of Planning & Environment
GPO Box 39 SYDNEY NSW 2001

A further submission

Dear Manager and staff assessing my submissions,

Item 265

Another reason to Scrap the M1 – M2 tunnel

265. 2013 6 Mar Ray Kearney PhD's **57 page** Submission to the 2013 Federal Senate Standing Committee Inquiry on:

'The impacts on health of air quality in Australia'

clearly shows diesel exhaust (p4) is a major problem globally and Australian governments are trying to avoid putting in place enforceable legislation instead of guidelines.

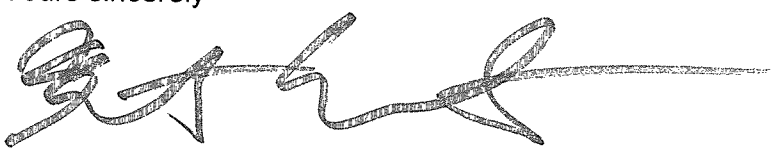
Pages 42/43; *LANE COVE TUNNEL AIR QUALITY IMPACT ASSESSMENT ALTERNATIVE VENTIALATION OPTIONS*

RECOMMENDATION
*It is recommended that the **RTA** investigate options for reducing the carcinogenic health risk at both the ground-level and elevated receptors. These options may include, but should not be limited to, increasing the stack height and/or exhaust velocity. Increasing the exhaust velocity may be achieved by segmentation of the stack so that the higher exhaust velocities may be achieved at less than maximum flowrate. Nick Agapides 26 June 2002.*

As the RTA is the EIS delegated sponsor, I question the integrity of the RTA to be able to make and impartial EIS that withstand informed objections. Why wasn't this study in the EIS?

Another political problem:
The Kearney paper related to a 2013 Senate Inquiry. Where is there evidence that any national NSW senator or politician has raised Kearney's paper to question risks from diesel air pollution?

Yours sincerely



Peter Waite

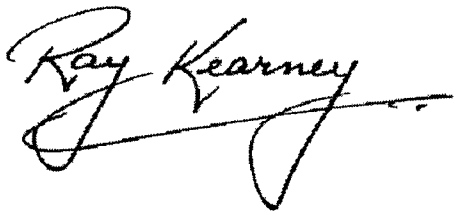
2013 Federal Senate Standing Committee Inquiry

'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 - 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.



Ray Kearney PhD, OAM

Date: 6th March, 2013

Hon. Assoc. Professor (Retired)
Dept. Infectious Diseases & Immunology
The University of Sydney

Correspondence:

24 Alder Avenue,
Lane Cove West
NSW 2066

Phone: (02) 94285336 (h)
E-mail: ray.kearney@sydney.edu.au

2013 Federal Senate Standing Committee Inquiry

'The impacts on health of air quality in Australia'

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.

For further information, contact:

Committee Secretary
Senate Standing Committees on Community Affairs
PO Box 6100
Parliament House
Canberra ACT 2600
Australia

Phone: +61 2 6277 3515
Fax: +61 2 6277 5829
Email: community.affairs.sen@aph.gov.au

Table of contents

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 PM2.5 particles, the National Environment Protection Council (NEPC), has NOT implemented a STANDARD for carcinogenic Particulate Matter (PM) of 2.5μm 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)

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 PM_{10}, $PM_{2.5}$ and $PM_{1,}$ are as follows:

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

***"$PM_{2.5}$ measurements** also provide information mainly on particles generated by mechanical process, but the contribution from combustion process (N + A) modes becomes significant. Thus interpretation of $PM_{2.5}$ data could become very complex in order to distinguish the contribution from different type sources. It follows that the application of this parameter as bases for standards may not adequately facilitate control of particle emissions and concentrations."*

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

A serious and fundamental defect in monitoring air pollution is the <u>failure</u> 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 ($\mu g/m^3$) 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, <u>reduction in melatonin</u> (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 <u>long-term exposure</u> 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: <u>levels that can be thousands of times below public safety limits.</u> 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 <u>synergistic or additive</u> in causing health impacts.

11. The WHO has stated that for particle matter (PM), the PM_{10} is a 'marker' and there is no threshold below which there is no health impact. Nor are PM_{10} levels relevant for particles in tailpipe exhaust. The current 24-hour PM_{10} standard of 50µg/M^3 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 0.01 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 /M^3 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., PM_{10} of 50µg/M^3/24 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 dickering 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 PM_{10} 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 dickering 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 2.5μm 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

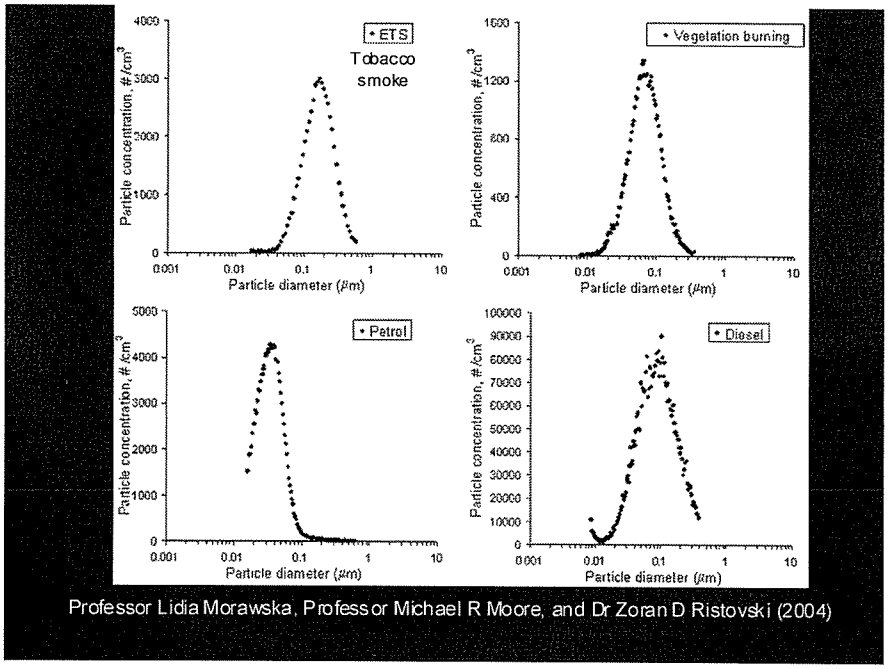
	Fine Mode (<PM 2.5)	**Coarse Mode PM10**
Formed from:	Gases	Large solids/droplets
Formed by:	Chemical reaction; nucleation; condensation; coagulation; evaporation of fog and cloud droplets in which gases have dissolved and reacted.	Mechanical disruption (e.g. crushing, grinding, abrasion of surfaces); evaporation of sprays; suspension of dusts.
Composed of:	Sulphate, $SO_4^{=}$; 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 peaks[1] - 0.03μm and 0.1μm. 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 PM2.5 or less – only a 'guideline'.

See also diagram below.



Morawska &Thomas[2] report that only 3% of combustion particles between 0.1μm and 1μm are present in PM_{10} measurements. Despite this research conducted for the Queensland Environment Protection Agency, the RTA continues to claim that PM_{10} measures all particles less than 10μm in size. Monitoring by NSW EPA excludes most particles 1μm and less. About 90% (by mass) of diesel particulates[3] are less than 1μm and are missing in the PM_{10}/M^3 measurements in most EIS Reports. As a consequence, PM_{10} measurements of air quality and those of air-streams vented from tunnel stacks are seriously under-estimated.

Morawska & Thomas[4] concluded: "*PM_{10} measurements provide information almost entirely on particles generated from mechanical processes. In an urban environment, this could mean particles re-suspended by vehicular traffic and mechanical wear and tear of tyres,* ***but not on emissions of motor vehicles.***"

---

[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 PM_{10} measurement is not an instrument for evaluating traffic emissions, the air pollution data and the calculated health-risks are also underestimated. The above authors conclude that PM_1 measurement provides very good information about contributions from the combustion engine, and distinguishes it from suburban background.
The fact that PM_1 contributions are about two-fold greater in traffic aerosol than in ambient air[5] is a serious deficiency in pollution monitoring by the RTA. Use of PM_{10} data serves to hide exceedances of air quality standards. By using PM_{10} measurements, the bulk of tail-pipe particulates are excluded and what is analysed is mainly re-suspended dust from the road surface[6]. The exclusion of combustion particles suppresses data of the enormous surface area (> 100 fold) of toxic respirable particles missing from PM_{10} measurements.
Several overseas countries have a standard for PM_{10} and $PM_{2.5}$. Australia has only now introduced a 'guideline' for $PM_{2.5}$ additional to a standard for PM_{10}. It is misleading to imply PM_{10} is stringent and includes all particles as fine as $PM_{2.5}$ and PM_1.

A major flaw in monitoring pollution from tunnel stacks is that the fine particulate matter of less than one micrometre (<PM_1), 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 *ar*[redacted] 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 0.5um 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 1μm (Brown *et al.*, 2000). NSW 'EPA' disclosed that its monitoring of atmosphere pollutants does **not** include those particles 1μm or less. (International Workshop on Tunnel Ventilation, June 2000). Therefore, the 90% of diesel emission is not included in the mass of PM_{10}/M^3 recorded in routine air-quality monitoring.

[redacted]

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.

