Health Issues Relevant to the Proposed Basin Reserve Flyover: Information for the Basin Reserve Schools

This report provides general information to assist the schools in the Basin Reserve area to consider health effects from the proposed Basin Reserve Flyover. Schools would need to obtain expert advice to assess the direct impact the Flyover might have on their specific school.

Introduction

Over the past two decades a sizable body of research has identified that health effects occur in proximity to major roads. Air pollution, noise and contamination of surrounding vegetation and soil is has been well documented and these impacts have a significant effect on health. This report seeks to provide a brief summary of main findings and highlight some of the process and policy issues which have been prevalent in other applications for resource consent made by the NZTA. Further details of research findings have been attached as an appendix.

A recent literature review prepared for the Board of Inquiry into the Kapiti Expressway (O'Sullivan 2012) identified that air pollution produced by traffic volumes is associated with a number of significant health effects. While some of the studies reviewed incorporated traffic volumes higher than would be expected in New Zealand, many did have traffic flows consistent with New Zealand conditions and several were conducted in local areas. A Christchurch study revealed that all cause mortality has been shown to increase with exposure to traffic pollutants with respiratory mortality most affected. Traffic pollutants associated with diesel trucks have been shown to increase respiratory disease, particularly asthma, with a strong case being made for nitrogen dioxide (NO2) as a causative factor in this disease (Janssen, 2003; WHO, 2005; Gauderman, 2005; O'Connor, 2007; 2008; Brugge et al 2007). Children appear to be differentially affected by exposure to traffic pollutants and evidence also suggests that living next to busy streets in infancy, while lungs are still developing, is a causative risk factor for asthma. Low birth weights are also associated with exposure to traffic pollution.

Life time exposure to traffic pollutants is a focus of concern for the Public Health sector. Long term effects when studied through cohort studies and other time serious investigations have clearly established the link between these ongoing exposures and poor health. The link between traffic pollutants and adverse health effects does not appear restricted to major freeways. The Netherlands study conducted by Hoek et al (2002) established that major urban roads can also be associated with adverse health effects. This large study found participants were almost twice as likely to die of a pulmonary or cardiovascular event if they lived next to a major road. Rapid elevation of blood pressure has been found in association with exposure to particulate matter, which is also fine enough to pass through the lungs and cause macrophage damage to the heart. The development of atherosclerosis is also associated with exposure to traffic pollutants. Therefore residential properties and schools in the vicinity of the proposed Basin Flyover and tunnel upgrades are likely to be affected by reduced air quality from this major road. Further research findings on air quality in relation to major roads can be found in appendix A to this report.

Noise from this project may also cause significant impacts, both in relation to construction noise and operational noise when the Flyover is complete. Children are more affected by background noise such as traffic noise and this has a detrimental effect on learning and ability to understand speech (Kujala & Brattico & Johnson (2000). Maxwell and Evans (2000),

Hygge 2003, Lercher et al 2003, Kempen et al 2012. Kozou et al (2005) found the more varied the background noise the greater the effect on cortical discrimination processing. Hence, noise from a variety of vehicles can significantly disrupt cortical auditory processing. Monotonous background noise appears more easily ignored. Noise is also known to raise blood pressure and cause other physiological stress reactions (Fyhri & Aasvang, 2010; Willich et al 2005), which can result in adverse health impacts for both students and teachers.

Further details of research findings on noise can be found in appendix A of this report.

Pertinent to any discussion of the Basin Flyover and tunnel upgrade in relation to the Basin Reserve schools is the location of tunnel vents, which extract a high concentration of vehicle emissions. Visually these vents cannot be described as attractive, rapidly accumulating deposits of black carbon and are reminiscent of early industrial smoke-stack architecture, as illustrated by the artists impression of the Waterview project ventilation stack below.

These vents discharge large concentrations of particulate matter, nitrous oxides, carbon monoxides and benzene into the surrounding area. (See table of emissions and their effects in Appendix A) Note that there is no safe level of benzene, hence no air quality guidelines associated with it. In the Waterview project, particular concern was expressed to the Board of Inquiry regarding the location of these vents alongside the playing fields of Waterview primary school, seen in the above illustration. Despite the acknowledgement that regional air quality guidelines for PM2.5 were regularly exceeded, NZTA's public health expert, Dr David Black, assured the board of inquiry that: "This proposal is not a concern in the overall protection of public health."

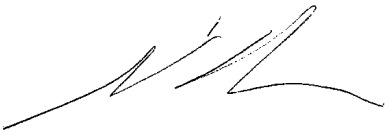
The Board however disagreed, and required that the location of the vents be moved away from residential areas as a condition of granting resource consent for the project.
Further technical details can be found at:
http://www.nzta.govt.nz/projects/completing-wrr/docs/docs-enquiry/board/7s42a-final-air-quality-review.pdf

That document is a technical report on air quality produced by the aptly named Emission Impossible consultancy for NZTA. However, an independent report questioned the validity of the technical data submitted:
http://www.aktnz.co.nz/2011/01/19/report-slams-official-waterview-claims/

As was also found at the Board of Inquiry into the Kapiti Expressway, air quality effects appeared to be underestimated. Any estimates for the Basin Flyover project should be carefully examined, therefore, as they may be based on a simple snapshot study over a small area. These technical reports also tend to omit any analysis of long term health impacts and ignore cumulative effects. The inner city bypass, hailed as a solution to reduced traffic congestion and improved air quality is now one of the most congested and polluted areas of Wellington city and this air quality within the same airshed may also combine with increased emissions to exceed guidelines (see appendix B). In addition, Riddiford St has one of the highest levels of nitrous oxides in the Wellington region (Mitchell, 2012), which may also contribute to cumulative effects in this area.

The Basin Reserve Schools should also be aware that health effects occur at levels below air quality guidelines, which are to some extent a lagging indicator of air quality and health. Recent claims by NZTA that these guidelines will protect the most vulnerable are not supported by the epidemiological evidence, nor is there any evidence to support the agency's claim that these roading projects will result in less traffic congestion and improved air quality. Analysis of other projects such as the inner city bypass (Appendix B) indicate that the result is the reverse of NZTA's claims, with more congestion and reduced air quality apparent.

