Submission
No 107

INQUIRY INTO CROSS CITY TUNNEL

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Theme:

Summary

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Residents Against Polluting Stacks

The Director
Joint Select Committee on the Cross City Tunnel
Parliament House
Macquarie Street
Sydney NSW 2000

29.5.06

Dear Mr Johnston

Submission to the Inquiry by Select Joint Committee of the NSW Legislative Council on the Cross City Tunnel

Residents Against Polluting Stacks (RAPS) wishes to make a second submission to the continuing Inquiry into the Cross City Tunnel (CCT) in relation particularly to the following terms of reference:
(d) the public release of contractual and associated documents connected with public private partnerships for large road projects,
(e) the communication and accountability mechanisms between the RTA and Government, including the Premier, other Ministers or their staff and the former Premier or former Ministers or their staff,
(f) the role of Government agencies in entering into major public private partnership agreements, including public consultation processes and terms and conditions included in such agreements, the role of Government agencies in relation to the negotiation of the contract with the Lane Cove Tunnel Consortium,
(h) the extent to which the substance of the Lane Cove Tunnel contract was determined through community consultation processes,
(i) the methodology used by the Roads and Traffic Authority for tendering and contract negotiations in connection with the Lane Cove Tunnel, and
(j) any other related matters.

We welcome the first and second inquiry reports and hope that their recommendations will be fully implemented, in particular those relating to greater transparency and accountability of PPPs, and the recommendation that the Government ensure motorists are advised to take appropriate precautions against possible adverse air quality in tunnels. We also welcome the extension of the Inquiry to issues relating to the Lane Cove tunnel project, as it mirrors some of the key problems identified with the Cross City project, and its predecessor, the M5 East.

As stated in our first submission to the Inquiry, our experience with tunnels has led us to have grave concerns about the standards and frameworks used to approve and regulate the increasing number of public/private road tunnels in our city, and the RTA's and regulatory authorities' approach to dealing with serious health, environmental and safety risks these tunnels pose to drivers and residents alike. We believe that they have demonstrably failed to exercise appropriate care, skill and foresight, as they have failed to act to provide the appropriate protections and accountabilities to the public. They have also failed to deliver ecologically sustainable infrastructure projects that represent value for money, and we are alarmed at the escalating economic, environmental and political costs of such poor processes and outcomes.

In relation to ventilation design alone, with the three (M5 East, CCT and LCT) tunnels, well over $140 million have been wasted in unnecessarily complicated and unsafe tunnel designs which place both drivers and residents at serious risk.

Our first submission documented some of the fundamental systemic flaws in the environmental assessment, consultation and regulation processes, namely:

- Community consultation and expert assessments is controlled by the RTA for the RTA
- The role of 'watchdog' agencies is severely compromised
- Inappropriate benchmarks are used in assessing impacts to facilitate approval

- There is no independent scrutiny of changes/modifications made following the EIS process and contracts are negotiated in secret, with changes self-approved
- The conditions of approval set are often vague, unenforceable, unenforced and yet seemingly set in stone.

We provided some specific examples of how these flaws apply to the Lane Cove tunnel in the areas of health and environmental risk assessment, and tunnel filtration.

Since the first inquiry, there have been a number of new developments which we consider relevant to the Inquiry's terms of reference, some of which have only come to light as a result of further release of documents by the order of the Legislative Council:

- Claims relating to the adequacy of current air quality standards
- Debate over costs and viability of filtration

Conflicting claims about the adequacy of current air quality standards

There have been a number of recent claims made by the RTA and Roads Minister Roozendaal in Parliament that "Sydney's road tunnels, including the M5 East, continue to meet some of the strictest health and environmental standards in the world" (Hansard, 6.4.06). The community believes that such claims are highly misleading and un-supportable.

Community demands for reduced in-tunnel and external exposures are informed by considerations which go far beyond a simplistic feeling that less must be better, valid though that position may be. They observe that:

- the conditions for in-tunnel pollutant exposures are defined by limits set in 1995, representing, in the words of the PIARC document , 'the art of tunnel ventilation over the last 15 years'. Thus, they are out of date.
- The PIARC tunnel 'limits' date from before the explosive increase in knowledge about the adverse impacts of vehicular emissions and, in particular, particulate matter. Thus, they do not represent modern medical knowledge.
- Limits for external emissions are based on NEPM goals when the NEPM specifically states that such goals are unsatisfactory for the assessment of point sources such as tunnels.[1]
- The limits for particulate matter based on PM10 measurements are inappropriate because the differences between the composition and potential harm of particles from vehicle emissions when compared to those in the ambient air.[2]
- Effective limitation of particulate emissions inside the tunnel is based on a flawed use of a visibility measurement when the particles capable of causing the greatest harm are effectively invisible because they are smaller than the minimum wavelength of visible light.
- The measurements of ambient levels of particulate matter intended to protect the public are in fact incapable of giving meaningful information of use in the prediction of likely health impacts.[3]
- Reductions in particulate emissions (nominated as PM10) resulting form the 'EURO' series of design improvements for diesel engines have not led to proportionate reductions in adverse health impacts from these emissions as, in many cases, they have actually resulted in increased numbers of finer and finer, more harmful particles.[4]

Growth in medical knowledge

Since the publication of the 'Six Cities Study ' by Dockery and Pope in 1993[5]. there have been literally hundreds of articles in peer reviewed journals dealing with aspects of vehicle and particulate pollution and the adverse effects caused by both long and, more recently, short term exposures to these pollutants.

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[1] *"Conversely, the air quality of some localised areas within major airsheds are dominated by local activities such as that experienced in a road tunnel or a heavily trafficked canyon street. Air quality management in these areas is complex and needs a different approach to that directed at meeting ambient standards intended to reflect the general air quality in the airshed".*
p13 from NEPM 1998

[2] "The NEPM PM10 standard....is a legislative entity and applies only to the ambient background, and a population of 25,000 people. It does not and should not, be applied to a point source such as a tunnel stack from which an entirely different composition of pollutant arises." Executive Director of the National Environment Protection Council, Dr. Bruce Kennedy,

[3] Guidelines for Concentration and Exposure-Response Measurement of Fine and Ultra Fine Particulate Matter for Use in Epidemiological Studies. Schwela, D., Morawska,L., Kotzias, D. WHO, European Commission. EUR 20238 EN. 2002

[4] Kittelson,D B, Watts, Jr.W. F (1998) Review of Diesel Particulate Matter Sampling Methods. Supplemental Report # 2 EPA Grant Review Of Diesel Particulate Matter. http://www.me.umn.edu/centers/cdr/reports/EPAreport2.pdf

These impacts are shown to be affecting all stages of the human life cycle form before birth, through childhood and young adulthood into old age. See 'Medical Evidence about impacts of vehicle pollution' (Appendix A)

A summary of concerns and issues relating to particulate matter and specifically diesel exhaust is provided as Appendix B 'Why is diesel exhaust so damaging to health?'

The fundamental question at which the public seems to be at odds with tunnel designers and operators relates to what are acceptable pollutant concentrations.

RAPS believes that the standards currently used to control in-tunnel air quality and local air quality changes related to tunnel emissions are inadequate, inappropriate and fail to provide appropriate protection to the public both as tunnel users and as residents living close to the tunnel stacks and portals.

The determination of what are adequate air quality standards has a crucial bearing on the arguments surrounding the provision and cost of filtration systems for tunnels.

Tunnel concentration limits.

The concentration and operational limits for air quality in Sydney's tunnels are based on recommendations published by the Committee on Road Tunnels of PIARC, the World Road Association in 1995, entitled Road Tunnels : Emissions, Ventilation, Environment

The only numerical limit of concentration applied to the M5 and other Sydney tunnels relates to carbon monoxide:

70. The tunnel ventilation system(s) must be designed and operated so that the World Health Organisation (WHO) 15-minute carbon monoxide (CO) goal of 87 ppm is not exceeded under any conditions.

The corresponding conditions for the Cross City and Lane Cove tunnels are more complex (but less strict)

This standard is supposed to provide protection against exposure to the other tunnel pollutants such as nitrogen dioxide and fine particulate matter.

It is our contention that it does not.

The progressive reduction of carbon monoxide as a component of vehicle emissions has led to carbon monoxide concentration to become less and less satisfactory as a pollution marker in tunnels.

Improvements in motor design and the introduction of catalytic converters to petrol engine cars have led to a significant change in the relative proportions of the various harmful exhaust gas components. Based on PIARC emission figures the changes are as follows:

Component	Petrol cars			Diesel cars			Diesel trucks 10 tonne		
At 60 km/hr	Pre Catalytic converter	With catalytic converter	%	Pre 'EURO'	EURO 2	%	Pre 'EURO'	EURO 2	%
CO g/hr	341	59	17.30%	51	14	27.45%	244	83	34.02%
NOx g/hr	91	14	15.38%	44	23	52.27%	528	338	64.02%
'Turbidity' m^2/h	--	--	--	43	17	39.53%	157	47	29.94%

Many of these 'improvements' have only become effective since the approval of the M5 tunnel.

As a direct consequence, the proportion of the other components in relation to carbon monoxide has increased and for any given carbon monoxide concentration, the relative proportion of the other components have increased between 2 and 4 fold.