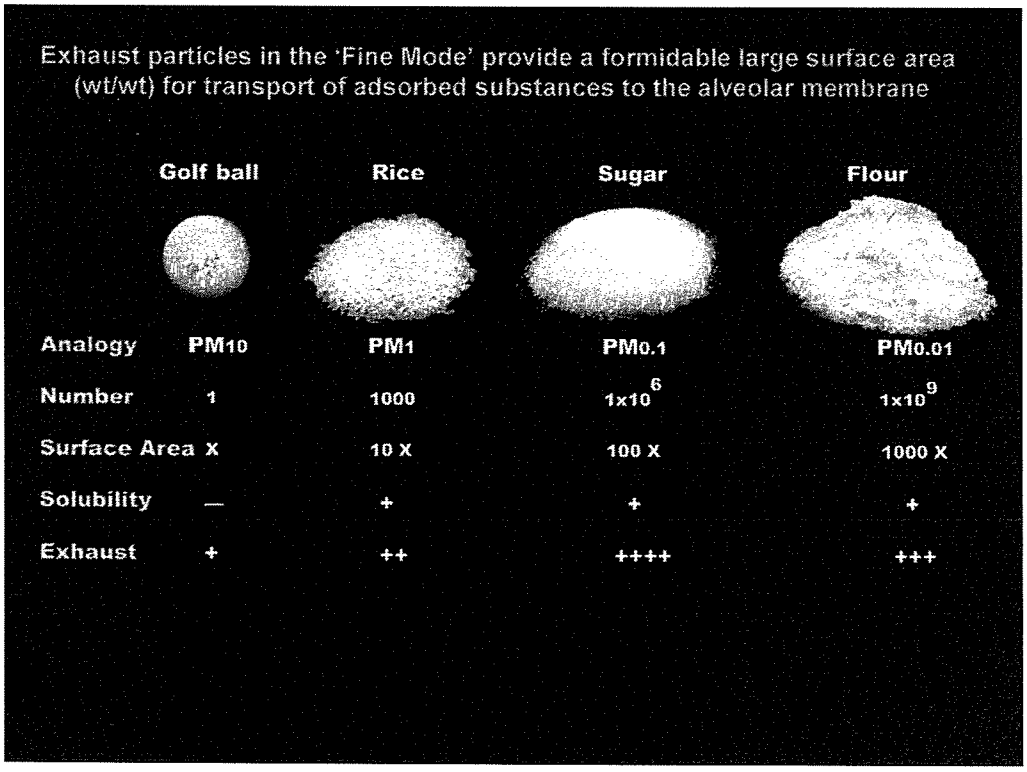
Contemporary risk management is about trying to get the levels as low as possible. We should not work up to a standard, we work down to a risk – that is not what has happened in this process. **A solution is to install filtration in the vehicle tunnel stacks in residential areas.**

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

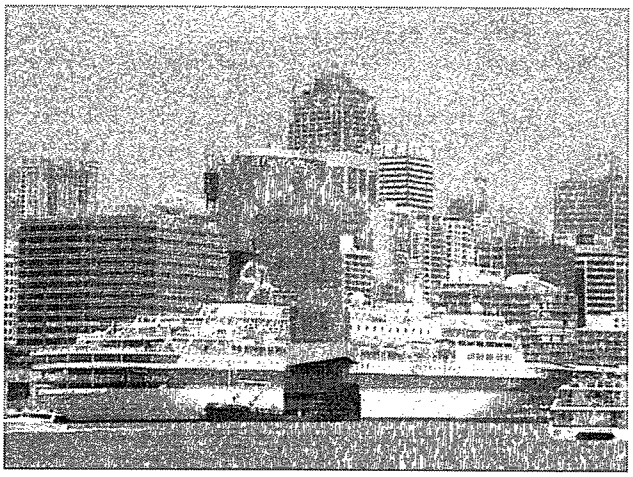
The lack of an identifiable safe threshold level for particulate exposure points to the need to reduce ambient particulate concentrations to as low a level as practical. This means that e.g., *NSW Planning* needs to acknowledge there are feasible, practicable and warranted measures to reduce toxic emissions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

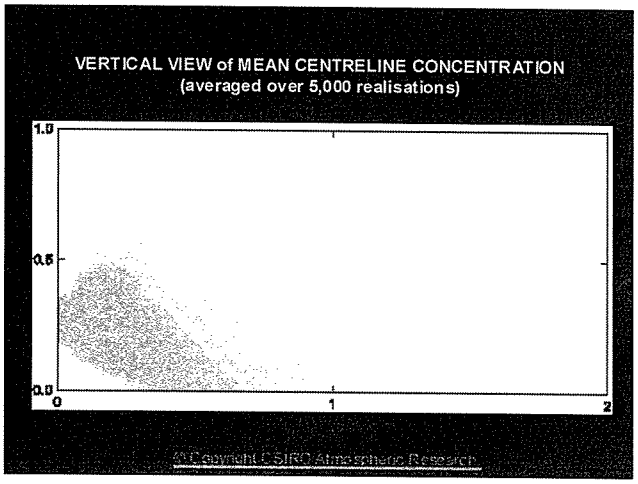
The 24 hour **averages** of PM_{10} do not identify *short-term pollution sources* e.g., a.m. and p.m. peak periods.

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 PM_{10} mass/M^3 measurements are made.



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 PM_{10} 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:

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

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

(c) **Coarse**, C, particle (>1.0μm). Almost all particles in the coarse particle mode 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 PM_{10}, $PM_{2.5}$ and PM_{1} are as follows:

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

*"**$PM_{2.5}$ measurements** also provide information mainly on particles generated by mechanical process, but the contribution from combustion process (N + A) modes becomes significant. Thus interpretation of $PM_{2.5}$ data could become very complex in order to distinguish the contribution from different type sources. It follows that the application of this parameter as bases for standards may not adequately facilitate control of particle emissions and concentrations."*

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

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 PM_{10} 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 PM_{10} instrument for measuring emission pollution from the tunnel stacks.

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

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

From L. Morawska and S. Thomas (2000)

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

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

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

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

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

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

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

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/m^3). 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 PM2.5 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. PM2.5 particles are **soluble** in the lungs, off-loading their cancer-causing cargo.

The bulk of the PM2.5 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
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."	

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 PM_{10} monitors) enhance allergic inflammation.

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

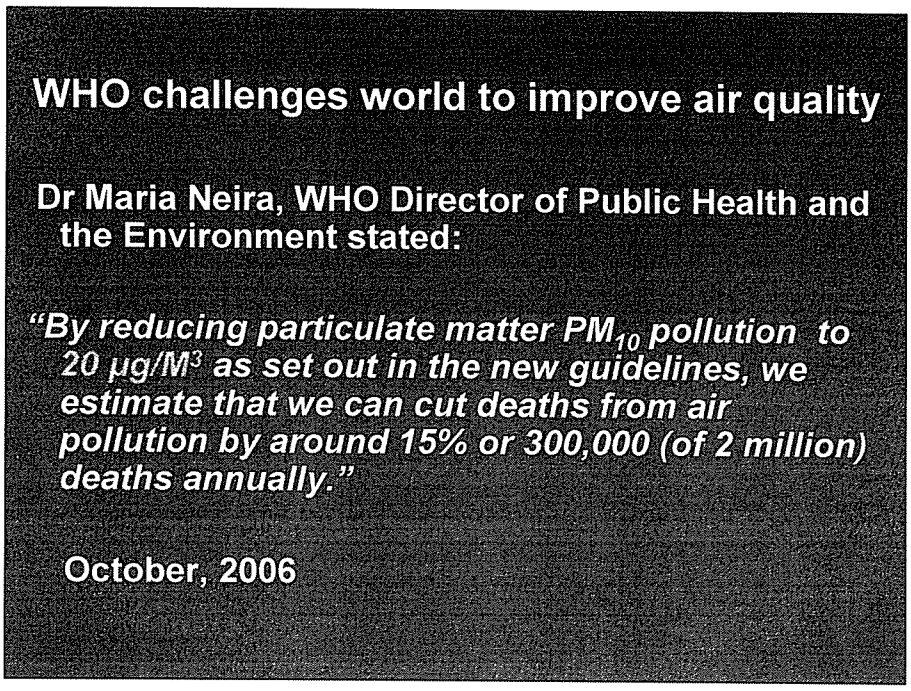
Government/consultant Air Quality Reports and EIS's do not provide evidence of the size of the 'at risk' population (e.g., asthmatics, bronchitics, cardiac patients, children < 5 years). The extent to which a 'risk' population is not quantified e.g., in the stack precincts, it is impossible to undertake specific risk assessments, only guesses are offered to [redacted] 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 M^3 of air daily. Government/consultant Air Quality Reports and EIS's fail to provide a thorough[redacted]e 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 $50\mu g/M^3$.



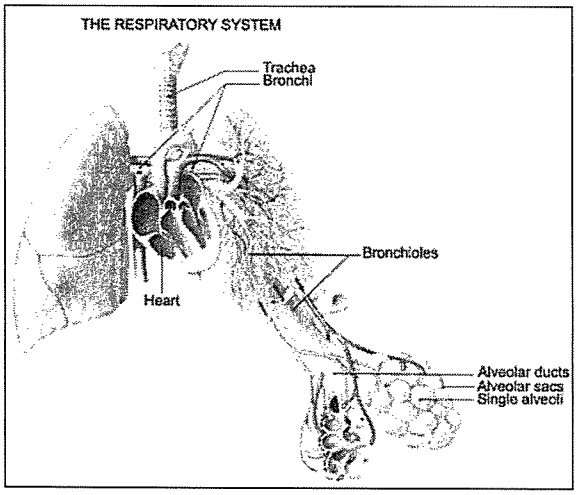
2 (i) Health impacts from short-term exposure during peak-hour are hidden

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**.

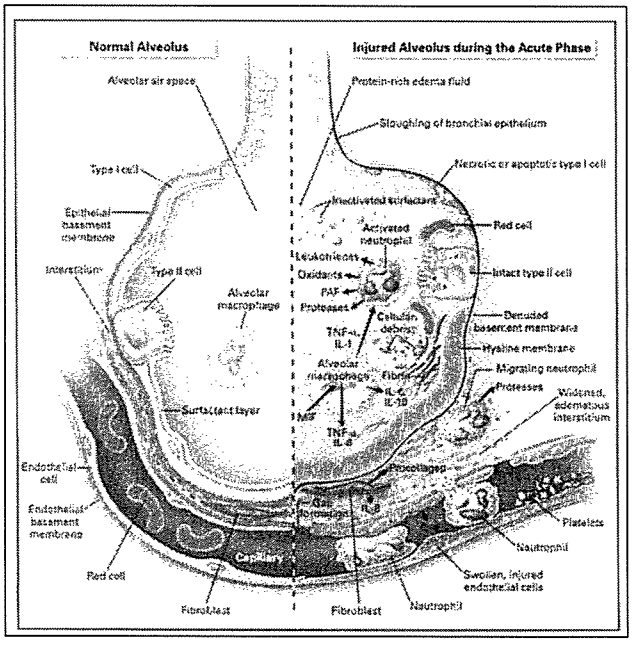
2 (j) Adverse impacts on health and well-being

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:

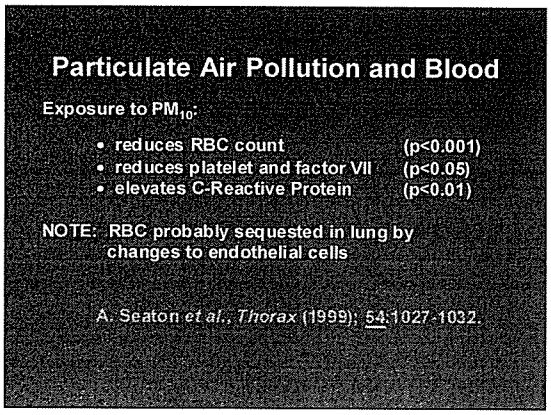
a. inflammatory lung diseases e.g., asthma, bronchitis and alveolitis
b. increased cardio-vascular disease
c. increased risk of myocardial infarction in susceptible persons
d. risk for exercise-induced heart damage
e. limited blood flow and increased blood clotting
f. increased mucous production and airway hyper-responsiveness
g. 1in 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

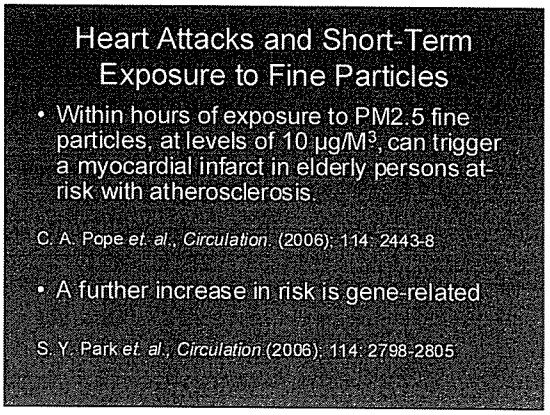


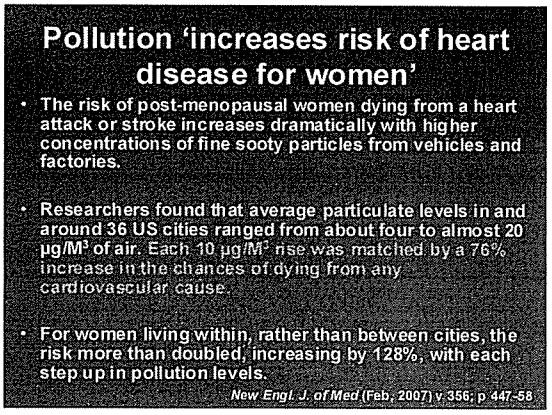
Lung and Heart Relationships in Gas Exchange

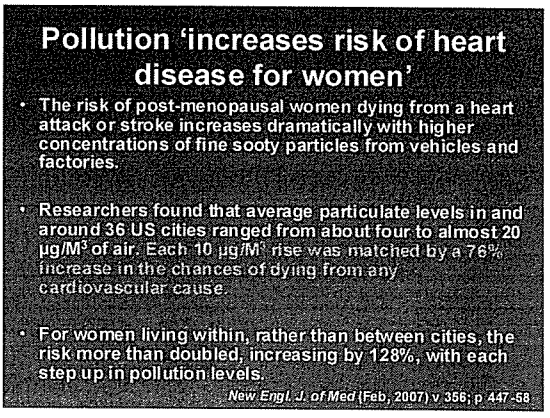


Normal Alveolus and Effect of Acute Inflammation









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/m^3 increase in long-term PM2.5 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 air-borne particulate matter (PM) and patient survival after hospital admission for acute coronary syndrome (ACS) found death rates increased with increased exposure to PM2.5.

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

The study found that for every 10μg/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 μg/m^3, compared to patients exposed to PM2.5 levels of 10μg/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.

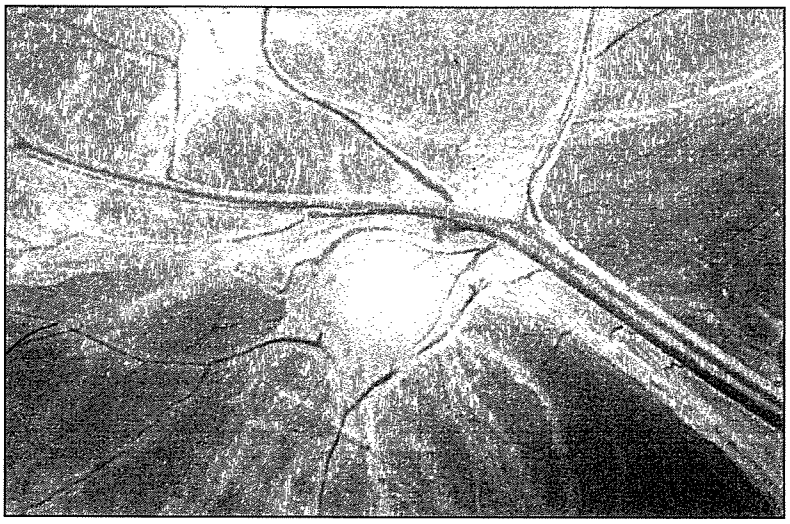
There have been too many studies world-wide which directly link vehicle emissions with mortality and morbidity for government to engage in a study where they would not be able to find the associations between vehicle emission and community health These strategies were used to hide the public health hazards of asbestos for over a century [redacted]es [redacted] 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:

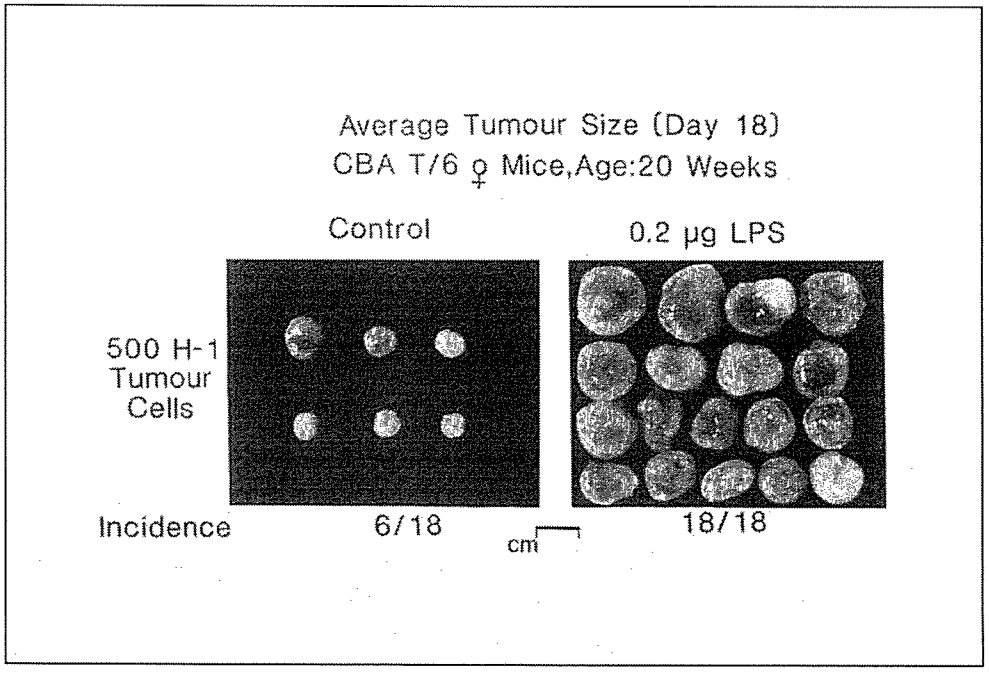
2 (n) Examples of the multi-factorial nature of cancer development.

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,

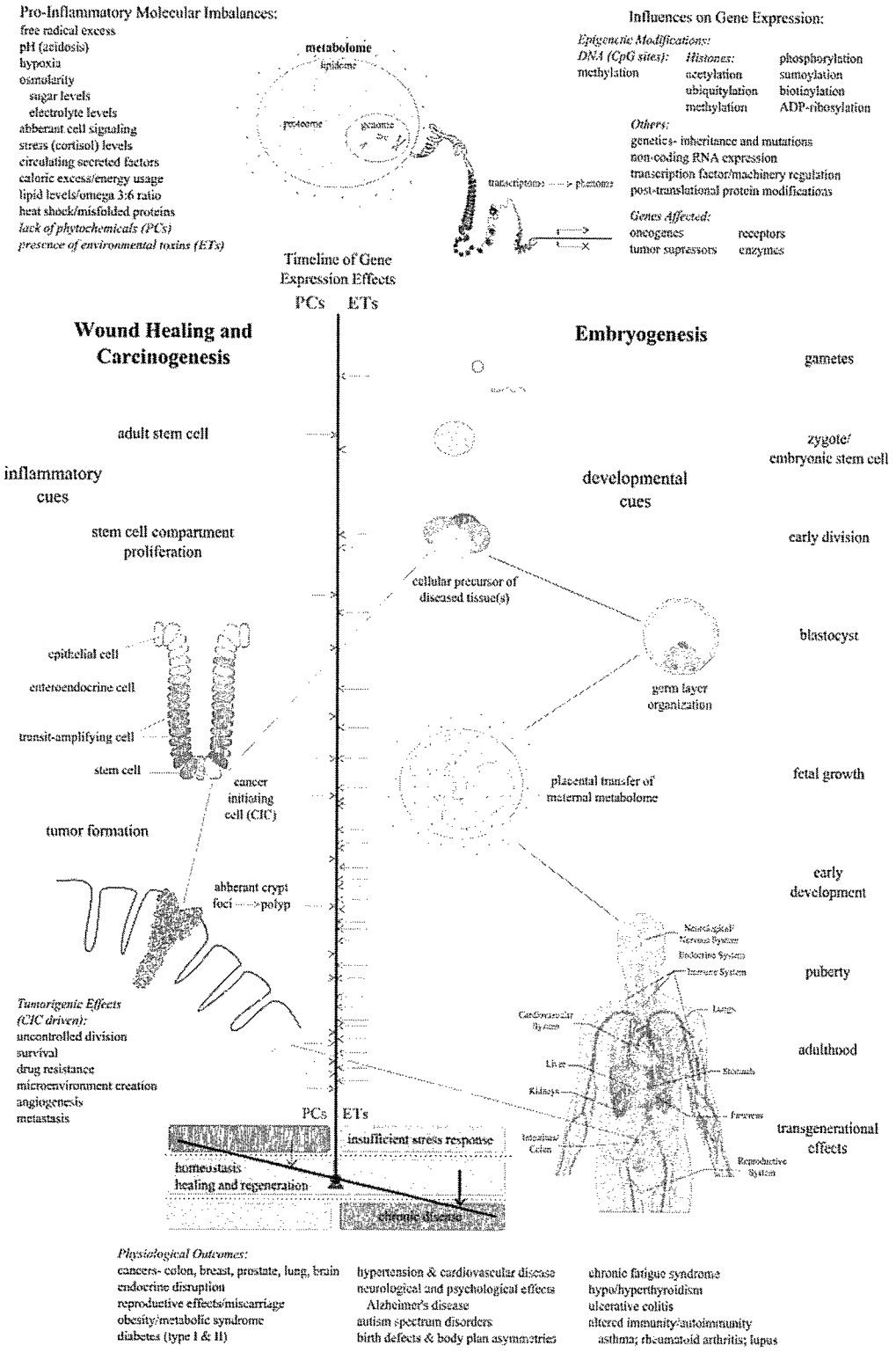
UV-radiation, asbestos or tobacco smoke. Surgery (tissue injury induced-inflammation) to remove a primary tumour is long documented to be a potent accelerator of any residual secondary tumour deposits.
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3365481/
http://jcb.rupress.org/content/192/4/547.full



