Lung Research Centre, UoM

A/Professor Louis Irving

Submission Overview

The Lung Health Research Centre (LHRC) of the University of Melbourne represents combined expertise in respiratory biology, respiratory medicine, epidemiology, public health, pharmacology and oncology. We welcome the opportunity to comment on the West Gate Tunnel Project, with specific reference to Technical Report J, the Human Health Impact Assessment (HIA).

We acknowledge that the West Gate Tunnel Project EES is one of the first environmental assessments of a major road project in Victoria to include a specific HIA. Whilst we very much support this approach, we are concerned the HIA does not reflect the most recent evidence, and that there are additional medical and biological considerations that should be taken into account.

In particular, there is now very strong data demonstrating the immediate and long term adverse effects of traffic related air pollution, highlighting a greater magnitude of impact than previously thought, and showing that there is no safe lower limit of exposure (New England Journal of Medicine (NEJM), July 2017). Therefore health impact assessments should aim at ensuring the cleanest air possible.

The HIA did not consider the impact of various pollution mitigation strategies despite proven success. If incorporated into the project these strategies may significantly abate public exposure to emissions at road surface level and capture the toxic pollutants before they enter Melbourne’s air-shed.

Our submission has drawn on international research and a review undertaken by the Woolcock Institute of Medical Research, Centre for Air Quality and Health Research and Evaluation (CAR) “Review of the health impacts of emission sources, types and levels of particulate matter air pollution in ambient air in NSW” (Hime et al). This was produced for the NSW Environmental Protection Authority and NSW Ministry of Health, Environmental Health Branch in December 2015, and is a comprehensive, rigorous selection of evidence that provides the best available summary of the health impacts of vehicle related particulate emissions pertinent to Australian use.

We agree that this road project will likely result in improvements for some areas within the project boundary, however there will also be disadvantages and environmental inequities for other areas both within the project boundary and further afield. We believe these impacts are currently under-estimated. Consideration of all the available evidence and relevant health end-points, along with a greater application of the precautionary principle is likely to substantially improve the public health outcomes associated with this project.
We advocate for major revisions with inclusions of recent evidence, broadening the range considered health endpoints and full consideration given to internationally used mitigation strategies with associated cost benefit analyses.

**Key recommendations**

- Update the HIA to include the full range of immediate and long term adverse health consequences as provided by the most recent data (eg NEJM July 2017)
- Incorporate the most recent data regarding the immediate and long-term adverse health impacts of traffic air pollution to update the relative risks used in hazard calculations in the HIA.
- Include the health benefits and cost effectiveness of tunnel filtration
- Include consideration of world’s best practice road side mitigation strategies
- Revise assessments of in tunnel air quality to account for the health impacts of PM2.5 and ultrafine particles.
- Include ongoing consultation with the Department of Health and Human Services

**Background**

Australian air quality compares favourably to other countries, and therefore the issue of air pollution is often overlooked. There is a general lack of awareness that even with relatively low levels, there are significant health impacts on a par with obesity (Skamp K, 2007) (ABS, 2009).

There are 3430 annual Australian deaths attributed to ambient pollutants; Particulate matter and ground level ozone with associated costs estimated to be up to $A17.8 Billion (HEI, 2017). Road vehicles contribute the largest proportion of air pollutants of which the Australian population is exposed to. In 2015, vehicle emissions were estimated to be responsible for more deaths (1715) than our national road toll (1205) (BITRE, 2017).

The trajectory of public exposure to vehicle emissions in Australia is currently very concerning. Globally, many countries are adopting stringent policies to protect the public from vehicle emissions. Australia is bucking this trend, and is in the minority of OECD countries to report a substantial increase (68 per cent) of air pollution related deaths between 2005 – 2010 (OECD, 2010). In the absence of any significant policy change, it’s reasonable to expect this increasing trend has continued to current day. Our fuel quality is amongst the worst in the world with a sulfur content of 30ppm. In the EU, much of Asia and the States, the legislated maximum sulfur content is 10 ppm. When the UK transitioned to ultra-low sulfur diesel in 2007, there was a correlated decrease of over 30 per cent of ultrafine particles (Jones et al, 2012).
Diesel vehicle emissions contain a much higher number of particles compared to petrol cars. (WHO, 2013c). Much of Europe is now back peddling away from diesel with bans or heavy fines placed on diesel vehicles driving through major cities such as Paris, London and Munich. In stark contrast, Australian diesel vehicle ownership has increased by 60 per cent from 2011 – 2016 (ABS, 2016). The recent VW emissions scandal highlighted the difference between laboratory emissions and real life emissions. The European Joint Research Centre has released data that suggests real life NOx emissions of Euro5 diesel cars are four fold higher than threshold limits suggest (Cames and Helmers, 2013). When these European cars are using Australian fuel, pollution reducing vehicle technology is degraded resulting in increased emissions and underestimated health impacts (Schofield et al. 2017). The health impact report is based on data provided by the traffic modelling and air quality reports which do not appear to have adequately accounted for these Australian specific conditions and trajectories.