Hence the control of the tunnel on the basis of a carbon monoxide concentration which may have been appropriate in the 1990's, leads inevitably to an increase in the other critical pollutants by a factor of up to 4 in 2006.

As detailed in our previous submission to the inquiry, we find it alarming that the regulatory authorities such as NSW Health, DEC and Planning have continued to approve the use of outdated and inappropriate standards for assessing health risks and regulating pollution standards. These issues are discussed in more detail in the document "Regulation of pollutants in Sydney tunnels" (Appendix C).

[5] 5 Dockery, D.W., Pope, C.A., Xu, S. and Spengler, J.D., et al; An Association Between Air Pollution and Mortality in Six U.S. Cities, 329 NEJM 1753-59 (1993).

There are no limits set for fine (PM2.5) particles in tunnel projects, either inside tunnels or outside their outlets. In the case of the M5 East, particles inside the tunnel have been documented at 10 times the levels of ambient air limits.[6] The 2003 Department of Health study of in-tunnel exposure in the M5 East showed particles above levels known to be harmful, especially in combination with nitrogen dioxide.

Rather than impose stricter conditions in relation to particles and nitrogen dioxide, as occurred in Sweden following similar studies, the conditions for the Cross City and Lane Cove tunnels allow the RTA to maintain higher in - tunnel levels of pollutants by setting inappropriately high in-stack limits for particles (up to 1600 $\mu g/m^3$ PM10), greater than those currently experienced in the M5 East.

To this day, NSW Health has still not released the results of its review of the M5 East Health study undertaken in 2003 on the effect of exhaust emissions on residents, which was strongly criticised by independent experts, including the independent NSW Health appointed peer reviewer. The recent announcement of a NSW Health study into the effects of the Lane Cove tunnel on residents, and in particular children in our view represents another 'far too little, too late', or at best a missed opportunity and more precious mis-spent public resources, which should be diverted to reducing the pollution rather than documenting the effects on children and vulnerable members of the community.

RAPS conclusion

RAPS concludes, with some considerable reason, that the current attitudes to vehicle and especially particulate pollution, evident in what appears to be the policy of the current government, is unsupportable and dangerous. The exposure of motorists and those affected by stack emissions to such harmful pollution, when there appears to be a relatively simple, effective and economical solution appears irresponsible, and to demonstrate a failure in the government's duty of care.

Community members are aware of the frequent and well researched findings of responsible health experts which put the number of deaths caused by air pollution and mainly by vehicular pollution higher than those caused by motor vehicle accidents and note the disproportionate amount of money and effort applied to the solution of these problems.

Given the extreme cost of provision of health care to those impacted by the effects of vehicle pollution, they see it as irresponsible to ignore the beneficial impacts of the removal of a major cause of illness and death in the community.

Debate over costs and viability of filtration

There have been a number of unsupported and unsupportable claims made in relation to the viability and costs of filtration, for example statements that filtration would cost $1 Billion, and would not be effective. (see for eg Min Roozendaal's statements in Hansard 6.4.06).

The arguments in relation to the viability and utility of filtration have been well canvassed in three previous inquiries relating to the M5 East.

Reports carried out for the RTA, such as that recently completed by Mr Child,[7] have found that "*technology for the removal of airborne particles, nitrogen dioxide, to some extent nitric oxide, and volatile hydrocarbons has now been either established or satisfactorily demonstrated in road tunnel applications.*" and that "*A potential basis for the cost-effective use of emission treatment technology in road tunnels has been identified. Confirmation of this potential will require a more detailed consideration of the specific circumstances associated with individual tunnels.*"

Not only has the RTA actively resisted any treatment of tunnel air, it has also refused to make provision for the installation on in-tunnel, as opposed to in-stack treatment systems, despite clear admissions by its own senior managers that in-tunnel treatment systems constituted best practice for environmental as well as energy costs reasons.

The last call for papers revealed that in Jan 2004, as a result of the problem with portal emissions from the M5 tunnel, the RTA , in cooperation with the tunnel operator, prepared a plan to fit filtration to this tunnel.

Now debate seems to be over cost. It is worth observing that it is common practice to inflate costs and difficulties of things you don't want to do, and understate costs and difficulties of what you do want to do.

As with PPP, costs of filtering or not should take into account public interest costs, costs of illness that are 'privatised' due to government inaction.

[6] General Purpose Standing Committee No. 5 Inquiry into the M5 East Tunnel 2002, p67

[7] Child,N. (2005) M5 East: a review of emission treatment technologies, systems and applications. http://www.rta.nsw.gov.au/constructionmaintenance/downloads/2004_10_childrepfiltration_dl1.html

What constitutes 'cost' in reference to filtration.

In defining the cost of fitting filtration in a tunnel there is the potential for a genuine difference of opinion. The possible cost basis are:

- What must be paid to the supplier/ manufacturer/ installer of the equipment to supply; install and commission the equipment. in a space prepared to the supplier's specification with basic services available.
- The above costs plus the costs involved with providing the space and services required.
- The above costs plus the 'on-costs' including RTA and main contractors mark-up (which can be as high as 28% for the M5)
- The 'whole of life costs' including running and maintenance costs and , in some cases, the eventual decommissioning and removal of the equipment.

RAPS and other community organizations have, in most cases, used the first cost criteria, while the RTA appears in most cases to have used the last criteria. The community organizations have always tried to make clear the basis of their claims and the fact that they are an incomplete estimate because:

1. The manufacturer's cost was the most accessible data available.
2. It was felt that the manufacturers' cost was what most closely corresponded to the public's conception of what would represent a true cost, as the government was 'theirs'.
3. The cost of providing the space for the filtration equipment was determined to a great extent by the stage of the project at which it was provided, the cost being minimal if the space was provided during the initial excavation of the tunnel but could be very large if the work was carried out in an operational tunnel. As this cost was a consequence of the failure of the RTA to respond appropriately to the community desire for filtration it was not considered germane to the cost discussion.
4. The extent of the RTA and manufacturers mark-up was and in most cases still is unknown to the public and is regarded as a flaw in the contractural arrangements entered into by the RTA.

Although 'whole of life' costing is an appropriate consideration in the assessment of the value of a particular project, it does not constitute, in any way, what the public would regard as the 'cost' of a piece of infrastructure, be it a filter system or the Harbour Bridge.

What is the appropriate type of filtration for tunnels.

Of the three main pollutants which occur in vehicle exhaust, effective, established technology exists to remove particulate matter and recently developed equipment appears able to effectively reduce nitrogen dioxide levels, either by completely removing it (absorbed onto a solid substrate) or by converting it to relatively harmless nitrogen oxide. Electrostatic precipitator technology is much cheaper to install and run and occupies much less space than does nitrogen dioxide removal technology.

No technology exists to effectively remove carbon monoxide from tunnel atmospheres.

Such health based research as exists, which refers specifically to tunnel exposures, suggests that nitrogen dioxide and particulate matter interact inside a tunnel in an additive way over relatively short exposure times and that the ill effects experienced are the result of this additive impact.[8]

This has led the conclusion that there is significant benefit to be achieved by the removal of particulate matter alone inside a tunnel. This conclusion is validated by over 30 years of experience of the beneficial effects from the use of electrostatic precipitators in tunnels in Japan.

Nitrogen dioxide, in association with particulate matter, becomes a concern outside the tunnel, either in the stack or portal emissions.

It is on this basis that RAPS has made a series of suggestions as to how the conditions inside the M5 tunnel may be significantly improved. All of these suggestions involve the permanent removal of varying proportions of particulate matter, inside the tunnel and in portal emissions, and nitrogen dioxide in portal emissions.

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[8] Bjornback, M.; Bylin, G, *et al.* Impact of NO2 on Health: exposure in Road Tunnels. (2000) PIARC Committee on Road Tunnels WG No2 "Pollution, environment and ventilation".
M. Svartengren, V. Strand, G. *et al.* Short-term exposure to air pollution in a road tunnel enhances the asthmatic response to allergen. Eur Respir J (2000) 15:716-724:

Early cost estimates for filtering the M5 Tunnel

As part of the conditions of approval, the RTA was required to prepare detailed plans for the construction of electrostatic precipitators prior to the opening of the tunnel.

The RTA commissioned Flagstaff, a consultant engineering firm to prepare these plans. The equipment considered was that manufactured by CTA Norway, however the costs used by Flagstaff did not correspond to those provided by CTA to the second inquiry. This was because there was a large element of double counting in the Flagstaff estimates (the waste water plant, mechanical and electrical installation, cabeling and ESP controls) and because the plant was significantly over-size.

The proposed installation was sufficient to treat 850 cubic meters of air per second and the cost estimate was just less than $37 million. This cost included $3.3 million in 'contingency', $2.2 million in project management and site 'overheads' $1.6 million in 'profit' , in addition to the other double countings.

These problems notwithstanding, this is the only known systematic attempt to estimate the cost of actually installing electrostatic precipitator filtration in a Sydney tunnel. As it examined an installation under the most difficult situation, in an already operating tunnel it at least sets a maximum cost.

This estimate was the basis of later extrapolations as to the cost of filtering 'all' of Sydney's tunnels even though it only referred to the cost of installing electrostatic precipitation, not electrostatic precipitation and nitrogen dioxide removal.