While improvements in fuel efficiency and engine technology have lead to energy savings, these have not translated into fewer health impacts. In fact, concentrations of nitrogen dioxide have increased with the use of catalytic converters and the removal of lead from petrol has introduced another pathogenic additive, that of benzene, known to cause luekemia and bone marrow suppression. Diesel is also now acknowledged as a known carcinogen with increased rates of cancer associated with exposure to traffic pollution. There is even emerging evidence that inflammatory processes associated with exposure to traffic pollution are a contributory factor in neurodegenerative diseases such as dementia. Increasing concentrations of nitrogen dioxide have serious implications for the development of asthma in children, exposure to other pollutants is known to affect the unborn child and older residents are also more vulnerable to the effects of degraded air quality from proximity to motorways.



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Dr. Marie O'Sullivan has a PhD in psychology. Since 2008 she has worked as a lecturer in public health at the Wellington School of Medicine (part of the University of Otago's Wellington campus). In that position she has taught and tutored in public health at post graduate level, with specific input into public health policy, health promotion and environmental health. She has also supervised 4th year medical students in public health projects. She currently supervises research work in public health.

Dr O'Sullivan's academic background prior to working in public health was in Neuropsychology and she retains a research interest in aging and disability. She has also spent a number of years as a Treasury analyst in social policy and local government.

Dr. O'Sullivan has provided evidence and acted as an expert witness to the hearings for the recent Boards of Inquiry into the NZTA proposals for Transmission Gully, and for the Mackay's to Peka Peka (Kapiti) Expressway.

Appendix A: Literature Review of Air Quality and Noise:

Evidence relating to proximity to Major Roads [1]

The WHO defines air pollution as made up of primary and secondary air pollution. Primary air pollution consists of sulfur dioxide, nitrogen oxide, carbon monoxide, volatile organic compounds (VOC) and particulate matter. Secondary pollution arises from the chemical reactions of primary pollutants, often with the natural compounds found within the environment. They can include nitrogen oxide, ozone, and secondary particulate matter. Major sources of pollution come from the combustion of petrol or diesel producing an exhaust gas which contains a number of pollutants potentially harmful to health. These typically include Carbon monoxide (CO), Nitrogen dioxide (NO2), Volatile organic compounds (VOC) and fine particulate matter (PM10, PM2.5). Other chemicals are also present as detailed in table 1.

Table 1: Impact of Motor Vehicle Emissions On Health

Contaminant	Formation	Absorption and effects in lung	Health effects
Carbon monoxide (CO)	Gas formed from incomplete combustion of fuels such as petrol and diesel	- Absorbed into the bloodstream - Reduces the oxygen-carrying capacity of blood (which can adversely affect the brain and heart)	Increased hospital admissions & mortality from cardiovascular disease Headaches Dizziness, Disorientation Visual disturbances Stress, anxiety Acute death after very high exposure
Nitrogen dioxide (NO_2)	Gas produced during fuel combustion	- Directly affects the lung. - Impairs the lung's immune defence mechanisms	- Increased frequency of coughing, wheezing & breathlessness - Increased susceptibility to infections & asthma attacks - Increased severity of asthma attacks - Increased reactivity to natural allergens - Stress, anxiety, irritability
Fine particles (eg. PM_{10} which is particulate matter smaller than 10 μm)	Solid particulate matter produced from emissions	- Penetrates into the lungs - contains chemical and biological (e.g. pollen) contaminants in solid form - lung cancer risk may be due to components of the particles or gaseous hydrocarbons in the emissions	- Exacerbates respiratory conditions such as bronchitis and asthma - Increased mortality - Increased hospital admissions for respiratory and cardiovascular disease - Increased frequency of respiratory symptoms - Reduced resistance to infection - Increased school absences, lost work days and restricted activity days – exacerbating stress and anxiety - Increased risk of myocardial infarction and cardio-respiratory death - increased risk of lung cancer
Benzene	Produced by fuel consumption	Known carcinogen	- Bone marrow suppression - Leukaemia
Lead	Leaded petrol was used in New Zealand until 1986	Absorbed via lungs into blood stream	Negative impact on children's neurocognitive functioning

Sources: USEPA (1998); WHO (2001)

(Source: Kjellstrom T & Hill S, (2002). New Zealand Evidence for Health Impacts of Transport, a background paper prepared for the Public Health Advisory Committee.)

[1] From: O'Sullivan, (2012). An Equity-focussed Health Impact Assessment of the Proposed McKays to Peka-Peka Expressway: Available online: www.Savekapiti.co.nz

In New Zealand each year there are a total of 1,921,000 restricted activity days, that is the number of days lost due to people being exposed to air pollution. A 2005 study in Christchurch found that a 10 microgram increase in 24hr PM10 was associated with a 1% increase in all cause mortality, 4% increase in respiratory mortality and a 3% increase in hospital respiratory admissions of adults and children. Kjellstrom and Hill (2003) showed that lifetime exposure to PM10 increases total mortality by 1.6%. More recent analysis from the HAPINZ study (Fisher, Kjellstrom, Kingham, Hales & Shrestha, 2012) suggests that mortality increases per 10ug/m3 PM10 may be as high as 5-10%. Exposure to pollutants is largely beyond the control of the individual and so the state has a responsibility to consider the control of exposure to air borne pollutants in the planning and implementation of a major road.