Living in close proximity to high traffic volume is associated with a range of adverse health outcomes including;


The health impacts of traffic emissions are summarised as:

On-road vehicles “Sufficient and strong evidence of increased risk of mortality, CV and respiratory morbidity from epidemiological studies. Toxicological studies provide mechanistic evidence.”(Hime et al, 2015)


The HIA considers proximity to roads in terms of residences; however location of work to busy roads and time spent commuting in traffic (drivers, commuters, cyclists and pedestrians), are also critical in dictating personal exposures (WHO, 2005). Cyclists utilising the veloway are likely to get a much higher dose of air pollution than that detailed in the report, as are the residents in closer proximity to Millers Rd compared to the residents who
live further away. Using aggregated data and weighted averages for risk calculations is not particularly informative to community members who are making decisions regarding their residence, internal ventilation options or exposures to their children at the local childcare centre.

Our National Environmental Protection Measures (NEPM) legislation discusses ‘equivalent protection’ and the goal that “all Australian’s enjoy the benefit of equivalent protection from air”. This is difficult to achieve. The ultrafine particles and air toxics which largely comprise vehicle emissions have high variability across the air-shed. Busy urban roads inevitably create air pollution ‘hot spots’. Most people cannot easily change the location of their homes, workplaces, schools or childcare centres, so when large road projects such as this one have the ability to significantly alter a community’s air pollution exposure every possible mitigation strategy should be comprehensively considered in terms of the net benefit to the community and associated health savings and then weighed up against the practicalities and costs of implementation.

This is an area of Victoria which, due to its location is already very vulnerable to air pollution. Hospital data clearly indicates Maribyrnong is a known ‘hot spot’ for emergency hospital presentations for paediatric respiratory disease (acsqhc, 2015). We consider it highly likely there is a correlation between this and the high number of diesel freight trucks recorded through the area. It is important to note this concern is not only felt by the affected community but by the wider health and academic community. This project represents an opportunity to abate some of these impacts by re-directing freight underground or to other surface roads. To capitalise on these gains, it’s essential the flow-on issues to adjacent communities are minimised to the fullest possible extent and the opportunity is taken to capture the tunnel emissions from the point source at the vent stacks. Without capture the emissions disperse into the local air-shed, where they are mixed and diluted before joining the regional air-shed. Regardless of dilution, they still pose a threat to public health, as there is no safe level beyond which impacts do not occur (Barnett, 2014). Therefore, we not consider the dispersal method currently reflects world’s best practice.

Given recent examples of public health failures in vulnerable areas we believe no rock should be left unturned when examining how we may minimise this community’s exposure to vehicle emissions. With appropriate conceptualisation and planning this project may present an opportunity to reduce air pollution exposure not only to various parts of Yarraville, but to the larger part of the community contained within the project boundary and beyond. Ideally, rather than merely utilise historic methods, this project should set a new benchmark for the rest of Australia to follow, closing the gap between our current practices and world’s best practice.
**Evidence used versus current available evidence**

Considering all available evidence the risks adopted for the major health endpoints in the HIA are too conservative and may have resulted in underestimations of the health risks (and benefits) to the community.

The health risks used in the HIA are compared against those in the CAR report in Table 1. The shaded section represents health endpoints which cannot be considered as a direct comparison (as endpoints measured differ) but serve to highlight the broader range of evidence available, relative to the category.

**Table 1. Comparison of HIA Health Impact Relative Risks and comparable risks cited in the CAR review. (Hime et al, 2015)**

<table>
<thead>
<tr>
<th>Health Impact</th>
<th>HIA Adopted Relative Risk (as %) and literature cited</th>
<th>CAR review (Hime et al, 2015). Literature cited with respective Relative Risks (as %)</th>
</tr>
</thead>
</table>
| Mortality, all causes              | 6% Krewski et al. 2009                               | 14% Beelan et al. 2014  
14% Lepeule et al. 2012  
26% Puett et al. 2009 (female nurses)*  
9.8% Hart et al. 2011 (Male truck drivers) |
|                                    | 14% Krewski et al. 2009                               | 26% (Cardiovascular) Lepeule et al. 2012  
37% (Lung cancer) Lepeule et al. 2012  
102% (Coronary disease) Puett et al. 2009 (female nurses)* |
| Cardiovascular mortality           | 65 yrs + 0.8% Bell M. L, et al. 2012                 | --                                                                                                                                       |
| Cardiovascular hospital admissions | 65 yrs + 0.8% Bell M. L, et al. 2012                 | 1.03% Dai et al. 2014  
0.86% Samoli et al. 2013  
0.85% Zanzobetti & Schwartz 2009 |
<p>| Respiratory hospital admissions    | 0.4% Bell M. L, et al. 2012                          |                                                                               |
| (65 yrs+)                          |                                                      |                                                                               |
| Mortality, all causes              | 0.94%                                                 | 1.18% Dai et al. 2014                                                         |</p>
<table>
<thead>
<tr>
<th>Health effect</th>
<th>Study 1</th>
<th>Study 2</th>
<th>Study 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>All-cause mortality</td>
<td>Zanzobetti &amp; Schwartz 2009</td>
<td>0.55% Samoli et al. 2013</td>
<td>0.98% Zanzobetti &amp; Schwartz 2009</td>
</tr>
<tr>
<td>Cardiovascular deaths</td>
<td>Zanzobetti &amp; Schwartz 2009</td>
<td>1.21% Franklin et al. 2007</td>
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<tr>
<td>Respiratory mortality</td>
<td>1.92% Zanzobetti &amp; Schwartz 2009</td>
<td>1.71% Dai et al. 2014</td>
<td>1.91% Samoli et al. 2013</td>
</tr>
<tr>
<td>Respiratory infections in children</td>
<td></td>
<td></td>
<td>1.68% Zanzobetti &amp; Schwartz 2009</td>
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<tr>
<td>Atherosclerosis</td>
<td></td>
<td></td>
<td>1.78% Franklin et al. 2007</td>
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<td>Ischaemic heart disease</td>
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<td>Complications of diabetes</td>
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<tr>
<td>Respiratory-related mortality</td>
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<tr>
<td>Asthma symptoms</td>
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<tr>
<td>Reduced lung function in children</td>
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<tr>
<td>Reduced lung function in susceptible adults</td>
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<tr>
<td>Respiratory infections in children</td>
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<tr>
<td>Cancer</td>
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<td>Lung cancer mortality</td>
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<tr>
<td>Neurological</td>
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<tr>
<td>Neurological disorders in adults</td>
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<tr>
<td>Impaired cognitive function</td>
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<tr>
<td>Development</td>
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<tr>
<td>Lung development</td>
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<tr>
<td>Neurological development in children</td>
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</table>

