Letter of Medical Evidence opposing NorthConnex tunnel portal and stack placement in residential suburb

We are writing to you regarding the planned NorthConnex tunnel and associated northern ventilation stack, proposed for the densely populated *residential suburb* of Wahroonga, NSW. This project will emit *unfiltered* exhaust fumes from approximately 5000 trucks and 9000 cars per day into an area which has a high density of schools, hospitals and aged care facilities, and will be less than 50 metres to the nearest house.

As health professionals, we feel that this project will have a *major negative impact on the health of the surrounding community.*

The NHMRC (National Health and Medical Research Council) states that the great advantage of tunnels is that their portals and stacks can be *deliberately sited away from residential areas.* These recommendations are also found internationally.

International air pollution experts state that there is no safety threshold to the amount of air pollution causing health impacts, hence *there is no "safe level"*. The smallest amount of air pollution will have a corresponding amount of health impact. Even low dose exposures to particulate matter have been demonstrated to have significant health risks.

There are numerous, well documented health risks associated with air pollution exposure, as found around tunnel portals, tunnel stacks and associated on-ramps. As such we would like to raise our concerns regarding this proposal.

The following facts regarding air pollution are researched and documented in the scientific literature:

- There is an increased *risk of death in people* exposed to particulate matter*, even when exposure is within concentration ranges well below the present European standards.*
- Air pollution causes *Lung Cancer and is associated with Bladder cancer.*
- In 2010, 223,000 deaths from lung cancer worldwide resulted from air pollution according to the World Health Organisation (WHO)
- WHO classifies diesel exhaust fumes as a *carcinogen (cancer causing)*, belonging in the "same deadly category as asbestos, arsenic and mustard gas"
- *Ultrafine particles* (median diameter <0·1 micrometers) are more toxic when inhaled than other measurable particles. They are greatly absorbed into tissues and the circulation and are important factors in determining *cardiopulmonary toxicity.*
- Both short- and long-term exposures to particulate matter are associated with a host of *cardiovascular diseases, including heart attacks, arrhythmias, strokes and increased risk of death* from the above cardiovascular causes
- Children show *reduced lung function* growth which persist later into life, even when exposure stops, i.e. the damage for growing lungs is permanent.
- Children have been found to suffer from symptoms of *bronchitis* following exposure
- Residents living around tunnel ventilation stacks report more *upper and lower respiratory symptoms and have lower lung volumes.*

- *Low birth weights* are more common in pregnant women exposed to traffic pollution.
- Exposure to traffic-related air pollution during pregnancy and during the first year of life is associated with *autism.*
- Higher levels of long-term pollution are associated with significantly faster cognitive decline i.e development of *dementia*
- Outdoor pollutant levels correlate with those measured indoors in houses exposed to air traffic pollution.

Traffic emissions contain substances that are not accounted for in standard pollution modelling. These include ultrafine particles and other *unmeasured substances.* Dozens of compounds can be detected in vehicle exhaust. While the adverse effects of these exhausts have been extensively studied surrounding open roadways, the hazards to local residents and commuters resulting from the presence of ultrafine particles are less well known. *It is these ultrafine particles and unknown substances that potentially pose a great health risk.*

We are very concerned that any modelling of air quality and drawing conclusions on their resultant health impacts drawn from this modelling prior to construction will be inaccurate, *as little scientific evidence exists for long term health impacts of unmeasured particles.*

As health professionals, we strongly oppose the construction of a major source of air pollution at the proposed site. This is a residential neighbourhood. We are of the opinion that there will inevitably be negative long and short term health impacts in the surrounding area.

We strongly feel that an alternative solution needs to be found.

Please find attached a detailed review of the literature, outlining the medical evidence for serious potential health impacts of such a project. Also attached are signatures from health care professionals from a multitude of specialties.