The filtration trial

Minister Scully announced in March 2004 that there would be a 'trial' of filtration and gas treatment technologies. The volume of air to be treated was 50 cubic meters per second, insufficient to make a significant difference in any Sydney tunnel. After an extensive selection process, it was announced that 3 firms had been short listed for further consideration. These firms were Panasonic, Siemens Filtrontec and Kawasaki. The respective "proponent costs' (ie the quote price) were $7.5 million, $7million and $5.5million respectively.

Documents contained in those tabled in the Legislative Council recently show how these costs became 'inflated' to $19.6 million, $18.7 million and $16.3 million through the addition of various operational and annual running costs and two cumulative profit mark-ups of 28% each.

Part of RTA document 0484

Pilot Filtration Plant

Item	Panasonic	Siemens	Kawasaki
Operational Consumables	$2,000,000	$2,000,000	$2,000,000
Operational	$338,500	$238,500	$255,000
RTA Project Managment	$240,000	$240,000	$240,000
Maintenance	$1,880,000	$1,880,000	$1,880,000
Refurbishment	$160,000	$160,000	$160,000
Comparative Proponent Costs	$7,481,379	$6,997,545	$5,424,560
TOTAL	$12,099,679	$11,516,045	$9,959,560
28% Mark Up (Excl RTA) Sub Con	$3,320,766	$3,224,493	$2,788,677
28% Mark Up (Excl RTA) Main Contractor	$4,223,001	$4,127,351	$3,569,506
TOTAL COST INCLUDING MARKUP	$19,643,646	$18,867,888	$16,317,743
Average Annual Cost	$1,964,364.58	$1,886,788.81	$1,631,774.31

The community is at a loss to know what to think about this 'trial' which is still claimed to be in progress, however it is clear that these prices do not provide a reliable estimate for the cost of fitting effective filtration to a tunnel.

Recent cost information

In April 2005 the Madrid City council announced that, as a response to community desires (and, it should be noted, significant pressure from the European Government about the extent of air pollution in Madrid), the tunnels of the Calle

30 ring-route then under construction would be filtered. The ring route is approximately 100 km long, of which 55 km is in tunnels.

The filtration of the first tunnel, the By-Pass Sur, will be installed in the stacks of the tunnel as the construction of the tunnel had already commenced. The filtration was to remove in excess of 80% of both particulate matter and nitrogen dioxide from the stack exhaust. It is expected that later tunnels will use in-tunnel filtration.

As a result of a call for expressions of interest from capable providers of filtration equipment, two companies, CTA and Aigner (an Austrian firm) were awarded contracts, each for one tube of the tunnel.

Through its contacts with CTA, RAPS were able to obtain details of the initial proposal made to the Madrid authorities and the final contract price. It is expected that further details will be forthcoming.

We have attempted to obtain similar information from Aigner but as yet we have been unsuccessful.

This is a summary of information received from Mr Anderl on the 23rd May.

CTA has been awarded the contract for one tube and Aigner, an Austrian firm, the other. The contract process is now finalised.

Excavation and general fit-out is well advanced and the fitting of the 4 filtration and gas treatment stations has started. The filter station work is planned to be finished by next February.

The CTA contract is for two filtration and gas cleaning stations treating approximately 700 and 400 cubic meters per second for the removal of particles, nitrogen dioxide and hydrocarbons.

Both companies will use gas cleaning based on the catalytic system developed by ABB in Norway for the Laerdal tunnel.

The CTA contract is for (Euro) 11,262,250, roughly equivalent to $A 18 million.

This information has been passed to the RTA along with the call for expressions of interest by the Madrid City Council and the technical details provided as a part of the CTA quote.

The contract to CTA involves the supply, installation, commissioning and, it is believed, 2 years maintenance of the equipment in the space made available in the tunnel.

From this we hold that the cost of filtration (on the basis of the first option for assessing cost) is equivalent to $A1.6 million per 100 cubic meters of air treated per second. This is generally consistent with the costs for CTA equipment quoted previously, taking into consideration changes in exchange rates.

The M5 tunnel uses about 900 cubic meters of air per second for ventilation. The RAPS suggestion to improve conditions inside and outside the M5 tunnel by more than 50% would require 700 cubic meters per second of cleaning capacity.

Conclusion

We would suggest that the only way to actually determine the cost of fitting filtration in any tunnel is on the basis of a call for expressions of interest to meet a real and well defined equipment and environmental outcome goal.

The notice issued by the Municipality of Madrid would form a good basis to begin. It could be provided to the inquiry if necessary. It clearly defines what the Madrid government was attempting to achieve and appears to have succeeded in reaching this goals.

We welcome this opportunity to make a submission to this extended inquiry and we hope that your deliberations and findings will lead to improved outcomes for all tunnel users, for those who live near the tunnels and for the general public in regard to the cost of these major pieces of infrastructure.

On behalf of RAPS (Residents Against Polluting Stacks)

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Appendix A

Medical Evidence about impacts of vehicle pollution

Since about the year 2000, there has been an explosive growth in research reporting adverse health impacts resulting from exposure to vehicle pollutants and especially, particulate matter. Starting mainly with epidemiological studies, this research has progressed to the explanation of some of the detailed physiological mechanisms causing the ill effects. The outstanding recent insight is the identification of the crucial impacts of ultra fine and 'nano' particles, the size and solubility of which make them uniquely harmful. From this it follows that the measure, PM10, commonly used in regulation, is actually inappropriate as it under-represents these particles which are less than 1 micron in diameter.

Some recent medical research on the impact of particulate matter in children and healthy adults.

Particulate air pollution and acute health effects Seaton,A., MacNee,W.,Donaldson,K.,Godden,D., *Lancet* 1995 345:176-78
Summary Epidemiological studies have consistently shown an association between particulate air pollution and not only exacerbations of illness in people with respiratory disease but also rises in the numbers of deaths from cardiovascular and respiratory disease among older people. We propose that the explanation lies in the nature of the urban particulate cloud, which may contain up to 100000 nanometer-sized particles per mL, in what may be a gravimetric concentration of only 100-200 $\mu g/m^3$ of pollutant. We suggest that such ultra-fine particles are able to provoke alveolar inflammation, with release of mediators capable, in susceptible individuals, of causing exacerbations of lung disease and of increasing blood coagulability, thus also explaining the observed increases in cardiovascular deaths associated with urban pollution.

Inhalation of Fine Particulate Air Pollution and Ozone Causes Acute Arterial Vasoconstriction in Healthy Adults Brook RD, Brook JR, Urch BU, Vincent R, Rajagopalan S, Silverman F. *Circulation* 2002;105:1534–6.
Fine particulate air pollution increases the risk of cardiovascular events. This study sought to determine the effects of fine particles and ozone on vascular function. 25 healthy adults underwent a randomized double-blind cross over study comparing the vascular response to a 2-hour inhalation of 150 g/m3 of fine particles (CAP) plus 120 ppb ozone to inhaled filtered air
Conclusion: Short-term inhalation of fine particulate air pollution and ozone at concentrations that occur in the urban environment causes acute coronary artery vasoconstriction.

Increased Particulate Air Pollution and the Triggering of Myocardial Infarction Annette Peters, PhD; Douglas W. Dockery, ScD; James E. Muller, MD; Murray A. Mittleman, MD, DrPH. (*Circulation.* 2001;103:2810-2815.)
Background—Elevated concentrations of ambient particulate air pollution have been associated with increased hospital admissions for cardiovascular disease. Whether high concentrations of ambient particles can trigger the onset of acute myocardial infarction (MI), however, remains unknown.
Results—The risk of MI onset increased in association with elevated concentrations of fine particles in the previous 2-hour period. In addition, a delayed response associated with 24-hour average exposure 1 day before the onset of symptoms was observed. Multivariate analyses revealed an estimated odds ratio of 1.48 associated with an increase of 25 mg/m3 PM2.5 during a 2-hour period before the onset and an odds ratio of 1.69 for an increase of 20 mg/m3 PM2.5 in the 24-hour period 1 day before the onset (95% CIs 1.09, 2.02 and 1.13, 2.34, respectively).
Conclusions—This study suggests that elevated concentrations of fine particles in the air may transiently elevate the risk of MIs within a few hours and 1 day after exposure.

Exposure to Traffic and the Onset of Myocardial Infarction Annette Peters, Ph.D., *et al.N Engl J Med* 2004;351:1721-30.
An association was found between exposure to traffic and the onset of a myocardial infarction within one hour afterward (odds ratio, 2.92; 95 percent confidence interval, 2.22 to 3.83; P<0.001). The time the subjects spent in cars, on public transportation, or on motorcycles or bicycles was consistently linked with an increase in the risk of myocardial infarction. The subject's use of a car was the most common source of exposure to traffic; nevertheless, there was also an association between time spent on public transportation and the onset of a myocardial infarction one hour later.
Conclusion: Transient exposure to traffic may increase the risk of myocardial infarction in susceptible persons.