*Females are generally more susceptible to the cardio-pulmonary impacts*

**A broadened consideration of health endpoints is required**

The range of considered health endpoints is does not reflect the current full range of evidence. We advocate the HIA be revised to include the health endpoints listed in Table 2.

Table 2. Health effects attributed to exposure to ambient PM (Hime et al, 2015)
Respiratory Impacts in Children

One of our largest health concerns arising from this project are the deleterious impacts of vehicle emissions on children’s lungs. Children are particularly vulnerable to air pollution due to the large surface area of their lungs compared to the rest of their body, reduced ability to excrete toxins, higher respiratory rate, and larger proportion of time spent outdoors. School children in comparison to other community members have been shown to have higher cumulative black carbon exposures (Barrett, MRC-PHE). School hours and in particular the commute to and from school are a key periods of exposure to pollutants (Mazaheri et al, 2014).

Surprisingly some of the included tables in the HIA omit respiratory emergency admission data from children 0 – 5yrs yet this is the age group most vulnerable to the respiratory impacts of air pollution.

The HIA does not consider ‘road side asthma’ as it states there is no evidence available to base estimates on. There is now substantial evidence available that examines the relationship between vehicle emissions and children’s asthma, much of it including proximity to major roads.

Gasana et al published a large meta-analysis of motor vehicle pollution and asthma in children 2012 with the following results:

- increased exposure to NO2 is positively associated with new-onset asthma (meta-OR 1.05 95% CI 1.00 – 1.11)
- increased exposure to sulfur dioxide is positively associated with a higher prevalence of wheeze in children. (meta-OR 1.04, 95% CI: 1.01-1.07)
- increased exposure to PM is positively associated with a higher incidence of wheeze in children (meta-OR 1.05, 95% CI: 1.04-1.07)

“Living or attending schools near high traffic density roads exposes children to higher levels of motor vehicle air pollutants, and increases the incidence and prevalence of childhood asthma and wheeze” (Gasana et al, 2012)
A recent systematic review and meta-analysis examining the influence of childhood traffic-related air pollution (TRAP) exposure on asthma, allergy and sensitisation found:

- Increased longitudinal childhood exposure to PM2.5 and black carbon was associated with increasing risk of subsequent asthma in childhood (PM2.5: OR 1.14, 95%CI 1.00 to 1.30 per 2 µg/m3 and black carbon: OR 1.20, 95%CI 1.05 to 1.38 per 1 x 10^5 m^−1)
- There was an association between TRAP and the development of asthma up to 12 years of age
- Increasing exposure to PM2.5 was associated with sensitization to both aero- and food allergens (Bowatte et al, 2015)

The relationship between vehicle emissions and asthma and allergic disease is of particular note given this project falls within an area of Victoria known for high pollen counts. The recent evidence pointing to supra-additive impacts of pollen combined with vehicle emissions and the rising epidemic of both asthma and allergies in children merit attention and discussion in the HIA.

Most of the evidence discussed considers the negative health impacts; however there is also evidence which demonstrates the positive gains to be made as air quality improves.

Decreases in PM following interventions have been shown to reduce respiratory hospital admissions of children (Pope III, 1989), and the prevalence of bronchitis in children (Heinrich, 2003).

The Californian EPA is an international leader in establishing legislation and best practice methods to reduce children’s exposure to roadside air pollution. The successful implementation of air pollution mitigation strategies successfully resulted in a downward trend in air pollution over the past two decades in Southern California which has been associated with significant health improvements (Gauderman, 2015). The most notable to date has been the significant improvement in lung function over 3 cohorts of children (n=2120) corresponding to the time periods 1994 – 1998, 1997 – 2001 and 2007- 2011. Declining levels of nitrogen dioxide and PM were associated with significant improvements in lung function growth and persisted after adjustment for potential confounders. Improvements were noted in children both with and without asthma. Children with clinically low lung function declined from 7.9% to 6.3% to 3.6% across the three time periods as air quality improved (p = 0.001). The benefits of improved lung development in children extend throughout their lives. A healthy lung function in adulthood has been associated with a reduced risks of cardiovascular disease and a lower mortality rate (Georgiopoulou, 2011, Schunemann, 2000).
The mitigation measures adopted by the Californian EPA have resulted in long-term substantial health and associated economic gains.