The type of vehicle traffic also affects the concentration of air pollutants. Janssen et al (2003) found truck traffic increased indoor and outdoor PM2.5 concentrations and soot more than car traffic while Ciccone et al (1998) found average NO2 concentrations were higher in streets with high truck volumes than in streets where trucks were reported as infrequent. Truck traffic flow in several centers throughout Italy was recorded within a mean range of 98-167 vehicles per hour, giving an estimated 2,353 – 4008 trucks per day. These levels are comparable with New Zealand conditions. Ciccone also found that a high frequency of trucks was associated with an increased risk of respiratory problems, with strong associations found with increased bronchitis (odds ratio (OR) 1.69), bronchiolitis (OR 1.74), pneumonia (OR 1.84) and severe wheezing limiting speech (OR 1.86). Persistent phlegm also showed strong associations with frequency of truck traffic, with an odds ratio of 1.68. Hence residents were 68% more likely to experience persistent phlegm with exposure to truck traffic (if symptoms were the same as residents not exposed to truck traffic, the odds ratio would have been 1.) .

Traffic movement is also a factor in air pollution. Ryan et al (2005) sampled for PM2.5 and black carbon in traffic and found that during stop-and-go traffic, levels of air pollutants increased significantly. In a review of 21 studies, results showed traffic that was not traveling at a constant speed (i.e. constantly accelerating and decelerating) was associated with higher emissions of air pollutants compared to traffic traveling at a constant speed. Hence air pollution from traffic is likely to be higher around areas where interchanges and intersections occur, where vehicles are exiting and entering the motorway with acceleration and deceleration occurring.

Speeding traffic also contributes to air pollution, along with increased noise levels. Long straight roads encourage higher speeds and drivers tend to drive to road conditions rather than imposed speed limits (Hanworth & Rechnitzer, 1993; Brown & Cotton, 2003). These conditions can also include the speed of other drivers (Haglund& Aberg, 2000). Long straight roads also lower perception of crash risk (Brown & Gould 2012). In addition, speeding drivers are optimistic regarding the chances of a crash or injury to others (Delhomme, Verlhiac & Martha, 2009).

Pedestrians are also not exempt from exposure to traffic related pollution. Briggs, de Hoogh, Morris and Gulliver (2008), found mean exposures to PM10 – PM 2.5 while walking in London were greatly in excess of those found from driving the same route. Mean exposures for walking trips were greater than driving by a factor of 4.7 for PM10. They concluded that more effort is needed to increase the separation between road vehicles and pedestrians to avoid the negative health effects associated with traffic related pollution. These results are supported by other studies showing pedestrian exposure to traffic pollutants in the city of London is high (Kaur et al 2005; Wang et al, 2011).

Prevailing winds are also known to effect concentrations of pollutants. Zhu et al (2002a; 2002b) measured wind speed and direction, traffic volumes and pollutant gradients downwind of a highway and found concentrations decreased exponentially between 17-150m downwind, while at 300m, concentrations matched those of upwind sites. Zhang et al (2004) found that condensation, evaporation and dilution alter size distribution of particles, which grew larger 30-90m downwind. Beyond 90m, there was continued particulate growth as well as particulate shrinkage due to evaporation. Thus, there is no one-to-one direct linear drop off in chemical pollutant gradients as distance from the highway increases. Hitchins et al (2000) found the distance at which pollutant gradients decreased by 50% varied from 100-375m dependent on wind speed and direction.

Use of noise bunds along highways produces an impact on air pollution levels and their distribution. These structures create a surge of air pollutants or "hot zone" further downwind of the highway, approximately 80-100m from the road (Bowker, 2007; Baldauf, 2008; Heist, 2009). These concentrations then carry further at distances of 250-400m, compared to 150-200m at sites with no noise barriers. Hence, residents within 400m of highways with noise bunds have been shown to experience worsening air pollution (Ning et al 2010). While 200m is generally regarded as the yardstick for extinction of air pollutants from highways, other studies suggest a wider margin of safety is needed. Gauderman (2007) found reduced lung function in children living 500m from a freeway. Janssen et al (2003) found children attending 24 schools within 400m of major roads had increased respiratory symptoms in response to truck traffic.

Health equity is concerned with the fact that not everyone is affected in the same way by the same impacts. Peled (2011) in a review of high risk factors for health effects related to air pollution, concluded that older people, young infants, people with pre existing allergies, pulmonary and heart disease, pregnant women and new born babies along with low socio-economic populations are all considered populations at risk. Brugge, Durant and Rioux (2007) in a review of epidemiological evidence for health risks associated with living near major highways, concluded that the weight of evidence established an elevated health risk. They cite a number of studies which found elevated risk for development of asthma and reduced lung function in children living within 30m of major roads (Brunekreef et al 1997; Venn et al 2001; Kim et al 2004; McConnell et al 2006). Studies of particulate matter have also showed strong associations with cardiac and pulmonary mortality (Miller et al., 2007) with a risk ratio of 1.76 (95%CI, 1.25-2.47). Some evidence was also found for an association between lung cancer and highways (Dockery et al 1993; Pope et al 1995). More recently studies have established stronger evidence for the association of traffic pollutants and lung cancer. Particulate matter contains polycyclic aromatic hydrocarbons (PAH) many of which are known to be carcinogenic and increase risks of lung cancer (Slezakova et al 2011). In 2002 the US Environmental Protection Agency also classified diesel exhaust as a carcinogen. In a recent Canadian study, a dose response relationship was found between cumulative exposure to diesel engine emissions and lung cancer (OR 1.68 for large cell carcinoma), Villeneurve et al 2011). Similarly, Olsson et al (2010) reported a 30% increase in relative risk of lung cancer for those exposed to diesel engine emissions. It is the level of exposure rather than the conditions in which it occurs that appear to increase risk. Cancer typically takes many years to develop, hence a lifetime exposure carries significant risk.

Hoek, Brunekreef, Goldbohm, Fischer & Brandt (2002) investigated traffic related air pollution in the Netherlands using a random sample of 5000 people. Long term exposure to traffic related air pollutants was estimated based on the participants home address.

Participants within 100 meters of a freeway or 50 meters from a major urban road were compared with those who lived further away. Exposures to pollutants were not large and comparable to NZ readings. These were estimated at black smoke concentrations of 4.4.ug/m3 and NO2 at 11 ug/m3 for participants in close proximity to a freeway. For participants near a major road, concentrations of 13 ug/m3 for black smoke and 8 ug/m3 for NO2 were used. Mortality was assessed over 8 years.