The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age W. James Gauderman, *et al. N Engl J Med* 2004;351:1057-67.
Background: Whether exposure to air pollution adversely affects the growth of lung function during the period of rapid lung development that occurs between the ages of 10 and 18 years is unknown. In this prospective study, 1759 children (average age, 10 years) from schools in 12 southern California communities and measured lung function annually for eight years. The communities represented a wide range of ambient exposures to ozone, acid vapor, nitrogen dioxide, and particulate matter.
Conclusion: The results of this study indicate that current levels of air pollution have chronic, adverse effects on lung development in children from the age of 10 to 18 years, leading to clinically significant deficits in attained FEV (forced expiratory volume) as children reach adulthood.

Air pollution and childhood asthma emergency hospital admissions: Estimating intra-city regional variations. Erbas,B., Kelly,A.M. *et al. International Journal of Environmental Health Research* Feb 2005; 15(1): 11 – 20
Abstract: In recent years childhood asthma has increased. Although the precipitants of childhood asthma are yet to be established possible contributing factors are local ambient air pollutants. This study aims to assess associations of regional ambient air pollutants on emergency department childhood asthma presentations across four regions of the city of Melbourne, Australia.. There was consistent associations between childhood ED asthma presentations and regional concentration of PM10, with a strongest

association of RR= 1.17 (95% CI 1.05 to 1.31) in the central district of Melbourne. NO2 and Ozone was associated with increased childhood asthma ED presentations in the Western districts. This study suggests that regional concentrations of PM10 may have a significant effect on childhood asthma morbidity. In addition, ozone may play a role however, its effect may vary by geographical region.

American Academy of Pediatrics Policy statement: Ambient Air Pollution: Health Hazards to Children Michael W Shannon; Dana Best; et al *Pediatrics;* Dec 2004; 114, 6;
ABSTRACT. Ambient (outdoor) air pollution is now recognized as an important problem, both nationally and worldwide. Our scientific understanding of the spectrum of health effects of air pollution has increased, and numerous studies are finding important health effects from air pollution at levels once considered safe. Children and infants are among the most susceptible to many of the air pollutants. In addition to associations between air pollution and respiratory symptoms, asthma exacerbations, and asthma hospitalizations, recent studies have found links between air pollution and preterm. birth, infant mortality, deficits in lung growth, and possibly, development of asthma. This policy statement summarizes the recent literature linking ambient air pollution to adverse health outcomes in children and includes a perspective on the current regulatory process. The statement provides advice to pediatricians on how to integrate issues regarding air quality and health into patient education and children's environmental health advocacy and concludes with recommendations to the government on promotion of effective air-pollution policies to ensure protection of children's health.

Partial list of articles about health impacts of PM10 and NO_2 published before 2000

1. Higgins IT. Epidemiological evidence on the carcinogenic risk of air pollution. IARC Sci Publ 1976;13:41–52.
2. Laurie, R.D., and Boyes, W.K., Neurophysiological Alterations Due to Diesel Exhaust During the Neonatal Life of the Rat, Environ Int., (1981) :5:363-8;
3. Laurie, R.D., Boyes, W.K., and Wessendarp, T., Behavioral Alterations Due to Diesel Exhaust Exposure, Environ Int., (1981). a:5:357-61;
4. Pepelko, W.E. and Peirano, W.B., Health Effects of Exposure to Diesel Engine Emissions: a Summary of Animal Studies Conducted by the US EPA's Health Effects Research Laboratories at Cincinnati, Ohio, J. Am. Coll. Toxicol. 1983:2(4):253-306.
5. Pershagen G. Air pollution and cancer. In: Pershagen G, Vainio H, Sorsa M, McMichael AJ, eds. Complex mixtures and cancer risk. Lyon: International Agency for Research on Cancer, 1990:240–51.
6. Folinsbee U. Does nitrogen dioxide exposure increase airway responsiveness? Toxicological and Industrial Health 1992 ; 8(5) : 273-283 ;
7. Pope, C.A., and Dockery, D.W., Acute Health Effects of PM10 Pollution Symptomatic and Asymptomatic Children, American Review of Respiratory Disease, Vol. 145, (1992), pp. 1123-1128;
8. Pope, C.A., Thun, M.J., Namboordiri, M.M. and Dockery, D.W., et al.; Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of U.S. Adults,. 151 American Journal of Respiratory and Critical Care Medicine (1995). Available online at http://ajrccm.ats journals.org/ search.html;
9. Dockery, D.W., Pope, C.A., Xu, S. and Spengler, J.D., et al; An Association Between Air Pollution and Mortality in Six U.S. Cities, 329 New England J. Medicine 1753-59 (1993).
10. Cohen, A.J., and Higgins, M.W.P., Health Effects of Diesel Exhaust: Epidemiology, Diesel Exhaust : A Critical Analysis of emissions, Exposure and Health Effects, pp. 251-292, Health Effects Institute, Cambridge MA., (April 1995).
11. Katsouyanni K, Touloumi G, Spix C, et al. Short term effects of ambient sulphur dioxide and particulate mater on mortality in 12 European cities: results from time series data from the APHEA project. BMJ 1997;314:1658–63.
12. Rusznak, C.; Bayram,H.; Devalia,J.L.: Davies,R.J. Impact of the environment on allergic lung diseases. Clinical and Experimental Allergy. 27 sup1(1997). pp.26-35.
13. Woodruff, T., Grillo, J. and Schoendorf, K., The Relationship Between Selected Causes of Postneonatal Infant Mortality and Particulate Air Pollution in the United States, Environmental Health Perspectives, Vol. 105, (1997). pp. 608-612.
14. Lewis, P.R.; Hensley, M.J. *et al* Outdoor air pollution and children's respiratory symptoms in the steel cities of New South Wales. MJA. (1998) 169: pp459-463.

15. Strand V. Effects of nitrogen dioxide on airway responsiveness in allergic asthma Stockholm 1998. Doktorsavhandling

16. Perera FP, Whyatt RM, Jedrychowski W, Rauh V, Manchester D, Santella RM. 1998. Recent developments in molecular epidemiology: a study of the effects of environmental polycylic aromatic hydrocarbons on birth outcomes in Poland. Am J Epidemiol 147:309–314.

17. Liao,D., Creason, J., Shy,C., Williams, R., Watts, R., and Zweidinger, R., Daily Variation of Particulate Air Pollution and Poor Cardiac Autonomic Control in the Elderly, Environmental Health Perspectives, Vol.107, No.7. (July 1999);

18. Lipsett M, Campleman S. Occupational exposure to diesel exhaust and lung cancer: a meta-analysis. Am J Public Health 1999;89:1009–17.

19. Neas LM, Schwartz J, Dockery DW. A case-crossover analysis of air pollution and mortality in Philadelphia. Environ Health Perspect 1999;107:629–31.

20. Norris, G., Young Pong, N., Koenig, J., Larson, T., Sheppard,L. and Stout, J., An Association Between Fine Particles and Asthma Emergency Department Visits for Children in Seattle, Environmental Health Perspectives, Vol. 107, No. 6, (1999), pp. 489-493;

21. Maynard RL, Howard B, eds. Particulate matter: properties and effects upon health. Oxford: Bios Scientific Publishers, 1999.

Partial list of articles about health impacts of particulate matter published after 2000

1. Bjornback, M.; Bylin, G, *et al.* Impact of NO2 on Health: exposure in Road Tunnels. (2000) PIARC Committee on Road Tunnels WG No2 "Pollution, environment and ventilation".
2. Brauer, M., Hoek, G., Van Vliet , P., et al., Air Pollution from Traffic and the Development of Respiratory Infections and Asthmatic and Allergic Symptoms in Children, American Journal of Respiratory and Critical Care Medicine, Vol.166 , (2002), pp. 1092-1098;
3. Brook, R.D., Brook, J.R., Urch, B., Rajagopalan, S., Silverman, P., Inhalation of Fine Particulate Air Pollution and Ozone Causes Acute Arterial Vasoconstriction in Healthy Adults, Circulation, Vol.105, (2002), pp. 1534-1536,
4. Bunn, H J, Dinsdale, D, Smith, T, Grigg,J. Ultrafine particles in alveolar macrophages from normal children. *Thorax* 2001;56:932–934
5. Cohen A. Outdoor air pollution and lung cancer. Environ Health Perspect 2000;108:743–50.
6. Dejmek J, Solansky I, Benes I, Lenicek J, Srám RJ. 2000. The impact of polycyclic aromatic hydrocarbons and fine particles on pregnancy outcome. Environ Health Perspect 108:1159–1164.
7. Donaldson K, Brown D, Clouter A, et al. The pulmonary toxicology of ultrafine particles. J Aerosol Med 2002;15:213–20.
8. Donaldson, K., et al. Ambient Particle Inhalation and the Cardiovascular System: Potential Mechanisms, Environmental Health Perspectives, Vol. 109, Supp.4;
9. Finkelstein, M., Jerrett, M., and Sears, M., Traffic, Air Pollution and Mortality Rate Advancement Periods, American Journal of Epidemiology, Vol. 160, (2004), pp. 173-177;
10. Friedman, Michael S.; Powell, Kenneth E., *et al.* Impact of Changes in Transportation and Commuting Behaviors During the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma. JAMA, Vol 285, (2001) pp897-905.
11. Fruin et al., "Fine particle and black carbon concentrations inside vehicles," 10th Annual Conference of the International Society of Exposure Analysis, Oct., 2000.
12. Garshick, E., Laden, F., Hart, J., Rosner, B., Smith, T., Dockery, D. and Speizer, F., Lung Cancer in Railroad Workers Exposed to Diesel Exhaust, Environmental Health Perspectives, Vol. 122, No. 15, (November 2004), pp. 1539-1543.
13. Gauderman, W.J., McConnell, R., Gilliland, F., London, S., Thomas, D., Avol, E., Vora, H., Berhane, K., Rappaport, E., Lurmann, F., Margolis, H.G., and Peters, J., Association Between Air Pollution and Lung Function Growth in Southern California Children, American Journal of Respiratory and Critical Care Medicine, Vol. 162, No. 4, (2000), pp. 1-8;
14. Ghio, A.J., and Devlin, R.B., (2001). Inflamatory Lung Injury After Bronchial Instillation of Air Pollution Particles, American