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)



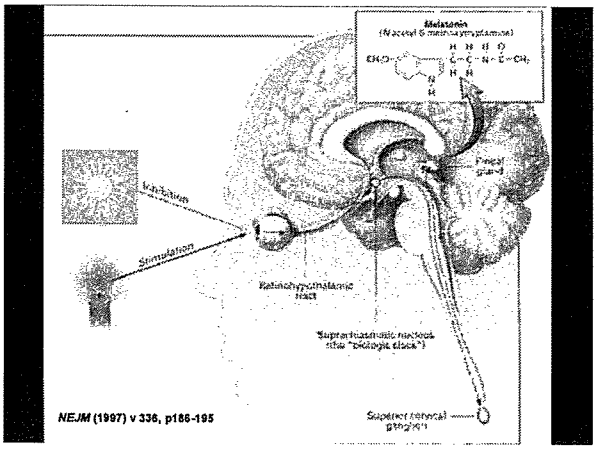
Demonstration by Dr. R. Kearney of acceleration of experimental tumour growth by inflammation induced by LPS. It would be expected diesel particles plus LPS would give greater acceleration of tumour growth.

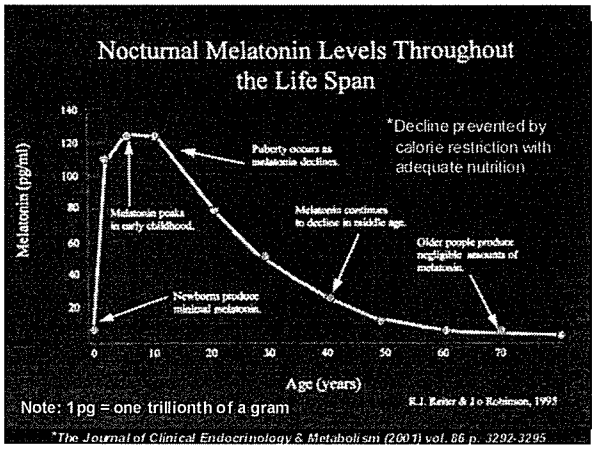


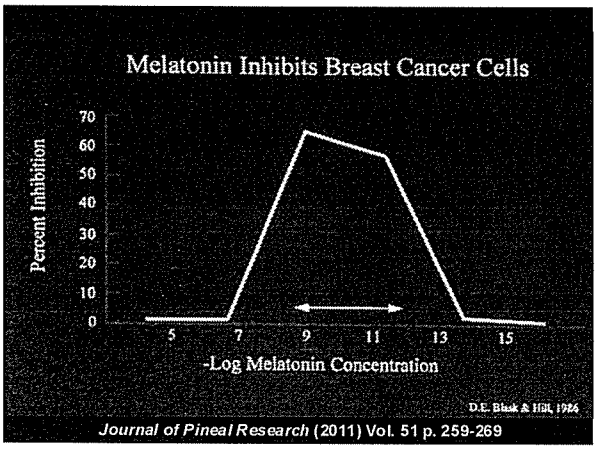
The

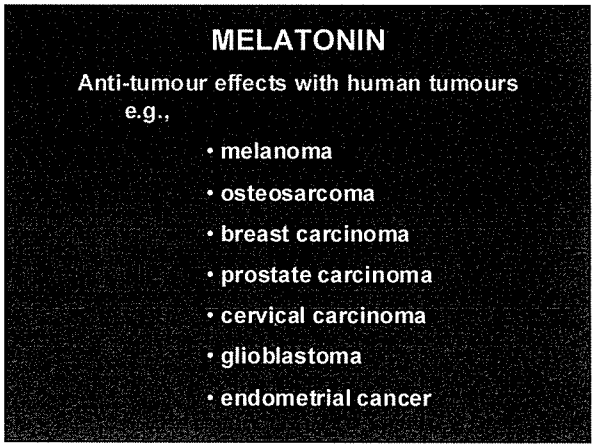
Dynamic epigenetic interplay of inflammation, environmental toxins, and phytochemicals theorized to lead to homeostatic imbalance and chronic disease in modernized societies; the manifestation and extent of which depends upon the hypothetical timing of critical gene expression changes in stem cells during embryogenesis, wound healing, and colon carcinogenesis (in this scenario), with other outcomes plausible.
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3365481/

2 (n) - 2 Properties of melatonin including its anti-cancer effects









Melatonin has many important biological properties including the prevention of cell death or injury from 'free radicals' generated in the mitochondria as a result of oxidative stress. The hormone also prevents initiation of cancer induced by chemical carcinogens. In established tumours, melatonin has anti-tumour effects in a dose-dependent 'bell-shaped' response. Melatonin is produced in picogram (a trillionth of a gram) amounts. Electro Magnetic Radiation (EMR) exposure is proven to diminish levels of melatonin.

"Obviously, melatonin's ability to protect DNA from oxidative damage has implications for many types of cancer, including leukemia, considering that DNA damage due to free radicals is believed to be the initial oncostatic event in a majority of human cancers [Cerutti et al., 1994]. In addition to cancer, free radical damage to the central nervous system is a significant component of a variety of neurodegenerative diseases of the aged including Alzheimer's disease and Parkinsonism.
In experimental animal models of both of these conditions, melatonin has proven highly effective in forestalling their onset, and reducing their severity [Reiter et al., 2001]."
http://www.bioinitiative.org/freeaccess/report/docs/report.pdf

Extra-pineal tissues and organs synthesize melatonin
These include:

- retina,
- ciliary body (inside eye),
- lens,
- Harderian gland (outside eye),
- brain,
- thymus,
- airway epithelium,
- bone marrow,
- gut (400x higher than in pineal gland),
- ovary,
- testicle,
- placenta,
- lymphocytes,
- skin

Note:

1. Extra-pineal melatonin synthesis is not subjected to light/dark regulation.
2. Locally generated melatonin is consumed by the tissues in which it is produced as a protective mechanism of oxidative stress.

Biological properties e.g.,

- Capacity to scavenge free radicals, especially the hydroxyl radical (e.g., reduces neuronal & heart damage). The reactive oxygen species (from the respiratory chain in mitochondria) can produce lipid peroxidation of cardiolipin (in mitochondria) which in turn promotes the detachment of Cytochrome C. The latter then interacts with the Caspase-9 enzymes to produce apoptosis. The ensuing cell death contributes to e.g., neurodegenerative disease. Melatonin blocks this process at multiple levels.

- Melatonin prevents DNA damage (especially in mitochondria) by free radicals

- Melatonin protects against cancer initiation; also has anti-cancer effects

- Melatonin binds calmodulin and blocks activation of oestrogen receptor-alpha

- Melatonin down-regulates glucocorticoid receptors and blocks effect of e.g., cortisol – which is lowest when melatonin levels are highest.

- inhibition (e.g., by beta-blockers) of melatonin production results in severe suppression of immune responses including production of antibodies

Activates:

- bone marrow cells

- natural killer (NK) cells

- antibody response e.g., anti-bacterial/viral (Th1) responses and suppresses allergic (Th2) responses

- antibody-dependent cell cytotoxicity

- T-cell proliferation (& increases size of thymus gland)
- monocytes, neutrophils and cytokine release (e.g., IL-1, IL-6 and TNFalpha)

Risks of melatonin deficiencies:

- Most medications prescribed to lower blood pressure may reduce serum melatonin levels

e.g., beta-blockers,
calcium channel blockers,
calcium antagonists

- An estimated 40% of individuals who take beta-blockers have sleep disorders
- Melatonin should be combined with statins to reduce the free-radical-mediated side effects of these cholesterol-lowering drugs.

Currently, melatonin appears to be a versatile anti-oxidative and anti-nitrosative agent, a molecule with immunomodulatory actions and profound oncostatic activity, and also to play a role as a potent neuroprotectant. It may be a useful therapeutic agent for treatment of CNS injuries. See article: http://onlinelibrary.wiley.com/doi/10.1111/j.1600-079X.2009.00703.x/full#ss3

Examples of other effects of melatonin:

Exercise and melatonin in humans: reciprocal benefits:

http://onlinelibrary.wiley.com/doi/10.1111/j.1600-079X.2011.00924.x/abstract

Melatonin supplementation ameliorates oxidative stress and inflammatory signaling induced by strenuous exercise in adult human males:

http://onlinelibrary.wiley.com/doi/10.1111/j.1600-079X.2011.00899.x/abstract

Melatonin treatment normalizes plasma pro-inflammatory cytokines and nitrosative/oxidative stress in patients suffering from Duchenne muscular dystrophy

http://onlinelibrary.wiley.com/doi/10.1111/j.1600-079X.2010.00752.x/abstract

Alzheimer's disease: pathological mechanisms and the beneficial role of melatonin

http://onlinelibrary.wiley.com/doi/10.1111/j.1600-079X.2011.00937.x/abstract

2 (n) - 3 Fluoride inhibits melatonin

There is now compelling documentation (see links below) especially in the last decade (fluoride was introduced in water etc some 30 years ago in NSW) to show fluoride does have a positive adverse impact such as on the pineal gland and melatonin levels. **The concern is that the cumulative effects of fluoride diminishes/inhibits the role of melatonin as an anti-cancer and neuroprotective hormone along with inhibiting medications such as beta-blockers, statins etc - to contribute to the rise in e.g., neurodegenerative diseases and cancer.** The following links raise concerns about fluoride that, on the balance of evidence, should not be added to water etc.

a) Fluoride and pineal gland/melatonin
http://www.fluoridealert.org/prof-statement.pdf
http://www.newjerseywatereducation.com/pdfs/Excitotoxicity.pdf
http://www.lewrockwell.com/miller/miller17.html
http://www.york.ac.uk/inst/crd/fluorid.htm
http://www.fluoridealert.org/limeback.htm

b) Bibliography of fluoride and effects
http://www.slweb.org/bibliography.html

c) Fluoride uptake and calcification in arteries (2012)
http://www.ncbi.nlm.nih.gov/pubmed/21946616
http://www.greenmedinfo.com/article/there-association-vascular-fluoride-uptake-vascular-calcification-and-coronary-artery

b) Fluoride not much better for caries (2010) e.g.,
http://www.theglobeandmail.com/life/health/ontario-fluoride-may-make-minor-difference/article1535873/

Melatonin should only be given to children under the supervision of a pediatrician who has sought the cause of the effect (e.g., insomnia) and ruled out other treatments or practical measures (e.g., fluoride exposure, or sleeping near an electric power box to the house – as was the case for our daughter some 25 years ago). If melatonin is indicated then a dose should be 0.3mg or less and given for not more than 4 weeks. Keep in mind levels of melatonin do affect time of onset of puberty. Pineal tumours (low or no melatonin) can lead to early precocious puberty. It is now hypothesized that early onset of puberty in females is a consequence of fluoride lowering melatonin levels as demonstrated experimentally.