Yours faithfully,

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Medical Evidence regarding adverse health effects of air pollution from tunnel portal and stack placement in residential suburb

There exists an overwhelming amount of medical evidence on the adverse health effects of air pollution, and as such, we have selected some of the most relevant and significant articles to present our concerns.

A recent study released in The Lancet, one of the most prestigious international medical journals, reported the adverse health effects in 367,251 people with long term exposure to air pollution. These people were followed for an average length of 13 years, during which 29,076 died.
The study found that there was a significantly increased risk of death in the participants exposed to particle matter. This risk was even found in individuals whose exposure was within concentration ranges well below the current European standard (1).

Another major study conducted by the American Cancer Society enrolled approximately 1.2 million adults in 1982 for an ongoing prospective mortality study.
Fine particulate and sulfur oxide--related pollution were associated with an increased risk of lung cancer and death from heart and lung diseases. Each 10-microg/m increase in fine particulate air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively (2).

A WHO press release in October 2012 stated that after thoroughly reviewing the latest available scientific literature, the world's leading experts concluded that there is sufficient evidence that exposure to outdoor air pollution causes lung cancer. They also noted a positive association with an increased risk of bladder cancer (3).
The most recent WHO data indicates that in 2010, 223,000 deaths from lung cancer resulted from air pollution (4).

Medical evidence is overwhelmingly clear that long term exposure to air pollution increases death rates.

A local study by Cowie et al looking at health effects of the Lane Cove tunnel in Sydney, NSW studied participants before and after the opening of the tunnel. The study found that residents living within 650m of the tunnel ventilation stack reported more upper and lower respiratory symptoms and had lower lung volumes in the first 2 years after the tunnel opened (5). There was also, unfortunately, no consistent evidence of improvement in respiratory health in residents living along the bypassed main road, despite a reduction in traffic from 90,000 to 45,000 vehicles per day.

A recent study using data from numerous international studies looked at dose response relationships for PM 2.5 (6). The results suggested a relatively steep exposure–response function at very low levels of exposure to PM 2.5 and a flattening out of cardiovascular risk at high exposure levels.
At very low levels of exposure excess mortality risks are similar for lung cancer and CVD mortality. A relative risk of 1.3 was found for cardiopulmonary disease secondary to PM2.5 levels of 24.5 micrograms/m3.
Current air quality modelling guidelines consider a level of less than 50 micrograms/m3 to be safe. This is equivalent to the risks associated with exposures to moderate to high levels of second hand cigarette smoke.
A potential explanation regarding the steep exposure–response for CVD mortality at low levels of exposure and the levelling off at high exposures is a saturation phenomenon whereby relatively low levels of exposure are capable of activating relevant biological pathways.

There is substantial and growing evidence that long-term exposures to PM2.5 from cigarette smoke, ambient air pollution, or both affect multiple physiologic pathways. Even low levels of exposure to PM 2.5 from second hand smoke and ambient air pollution have been associated with pulmonary and systemic oxidative stress, inflammatory vascular dysfunction, increased platelet activation and blood viscosity, atherosclerosis, IHD, and altered cardiac autonomic function

In eight different communities in Switzerland, lung function in adults was negatively associated with PM10, nitrogen dioxide, and sulphur dioxide all of which are pollutants arising from vehicle exhausts (7). The pollutants also increased symptoms of bronchitis (8). In children from ten Swiss communities, the same pollutants were found to be associated with symptoms of bronchitis (9).

In children living in 24 communities in Canada and the USA, significant associations were reported between exposure to fine particles and lung function and symptoms of bronchitis (10-12).

Exposure to particulate pollution is associated with reduced lung function growth in children (13), and even children relocating from high to low pollution areas (or vice versa) were shown to experience changes in lung function growth that mirrored changes in exposure to particulate matter (14).

Gauderman et al followed school children from the age of 10 for 8 years to observe the effects of air pollution on lung development. He showed that lung development is significantly affected through reductions in FVC, FEV1 and MMEF, as would be expected of the children had been exposed to maternal smoking(15).