Journal of Respiratory Critical Care Medicine, Vol. 164, (2001) p. 704-708;

15. Gold, D., Litonjua, A., Schwartz, J., Lovett, E., Larson, A., Nearing, B., Allen, G., Verrier, M., Cherry, R., and Verrier, R. Ambient Pollution and Heart Rate Variability, Vol. 101, No. 11, (21 March 2000), pp. 1267- 1273;

16. Harrison RM, Smith DJT, Kibble AJ. What is responsible for the carcinogenicity of PM2.5? Occup Environ Med 2004;61:799–805.

17. Hoek, G., Brunekreef, B., Goldbohm, S., Fischer, P. and van den Brandt, P., Association Between Mortality and Indicators of Traffic- Related Air Pollution in the Netherlands: a Cohort Study, The Lancet, Vol. 360, December 19, 2002, pp.1203-1209;

18. Hong, Y., Lee, J., Kim, H., Ha, E., Schwartz, J. and Christiani, D.C., Effects of Air Pollutants on Acute Stroke Mortality, Environmental Health Perspectives, Vol. 110, No. 2, (February 2002).

19. Hong, Y., Lee, J., Kim, H., Kwon, H., Air Pollution. A New Risk Factor in Ischemic Stroke Mortality, Stroke, Vol. 33, (2002), pp.2165-2169;

20. Kilburn, K.H., Effects of Diesel Exhaust on Neurobehavioral and Pulmonary Functions, Archives of Environmental Health, Vol. 55, No. 1, (2000), pp. 11-17.

21. Kim, J., Smorodinsky, S., Lipsett, M., Singer, B., Hodgson, A., and Ostro, B., Traffic-related Air Pollution near Busy Roads The East Bay Children's Respiratory Health Study, American Journal of Respiratory and Critical Care Medicine, Vol. 170, (2004), pp. 520- 526;

22. Knaapen AM, Borm PJ, Albrecht C, et al. Inhaled particles and lung cancer. Part A: Mechanisms. Int J Cancer 2004;109:799–809.

23. Krewski, D., Burnett, R.T., Goldberg, M.S., Hoover, K., Siemiatycki, J., Jerrett, M., Abrahamowicz, A. and White, W.H., Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Matter and Mortality; Special Report to the Health Effects Institute, Cambridge, MA (July 2000);

24. Levy D, Sheppard L, Checkoway H, et al. A case-crossover analysis of particulate matter air pollution and out of hospital primary cardiac arrest. Epidemiology 2001;12:193–9.

25. Lin, S., Munsie, J., Hwang, S., Fitzgerald, E., and Cayo, M., Childhood Asthma Hospitalization and Residential Exposure to State Route Traffic, Environmental Research Section A 88, (2002), pp. 73-81.

26. Mar,T., Norris,G., Koenig, J. and Larson, T., Associations Between Air Pollution and Mortality in Phoenix, 1995– 1997, Environmental Health Perspectives, Vol.108, No. 4, (April 2000).

27. Marr, L.C., Grogan, L.A., Wohrnschimmel, H., Molina, L., Molina, M., Smith, T., Garshick, E., Vehicle Traffic as a Source of Particulate Polycyclic Aromatic Hydrocarbon Exposure in the Mexico City Metropolitan Area, Environmental Science and Technology, Vol. 38, No. 9, (2004), pp. 2584-2592;

28. Nemmar, A., Hoet, P., Dinsdale, D.,Vermylen, J., Hoylaerts,M., and Nemery, B., Diesel Exhaust Particles in Lung Acutely Enhance Experimental Peripheral Thrombosis, Circulation. Vol. 107, (2003), pp.1202-1208.

29. Perera FP, Rauh V, Tsai WY, Kinney P, Camann D, Barr D, et al. 2003. Effects of transplacental exposure to environmental pollutants on birth outcomes in a multi-ethnic population. Environ Health Perspect 111:201–205.

30. Peters, A., and Pope, A.C., Cardiopulmonary Mortality and Air Pollution, The Lancet, Vol. 360, (October 19, 2002), p.1184,

31. Peters, A., Dockery, D.W., Muller, J.E., Mittleman, M.A., Increased Particulate Air Pollution and the Triggering of Myocardial Infarction, Circulation, Vol. 103, (2001), pp. 2810-2815;

32. Peters, A., Increased Particulate Air Pollution and the Triggering of Myocardial Infarction, Circulation, Vol. 109, (June 12, 2001);

33. Peters, A., Liu, E., Verier, R.I. et al., Air Pollution and Incidence of Cardiac Arrhythmia, Epidemiology, Vol. 11, (2000), pp.11-17.

34. Peters, A., Von Klot, S., Heier, A., Trentinaglia, I., Hormann, A., Wichmann, E., Lowel, H., Exposure to Traffic and the

Onset of Myocardial Infarction, NEJM, Vol. 351, No 17, (October 15, 2004);

35. Riediker, M., Cascio, W., Griggs, T., Herbst, M., Bromberg, P., Neas, L., Williams, R., and Devlin, R., Particulate Matter Exposure in Cars Is Associated with Cardiovascular Effects in Healthy Young Men, American Journal of Respiratory and Critical Care Medicine, Vol. 169, (2004), pp. 934-940;

36. Ro¨o¨sli M, Kunzli N, Schindler C, et al. Single pollutant versus surrogate measure approaches: do single pollutant risk assessments underestimate the impact of air pollution on lung cancer risk? J Occup Environ Med 2003;45:715–23.

37. Samet, J.M., Dominici, F., Zeger, S.L., Schwartz, J. and Dockery, D.W. National Morbidity, Mortality and Air Pollution Study, Part II: Morbidity, Mortality and Air Pollution in the United States; Health Effects Institute Research Report No. 94, Cambridge MA (June 2000);

38. Schwartz, J. The effects of particulate air pollution on daily deaths: a multi-city case crossover analysis Occup Environ Med 2004;61:956–961

39. Steffen, C , M F Auclerc, A Auvrignon, A Baruchel, K Kebaili, A Lambilliotte, G Leverger, Sunyer J, Schwartz J, Tobias A, et al. Patients with chronic obstructive pulmonary disease are at increased risk of death associated with urban particulate air pollution: a case-crossover analysis. Am J Epidemiol 2000;151:50–6.

40. M. Svartengren, V. Strand, G. *et al.* Short-term exposure to air pollution in a road tunnel enhances the asthmatic response to allergen. Eur Respir J (2000) 15:716-724:

41. Tolbert, P., et al. Air Quality and Pediatric Emergency Room Visits for Asthma in Atlanta, Georgia, American Journal of Epidemiology, Vol. 151, No. 8, (2000), pp. 798-810;

42. Weinhold, B., Pollutants Lurk Inside Vehicles: Don't Breathe and Drive? Environmental Health Perspectives, Vol. 109, No. 9, (September 2001);

Nanoparticles

1. Jani P, Halbert GW, Langridge J, et al. Nanoparticle uptake by the rat gastrointestinal mucosa: quantitation and particle size dependency. J Pharm Pharmacol 1990;42:821–6.

2. Colvin VL. The potential environmental impact of engineered nanomaterials. Nat Biotechnol 2003;21:1166–70.

3. Brumfiel G. Nanotechnology: a little knowledge. Nature 2003;424:246–8.

4. Donaldson K, Borm PJA. Particle paradigms. Inhal Toxicol 2000;12(suppl 3):1–6.

5. Donaldson K, Stone V, Clouter A, et al. Ultrafine particles. Occup Environ Med 2001;58:211–16.

6. Tran CL, Buchanan D, Cullen RT, et al. Inhalation of poorly soluble particles. II. Influence of particle surface area on inflammation and clearance. Inhal Toxicol 2000;12:1113–26.

7. Donaldson K, Tran CL. Inflammation caused by particles and fibres. Inhal Toxicol 2002;14:5–27.

8. Oberdorster G, Utell MJ. Ultrafine particles in the urban air: to the respiratory tract—and beyond? Environ Health Perspect 2002;110:A440–1.

9. Donaldson K, MacNee W. Potential mechanisms of adverse pulmonary and cardiovascular effects of particulate air pollution (PM10). Int J Hyg Environ Health 2001;203:411–15.

10. Donaldson K, Tran CL, MacNee W. Deposition and effects of fine and ultrafine particles in the respiratory tract. The European Respiratory Monograph 2002;7:77–92.