Cancer

The association between PM and lung cancer is stronger than stated in the report. The association with bladder cancer is ‘suggestive’ however the association with lung cancer is ‘causal’ and has been considered so for several years (IARC, 2012).

Lung cancer is Australia’s leading cause of cancer related mortality and causes more annual deaths than breast and prostate cancer combined (Cancer Australia, 2017). Reductions in cigarette smoking have reduced the incidence of squamous-cell lung cancer; however lung adenocarcinoma is increasing and affecting a growing proportion of ‘never smokers’ (Gabrielson, 2006). Approximately twenty per cent of lung adenocarcinoma cases in Australia occur in ‘never smokers’ (Couraud et al, 2012).

Lung cancer contributes 21 per cent of the health burden attributable to Australian urban air pollution (Begg et al, 2003) and should be included as a separate health end point in the quantitative assessment. It is potentially misleading to include lung cancer as a secondary health end point grouped in the short-term respiratory mortality category with a relative risk of 0.0192. Given the lag time from exposure to presentation of disease (15-30 years) lung cancer cannot be considered a short term impact. The most recent available systematic review and quantitative summary of the relationship between outdoor PM and lung cancer reports a relative life-time risk for lung cancer of 1.09, which is more informative to the community than the report’s value of 0.0192. The meta-relative risk for the specific sup-type of lung cancer most associated with air pollution (adenocarcinoma) is 1.40 (95% CI 1.07 – 1.83) (Hamra et al, 2014). Put simply, this equates to a 40% increased risk (relative to the current risk) of developing lung adenocarcinoma over the course of 60 years.

Diesel exhaust is particularly carcinogenic and there is evidence that the DNA damage and mutations caused by diesel pollution also occur in sperm (Somers, 2011), which may give rise to subsequent generations inheriting the mutagenic impacts of diesel vehicle emissions.

Forest Plot 1. Relative risks for lung cancer associated with a 10µg/m³ increase of PM2.5 (A) and PM10 (B). (Hamra et al.)
Cognitive Impairment

There is a rapidly growing body of evidence linking fine and ultrafine particles to cognitive impairment and neuro-degenerative impacts. To date, most evidence relates to children (neurodevelopment, intelligence, cognitive function, motor skills) and elderly (cognitive function, memory, alzheimers, vascular dementia and Parkinson’s disease). A recent meta-analysis of the link between air pollution and cognitive function concluded:

“exposure to a range of largely traffic-related pollutants has been associated with quantifiable impairment of brain development in the young and cognitive decline in the elderly.” (Clifford et al, 2016)
A Swedish study assessing the relationship between long-term exposure to vehicle emissions and dementia found positive associations with Alzheimer's disease with a hazard ratio of 1.43 (95% CI: 0.998, 2.05 for the highest vs. the lowest quartile). The estimates were similar for Alzheimer's disease (HR 1.38) and vascular dementia (HR 1.47). A sub-analysis excluding a younger sample that had been retested after only 5 years of follow-up suggested stronger associations with exposure than were present in the full cohort (HR = 1.71; 95% CI: 1.08, 2.73 for the highest vs. the lowest quartile) (Oudin et al, 2016).

A study of the 9.8 million elderly (>65 years) in the northeastern United States found long-term exposure of each 1µg/m³ increase in PM2.5 was associated with:

- **Dementia.** HR 1.08 (95% CI: 1.05, 1.11)
- **Alzheimer’s Disease.** HR 1.15 (95% CI: 1.11, 1.19)
- **Parkinson’s Disease.** HR 1.08 (95% CI: 1.04, 1.12) (Kioumourtzoglou et al, 2016)

"For every 1µg/m³ increase in PM2.5 the elderly population has an 8% increased risk of developing dementia or Parkinson’s disease, and a 15% increased risk of Alzheimer’s disease" (Kioumourtzoglou et al, 2016)

While there is currently not enough available epidemiological evidence to support a causal association; the nascent epidemiological studies, plausible toxicological mechanisms, animal studies and findings from brain imaging and autopsies all point in a perceptible direction and support our position that further judicious consideration of mitigating strategies is warranted (MohanKumar et al. 2008, Levesque et al. 2011, Costa et al. 2014, Calderon-Garciduenas et al. 2008 and Calderon-Garciduenas et al. 2004).

**Type 2 Diabetes**

Vehicle emissions may also be implicated in the modern epidemic of type 2 diabetes. A recent meta-analysis of 10 studies found (after adjustment for confounders) a positive association for both nitrogen dioxide and PM2.5 with an increased risk of type 2 diabetes. The five prospective studies resulted in a meta-HR of 1.13 (95% CI: 1.01, 1.22) for nitrogen dioxide and meta-HR 1.11 (95% CI 1.03, 1.20) for type 2 diabetes. This was further supported by the odds ratios reported in two cross-sectional studies. All studies after adjustment for confounders supported an association of PM, nitrogen dioxide with an increased risk for type 2 diabetes (Balti et al. 2014).