2 (n) - 4 Radiation from phone base stations decreases melatonin:

There is strong evidence from epidemiologic studies that long-term exposure to extremely low frequency (ELF, ≤ 60 Hz) magnetic fields (MF) and the radiofrequency (RF) electro-magnetic radiation (EMR) from base stations is associated with a **decrease** in melatonin production. These transmissions are continuous. Mobile phone base towers/stations can be more dangerous than phones. http://www.hese-project.org/hese-uk/en/niemr/cellfeedback.php

Human studies have found that **low** melatonin production is a likely risk factor e.g., for breast cancer.

The World Health Organization (WHO) has known for decades that diesel fumes are associated with certain cancers but only in June, 2012 did WHO declare **diesel fumes is a Class-1 Human Carcinogen.**

http://www.nhs.uk/news/2012/06june/Pages/who-classes-diesel-vehicle-exhaust-fumes-as-carcinogen.aspx

Therefore, a combination of a proven cancer initiator (e.g., **diesel fumes**) coupled with **co-factors** of decreased levels of melatonin (due to effects of e.g., EMR, fluoride, beta-blockers, either alone or in combination), inflammation, diet enriched with pro-inflammatory omega-6 fatty acids (e.g., linoleic acid) can **increase the risk of cancer**.

The Australian, Dr. Michael H. Repacholi, the Chairman and Founder of the WHO International Commission on Non-Ionizing Radiation and Protection (ICNIRP) has been rebuked on numerous occasions for not adopting 'Precautionary Principles' and ridiculed for not adhering to the principles of the Scientific Method. e.g., http://www.mast-victims.org/resources/docs/The-Laughing-stock-and-pursuit-of-Gro_Plot-issue7.pdf [redacted] The link to WHO generally ignores proven health impacts. http://www.who.int/peh-emf/en/ Similarly: www.commsalliance.com.au/mobile-phone-tower-information

The clear consensus of the **Bio-Initiative Working Group members** is that the existing public safety limits are **inadequate** for both Extremely Low Frequency (ELF) and radiofrequency radiation (RF). In today's world, everyone is exposed to two types of EMFs: (1) extremely low frequency electromagnetic fields (ELF) from electrical and electronic appliances and power lines and (2) radiofrequency radiation (RF) from wireless devices such as cell phones and cordless phones, cellular antennas and towers and broadcast transmission towers. http://www.bioinitiative.org/freeaccess/report/docs/report.pdf

We understand that in Australia, the mere fact that exposure limits are based around ICNIRP "research" and limits, which selectively chooses to consider only thermal effects of EMR as being dangerous, is in itself cause for significant concern. "It's like a 100-watt light bulb," the mobile-phone carriers' PR personnel often tell unsuspecting people about phone base stations.

Research into EMR continues to be conducted around the world and is continuing to find adverse effects on health. For example, in 2009 a Stockholm study found that 3G mobile phone radiation inhibited DNA repair for up to 72 hours after exposure. This result is typical, but governments and health authorities continue to either ignore or fail to act on the evidence that is piling up, both in the laboratory and in populations.

The human race has evolved against a background of very low level natural radiation - about 8Hz. The current artificial EMR is now billions of times higher than when our grandparents lived.

A study of the health of people living in the vicinity of mobile phone base stations found significant health effects on people living within 300 metres of mobile phone base stations. Conclusions include the recommendation:
"... it is advisable that mobile phone base stations not be sited closer than 300 metres to populations". Santini *et al. Pathol Biol* (Paris) [Pathologie Biologie (Paris)] 2002; 50: 369 –73.

There are numerous studies that show clear and significant ill-health effects from mobile base stations or masts. In this respect, therefore, we believe that any statement by industry or official sources e.g., www.mcf.amta.org.au/files/Mobile.InSite.May.2011.pdf that claims (or suggests) that:

(a) There is no evidence of ill-health effects from masts; or
(b) The overwhelming evidence is that masts do not cause ill-health effects;

Such assertions are *completely and blatantly untrue*!

The *Bio-Initiative Report* provided detailed scientific information on health impacts when people were exposed to electro-magnetic radiation *hundreds or even thousands of times below limits* currently established by the FCC and International Commission for Non-Ionizing Radiation Protection in Europe (ICNIRP).

The authors reviewed more than 2000 scientific studies and reviews, and have concluded that:

(1) the existing public safety limits are inadequate to protect public health; and
(2) from a public health policy standpoint, new public safety limits and limits on further deployment of risky technologies are warranted based on the total weight of evidence. A precautionary limit of 1 mW/m2 (0.1microW/cm2 or 0.614 V/m) was suggested in Section17 of the Bio-Initiative Report to be adopted for outdoor, cumulative RF exposure.

The existing standards are based on **thermal (heating) limits**, and do not address non-thermal (or low-intensity) exposures which are widely reported to cause bio-effects. Some are likely to lead to adverse health effects with chronic exposure. The existing ICNIRP and FCC limits for public and occupational exposure to ELF and RF are **insufficiently protective of public health.**

The history and politics of the Telecommunication Industry, EMR and its health impacts are similar to those of the fossil fuel industry dealing with air-quality standards! Claims by the mobile phone industry that the base stations are safe because the radiation falls off rapidly with distance are flawed. http://www.hese-project.org/hese-uk/en/niemr/cellfeedback.php

As with the failure to adopt a $PM_{2.5}$ standard for air-quality, biologically-based public and occupational exposure standards for extra-low frequency (ELF) and radiofrequency radiation (RF) are recommended to address bio-effects and potential adverse health effects of chronic exposure to ELF and RF. **These effects are now widely reported to occur at exposure levels significantly below most current national and international limits.** A biologically-based exposure limit should reflect current scientific knowledge of bio-effects and health effects, and impose new limits based on preventative action as defined by the Precautionary Principle.

Biologically-based exposure standards are needed to prevent disruption of normal body processes. Effects are reported for DNA damage (genotoxicity that is directly linked to integrity of the human genome), cellular communication, cellular metabolism and repair, cancer surveillance within the body; and for protection against cancer and neurological diseases. Also reported are neurological effects including impairment of sleep and sleep architecture, cognitive function and memory, depression, cardiac effects, pathological leakage of the blood-brain barrier and impairment of normal immune function, fertility and reproduction.

Melatonin production is lowered by exposure to certain EMR frequencies

The scientists involved in the 2007 *'Bio-Initiative' Working Group* project thoroughly documented the effect of EMR on melatonin. See link:
http://www.bioinitiative.org/freeaccess/report/docs/report.pdf

Human beings are bio-electrical systems. Our hearts and brains are regulated by internal bio-electrical signals. Environmental exposures to artificial EMFs can interact with fundamental biological processes in the human body. There are some credible articles from researchers reporting that cell tower-level RF exposures (estimated to be between 0.01 and 0.5 μW/cm2) produce ill-effects in populations living up to several hundred metres from wireless antenna sites.

This information now argues for thresholds or guidelines that are substantially below current FCC and ICNIPR standards for whole body exposure. Uncertainty about how low such standards might have to go
to be prudent from a public health standpoint should not prevent reasonable efforts to respond to the information at hand.

We are regularly told by the mobile phone industry that these base stations are safe because their microwave radiation falls off rapidly with distance and is far too low to generate significant heat. **Sadly, this is not true.** It is based on the false assumption that it is only their heating effect that

can cause damage and a serious misunderstanding of the ways in which living organisms use negative feedback to respond to changes in their environment, including the metabolic insults from mobile phones.

There are hundreds of scientific papers in peer-reviewed scientific journals showing biological effects from non-ionising radiation that may be **hundreds or thousands of times below the levels that cause significant heating.** See the *'Bioinitiative Report'*, 2007 - http://www.bioinitiative.org/ Claims by the mobile phone industry that the base stations are safe because the radiation falls off rapidly with distance are flawed. http://www.hese-project.org/hese-uk/en/niemr/cellfeedback.php

The following are **extracts** of the 2007 *'Bio-Initiative' Working Group* Report: http://www.bioinitiative.org/freeaccess/report/docs/report.pdf

EXECUTIVE SUMMARY

Melatonin Production

Melatonin is a hormone produced primarily by the pineal gland, located in the centre of the brain. Melatonin is evolutionarily conserved and is found in nearly all organisms. It has numerous properties which indicate that it helps prevent both Alzheimer's disease and breast cancer. There is strong evidence from epidemiologic studies that high ($\geq$ 10 milligauss or mG), long-term exposure to extremely low frequency (ELF, $\leq$ 60 Hz) magnetic fields (MF) is associated with a decrease in melatonin production (Section II.)

Alzheimer's Disease (AD)

Amyloid beta (Aβ) protein is generally considered the primary neurotoxic agent causally associated with Alzheimer's disease (AD). Aβ is produced by both brain and peripheral cells and can pass through the blood brain barrier.