Studies from across the world have consistently shown that both short- and long-term exposures to particulate matter are associated with a host of cardiovascular diseases, including heart attack, heart failure, abnormalities of heart rhythm, strokes and increased death from cardiovascular causes (16).

Evidence from cellular/toxicological experiments, controlled animal and human exposures and human panel studies have demonstrated several mechanisms by which particle exposure may both trigger acute events as well as prompt the chronic development of cardiovascular diseases. Particulate matter inhaled into the pulmonary tree may instigate remote cardiovascular health effects via three general pathways: instigation of systemic inflammation and/or oxidative stress, alterations in autonomic balance, and potentially by direct actions upon the vasculature of particle constituents capable of reaching the systemic circulation. In turn, these responses have been shown to trigger acute arterial vasoconstriction, endothelial dysfunction, arrhythmias and pro-coagulant/thrombotic actions (17).

In both short-term and long-term studies, air pollution has an effect on cardiac deaths and hospital admissions in addition to respiratory effects. Plasma viscosity, as well as heart rate and concentrations of C-reactive protein, were increased (18-20), all of which can contribute to an increased risk of cardiovascular events.
Studies in Boston, MA, USA, showed that nitrogen dioxide and PM2·5 were associated with life-threatening arrhythmia leading to therapeutic interventions by an implanted cardioverter defibrillator (21), and that PM2·5 concentrations were higher
in the hours and days before onset of myocardial infarction in a large group of patients (22).

Hoffman et al found that long-term residential exposure to high traffic is associated with the degree of coronary atherosclerosis. Participants living within 50m of a busy road had an odds ratio of 1.63 for developing coronary artery calcification compared with a control group (23).
Older subjects (greater than or equal to 60 years of age) and women were found by Künzli et al, to have a 15.7% stronger association between particle matter exposure and carotid intimal thickening, ie the risk of stroke. (24)

In a study of 1,705 Boston-area patients admitted to hospital with strokes, the risk of stroke was increased by 34 percent on days when traffic pollutants were classified by federal regulators as "moderate," which is defined as a minimal danger to health. These results suggest that exposure to $PM_{2.5}$ concentration generally considered safe by the US EPA increase the risk of stroke onset within hours of exposure (25).

One of the most commonly measured chemicals arising from car emissions is nitrogen dioxide. Associations between natural-cause and respiratory mortality have been found to be statistically significant for NO_2 and black smoke (26).

Giulia et al studied the effects of long-term exposure to both fine particulate matter (≤ 2.5 μm; $PM_{2.5}$) and nitrogen dioxide (NO_2) on risk of death (27). This large study of over 1.2 million subjects strongly supports that long-term exposure to NO_2 and $PM_{2.5}$ increases risk of death, especially from cardiovascular causes.

Traffic emissions contain substances that can be measured and that cannot be measured or are accounted for in standard pollution modelling.

Dozens of volatile and semivolatile organic compounds can be detected in vehicle exhaust, along with numerous metals and oxides of sulfur, nitrogen, and carbon. While the adverse effects of these chemicals have been extensively studied surrounding open roadways, the hazards to local residents and commuters resulting from the presence of tunnel emission chemicals are less well known (28).

It is the unknown substances that potentially pose a great health risk in themselves.

The recognition that ultrafine particles (mass median diameter <0·1 _m) are more toxic when inhaled than PM10 suggests that their ability to be absorbed into tissues and the circulation, and their greatly increased surface area, might be important factors in determining cardiopulmonary toxicity (29).

In the local study by Cowie et al, which looked at the health impacts on locals living near the Lane Cove Tunnel Stack, the study found that there was an increase in the number of adverse health effects among residents living around the stack. It also went on to suggest that these effects may have occurred due to unmeasured pollutants. (5)

Diesel particulates and ozone have been shown to increase the synthesis of the allergic antibody IgE in animals (30), and human beings (31), which would increase sensitisation to common allergens (32). By interacting together and with other environmental factors, particulates and gaseous air pollutants can have long-term effects on allergic individuals.