The study found that cardio-pulmonary mortality was associated with living near a major road , with a relative risk of 1.95. That is, participants who lived near a major road were almost twice as likely to die of a cardio-pulmonary event than those who lived at a distance from a major road.

Health Impacts on Children

Children are especially vulnerable to the effects of air pollution as their lungs and immune system are still developing (WHO 2005). Outdoor play means that they spend more time in the polluted environment and at times when peak traffic flows may occur. They are also more likely to have respiratory infections, increasing their susceptibility to air pollution and exacerbating their symptoms. Children living along the proposed route therefore, will have an uncontrollable increase in exposure to the air pollutants generated.

The findings from the Netherlands study (Hoek et al, 2002) mirror that of Edwards, Walters & Griffith (1994), who demonstrated that chronic respiratory disease in children is associated with distances from busy streets. Studies assessing the risk of developing asthma due to proximity to major roads have evolved over time to present an increasingly precise picture of health impacts. Early studies, frequently reliant on measures such as self-report, often failed to reach significance. However with the advent of GIS and methods which include air monitoring, modeling and proximity to traffic, more precise information has emerged. Evidence suggests health impacts of living next to major highways are greater for boys than girls (Venn, et al, 2001: Kim, et al., 2004; McConnell et al 2006) and that early childhood exposure, before the age of two is a major risk factor (McConnell, 2006).

The effect of traffic pollution on lung development is another health concern to be considered. A WHO report on the effect of air pollution on child health (WHO, 2005), found that Nitrogen Dioxide (NO2) and NO3 caused oxidative damage to developing lung tissue. NO3 was especially damaging to the epithelium of the airways, initiating inflammation and impairing future healing processes in the lung. Gauderman et al (2007) found from their study that over a period of 8 years, 10-18 year olds had reduced lung development if they were living within 500m of a motorway compared to those not living near a motorway. The average decrease in lung function (FEV1) was 81ml. Reduced lung development potentially causes problems such as reduced exercise tolerance and poor respiratory function. For children, healthy development in the early years is a critical factor in determining future health outcomes, with early lung damage likely to have detrimental consequences as the child matures.

While a great deal of emphasis is often placed on the location of schools in evaluation of roading projects, studies suggest that it is the exposure in the home living environment which increases risk. A Dutch study which measured PM 2.5, NO2, and Benzene for one year at 24 schools located within 400m of major roadways, showed lung function was not significantly associated with environmental measures (although truck traffic was associated with

respiratory symptoms) However, restricting the analysis to children living within 500m of highways generally increased the odds ratios, indicative of increased risk of health impacts. (Janssen 2003) Studies in France (Penard-Morand et al., 2005) also indicate that lung function is decreased in association with traffic-related air pollution. Some studies suggest that the finer the scale of monitoring, the more apparent the effects, such as measuring PM using personally carried monitors (Koenig et al 1993). Hence comparing respiratory symptoms with 25 hour averages of traffic pollution may yield little in the way of valid information.

Janssen et al (2003) investigated respiratory health of Dutch children from 24 schools situated within 400m of major roads. They found that exposure to heavy vehicle traffic (5-22,000 trucks per day) showed bronchial hyper-responsiveness and greater sensitivity to common allergens, particularly pollen. Respiratory symptoms increased for highways with high truck traffic but not high car traffic counts. While it was not established whether the hyper- responsiveness was a pre-existing condition prior to exposure to truck traffic, or caused by this exposure, other studies have established that exposure to traffic-related air pollution increases airway sensitivity (Annesi-Maesanos et al 2007; Bentayels et al., 2010; Farhat et al 2011; Ghosh et al 2012) making this a plausible explanation.

Ryan et al (2005) investigated infants living within 400m of bus and truck traffic and found a significantly increased prevalence of wheezing compared with unexposed infants (odds ratio 2.50). A recent study in Spain has highlighted the fact that particulate matter is not solely an air quality issue. It is also deposited in dust at roadside locations and leads to large amounts of hazardous pollutants available for resuspension and redistribution (Amato et al., 2009).

Many studies point to the higher levels of Nitrogen Dioxide (NO2) in traffic pollutants as a key factor in the development of asthma in young children, with particulate matter and NO2 consistently identified as adversely affecting respiratory health. Gaudemann et al (2005) found an odds ratio of 1.83 for outdoor NO2 and a lifetime diagnosis of asthma. That is, children living next to major highways were 83% more likely to have a lifetime diagnosis of asthma. Weinmayr et al (2010) found that exposure to particulate matter, specifically PM10, has a statistically significant effect on cough and asthma. A significant decrease in peak expiratory flow was recorded in the children studied. Hruba et al (2001) found that an increase in respiratory illness which included symptoms of asthma, pneumonia, bronchitis and hospitalisation in children aged 7-10 was associated with an increase in total suspended particulate matter (64% for PM10, 46% for PM2.5). Pennard-Morand (2005) reported a positive relationship between long-term background concentrations of SO2 and PM10 with an increased prevalence of asthma and exercise-induced bronchial reactivity.

Pennard-Morand (2005) also found an increased predisposition toward hypersensitivity or allergic reaction (known as atopy, shown by positive skin prick testing) associated with increased O3 exposure in school children. Janssen et al (2003) found elevated total IgE (an allergy related immunoglobulin) was associated with soot and NO2. Skin prick tests (SPT) showing reactivity to any allergen and to indoor allergens were both significantly associated with NO2, whereas SPT reactivity to outdoor allergens was associated with truck traffic and PM2.5. Early exposure has also been identified as contributing to the increased risk of allergic reaction. Gruzieva et al (2012) found that air pollution exposure during the first year of life was associated with an increased risk of sensitization to pollen by four years of age (OR 1.83). Exposures in children over 8 years of age showed the risk of food sensitization was increased (OR 2.30).

O' Connor et al's (2007) study showed increasing NO2 concentration was significantly related to a decrease in lung function (FEV1) of more than 10% below personal best. There was also an association between increased NO2 and increased occurrence of respiratory symptoms; including days with wheeze, tightness in chest, cough, nights with wakefulness because of asthma, days where the child slowed down or stopped play, number of school days missed.