1. There is longitudinal epidemiologic evidence that high peripheral blood levels of Aβ is a risk factor for Alzheimer's disease (AD). (Section III.A.)
2. There is epidemiologic evidence that extremely low frequency (ELF, $\leq$ 60 Hz) magnetic fields (MF) exposure up-regulates peripheral blood levels of Aβ. (Section III.A.)
3. There is evidence that melatonin can inhibit the development of AD and, thus, low melatonin may increase the risk of AD (Section III.B.)
4. There is strong epidemiologic evidence that significant (i.e., high), occupational ELF MF exposure can lead to the down-regulation of melatonin production. The precise components of the magnetic fields causing this down-regulation are unknown. Other factors which may influence the relationship between MF exposure and melatonin production are unknown, but certain medications may play a role. (Section II.)
5. There is strong epidemiologic evidence that high occupational MF exposure is a risk factor for AD, based on case-control studies which used expert diagnoses and a restrictive classification of MF exposure. (Section III.C.)
6. There are no epidemiologic studies of AD and radiofrequency MF exposure and only one of non-acute radiofrequency MF exposure and melatonin, so conclusions are not yet appropriate. (Sections III.D and II.)

Breast Cancer (BC)

The only biological hypothesis which has been epidemiologically investigated to explain the relationship between MF exposure and breast cancer is that high MF exposure can lower melatonin production. Which in turn can lead to changes in the various biological systems which melatonin influences, including increased estrogen production and subsequent deleterious interactions with DNA and decreased anti-proliferative,

antioxidant, DNA repair and immune response capabilities. Thus lowered melatonin production can be expected to lead to increased risk of breast cancer.

EMF & Melatonin: AD & BC (Davanipour & Sobel)
1. *In vitro* and animal studies have demonstrated that -
(i) melatonin is a potent scavenger of oxygen and nitrogen radicals that cause DNA damage,
(ii) melatonin interferes with estrogen's deleterious interactions with DNA, and
(iii) melatonin inhibits the development of mammary tumors. (Section IV.A.)

2. Human studies indicate that MF exposure can decrease melatonin production. (Section II)
3. Human studies have found that low melatonin production is a likely risk factor for breast cancer. (Section IV.B.)
4. Human studies have shown that light-at-night and night shift work reduce melatonin production and are both risk factors for breast cancer. (Section IV.D.)
5. Occupational studies indicate that high MF exposure increases the risk of breast cancer. This is particularly true for a recent, large, and well-designed study from Poland (funded by the NCI, administered for the NCI by Westat, and conducted by Polish scientists)....

II. ELF Magnetic Field EXPOSURE and MELATONIN PRODUCTION
Conclusion: Eleven (11) of the 13 published epidemiologic residential and occupational studies are considered to provide (positive) evidence that high MF-exposure can result in decreased melatonin production. The two negative studies had important deficiencies that may certainly have biased the results. There is sufficient evidence to conclude that long-term relatively high ELF MF exposure can result in a decrease in melatonin production. It has not been determined to what extent personal characteristics, e.g., medications, interact with ELF MF exposure in decreasing melatonin production.

VII. Conclusions
Conclusion: The constellation of relevant scientific papers providing mutually-reinforcing evidence for an association between power-frequency electromagnetic fields (ELF-EMF) and breast cancer is strongly supported in the scientific literature.

The question that arises is whether the combination of exposure to diesel plus ELF-EMF's that reduce the anti-cancer levels of melatonin a risk factor?

Conclusion -1: ELF at environmental levels negatively affects the oncostatic effects of both melatonin and tamoxifen on human breast cancer cells. Numerous epidemiological studies over the last two decades have reported increased risk of male and female breast cancer with exposures to residential and occupational levels of ELF. Animal studies have reported increased mammary tumor size and incidence in association with ELF exposure.
Conclusion -2: ELF limits for public exposure should be revised to reflect increased risk of breast cancer at environmental levels possibly as low as 2 mG or 3 mG; certainly as low as 4 mG.

In the last few decades, it has been established beyond any reasonable doubt that bio-effects, and some adverse health effects, occur at far lower levels of RF and ELF exposure where no heating (or induced currents) occurs at all. Some effects are shown to occur at several hundred thousand times below the existing public safety limits where heating is an impossibility.

It appears it is the INFORMATION conveyed by electromagnetic radiation (rather than heat) that causes biological changes - some of these biological changes may lead to loss of well-being, disease and even death.

The existing standards have been proven to be inadequate to control against harm from low-intensity, chronic exposures, based on any reasonable, independent assessment of the scientific literature. It means that an entirely new basis (a biological basis) for new exposure standards is needed. New standards need to take into account what we have learned about the effects of ELF and RF (all non-ionizing electromagnetic radiation) and to design new limits based on biologically demonstrated effects that are important to proper biological function in living organisms.

There may be no lower limit at which exposures do not affect us. Until we know if there is a lower limit below which bio-effects and adverse health impacts do not occur, it is unwise from a public health perspective to continue "business-as-usual" deploying new technologies that increase ELF and RF exposures, particularly involuntary exposures as is the case here.

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.

Both ELF and RF exposures can be considered genotoxic (will damage DNA) under certain conditions of exposure, including exposure levels that are lower than existing safety limits.

Very low-level ELF and RF exposures can cause cells to produce stress proteins, meaning that the cell recognizes ELF and RF exposures as harmful. This is another important way in which scientists have documented that ELF and RF exposures can be harmful, and it happens at levels far below the existing public safety standards.

There is substantial evidence that ELF and RF can cause inflammatory reactions, allergy reactions and change normal immune function at levels allowed by current public safety standards.

The lower limit for reported human health effects has dropped 100-fold below the safety standard (for mobile phones and PDAs); 1000- to 10,000-fold for other wireless (cell towers at distance; WI-FI and WLAN devices). The entire basis for safety standards is called into question, and **it is not unreasonable to question the safety of RF at any level.**

A cautionary target level for pulsed RF exposures for ambient wireless that could be applied to RF sources from cell tower antennas, WI-FI, WI-MAX and other similar sources is proposed. http://www.bioinitiative.org/freeaccess/report/docs/report.pdf

The recommended cautionary target level is 0.1 microwatts per centimeter squared (μW/cm2) (or 0.614 Volts per meter or V/m) for pulsed RF where these exposures affect the general public; this advisory is proportionate to the evidence and in accord with prudent public health policy. A precautionary limit of
0.1μW/cm2 should be adopted for outdoor, cumulative RF exposure. This reflects the current RF science and prudent public health response that would reasonably be set for pulsed RF (ambient) exposures where people live, work and go to school.
http://www.bioinitiative.org/freeaccess/report/docs/report.pdf

The scientific evidence is sufficient to warrant **regulatory** action for ELF; and it is substantial enough to warrant **preventative** actions for RF. The synergistic effect with diesel is plausible.

Other examples of Reports of health impacts of phone base antennae – can these effects be augmented with diesel fumes?

(a) Professor Franz Adlkofer, Verum-Foundation, at a conference at the University of Gelsenkirchen, 6 October 2007, reported his findings that DNA strand breaks occurred at 1/40 of the recommended exposure, indicating that UMTS (3G) has ten times the effect than GSM, and thus leads to a higher risk of cancer. http://www.hese-project.org/hese-uk/en/niemr/news.php?id=adlkofer

He noted the Interphone research, which shows an increase of brain tumors after 10 years of use, and Friedman (Israel) who observed the biological mechanism of cell damage by mobile phone radiation **far below the recommended limits**. There is no doubt, he said; UMTS is much more dangerous than GSM.

Prof. Adlkofer called for an immediate change in thinking, warning of a huge health problem to emerge in society that increasingly uses EMF technology. He named the mobile phone technology and its political justification a giant human experiment with neither control nor plan.

(b) Mobile phone base stations: Effects on well-being and health

Kundi M, Hutter HP

Abstract

Studying effects of mobile phone base station signals on health have been discouraged by authoritative bodies like WHO International EMF Project and COST 281. WHO recommended studies around base stations in 2003 but again stated in 2006 that studies on cancer in relation to base station exposure are of low priority.

As a result only few investigations of effects of base station exposure on health and well-being exist. Cross-sectional investigations of subjective health as a function of distance or measured field strength, despite differences in methods and robustness of study design, found indications for an effect of exposure that is likely independent of concerns and attributions.

(c) Neuro-behavioral effects among inhabitants around mobile phone base stations

Neurotoxicology. 2007 Mar;28(2):434-40. Epub 2006 Aug 1.
http://www.ncbi.nlm.nih.gov/pubmed/16962663
Abdel-Rassoul G, El-Fateh OA, Salem MA, Michael A, Farahat F, El-Batanouny M, Salem E.

Abstract

There is a general concern on the possible hazardous health effects of exposure to radiofrequency electromagnetic radiations (RFR) emitted from mobile phone base station antennas on the human nervous system.

Results The prevalence of neuro-psychiatric complaints as headache (23.5%), memory changes (28.2%), dizziness (18.8%), tremors (9.4%), depressive symptoms (21.7%) and sleep disturbance (23.5%) were significantly higher among exposed inhabitants than controls: (10%), (5%), (5%), (0%), (8.8%) and (10%), respectively ($P<0.05$). The NBTB indicated that the exposed inhabitants exhibited a significantly lower performance than controls in one of the tests of attention and short-term auditory memory [Paced Auditory Serial Addition Test (PASAT)]. Also, the inhabitants opposite the station exhibited a lower performance in the problem solving test (block design) than those under the station. All inhabitants exhibited a better performance in the two tests of visuomotor speed (Digit Symbol and Trailmaking B) and one test of attention (Trailmaking A) than controls. The last available measures of RFR emitted from the first mobile

phone base station antennas in Menoufiya governorate were less than the allowable standard level.

CONCLUSIONS AND RECOMMENDATIONS:

Inhabitants living nearby mobile phone base stations are at risk for developing neuro-psychiatric problems and some changes in the performance of neuro-behavioral functions either by facilitation or inhibition. Can these effects be augmented with diesel exposure is plausible.

So, revision of standard guidelines for public exposure to RER from mobile phone base station antennas and using of NBTB for regular assessment and early detection of biological effects among inhabitants around the stations are recommended.

(d) Epidemiological evidence for a health risk from mobile phone base stations

http://www.ncbi.nlm.nih.gov/pubmed/20662418
Khurana VG, Hardell L, Everaert J, Bortkiewicz A, Carlberg M, Ahonen M.