Short term and long term health impacts have been well studied internationally.
The findings of increased airway inflammation and symptoms in subjects after only 2 hours exposure at a heavily trafficked location indicate that even short-term exposures to traffic-related air pollution has adverse health effects (33).

Fischer et al found that outdoor pollutant levels correlated with those measured indoors in 36 houses exposed to air traffic pollution. A substantially larger contrast (about a factor two) was found for outdoor concentration of the particulate components BaP, total polycyclic aromatic hydrocarbons, absorption coefficient (`soot') and the gas-phase components benzene and total volatile organic compounds. The contrasts for these pollutants were substantially larger than the estimated contrast in average NO_2 (22%). (34)

Pregnant women exposed to sulphur dioxide from traffic pollution are more likely to give birth to low birth-weight babies. (35)
Exposure to traffic-related air pollution, nitrogen dioxide, $PM_{2.5}$, and PM_{10} during pregnancy and during the first year of life is associated with autism (36).
Effects on the elderly
Higher levels of long-term exposure to both $PM_{2.5\text{-}10}$ and $PM_{2.5}$ are associated with significantly faster cognitive decline, i.e. can accelerate the development of dementia. (37).

A study of 137 Brisbane school children at 25 schools by Mazaheri (38) analyzing alveolar concentrations of ultrafine particles concluded that children's exposure during school hours was more strongly influenced by urban background particles than traffic near the school. The study also found that the highest dose intensity occurred during outdoor times at school and when children were more active.
There are large numbers of children in the immediate area surrounding the stack, attending schools.
These background levels of ultrafine particles could be significantly affected by an unfiltered exhaust stack within close proximity of multiple schools.

Buonanno et al (39) studied particle concentrations at schools in several different urban locations. In general, children attend school during day time hours on weekdays when traffic intensity is high.
It has been proven, that outdoor pollutants are able to penetrate inside the buildings, influencing indoor concentration levels on the basis of traffic, meteo-climatic and urban characteristics with regard to airborne particles. Indeed, indoor pollutants were found to explain a number of health effects even at concentrations significantly lower than outdoors.

In a separate study, Buoanno et al (40) looks at the health effects of dose related particle exposure on children. Significant differences were found for asthmatics, children with allergic rhinitis and sensitive to allergens compared to healthy subjects.
At present, it is not known which particle size, morphology or chemical components are most strongly related to the negative effects on human health and further research in this field is required.
These effects have received more attention in relation to children, because they inhale a higher dose of airborne particles relative to lung size when compared with adults.
Nevertheless, the major difficulty facing epidemiological studies of ultra fine particles is mostly related to the estimation of individual exposure levels. The most common current approach assumes that each person in a given region has the same exposure level, which is often obtained from a few air quality monitors and reflects the mean concentrations in the entire urban area or community.
This approach could lead to significant errors in the estimation of individual exposure to air pollutants because the actual exposure is strongly related to the time activity of the individuals. Furthermore, the use of mean air pollution levels smoothes peak air pollution concentrations and thus, may result in unreliable estimates of exposure (Manigrasso et al., 2013).

Furthermore, several authors have suggested that short term fluctuations in aerosol concentrations of particles increase morbidity and mortality (Brugge et al., 2007; Strak et al.,)

A recent study by the OECD has found that Australia is amongst only 14 out of 34 developed countries in the world where deaths from air pollution have increased in the past 5 years. In between 2005 and 2010, the number of deaths from air pollution in Australia increased by 68 per cent. Evidence suggested that road transport was probably responsible for about half of all deaths from air pollution. The economic cost for Australia was about $5.8 billion in 2010, up from $2.9 billion just five years earlier (41).

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