11. Oberdorster G, Sharp Z, Elder AP, et al. Translocation of inhaled ultrafine particles to the brain. Inhal Toxicol 2004;16:437–45.

WHY DIESEL EXHAUST IS SO DAMAGING TO HEALTH

Mark Curran   March 2006

It is not long ago that diesel engines were regarded as being safer alternatives to the petrol engine and the 'smoke' was thought of as a minor irritant, important because it was unsightly, smelly and reduced visibility in tunnels.

Modern medical science has clearly shown that this is not the case and that diesel exhaust is one of the most dangerous and widespread of modern pollutants.

All of the exact mechanisms by which diesel exhaust causes harm are not yet known but it is clear that the harmful effects are related both to the size and composition of the particles in the exhaust. Diesel exhaust is now known to be a carcinogen[1,2] and particulate pollution has been fairly labelled as the 'new asbestos' for its ability to cause serious illness and death.

In 2000, the respected New England Journal of Medicine [342.pp406-13] listed the following health impacts from particulate air pollution

- Rhinitis and laryngitis Large particles are deposited in the nose, pharynx, and larynx.
- Tracheitis, bronchitis and bronchiolitis Particles larger than 10µ cleared by cilia. Smaller particles and fibers deposited in bronchioles and alveola ducts.
- Asthma and chronic obstructive pulmonary disease. Allergens and irritants are deposited in large airways causing chronic inflammatory changes
- Cancer Carcinogens (asbestos and polycyclic aromatic hydrocarbons) come into contact with bronchial epithelial cells, causing mutations on proto-oncogenes and tumor-suppressor genes and leading to malignant transformation.
- Interstitial disease Small particles and fibers are deposited in terminal bronchioles and alveoli. Penetration of interstitium results in fibrosis and formation of granulomas.

All particles are dangerous and there is no safe level of exposure, but weight for weight, toxicity appears to double with each halving in the diameter of the particles inhaled. "Nano' particles less than about 100 nanometers in diameter severely irritate the lung, irrespective of their composition and can carry adsorbed toxins directly into lung tissues, easily penetrating cellular membranes.[3]

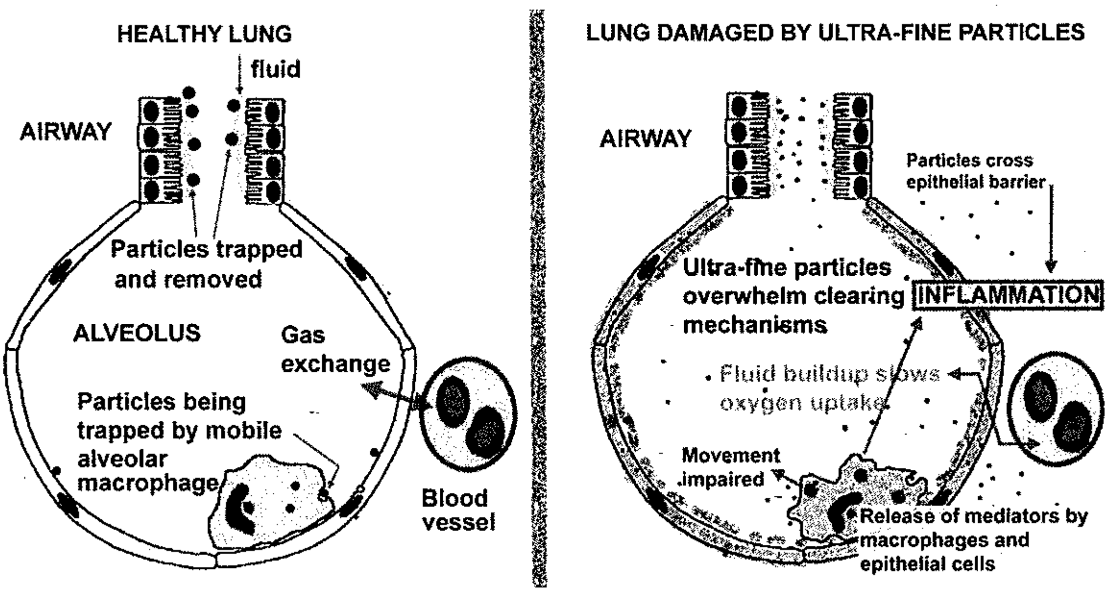


Diagram after Stone V. & Donaldson K. (1998) 'Small particles-big problem'. *Aerosol Soc Newsletter. 33.*

Diesel exhaust contains up to 100 times more ultra-fine and 'nano' particles than does exhaust from petrol engines.[4]

Proof of the benefits of reducing particulate matter levels.

By strictly regulating emissions from vehicles and a series of other measures, Tokyo reduced ambient particle levels by more than 50% between 1975 and 1998 (from >100$\mu g/m^3$ in 1975 to < 45$\mu g/m^3$ in1998) . The economic benefits of this reduction, mainly by its impact on health costs and including the value of wages not lost, has been estimated to have totalled $A 47.5 BILLION up to 1999.

Voorhees. A.S. Benefits Analysis of Particulate Matter control programs – A case Study in Tokyo. - J. Risk Research Vol. 8, 311-329 (June, 2005)

Growth of medical knowledge

Over 300 papers on the health effects of fine particles and vehicle emissions have been published in the last 10 years.

- In 1993 Dockery and Pope[5] published the findings of their '6 cities' study which clearly associated increases in particulate matter with increases in mortality and hospital admissions.
- In 2001, Friedman and Powell[6] showed that the reduction in vehicle emissions during the Atlanta Olympic Games significantly reduced childhood asthma attacks.
- In 2004 Ann Peters (*et al*)[7] published highly significant research which showed that short term exposure to vehicle pollutants could trigger the onset of myocardial infarctions (heart attacks).

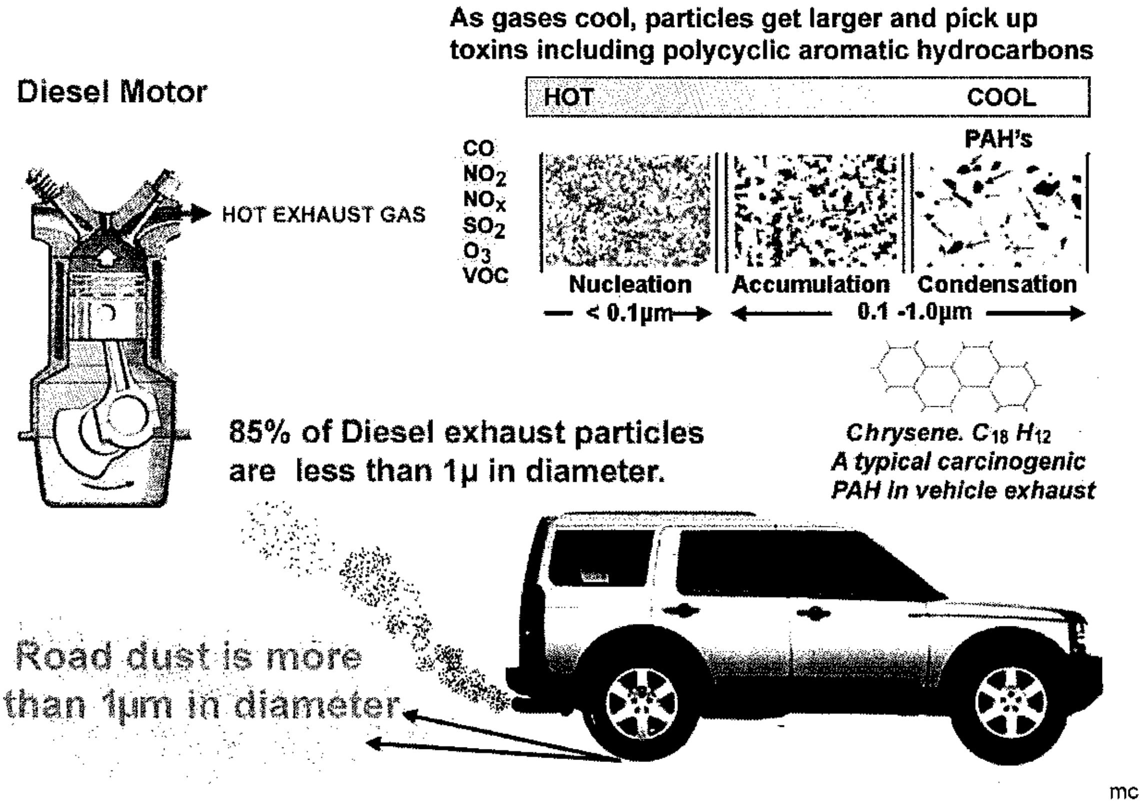
These are just landmarks in the steady progress of medical knowledge showing the adverse impacts of vehicle pollution and especially particulate pollution on all stages of the human life cycle, from before birth[8], through childhood[9] and early adulthood[10] to the causation of early mortality.[11]

In addition recent research carried out for the World Road Association (PIARC)[12] shows that particles interact with another significant pollutant, nitrogen dioxide in an additive way especially inside tunnels.