O'Connor et all (2008) investigated levels of air pollutants and inner city children with asthma. The majority of pollutant concentrations measured were below the National Ambient Air Quality Standards. Despite the low levels present, these were associated with respiratory health problems. The authors concluded that the associations found between Asthma and NO2 suggest that motor vehicle emissions may be causing excess morbidity in this population.

Nitrogen Dioxide (NO2) has been implicated in a number of studies examining asthma and traffic related pollution. While the use of catalytic converters has led to reduced volumes of some particulate matter they have also contributed to increased levels of NO2 over the past decade (Keuken et al, 2009; Kousoulidou et al 2008). McAdam et al, (2011) also found that downwind concentrations of NO2 were relatively stable with distance from the road. Thus the decay pattern seen with many other traffic pollutants does not appear present in dispersion of NO2. This study used a road with approx volume of 34,000 vehicles per day, comparable to New Zealand conditions.

A recent study in Melbourne Australia examined the effects of proximity to major roads and indoor air quality, until recently thought to be relatively unaffected by traffic pollution. The study found indoor NO2 was elevated in near road dwellings (less than 50m) relative to far road dwellings (greater than 300m) by approximately 4 ppb, in the absence of gas cooking (Lawson et al 2011). Other findings indicate that a similar increase of NO2 in outdoor areas increases the risk of lung cancer and childhood asthma (Brauer et al 2000l; Nyberg et al 2000). Hence traffic emissions have the potential to contribute a large proportion of indoor NO2 in dwellings situated close to major roads, with potential health risks to residents.

Air pollution is also know to affect cognitive function in children attending schools close to major rods. (Suglia et al 2008; Wang et al 2009). Associations have also been found between decreased cognitive function and long term concentration of black carbon (Suglia 2008). In a recent study carried out in Spain, further evidence has emerged linking NO2 exposure to cognitive deficits in children (Freire, et al., 2010). Other studies in toxicology lend further weight to the body of evidence pointing toward cognitive impacts on children from exposure to roadside pollutants. The inflammatory reactions and oxidative stress discussed in more detail below in relation to cardiac and neurodegenerative processes are also thought to be involved here (Oberdoister et al 2004; Calderón-Garcidueñas 2011).

Brugge et al (2007) reported elevated risk for asthma for children who moved to residences next to a highway before they were 2 years of age. This study and others lends some support to the conclusion that early childhood exposure to air pollutants may be key in the development of respiratory problems such as asthma and bronchitis. It also emphasizes that the early developmental environment is important in child health outcomes. A number of studies also show greater risk of other respiratory conditions and allergy resulting from highway exposure.

The early developmental environment begins prior to birth. Traffic pollution has also been associated with low birth weight and pre-term delivery. While early studies in this area were often inconsistent, the removal of confounders and improved methodology has resulted in more precise and consistent results. Evidence has been found for negative associations between birth weight and first-trimester exposure to carbon monoxide and particulate matter, second trimester exposure to carbon monoxide, particulate matter, sulphur dioxide (SO2), and ozone and third trimester exposure to carbon monoxide (US Environmental Protection Agency 2006 (EPA); Wilhelm & Ritz 2005; . Salam, Millstein , Li , Lurmann , Margolis, & Gilliand, 2003; Lee et al 2003; Hackley, Feinstein & Dixon, 2007). de Medeiros et al (2009) also reported that neonatal death risk increased if the mother lived close to a main road.

Currie, Neidell, & Schmieder, (2009) found carbon monoxide exposure and negative health effects on infants both before and after birth. These negative effects were present even at low ambient levels and the authors note a reduction in CO levels from 4ppm to 1pmm over the sample period (1989-2003) had an equivalent effect of getting a woman smoking 10 cigarettes a day to quit.

Yorifuji et al (2012) examined placenta to birth weight ratios and absolute weights of placenta and birth weight in 14,189 single live births between 1997-2008 in the Shizholean Prefecture of Japan. They found living within 200m of a major road was associated with a 48% increase in the placenta-birth weight ratio, (that is, higher placenta weight, lower birth weight) In addition proximity to major roads was associated with lower absolute birth weights. They also found maternal smoking was independently associated with lower birth weights.

The association of tobacco smoking with lower birth weight is by now well established (Mercer et al 2008). Also a cause for concern is the growing body of evidence which points to similar effects from proximity to major roads. Exposure to carbon monoxide levels of 4ppm have equivalent health impacts to that of smoking 10 cigarettes per day. Hoek et al (2002) in their Netherlands study found those residents living next to major roads were almost twice as likely to die from a cardio-pulmonary event. It is reported that 50% of all cigarette smokers are likely to die of a tobacco related disease (Boyle, 1997). Evidence from a number of sectors therefore indicates that living next to a major road carries similar risks to that of cigarette smoking.

Yorifuji et al (2012) also found evidence of effects of PM10 and ozone on health of infants at birth, though these findings were less robust then those for carbon monoxide. They estimated that a one unit decrease in mean CO levels in the first 2 weeks of life saves approximately 18 lives per 100,000 births, representing an estimated reduction in the probability of infant death of 2.5%. The authors further note that the method used in the study, that of proximity to air quality monitors, contained some noise, and that more precise measures would yield larger effects. The findings make a strong case for reduction in exposure to traffic pollution, either through tighter emissions standards, or greater separation of residential areas and major roads. These two methods are not mutually exclusive and could be achieved simultaneously.

102. Weight gain in children has also been associated with exposure to traffic in residential areas. Jerrett et al (2009) followed children aged 9-10 years up to age 18 who were exposed to traffic within 150m of their home in Southern California. He found significant positive associations with exposure to traffic and increased body mass index at age 18. He concluded

that the study identified traffic as a major risk factor for the development of obesity in children.