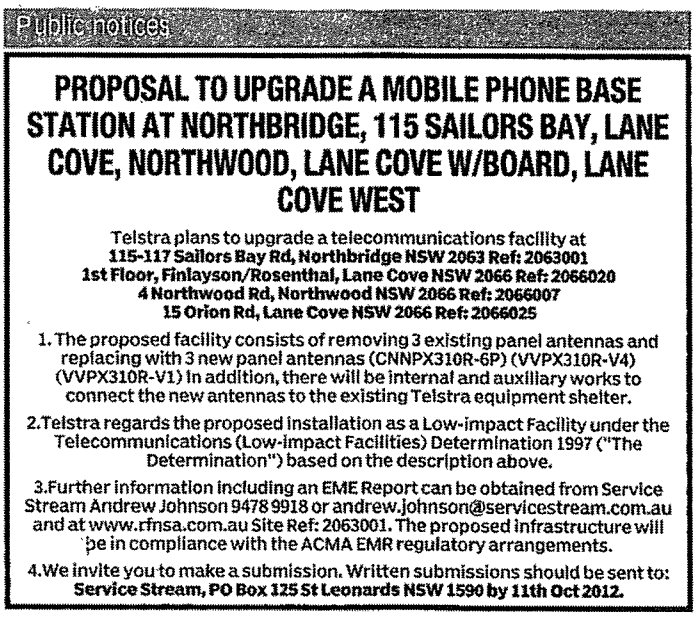
Abstract
Human populations are increasingly exposed to microwave/radiofrequency (RF) emissions from wireless communication technology, including mobile phones and their base stations. By searching PubMed, we identified a total of 10 epidemiological studies that assessed for putative health effects of mobile phone base stations. Seven of these studies explored the association between base station proximity and neuro-behavioral effects and three investigated cancer. We found that eight of the 10 studies reported increased prevalence of adverse neuro-behavioral symptoms or cancer in populations living at distances < 500 meters from base stations. None of the studies reported exposure above accepted international guidelines, suggesting that current guidelines may be inadequate in protecting the health of human populations. We believe that comprehensive epidemiological studies of long-term mobile phone base station exposure are urgently required to more definitively understand its health impact.

CASE EXAMPLE:

Telstra plans to upgrade a mobile phone base station at 15 Orion Rd., Lane Cove West, 2066

Nearby is the Western Stack of the Lane Cove Tunnel (LCT) that exhausts diesel fumes – a proven carcinogen for human beings. Other carcinogens e.g., benzene and 1,3-butadiene are also exhausted.

NSW Environment Protection Authority (EPA) determined there will be up to a 10-fold increase risk of cancer to "at risk receptors" from stack pollution. (see copy of document 2 pages on)



Public notices

PROPOSAL TO UPGRADE A MOBILE PHONE BASE STATION AT NORTHBRIDGE, 115 SAILORS BAY, LANE COVE, NORTHWOOD, LANE COVE W/BOARD, LANE COVE WEST

Telstra plans to upgrade a telecommunications facility at
115-117 Sailors Bay Rd, Northbridge NSW 2063 Ref: 2063001
1st Floor, Finlayson/Rosenthal, Lane Cove NSW 2066 Ref: 2066020
4 Northwood Rd, Northwood NSW 2066 Ref: 2066007
15 Orion Rd, Lane Cove NSW 2066 Ref: 2066025

1. The proposed facility consists of removing 3 existing panel antennas and replacing with 3 new panel antennas (CNNPX310R-6P) (VVPX310R-V4) (VVPX310R-V1) In addition, there will be internal and auxiliary works to connect the new antennas to the existing Telstra equipment shelter.

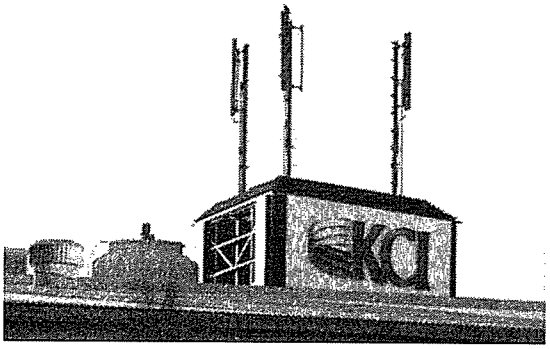
2. Telstra regards the proposed installation as a Low-impact Facility under the Telecommunications (Low-impact Facilities) Determination 1997 ("The Determination") based on the description above.

3. Further information including an EME Report can be obtained from Service Stream Andrew Johnson 9478 9918 or andrew.johnson@servicestream.com.au and at www.rfnsa.com.au Site Ref: 2063001. The proposed infrastructure will be in compliance with the ACMA EMR regulatory arrangements.

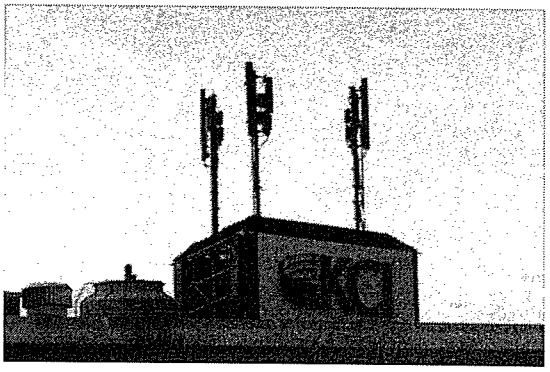
4. We invite you to make a submission. Written submissions should be sent to: **Service Stream, PO Box 125 St Leonards NSW 1590 by 11th Oct 2012.**

NORTH SHORE TIMES, Friday, September 28, 2012 113

However, are there increased risks to residents (including children at Lane Cove West Public School) exposed to such pollution when they also live within 500 metres of the phone base station at 15 Orion Road, Lane Cove West that Telstra upgraded its transmission capability?

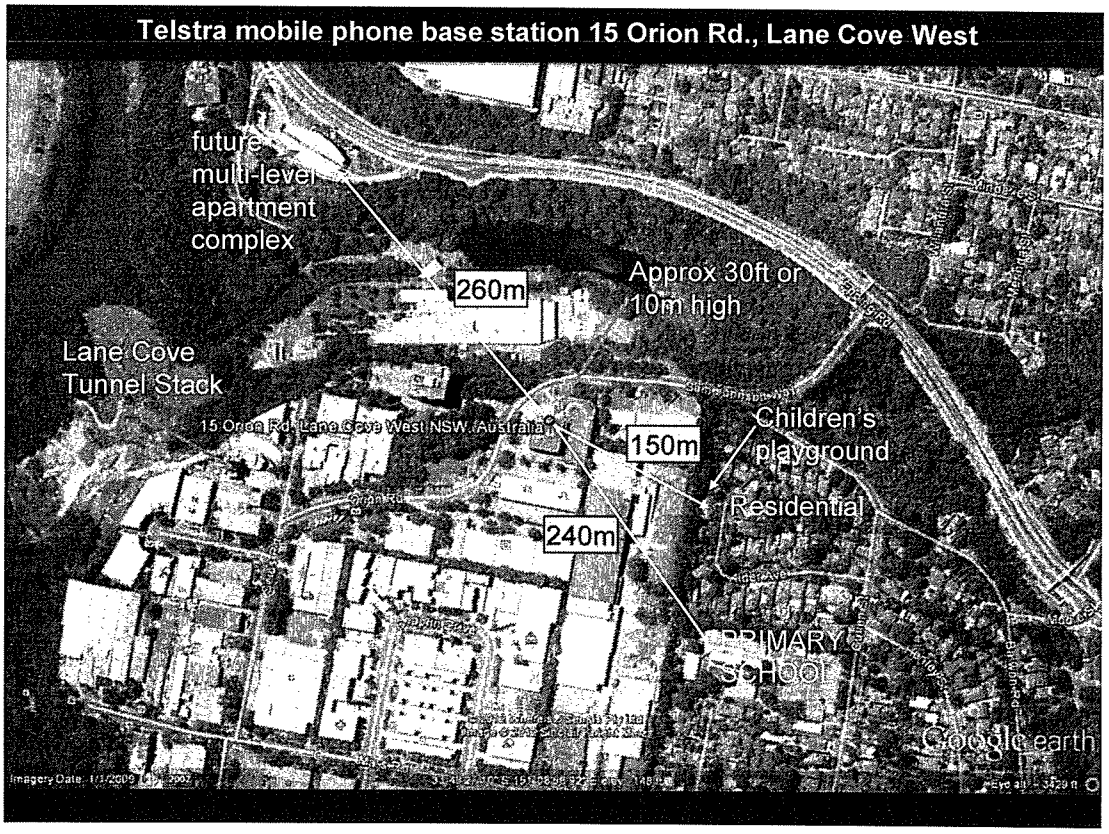


1.10.12

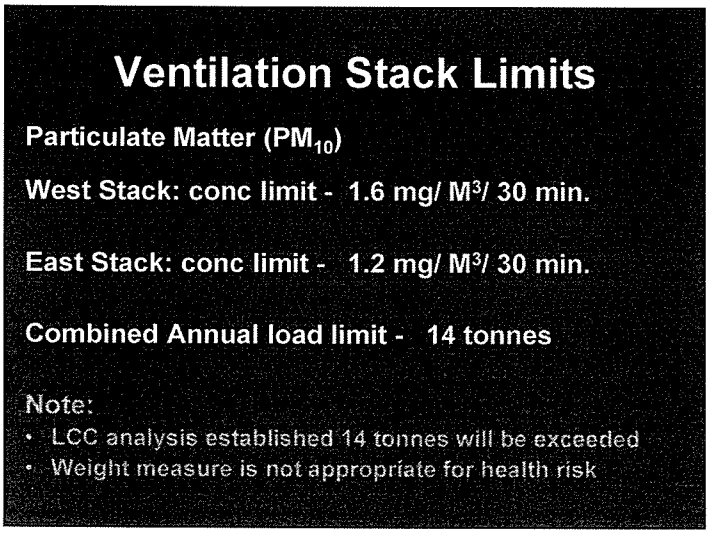


22.1.13 (upgraded towers)

Lane Cove Tunnel exhausts many carcinogens through the <u>western stack</u>

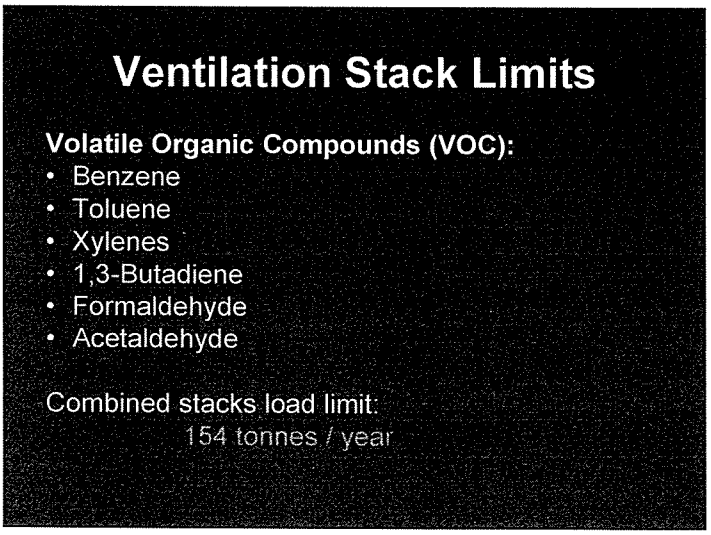


Locations of <u>tunnel stack</u> and site of <u>phone base station</u> relative to 'at risk receptors'



Noteworthy is the Ministers Conditions of Approval (MCoA) for the LCT allow:

Note that the MCoA allows 14 tonnes of PM10 each year to be discharged from the 2 stacks.