Particles in vehicle emissions consist mainly of carbon but also include fibers, metal fragments and soluble salts. The carbonaceous particles carry carcinogenic polycyclic aromatic hydrocarbons (PAH) and other volatile, and also carcinogenic, organic compounds (VOC) on their surface.

Diesel particles, which are mainly less than 1 μ (micron) in diameter and have an median diameter of about 0.2 μ (200 nanometers), are by far the most dangerous of the commonly found particles.

We are exposed to these pollutants at high concentrations in our cars or beside busy roads. In addition, every time we travel through an unfiltered urban tunnel our exposure jumps 10 fold. Tunnels might be good for traffic but unfiltered tunnels are bad for health!



[1] Lipsett M, Campleman S. Occupational exposure to diesel exhaust and lung cancer: a meta-analysis. Am J Public Health 1999;89:1009–17.
[2] Harrison RM, Smith DJT, Kibble AJ. What is responsible for the carcinogenicity of PM2.5? Occup Environ Med 2004;61:799–805
[3] Stone V. & Donaldson K. 'Small particles-big problem'. *Aerosol Soc* Newsletter1998;33.
[4] Kittelson,D B, (1998) Review of Diesel Particulate Matter Sampling Methods. Supplemental Report # 2 EPA Grant Review Of Diesel Particulate Matter. http://www.me.umn.edu/centers/cdr/reports/EPAreport2.pdf
[5] Dockery, D.W., Pope, C.A., Xu, S. and Spengler, J.D., et al; An Association Between Air Pollution and Mortality in Six U.S. Cities, 329 NEJM 1753-59 (1993).
[6] Friedman, M S, *et al.* Impact of Changes in Transportation and Commuting Behaviors During the 1996 Summer Olympic Games in Atlanta on Air Quality and Childhood Asthma. JAMA, Vol 285, (2001) pp897-905
[7] Peters, A., Von Klot, S., Heier, A., Trentinaglia, I., Hormann, A., Wichmann, E., Lowel, H., Exposure to Traffic and the Onset of Myocardial Infarction, NEJM, Vol. 351, No 17, (October 15, 2004);
[8] Dejmek J, Solansky I, Benes I, Lenicek J, Srám RJ. 2000. The impact of polycyclic aromatic hydrocarbons and fine particles on pregnancy outcome. Environ Health Perspect 108:1159–1164.
[9] Brauer, M., Hoek, G., Van Vliet, P., et al., Air Pollution from Traffic and the Development of Respiratory Infections and Asthmatic and Allergic Symptoms in Children, Am Journal of Respiratory and Critical Care Medicine, Vol.166, (2002), pp. 1092-1098;
[10] Riediker, M., Cascio, *et al.*, Particulate Matter Exposure in Cars Is Associated with Cardiovascular Effects in Healthy Young Men, Am. Journal of Respiratory and Critical Care Medicine, Vol. 169, (2004), pp. 934-940;
[11] Finkelstein, M., Jerrett, M., and Sears, M., Traffic, Air Pollution and Mortality Rate Advancement Periods, Am J Epidemiology, Vol. 160, (2004), pp. 173-177;.

[12] Bjornback, M.; Bylin, G, *et al.* Impact of NO2 on Health: exposure in Road Tunnels. (2000) PIARC Committee on Road Tunnels WG No2 "Pollution, environment and ventilation".

Regulation of pollutants in Sydney tunnels

The RTA routinely claims that the air quality inside their tunnels meets either the 'regulations', the 'standards' or 'international standards of best practice'. From the context of the RTA statements the regulations or standards refer to either the WHO goals for carbon monoxide and the provisions of the PIARC (World Road Association) Committee on Road tunnels.

The clear implication is that these standards or regulations for tunnel operation are nationally or internationally applicable or accepted and are appropriate for this purpose.

In fact there are no such standards specifically for tunnels and similar environments. The only 'regulations' which may be legally enforceable are the minister's conditions of approval for the project. These conditions refer to a number of 'goals', based either on the WHO or Australian NEPM.

The WHO goals are not standards, however they may be adopted by national and international legislation as standards. They have been given regulatory force by the European Union mainly in relation to ambient air quality and have been accepted as the basis for the Australian NEPM as ambient goals in some cases.

NEPM standards are legislative entities and apply only to the ambient background, and a population of 25,000 people. They do not and should not be applied to a point source such as a tunnel stack.

Generally, the NEPM clearly states that their goals should not be used for the assessment of other-than-ambient conditions and specifically refer to their unsuitability for use in the case of '*a road tunnel or a heavily trafficked canyon street. Air quality management in these areas is complex and needs a different approach to that directed at meeting ambient standards intended to reflect the general air quality in the airshed*'.

In practical terms, this caveat applies more to particulate matter and other pollutants than to carbon monoxide as the mode of action of carbon monoxide in the body is largely independent of other pollutants.

The unsuitability of carbon monoxide as a regulatory measure for tunnels arises from other considerations.

The difference between maintaining 'Health' and maintaining 'Safe Conditions'.

Providers of infrastructure such as tunnels have the responsibility to provide projects which are both safe and free from risk, especially to the health of both motorists and local residents.

There is a real difference between 'safety' and 'health' in the context of tunnel pollution. Safety is about "being free from danger". On the other hand, 'health' is about bodily and mental well-being.

Of all the toxic pollutants in the airstream of a traffic tunnel, the levels of carbon monoxide are crucial for 'safety' rather than 'health' since carbon monoxide is the only pollutant able to cause unconsciousness very quickly. Once carbon monoxide combines with haemoglobin that it takes up to 9 hours for the carbon monoxide to be displaced when the person breathes clean air, however urban motorists are not exposed to 'clean' air whilst driving. Hence there is also a potential 'safety' problem with multiple exposures, especially to professional drivers.

This is a subtle difference, even though carbon monoxide induced unconsciousness is also a health issue.

All the other pollutants have importance relating mainly to 'health', although the particle/nitrogen dioxide mixture in tunnel atmospheres can also provide a 'safety' problem through its ability to promote rapid onset of debilitating asthmatic symptoms (or even severe sneezing episodes).

Whilst the other gaseous pollutants and particles, either alone or in combination, can impact on health, especially in those persons with underlying risk-conditions e.g., heart and respiratory diseases, these do not normally present a 'safety' risk to a motorist in the tunnel as great as carbon monoxide, at least in the ACUTE phase.

The issues of the health impacts and the appropriate standards are complex for the other pollutants. Unlike the UK. there still is no standard for the highly toxic and carcinogenic 1,3-butadiene in the NEPM system in Australia, nor is there any health based standard for short term exposure to fine particles.

No attempt has been made to address the impacts of pollutants in combination, except to note that is a potential for such impacts.

Regulations Applying in Sydney Tunnels

Carbon monoxide.

The conditions of approval for Sydney tunnels refer to compliance with the WHO 15 and 30 minute 'goals' of 87ppm and 50 ppm. This is the only numerical goal specified for internal conditions in the M5 tunnel.

This is in spite of the fact that the PIARC documents on road tunnel ventilation state that carbon monoxide is no longer suitable as a sole determinant of tunnel ventilation. *"Classically most ventilation control systems are based on CO measurements in tunnel. Due to the decrease of CO emissions, these measurements are no longer sufficient to control the ventilation".* 'Pollution by nitrogen dioxide in road tunnels' PIARC (AIPCR, 05.09.B.2000)

Whereas previously the actions taken to provide 'safe' conditions, mainly in relation to carbon monoxide asphyxia, were thought to be sufficient to also protect motorists' health, this is clearly no longer the case.

The PIARC 1995 publication 'Road Tunnels: Emissions, Ventilation, Environment' upon which the M5 design is largely based notes: 'In this publication new design information and some references are given for dimensioning a longitudinal and a semi-transverse ventilation system.'

It contains the following table which is the basis of the 'regulations' the RTA claims to follow.

Table 2.3

Traffic situation	CO-concentration		Visibility	
	Design year dimensionnement		Extinction coefficient K	Transmission s (beam length: 100 m)
	1995	2010		
	ppm	ppm	10-3 . m⁻¹	%
Fluid peak traffic 50 - 100 km/h	100	70	5	60
Daily congested traffic, standstill on all lanes	100	70	7	50
Exceptional congested traffic, standstill on all lanes	150	100	9	40
Planned maintenance work in a tunnel under traffic	30	20	3	75
Closing of the tunnel	250	200	12	30

The wide range of 'permissible concentrations' is clearly not in the nature of a 'standard'.

The CO level is noted to correspond to the then-current WHO recommendation for short term exposures, however, the level falls to 70ppm under some conditions in 2010, lower than the current WHO recommendation of 87ppm.

The allowable concentrations are considerably higher than Australia is prepared to allow. In even the Sydney Harbour tunnel the CO limits were set at 100ppm/15 minutes and 150ppm/3 minutes.

Of significance to the Lane Cove tunnel is the observation in the same document ***"Design-year:** The design-year of a tunnel ventilation is usually the opening year, but it may also be 5 to 10 years later, when a traffic development is expected. The traffic composition and as average emission must be estimated for the design year."*
As traffic development is clearly expected for this tunnel then it follows that the 70 ppm CO limit should have been applied as it was to the new Brisbane tunnel. An even more stringent 50ppm /15 minute exposure limit is applied to the tunnel currently in planning in Melbourne.