Long Term Health Impacts

Health impacts include both short term and long term effects. Increased morbidity (as indicated by increased hospital admissions) has been directly linked to short term exposure to air pollutants. Ruckerl et al (2007) examined exposures related to hospital admissions in Chicago 1988-1993 and found an increase in PM10 level by 10 _g/m3 was associated with 1.27%, increase in hospital admissions for heart disease, 1.45% increase for chronic obstructive pulmonary disease (COPD) and 2.00% increase for pneumonia.

Long-term effects of exposure to traffic pollutants have also been found, with increased morbidity a feature of this exposure. Vascular damage in the form of accelerated progression of atherosclerosis has been found (Pope et al., 2002; Kunzli et al., 2005; Peters, 2005).
Long-term effects of air pollution on mortality are generally investigated by the use of cohort studies. Because conditions such heart disease and cancer can take 20-30 years to develop, these studies cover many years of exposure, involve large numbers of participants, and provide information on air pollution morbidity and mortality. A large-scale study based on 14-16-year mortality in 8,111 adults in six U.S. cities (HSCS Harvard Six Cities Study) demonstrated a close relationship between levels of PM2.5, cardiopulmonary mortality and lung cancer. An extended follow-up of the six cities study revealed that the increase in the relative risk of mortality averaged 16% per 10-_g/m3 increase in the PM2.5 concentration risk ratio 1.37). One of the most comprehensive studies, known as the American Cancer Society (ACS) study carried out from 1982 to 1989, linked individual health risks for residents of approximately 150 U.S. cities with ambient air quality in those cities (Pope et al., 2006). The study showed a risk ratio 1.31 for all-cause mortality due to increased levels of PM2.5 . A subsequent follow-up of 500,000 ACS participants through December 31, 1998 indicated that there was a 4% increased risk of all-cause mortality, a 6% increased risk of cardiopulmonary mortality, and an 8% increased risk of lung cancer death, per each 10-_g/m3 increase in PM2.5. A woman's health collective in the US examined 65,000 women respectively alongside data from 573 PM2.5 monitors and found high risk ratios for cardiovascular disease (1.76, 95% CI 1.35-2.47).

Long term effects from traffic related pollution also adversely impact on another vulnerable subgroup of society, that of older people. As western populations age, it is estimated that over 35% of the population will be aged over 65 by 2030. Kapiti is already over-represented with residents aged 65 and over, with the aged population on the Kapiti Coast now at 23% and likely to increase to 33% by 2030. As our population ages, health needs increase and many of these may be related to long term exposure to air pollution. As early as 1990 it has been established that atherosclerosis, associated with ischaemic heart disease increases with age. Fowkes et al (1991) found increases of 8.2 percent for men aged 40-49 years, 82.5% in men aged 70-79 and 76.7% in women aged 70-79. Cohort studies provide evidence that the effects of long term exposure to air pollutants result in cardiovascular mortality, coronary heart disease events, and stroke. Gill et al (2011) in a review of evidence concluded support exists for the existence of mechanisms which include systemic inflammation, autonomic nervous system imbalance, changes in vascular function, altered cardiac structure and development of atherosclerosis. Bentuyels et al (2010) examined the health of 2104 residents aged over 65 years living near major roads in France. They found proximity to air pollution

lead to increased symptoms of bronchitis in the elderly population . The effect was most significant for PM10 and SO2 pollutants.

In a recent review of the area, Simkhovich, Kleinmen and Kloner (2008) presented a body of evidence which indicates that ultra fine particles of air pollutants pass through the lung and directly transport to the vasculature and heart, where they can induce cardiac arrhythmias, decrease cardiac contractility and coronary flow. Further evidence for cardiac impact has come from a study by Brook & Rajagopalan (2009) who found that increases in fine particulate matter increased the risk for elevations in arterial blood pressure which were a likely contributing factor to the risk of myocardial infarction, stroke and heart failure. They conclude that rapidly triggered autoimmune nervous system imbalance occurs in response to inhalation of fine particulate matter, raising blood pressure within minutes of exposure. This occurs in present day environments, considered to be considerably lower than concentrations of 30 years ago. Further evidence suggests that in individuals with existing elevated blood pressure, the response is further exaggerated, leading to chronic elevation with even greater increases in blood pressure. The authors also note the temporal nature of the exposure-response relationship and the important issues this raises for further research. This is another area where merely examining 24 hour averages of air quality data may be inadequate. Hoek et al (2010), commissioned a panel of European experts to give an estimate of the likely concentration – response function. They estimated that a decrease in all-cause mortality with a permanent 1000 p/cm3 decrease in ultra fine particles was a median 0.30% with a 0.20% reduction in hospital admissions. Baseline concentrations were set at 20,000 particles making the reduction in the region of 0.5% of baseline. Hence a 10% reduction in particulate matter may reduce all cause mortality by 6%. The authors also note that there is a temporal correlation between particle mass and number and these are cumulative effects not captured by a single estimate of each particulate matter indicator.

Fine particulate matter is also capable of passing through the lung and crossing the blood brain barrier to the brain. Animal studies suggest that PM10 can cause brain inflammation, a process involved in the pathology of neurodegenerative diseases such as Alzheimer's disease (AD). Ranft, Schikovski, Sugru, Krutam & Kramer (2009) looked at exposure to fine particulate matter and cognitive impairment. A group of 399 women who had lived at the same address for 20 years participated in the study. The address was assessed for long term exposure to PM10 and proximity to major roads. A dose- response relationship was found between exposure to particulate matter and performance on neuropsychological tests. The authors conclude that impaired cognitive function was found in association with exposure to traffic-related particulate matter and that this exposure may be involved in the pathenogenesis of AD. Autopsy studies have also supported this conclusion. Higher cerebral beta-amyloid deposits have been associated with lifelong exposure to air pollution (Calderón-Garcidueñas, Solt, & Franco-Lira 2008; Calderón-Garcidueñas et al., 2011).