Note that the MCoA allows ***154 tonnes*** of highly toxic volatile chemicals to be discharged each year from the 2 stacks.

Cancer-risk already estimated to be up to 10-fold higher from stack pollution

Also for your information is the document (below) obtained by a 'Call for papers in Parliament'. The document (dated 26th June, 2002) asserts an increase in cancer risk is between 2-4 times (1 per million) at ground level but up to 10-fold higher for "at-risk receptors" (human beings) living near to the Lane Cove Tunnel western stack.

One can allege the risk is even higher with the presence of an upgrade of a phone-base station which is known to diminish anti-cancer melatonin levels.

LANE COVE TUNNEL AIR QUALITY IMPACT ASSESSMENT
ALTERNATIVE VENTILATION OPTIONS

(25)

ISSUE

HOF22460/HO4105

Air Technical Advisory Services Unit's (ATASU) comments on additional information required by the EPA to assess the Lane Cove Tunnel proposal.

BACKGROUND

Further to ATASU's memo (HOF19461) to Sydney Planning Section (SPS) dated 8 March 2002, the RTA has recently provided the additional information (HAS, 2002, Lane Cove Tunnel Air Quality Study: Supplementary Information on Air Quality Impacts from Lane Cove Tunnel Ventilation Options, 31 May 2002). John Goodwin of SPS has requested advice on the HAS, 2002 report.

CURRENT POSITION

The base elevation, stack height and elevation above sea level for each stack location are presented in descending order in Table 1, while a 3-dimensional plot of the terrain which shows the location of the various stack options is presented in Figure 1.

The results of the health risk assessment at each stack location due to combined exposure to benzene, 1,3-butadiene, acetaldehyde, formaldehyde and benzo(α)pyrene have been reported in HAS, 2002 and presented for ease of comparison in descending order in Table 1 and Figure 2. The cells highlighted yellow in Table 1 indicate that the carcinogenic health risks are above 1 in 1 million (1×10^{-6}). In general, the health risk assessment has been carried out in an appropriate manner. Air Technical Advisory Services Unit has used the results presented in Tables 2, 3, and 4 of the HAS, 2002 report to generate aggregate carcinogenic health risk estimates for a number of scenarios (i.e. different stack heights (i.e. 30 m and 36 m) and locations (i.e. A1, A2, A3, A4, A5, A6, W1, W2 and W3) at a group of ground-level and elevated receptors.

While the predicted carcinogenic health risks associated with exposure to all pollutants are between **2 and 4** times higher than 1×10^{-6} at ground level, they are up to approximately **10** times higher at elevated receptors located at 40 metres above ground-level.

The incremental increase in carcinogenic health risks associated with exposure to various air toxic compounds has been predicted to be above 1×10^{-6}. This level is commonly used within the EPA as a trigger for consideration about possible options for reducing the levels of pollutants predicted through the use of health risk assessment.

Table 1

Location	Risk 0 m	Location	Risk 20 m	Location	Risk 40 m	Location	Base elevation (m)	Stack height (m)	Elevation asl (m)
A2	2×10^{-6}	A1	5×10^{-7}	A3	2×10^{-6}	W2	41	36	77
W1	2×10^{-6}	A3	5×10^{-7}	A4	3×10^{-6}	W3	44	30	74
A1	2×10^{-6}	A2	6×10^{-7}	A6	3×10^{-6}	A6	32	30	62
A6	3×10^{-6}	A4	8×10^{-7}	W1	6×10^{-6}	A5	24	30	54
W2	3×10^{-6}	W2	1×10^{-6}	A1	6×10^{-6}	W1	20	30	50
A3	3×10^{-6}	A6	1×10^{-6}	W3	8×10^{-6}	A3	18	30	48
A5	4×10^{-6}	W1	3×10^{-6}	W2	1×10^{-5}	A4	12	30	42
A4	4×10^{-6}	W3	4×10^{-6}	A2	1×10^{-5}	A2	7	30	37
W3	4×10^{-6}	A5	-	A5	-	A1	4	30	34

RECOMMENDATION

It is recommended that the RTA investigate options for reducing the carcinogenic health risk at both ground-level and elevated receptors. These options may include, but should not be limited to, increasing the stack height and/or exhaust velocity. Increasing the exhaust velocity may be achieved by segmentation of the stack so that higher exhaust velocities may be achieved at less than maximum flowrate.

1. MSP

Nick Agapides/MATASU
26 June 2002

An analysis of Telstra's information shows that the upgrade will increase the threshold of health risk by **approx 50-fold** above the minimum level for health impact. This estimate is derived as follows:

The proposal calls for an increase in Electro-Magnetic Energy (EME) of currently a maximum 0.18% (of 100% limit) to approx **0.51% at 167.85m** (about a 3-fold increase in the upgrade). See Telstra's attachment I received from them on request.

The EME standard, as I understand, is based on the level of thermal heating – i.e., NOT the biological effects (health impacts) of EMR that are proven to occur up to 10,000 times lower than the existing limit (stated by Telstra as 100%). The 0.51% of 100% (at about 167metres) is about 200-fold less (100%/0.5%), based on 'heat energy' NOT health impacts that have been found to occur up to 500m at 100%/10,000 = 0.01%.

Telstra proposes an upgrade of 0.51% which is about 50 times (0.5%/0.01%) above the lower limits EME proven to cause health effects within 167m. (and up to 500m).

The evidence in the research literature confirms that **base stations should not be so close to residential areas and even more so if they are near an unfiltered stack of Lane Cove Tunnel.**

Note the following profound statement:

"The lower limit for reported human health effects has dropped 100-fold below the safety standard (for mobile phones and PDAs); 1000- to 10,000-fold for other wireless (cell towers at distance; WI-FI and WLAN devices). The entire basis for safety standards is called into question, and **it is not unreasonable to question the safety of RF at any level."**

The statement comes from the excellent Scientific Report found at:
http://www.bioinitiative.org/freeaccess/report/docs/report.pdf

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 PM_{10} known as the Tapered Element Oscillating Microbalance (TEOM) *under-estimated PM_{10} values*, below temperatures of about 15 - 17°C, by as much as 25%. The national automatic air quality monitoring networks employ mainly TEOM PM10 samplers, together with some TEOM PM2.5 instruments.

The TEOM instruments 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 PM_{10} 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

Particulate Matter (PM_{10})

West Stack: conc limit - 1.6 mg/ M^3/ 30 min.

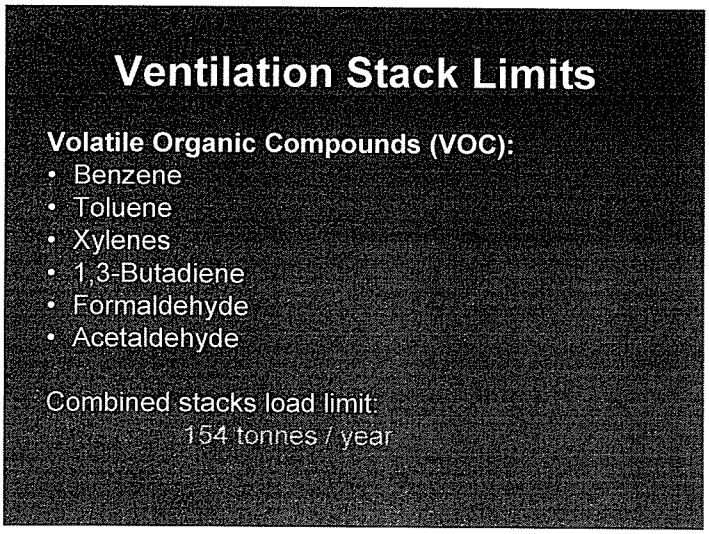
East Stack: conc limit - 1.2 mg/ M^3/ 30 min.

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.



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 (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!

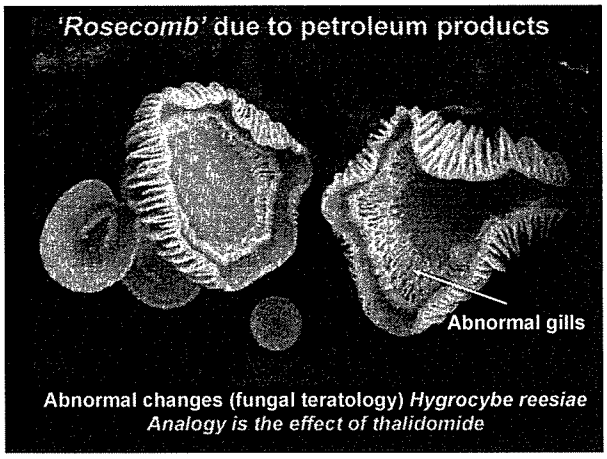
According to new information by teams of citizen scientists and experts, the Earth is now entering the sixth mass extinction event in its four-billion-year history. This die-off is the only

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 that 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
CONTACT: John Flannery (02) 6270 5477 / (0419) 494 761

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 CO_2-equivalent greenhouse gas emissions by approx. 3.6 million tons in the USA in 2001. i.e., equivalent to removing 520,000 cars from roads.
- Tripling the use of ethanol in USA would triple the greenhouse gas benefit.
- Ethanol fuels not only enhance energy security and boost rural economies, but can reduce harmful air pollution and greenhouse gas emissions.
- Biofuels can cause a renewal in agriculture and rural Australia and benefit the entire national economy.

What are the advantages to using ethanol-blends?

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

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 ($\mu g/m^3$) 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 $\mu g/m^3$), 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.