*Forest Plot 2. Association between gaseous air pollutants [NO2 and NOx] and diabetes mellitus (Balti et al.)*
Stroke

Globally, nearly one third of all strokes are attributable to air pollution (Feigin et al. 2016).

The researchers involved in the Global Burden of disease study 2013 stated

“The most alarming finding was that about a third of the burden of stroke is attributable to air pollution. Although air pollution is known to damage the lungs, heart, and brain, the extent of this threat seems to have been underestimated” (Feigin et al. 2016)

The conclusion from the CAR report:

“Gaseous and particulate air pollutants have a marked and close temporal association with admissions to hospital for stroke or mortality from stroke. Public and environmental health policies to reduce air pollution could reduce the burden of stroke.” (Hime et al. 2015)

Developmental and reproductive effects

(Section 4.6.2 of the CAR report)

“Exposure to air pollution (and specifically PM) during pregnancy appears to retard foetal growth, as evidenced by associations between exposure and low term birth weights, albeit with considerable variability in results from different regions (Wilhelm and Ritz 2005, Heinrich and Slama 2007, Millman et al. 2008, Parker et al. 2011, Dadvand et al. 2013). Exposure to PM2.5 from traffic has been associated with low term birth weights in Los Angeles (Wilhelm et al. 2012). Exposure to PM during pregnancy has also been associated with an increased risk of pre-term birth (Wilhelm and Ritz 2005, Rappazzo et al. 2014). It is not clear whether PM directly affects the foetus or whether effects on the health of the
mother are responsible for these adverse birth outcomes. However, the presence of PAHs attached to DNA in umbilical cord blood indicates that air pollutants can transfer to the foetus (Tang et al. 2006). High PAH-DNA adducts in cord blood have been associated with decreased body weight in the first few years of childhood (Tang et al. 2006). Pre-natal exposure to PAHs in air has been associated with morphological changes related to cognitive deficits and behavioural problems in the brains of children aged 7-9 years (Peterson et al. 2015).

Lung development continues well into childhood and postnatal exposure to PM2.5 is associated with impaired lung growth and decreased lung function later in life (Gauderman et al. 2004, Schuepp and Sly 2012). Children who moved to locations with higher ambient PM have demonstrated a slowing in lung function development, whereas children who have moved to areas with cleaner air have demonstrated an increase in lung function development (Avol et al. 2001).

Pre- and post-natal exposure to diesel exhaust particulates in animals has been associated with a variety of developmental and reproductive effects including retarded growth of offspring, abnormal development of the female and male reproductive systems, altered sperm development and, increased mutation rates in male germ line cells (Ema et al. 2013). It has been suggested that inhalation of PM could result in heritable mutations by causing mutations in sperm (Samet et al. 2004), however there is no human evidence for this. There is the suggestion that exposure to air pollution may be associated with a reduction in sperm quality (Selevan et al. 2000) and this could reduce fertility rates. This effect may be related to the oxidative stress effects of PM as increased oxidative stress is associated with decreases in sperm motility (Ruckerl et al. 2011).”

In summary, recent evidence has elucidated a far broader and rapidly expanding range of health impacts than those currently considered in the HIA.

**Vehicle Emissions versus general ambient air pollution**

PM emitted by traffic is likely to be more toxic than the general mixed PM measured by static ambient pollution monitors (WHO 2005).

Ambient fine particulate matter (PM2.5) is comprised of all particulate matter up to 2.5 micron in diameter. Particulate matter generated from vehicle matter is ultrafine (PM0.1) and has a different composition compared to larger non-anthropogenic particulate matter.

The ultrafine fraction of PM2.5 has

- The ability to penetrate the skin and enter the lymph system (Oberdorster et al. 2005).
• Greater deposition through the lungs and can translocate into the blood stream and central nervous system allowing them to reach the brain and other organs (Oberdorster et al. 2005).

• A higher proportion of elemental carbon (Grahame et al. 2014)

• A very large, chemically reactive surface area which can bind and act as a carrier for other vehicle associated toxics including heavy metals, toxicologically active PAHs and semi VOCs (WHO, 2013c)

Using background PM2.5 measurements from static monitors does not necessarily provide an accurate account of public exposure to road side pollutants for two reasons:

1. There is a high degree of spatial variability in vehicle emissions making it difficult to capture ‘hot spots’. These areas of much higher levels can be further exacerbated by street canyon effects and building designs. There is significant elevation of heavy metals and PAHs which is not captured by monitors and yet, likely to contribute to the health risks associated with proximity to traffic (Lai et al. 2005).

2. Vehicle emissions are largely comprised of ultrafine particles (PM0.1 – 1.0). As PM2.5 measurements are mass based (weight) increases of ultrafine particles make little difference to the overall measurement compared to larger non-anthropological particles such as dust and salt. A better representation would be obtained by confining measurements to particles <1 μm (Morawska et al.2008a) or using the total particle number concentration (PNC).