It has been argued by some industry analysts that air quality issues have declined with improvements in fuel composition and, in particular, since lead was removed from petrol. Countering this argument, Lipfert et al. (2003) note that the decline in regulated tailpipe emissions since the mid 1970's has not been matched by corresponding reductions in traffic-related mortality risks. They point to other factors involved, such as particulate matter from brake, tire, and road wear as contributing to mortality increases. While tailpipe emissions overall have reduced, levels of benzene have increased with the introduction of unleaded fuel. Benzene is known to cause bone marrow suppression and is associated with Leukemia

(Kellstrom 2003). There is no established safe level of benzene and it remains a substance capable of endangering health at low levels.

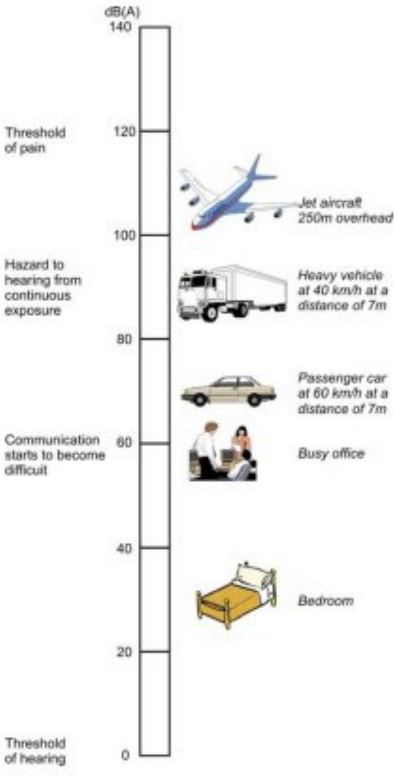
Factors such as traffic type, vehicle movement, wind and environmental factors all play a part in generation of air pollution and levels of exposure. Respiratory illnesses such as asthma, bronchitis, wheezing and cough as well as allergic reactivity have been associated with increased air pollution. The correlation of particulate matter and Nitrogen Dioxide (NO2) with respiratory illness seems to be the strongest evidence for adverse health effects. Lung development occurs during childhood, so if it is adversely affected by air pollution there can be flow on effects to adulthood. In addition, long term effects from prolonged exposure to traffic pollutants can cause heart disease, vascular damage, stroke and possibly even cerebral neurodegeneration.

Noise

> "Unnecessary noise is the most cruel abuse of care which can be inflicted on either the sick or the well,"
>
> Florence Nightingale, 1859, from *Notes on Nursing*

Noise & Sleep Deprivation

Road traffic noise has a negative impact on sleep, causing a physiological reaction because of the need for continued noise processing, which leads to primary sleep disturbance, which in turn impairs daytime functioning and can lead to mental health problems and cardiovascular disease. Night time traffic flows decrease relative to day time while noise levels are perceived as louder and the resultant intermittent noise pattern causes sleep disturbance not generally experienced with continuous traffic noise (Greifahn & Sprung 2004)



The WHO (2009) guidelines state that under 30 decibels (db) no substantial health effects are normally observed. Between 30-40 db, sleep disturbance increases with vulnerable groups such as children and elderly affected. Within the range 40-55 db, there is a sharp increase in adverse health effects. At 55 db and over, adverse affects widely occur and a high percentage of the population is annoyed by these noise levels.

It is estimated that 30% of the European population are exposed to road traffic noise levels exceeding 55 db, which are capable of producing sleep disturbance. In the US it is estimated that 30% of the population is sleep deprived, sleeping less than 6 hours per night. The WHO recommends 7-8 hours sleep over a 24 hour period. (Chang et al., 2010).

Figure 6: level of common sounds on the db(A) scale

Sleep disturbance is more than a minor annoyance from a health perspective. It increases daytime sleepiness, reduces performance and negatively impacts on mood. Reduced

performance in reaction time tests have been recorded along with changes in cortisol levels and endocrine changes, including levels of catecholamines (noradrenalin, adrenaline) and glucocorticoids (steroid hormones involved in carbohydrate metabolism).

Sleep deprivation affects major neuro-endocrine stress systems, principally the autonomic simpatho-adrenal system and hypothalamic-pituitary –adrenal axis. Over time chronic sleep restriction can induce changes similar to those seen in stress disorders such as depression. Insufficient sleep may sensitize individuals to stress related disorders and increase the risk of diseases linked to stress such as cardiovascular disease and mood disorder (Sforza, 2004; Mierlo et al 2008). Restricted sleep causes a cumulative, dose-dependant reduction in performance capacity which continues to decrease as time awake increases (Gander, 2011). With sleep restriction, performance on complex tasks decreases rapidly while simple tasks may remain unaffected. This has safety implications. Gander (2011) cites the example of friendly fire operations during Operation Desert Storm whereby sleep deprived tank crews were able to successfully fire on and destroy friendly vehicles, causing casualties.

Sleep deprivation is also associated with changes in cognition, emotion and behavior consistent with mild prefrontal lobe dysfunction. Killgore et al (2008) examined sleep deprived participants and found reduced self regard, along with reduced assertiveness, sense of independence and self actualization, reduced empathy toward others, lower stress management skills and reduced impulse control, reduced coping strategies such as positive thinking and increased esoteric thinking, such as a greater reliance on superstition.

Noise and poor sleep has also been linked to cardiovascular problems. Fyhri & Aasvang (2010) give evidence from a study in Norway which supports a noise-stress-health model involving increased noradrenalin concentrations, hypertension and increased risk of myocardial infarction. Individuals who are sensitive to noise are more likely to experience sleep disturbance from road traffic noise (Fyhri & Aasvang 2010) Higher blood pressure produces an increased risk of cardiac problems. One European study of 32 Berlin hospitals with 4,115 patients, found that chronic noise increased the risk of heart attack by 50% for men and 75% for women. (Willich et al 2005). In a further study Willich et al., (2006) found noise levels above 50 decibels at night increased the risk if cardiovascular disease in women, with an odds ratio of 2.62, (adjusted OR 3.50)

Ohrstron & Skanberg (2004) examined sleep disturbance from road traffic noise in both laboratory and community settings. In a laboratory setting, perceived sleep quality decreased by 22% during nights with exposure to road traffic noise compared to a quiet night with no noise. In a home setting, the same pattern of results was observed. Pirrera et al (2011) investigated methods of noise assessment in the study of nocturnal noise during sleep. They concluded that use of averages for noise exposure might not be sufficient to reflect the actual noise levels during the sleep period. It is noted that variable noise has adverse impacts and averaging this exposure gives a false picture of events. For both noise and air quality measurements therefore, use of averages do not adequately reflect the dose-response relationships at peak periods.