The crucial part of Condition 70 which relates to operation states *"The tunnel ventilation system(s) must be designed and operated so that the World Health Organisation (WHO) 15-minute carbon monoxide (CO) goal of 87 ppm is not exceeded under any conditions."*

An attempt has been made by the RTA and Dept of Planning to hold that the true meaning of this condition is that no tunnel user may be exposed to a carbon monoxide concentration for more than 15 minutes, on the grounds that the WHO goal is an exposure goal.

The WHO publication Environmental Health Criteria 213 - CARBON MONOXIDE - (SECOND EDITION) states :

Recommended WHO guidelines

The following guideline values (ppm values rounded) and periods of time-weighted average exposures have been determined in such a way that the carboxyhaemoglobin level of 2.5% is not exceeded, even when a normal subject engages in light or moderate exercise:
100 mg/m3 (87 ppm) for 15 min
60 mg/m3 (52 ppm) for 30 min
30 mg/m3 (26 ppm) for 1 h

10 mg/m3 (9 ppm) for 8 h

The interpretation of condition 70 as requiring a person to have been exposed to the stated concentration of carbon monoxide for the stated time must be rejected for the following reasons:

- The community had been given to understand during community consultation that the condition would be interpreted as written and that the occurrence of a carbon monoxide concentration in excess of 87ppm for a time in excess of 15 minutes (or its equivalent) would be a breach of the condition.
- The words 'under any conditions' have a clear 'plain English' meaning. The tunnel must be operated in such way that a carbon monoxide concentration in excess of 87ppm for a time in excess of 15 minutes (or its equivalent) does not occur under any circumstances.
- The prohibition of the occurrence of a carbon monoxide concentration in excess of 87ppm for a time in excess of 15 minutes (or its equivalent) is consistent with good safety practice, which holds that the best way to control an unsafe condition or situation is to prevent the unsafe condition or event from ever occurring. This is consistent with the use of the words 'under any conditions' .
- The reinterpretation of the condition as one requiring exposure leads directly to the possibility of creating an unsafe condition. The occurrence of a particular concentration inside the tunnel is necessarily a result of a situation in existence before exposure has occurred. Once the required concentration is in existence in the tunnel then the exposure of a person depends on the ability of the operator to control access of motorists or workers or to remove them from the scene. It is clear that neither of these actions can be guaranteed under every circumstance.
- If the 'exposure' interpretation were to be strictly adhered to then it would be allowable for the operator of the tunnel to maintain carbon monoxide far above the 'safe' 87ppm concentration if it is held that the tunnel management systems are such that no motorist should be in the tunnel for the required period of time. As an example, if it could be claimed that no motorist could be in the tunnel for more than 7 minutes then the carbon monoxide level could be held at 185ppm. This is clearly unacceptable and contrary to good safety practice.
- There is no apparent explanation for the use of the words 'under any conditions' other than to make it clear that the carbon monoxide level must be kept below 87ppm over a 15 minute average at all times and during all situations and occurrences which occur while the tunnel is in operation.

Not only does the 'plain English' interpretation of the condition ensure safe conditions at all times but the reinterpreted condition is inconsistent with good practice and can lead directly to inherently unsafe situations.

Administrative and operational convenience must not be allowed to force the acceptance of an operating protocol which is inherently unsafe. In addition it is far from clear that it is possible to correctly estimate exposure or to rule out the possibility that someone has been exposed.

Fundamental flaw in the 'interpretation'

The whole of the reinterpretation of Condition 70 ignores the fact besides being designed to protect against excessive exposure to carbon monoxide it is also being used (incorrectly) to attempt to ensure safe levels of other pollutants.

The PIARC documents clearly note this anomaly in the use of carbon monoxide as a marker and suggest that other components should also be controlled, but in the case of the M5 they are not, so the carbon monoxide level is the sole regulatory protection. In fact, PIARC does not use the concept of exposure in its 1995 document, upon which the tunnel design is based. Under 'Admissible Concentrations', it notes:

"Table 2 3 gives CO design-values, taken in conjunction with the maximum traffic and the type of traffic regime. The 100 ppm value corresponds to the WHO recommendation for short term-exposures . In order to avoid excessive fresh air demands for a rarely occurring congested traffic, a higher CO concentration can then be admitted."

- The reference to *' the 100ppm value'* is followed immediately by the suggestion *'a higher CO concentration can then be admitted.'* (Note - it is not an exposure which is referred to.)
- Clearly the carbon monoxide design values are used in the sense of concentrations not in the sense of personal exposure, otherwise the provision "*a higher CO concentration can then be admitted*" would lead inevitably to an exceedence of the WHO goal, an inconceivable suggestion.
- Nowhere in this otherwise comprehensive document is there any method given by which measured carbon monoxide concentrations can be used to estimate motorists' exposure, yet this is a mathematically and technically difficult procedure requiring constant tunnel supervision and a high level of information relating to traffic speeds and driver behaviour.

Reinterpreting the carbon monoxide goal as an exposure goal rather than a measured concentration undoubtedly weakens the protection provided for carbon monoxide and this weakening also applies to other components, for which carbon monoxide is acting as a marker or proxy, perhaps to a greater extent.

Thus it is unsafe and contrary to accepted international practice to reinterpret or to actively use an exposure approach to the carbon monoxide goal in the regulation of any tunnel.

Other components

No other components of tunnel atmospheres are subject to the conditions of approval for the M5.

The only PIARC recommendation which relates in any way to particles is the reference to visibility where the requirement is related to the distance required for a vehicle to stop in front of a stationary object. This recommendation clearly does not consider smoke/particulate matter as a health hazard.

The 1995 PIARC document notes that *'Usually the fresh air demand for diesel-smoke dilution is quite higher* (sic) *than for the CO-dilution, but in countries where the N02 limits will be adopted the fresh air demand is even higher than for diesel smoke dilution.'*

It appears to be RTA policy that the visibility levels should be kept so that the extinction coefficient (K) does not exceed the lowest figure noted (K = 0.005 m^{-1}) This is equivalent to a smoke concentration which will absorb 40% of a beam of light in 100 meters. This level is probably the 'regulation' the RTA claims to meet, but the conditions of approval do not set a specific figure.

As the smoke concentration increases the proportion of light absorbed becomes higher. The document notes that even when there is enough visibility for a car to stop safely, the conditions inside the tunnel will be 'most uncomfortable'.

The 1995 PIARC document notes that *'in a few countries, nitrogen dioxide is taken into account and the threshold values following the WHO recommendations are considered.*

The later 2000 PIARC discussion document 'Pollution by nitrogen dioxide in road tunnels' examines the effect of nitrogen dioxide (reflecting, mainly, the concerns of the French about this gas) however the Swedish experimental work described clearly shows that nitrogen dioxide interacts with particulate matter and that, under tunnel conditions, it is likely that particle levels equivalent to 100 $\mu g/m^3$ PM2.5 are equivalent to NO_2 concentrations of around 200 $\mu g/m^3$ and that the two components interact additively inside tunnels.

Other research on nitrogen dioxide carried out in the same laboratory but not reported in the PIARC document demonstrates the ability of repeated, relatively short (15 minute) exposures to nitrogen dioxide to act cumulatively over periods between 24 and 48 hours.

Situation in Sydney tunnels

In relation to particulate matter, an anomaly became evident during planning for the Cross City tunnel.

An examination of the pollution data from the M5 tunnel showed that the particulate matter concentrations measured by several different methods inside the tunnel and in the stack did not correspond to the levels calculated using a relationship developed by PIARC between visibility and PM10 concentrations, using European data. This relationship suggests that a visibility extinction coefficient of K = 0.005 m^{-1} is equivalent to a PM10 concentration of 1000$\mu g/m^3$.

In fact a series of measurements carried out in the M5 showed that the PIARC relationship did not hold in the M5 (and presumably in other tunnels), and that this level of visibility was , in fact equivalent to a PM10 level of more than 2000 $\mu g/m^3$. The study noted "Experience by the RTA indicates that the haze in the M5 tunnel becomes unacceptable when the extinction coefficient as reported in the stack opacity monitor exceeds 0.003 m^{-1}.

It is observed that the concentration of PM10 in the stack during daytime, working-day hours almost always exceeds 1000$\mu g/m^3$. This means that the concentration of PM10 exceeds 1000$\mu g/m^3$ in at least one of the tubes. On the basis of the PIARC visibility/PM10 relationship this means that the K factor would be greater than 0.005 m^{-1} in at least one of the tubes during most of the day. This is not the case.

The most likely explanation is that the 'haze' on which the PIARC relationship is based contains a higher proportion of particles with diameter larger than 10 microns than does the 'haze' in the M5. Though annoying, these larger particles have a much less severe impact on human health than do smaller particles. In fact, it is likely that those particles which have the greatest impact on health are not detected by measuring techniques using light absorption at all, as they are significantly smaller than the shortest wavelength of visible light (ca 400nm) and are effectively invisible to the measuring instrument.

It is clear that the control of 'visibility' at the levels envisaged by PIARC gives rise to PM10 levels almost twice those which would be experienced under the same control in Europe. There is no reason to expect that it would not also be twice as harmful to motorists.