The 2008 NSW Air Emissions Inventory calculated on road vehicles contributed 13.2% of total PM2.5 emissions in the Sydney region. Interestingly, as a comparison, the contribution of motor vehicle emissions in Melbourne in 2016 to PM2.5 is reported as 31%.(EPAV, 2016)

When discussing particles by number rather than mass, in urban environments, the contribution of vehicle emissions is estimated to be 90% (Knibbs et al 2011).

**Table 3. Concentrations of PM around roadways (CAR report)**

<table>
<thead>
<tr>
<th>PM pollutant</th>
<th>Increase above urban background at roadside</th>
<th>Distance from roadside to reach urban background levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ultrafine PM (PM0.1)</td>
<td>4-7 x</td>
<td>200-900 metres</td>
</tr>
<tr>
<td>PM2.5</td>
<td>Marginally above background</td>
<td>Essentially no variation with distance</td>
</tr>
<tr>
<td>PM10</td>
<td>1.3-1.4 x</td>
<td>Approximately 100 metres</td>
</tr>
</tbody>
</table>
This questions the methodology of taking background PM2.5 levels from static monitors situated away from heavy traffic, calculating the contribution the project makes (which is almost all vehicle generated) and using the sum difference to calculate the health impacts. We accept this remains the most practical available option, however suggest it would be more informative for the project stakeholders to also have some account as to the differences between vehicle emissions and general background PM2.5 including a discussion on how this may skew health risk calculations. In terms of project monitoring ultrafine particles should be accounted for in multiple locations (with a focus on sensitive receptors) prior to project commencement, during construction and at regular intervals subsequent to project completion.

**In tunnel air quality**

Using visibility to dictate the in-tunnel threshold of PM is not appropriate from a health perspective. The in-tunnel air quality thresholds outlined by the international PIARC guideline were developed with northern hemisphere conditions in mind. Vehicles often use studded tyres (to prevent slipping on icy roads) which in turn creates more surface abrasion. This results in a much higher proportion of larger light scattering particles creating issues with visibility.

Concentrations of ultrafine particles have been found to be significantly higher within tunnels than urban background levels (Morawska et al. 2008b) exposing commuters to possible inflammation, oxidative DNA damage (which can give rise to carcinogenesis and mutagenesis), reductions in lung function and, irregular heart rates (Knibbs et al. 2011). Evidence suggests repeated exposures have cumulative effects with PM0.1 exposure over 5 days causing more inflammation than same-day effects (Morawska et al. 2004).

We support the report’s recommendation to warn drivers to wind up windows and reticulate circulation, however strongly suspect this alone does not provide adequate protection for regular tunnel users from the health impacts of ultrafine particles. Anecdotally, despite following this advice when using the Burnley tunnel, there is still a noticeable odour that penetrates the car cabin particularly during heavier traffic periods. If an odour penetrates the cabin, it is likely that ultrafine particles have also penetrated the cabin and are now trapped inside the cabin for the driver and passengers to inhale for the duration of the journey.

**Strategies to mitigate the public health impact**

“On current evidence, if a source of PM is to be targeted for emissions reduction policies, it should be on the basis of:

1. the source is a large contributor to ambient PM mass in the community and therefore regulation specific to the source will help to significantly reduce exposure to total ambient PM;
2. the source of PM emissions occurs in close proximity to people and therefore it could be expected that the emission source is responsible for considerable exposure to PM; or,

3. the source of PM is a combustion source and therefore the health benefits from exposure reduction are likely to be significant” (Hime et al. 2015)

**Filtration of Point Source**

The argument that filtration is used purely as a means of improving tunnel visibility is no longer valid. As the evidence of health impacts mounted and advancements were made to the technology, tunnels are now able to be fitted with filtration equipment that captures most of the harmful vehicle generated PM and NOx rather than allowing it to be dispersed through the vent stacks.

It is no longer appropriate to rely on the ‘dispersal’ method. Dispersal is not equivalent to removal: When dispersed particles enter the local and regional air-shed where they can still be consumed by humans. Once dispersed and exposed to sunlight, particles undergo photo-chemical transformation. These secondary particles have a higher oxidative capacity which is likely to be associated with greater health impacts (WHO 2013c, Stevanovic et al. 2013, Zielinska et al. 2010). Filtration would capture these emissions prior to these reactions and remove them thus reducing the overall amount of biologically active particles in the local and regional air-shed.

The argument that the overall impact compared to the total background air pollution is invalided for reasons previously discussed in this paper chiefly;

1. Vehicle emissions are an especially noxious sub-set of ambient particulate matter

2. There is no level beyond which negative impacts do not occur, therefore any reduction however small, will likely result in public health gains.

To date, the M5 East tunnel in Sydney remains the only Australian tunnel fitted with filtration technology. Unfortunately the technology selected were not considered to be the best available option, and combined with the unusual tunnel design, expensive retro-fit and errors in traffic modelling during the planning approval process the trial was deemed a failure. Yet, the filters are still utilised during heavy traffic periods and are reported to remove 68 per cent of PM10.

The Calle 30 is a 4km tunnel through urban Madrid and has filters that successfully remove the majority of particulates and nitrogen dioxide from tunnel emissions prior to entering the air-shed.
Table 4. Measurements (indicative only) taken during a visit to the Calle 30 tunnel using a FLUKE 983 Handheld particle counter. (Courtesy of Mr Mark Curran).

<table>
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<th>Time</th>
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<th>0.5um</th>
<th>1.0um</th>
<th>2.0um</th>
<th>5.0um</th>
<th>10.0um</th>
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<td></td>
<td>In front of Filter</td>
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<td></td>
<td></td>
<td></td>
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<td>02.01.2008</td>
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<td></td>
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<td>02.01.2008</td>
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<tr>
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<td>99.7%</td>
<td>99.8%</td>
<td>99.6%</td>
<td>99.6%</td>
<td>100%</td>
<td>100%</td>
</tr>
</tbody>
</table>

The cost and energy requirements of filters installation are considerable; however there is evidence from Japan and Norway that demonstrates these can be offset by reductions in energy used to propel the ventilation turbines and over time the cost of filter installation (particularly when incorporated from the outset) can result in net overall savings. This is without factoring in health economics (NHMRC, 2007).

In 2014, the Victorian government appointed expert assessment panel for the proposed EW link considered the issue of filtration for the proposed EW link tunnel.

The panel’s report referred to a review on the international practice on the use of pollution control equipment in tunnels (CETU, 2010 (Document 100); Norwegian Public Roads Administration, 2004) which listed the major reasons for requiring pollution control equipment as:

- Location in a densely populated area
- The sensitivity and quality of the existing environment
- Community concern about existing air quality in the vicinity of the ventilation stacks
- Length of the tunnel (> 1 km)
- A high proportion of heavy vehicles using the tunnel

“Given the location of the ventilation stack and the condition of the existing air environment, it could be considered that pollution control equipment would be considered as best practice emissions control as required under the SEPP (AQM).” (East West Link Project, Assessment Committee Report, 2014)
Reducing road side pollution exposure

The Californian EPA have emerged over the past two decades as world leaders in establishing mitigation measures designed to protect children from the impacts of vehicle emissions. Much of their policies are summarised in a booklet “Best Practices for Reducing Near-Road Air Pollution Exposure at Schools” the content and background of which is particularly pertinent to the area of Melbourne this project falls within. The policies outlined in this book contrast rather poorly to the VCAT approved planning of a childcare centre on the corner of Francis St and Williamstown Rd. This is an area already exposed to truck emissions that breach air quality thresholds (EPAV, 2001) and the future projections in the EES add an additional 5000 trucks per day, passing two metres away from this childcare centre. In California, the legislated distance for a new school or childcare centre is a minimum of 300m from sources of truck traffic emissions.

Some of the useful policies in the booklet include:

- Upgrade filtration systems used in classrooms.
- Locate air intakes away from pollution sources.
- Provide training to school staff and students on indoor air quality and ventilation.
- Avoid strenuous activities, such as physical education class and sports, during peak traffic times.
- Reduce car and bus idling, upgrade bus fleets, and encourage active transportation like walking and biking to school.
- Consider improvements to site layout, such as locating classrooms further from the roadway.
- Consider installation of solid and/or vegetative barriers.

Green Infrastructure

Currently, there is much research, government funding and discussion around ‘green infrastructure’ and the creation of ‘sustainable cities’ and ‘healthy cities’. Much of this is occurring in Melbourne. This project is a major piece of infrastructure that will change a significant part of Melbourne city, and yet none of the current research appears to have been translated and applied into the planning.

Trees and vegetation barriers have long been known to abate air pollution, however current research highlights this depends on a number of variables such as the type of vegetation, metrological conditions and the built space around the emissions.

Researchers working for the iSCAPE project (improving the Smart Control of Air Pollut ion in Europe) project are currently performing extensive investigations designed to elucidate the best type of green barrier for various road situations. A recent review summarising studies to date found:

- In a street canyon environment, high-level green infrastructure (i.e. trees) generally has a negative impact on air quality while low-level dense vegetation with complete coverage from the ground to the top of the canopy (i.e. hedges) hinder the air flow underneath and hence generally show a positive impact.
- In open road conditions, vegetation barriers have a positive impact on air quality with thick, dense and tall vegetation. Studies observed considerable pollutant removal through
designing vegetation barriers closer to the pollutant source and plume's maximum concentration. In excess of a 50% reduction was observed with a 10 m thick green belt for numerous pollutants.

- The combination of vegetation and solid passive air pollution control measures has the potential to maximise the reduction in pollutant concentrations and improve personal exposure conditions, more than that achieved by any individual intervention in both street canyon and open road conditions.
- Only a small number of studies investigated air quality improvements for green roofs and greenwalls. Reported reduction in air pollutants with green walls ranged up to 95% (Abhijith et al. 2017)

The public health benefits of green infrastructure are highly applicable to this project, and with the available resources in Melbourne, we advocate for this project to include consultation on this topic and how it may best be utilised to benefit the sensitive receptors within the project boundary.
This project must comply with Victorian SEPP (AAQ) legislation. The policy aims of this legislation are:

- Ensure that the ambient air quality standards in SEPP (AAQ) (which reflect the AAQ NEPM standards) are met

- **Drive continuous improvement in air quality and achieve the cleanest air possible having regard to the social and economic development of Victoria**

- Support Victorian and National Measures to address the greenhouse effect and depletion of the ozone layer.

In summary, in order to adequately address the health impacts of this project, and comply with stated intentions of applicable state policies we recommend greater consideration be given to available mitigation strategies.
**Precautionary Principle**

We advocate for an improved application of the precautionary principle throughout the HIA.

The underpinning methodology for setting the NEPM air quality standards in Australia states “*In the risk management phase, regulators consider the results of the risk assessment stages outlined above, apply the precautionary principle and take into account social and economic factors*.”

We believe the same approach should apply in air quality related health risk assessments for large government projects.

The recent explosion of evidence relating to vehicle emissions and health renders it unacceptable to merely adhere to NEPM thresholds. Whether it is possible to attach a calculation or not, every angle should be considered and thoroughly explored in determining how public exposure to the vehicle emissions associated with this project may best be minimised.

Successful utilisation of the precautionary principle would involve applying conservatism (towards over-estimating health impacts) at every point and then addressing every possible way in which these impacts may be reduced. Particular consideration should be given to sensitive receptors such as the planned child care centre on Williamstown Rd and the Emma McLean kindergarten.

There is no level beyond which the public are safe from impacts, therefore any reduction in public exposure to vehicle emissions (albeit at the road surface level, within the tunnel, or the dispersal and contribution to the greater general air-shed) will likely result in health benefits (WHO 2013 recommendations) therefore it stands to reason that in a report such as this, all mitigation strategies available to the affected communities should be fully considered with associated cost-benefit analyses.

**Summary**

The HIA should offer an important opportunity to protect the health of tunnel users and the surrounding communities. To achieve this, the report needs to include the most recent scientific information that demonstrates the significant immediate and long-term adverse health effects of traffic pollution.

This includes that there is no safe lower limit of traffic pollution, and there are some individuals who are at particular risk, including children, the elderly, people with underlying cardio-respiratory comorbidities, those with genetic susceptibility and those with socio-economic disadvantage.

The projected demographics for population growth, industry growth and associated demands for transportation through this area of Melbourne will likely further worsen traffic related health impacts in this area.

In our expert opinions, the current HIA substantially underestimates the health impacts of the proposed tunnel.
Finally, a number of air quality initiatives, including those in Southern California have clearly demonstrated the immediate and long-term health benefits of policy aimed at achieving cleanest possible air. The West Gate Tunnel Project should strive for this.

Appendix A

- The charts used in Sections 5.7 – 5.10 are not recent, highly aggregated and not likely to generate meaningful analytics around high traffic areas. SA1 data would be more appropriate.

- Table 5.5 is also aggregated at a high level. If the LGA Maribyrnong has 83,515 (according to last Census) and 10.7% of those have asthma, then the real issue is if those 10.7% live within 100m of main traffic corridors in Yarraville and Seddon for example. The aggregation level does not reflect the localised impact.

- Pairing the recent census data with health impacts from 2003-2007 is likely to underrepresent the applicable (traffic emission related) health impacts. The assumption that it over represents the health impacts as general health is on an improving trend is somewhat irrelevant given the health impacts of concern are traffic related, and not general health. Keeping in mind air pollution related mortality in Australia is increasing rather than decreasing, and that due to population growth the number of people exposed in the census year is significantly higher than the years the health data was collected, we find this methodology and assumption incongruous.

- Ground level ozone, a by-product of vehicle emissions is a significant contributor to the health impacts of vehicle emissions and should be included in the HIA. As the severity, duration and frequency of heatwaves is increasing in Victoria (SoE report, 2013), ground level ozone will increase (process is catalysed by sunlight). Ground level ozone, particulate matter and high temperatures interact to have additive impacts on mortality and respiratory disease (Bernstein and Rice, 2013).

- The risk analysis is based on methodology established in 2004 and is possibly outdated. Rather than mortality risks, assessing risk on Disability Adjusted Life Years (DALYs) is possibly a more appropriate method.

- We consider it incongruous that when discussing the lung cancer incidence, the City of Melbourne is discussed, however for Maribyrnong the discussion moves to ‘cancer’ (presumably ALL cancers?)

- There are inconsistencies for asthma prevalence discussions and tables. Some sections include only adult asthma related ED admissions and in another section children are referred to but only aged 5 and greater. As the age group most susceptible to vehicle emission
respiratory impacts is 0 – 5 years, we suggest it needs to be included to provide the most informative background information. We disagree with the conclusion that asthma incidence is consistent with the national rate. Maribyrnong has the highest rate of respiratory related ED admissions in children in Victoria and is listed as a national ‘hot spot’ for asthma in children in the Australian Atlas of Healthcare Variation.

- The HIA mentions the PAHs are below ‘health related thresholds’. We are unclear which thresholds are being referred to. The NEPM PAH thresholds are not health related, the thresholds are set at intervention levels to serve as a red flag for when interventions are required.

- If residents adjacent to Francis Rd will experience improvements, it stands to reason that residents adjacent to streets with a projected increase in pollution will experience deteriorations. If the improvements are mentionable, then we believe the deteriorations are more than insignificant.

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