While perceived sleep quality can often be inaccurate, a number of studies using sleep recording devices have shown that road traffic noise has a detrimental effect on sleep quality. A (2006) study in Sweden used sleep logs and wrist-actography for children and parents to examine road traffic noise and sleep quality relationships. They found a significant exposure–effect relationship between noise levels from traffic and sleep quality, awakeness and tendency to keep windows closed at night. For children a significant exposure-effect was

found alongside daytime sleepiness. Gee and Takeuchyi (2004) found that residents who reported traffic stress had lower health status and more symptoms of depression. High traffic counts were associated with the lowest health status and greatest depressive symptoms. The authors note that traffic stress may be an important factor in the well being of urban populations and an examination of factors on only one level , such as an economic level or individual level, alone, may understate the effects on the social environment.

Effects on Children

Long term exposure to road traffic noise among primary school children has been well documented over the past 30 years (Evans et al 1995, 1998; Hygge et al 1997, 2003; Lercher et al 2003; Shield et al., 2008; Van Kempen 2012).

Both children and elderly are more affected by background noise such as traffic noise and this has a detrimental effect on ability to understand speech (Kujala, Brattico & Johnson, 2000). Children's learning is also affected by noise. Maxwell and Evans (2000), Hygge (2003), Lercher et al (2003), and Kempen et al (2012) have all reported adverse affects on learning in conjunction with noisy environments. Maxwell & Evans (2000) installed sound absorbent panels in children's classrooms and found recognition skills increased markedly compared to a control group in a noisy classroom. Children with disabilities are differentially affected by exposure to noise and perform worse than non impaired children in noisy classroom environments. Kozou et al (2005) found the more varied the background noise the greater the effect on cortical discrimination processing. This finding is directly relevant to consideration of traffic noise, which is variable in tone, intensity and frequency. Hence, cars, trucks buses and motorbikes may all produce a cacophonous quartet of variable noise which disrupts speech-sound discrimination . Speech is normally lateralized to the left auditory cortex, while background noise levels can cause speech perception to predominate in the right hemisphere (Kujala, Brattico & Johnson 2000). This right lateralization may be the auditory equivalent of a right handed person forced to write with their left hand, with a similar level of reduced accuracy in auditory processing.

However, other studies indicate that cognitive changes can occur at noise levels much lower than guidelines set by WHO. Trimmel, Altzsdorfer, Tupy & Trimmel (2012) found even background neighborhood noise of 45 db produced impaired performance on computer based learning tasks compared to a control group exposed to 35 db. Higher levels of noise are known to increase stress (Cohen et al., 1986; Evans, Hygge & Bullinger 1995) which has an adverse impact on most cognitive function. Reading, memory and learning are all processes known to be affected by exposure to noise. It is thought that noise disrupts the immediate cognitive processing of material to be learned within working memory systems (Trimmel et al 2012).

Pregnant women are more vulnerable both to the effects of reduced air quality and sleep disturbance from traffic noise. Reduced air quality has been associated with low birth weights and preterm births (US Environmental Protection Agency 2006 (EPA); Wilhelm & Ritz 2005; . Salam, Millstein , Li , Lurmann , Margolis, & Gilliand, 2003; Lee et al 2003; Hackley, Feinstein & Dixon, 2007). de Medeiros et al (2009) Similarly, sleep deprivation in mothers is also associated with low birth weights and pre-term delivery. Pregnant women in particular need adequate rest to encourage normal development of the child and promote the stamina they need for delivery. Sleep deprivation during pregnancy has been associated with longer labor, elevated perception of pain discomfort, higher caesarian rates, pre-term delivery and high levels of pro-inflammatory serum cytokines, associated with post-partum

depression. Pre-term deliveries are also commonly associated with low birth weights. In addition, they take longer than full term babies to sleep through the night, increasing the sleep deprivation of the mother and increasing the risk of post partum depression (Chang et al 2010).

Effects on Elderly

Background traffic noise, which can vary widely, presents challenges for elderly people who wear battery activated hearing aids (BAHA's). Such variations in the signal to noise ratio have a significant impact on speech discrimination, for both unilateral or bilateral hearing impairments. With normal hearing conditions, background noise can result in unwanted distortions of speech sounds, making comprehension difficult (Plomp. 1986; Schilling, Miller, Sachs & Young, 1998). This distortion effect is intensified for people with hearing loss. Speech recognition thresholds in listeners with hearing loss are up to 12 db higher for one voice when a competing voice is present, compared to those with normal hearing (Hygge et al., 1992). Hence, an elderly listener with a hearing aid in conditions of 50 db of background traffic noise may require the speaker to virtually shout in order for speech recognition to be accurate.

While background noise that approximates speech is easier to accommodate, such as listening in a room full of people talking, intermittent traffic noise presents more of a challenge to a listener with a hearing device. The fluctuating nature of traffic noise creates further distortion in signal to noise ratios which make speech comprehension difficult to anyone with a hearing impairment (Brown & Bacon 2010). Furthermore, attuning hearing devices to background traffic noise presents further challenges (Bach, Anemuller & Collmeier, 2011). Background noise as low as 4 db is also known to interfere with speech discrimination in people with cochlear implants (Brown & Bacon, 2010).

Most people who require a hearing aid will have difficulty understanding speech where there is background noise present. However, as with other areas of perception, speech perception has wide individual variation and is also impacted by individual ability to tolerate background noise, which is found to be independent of level of hearing loss. This is referred to as Acceptable Noise Levels Ratios (ANL) and these will vary amongst users of hearing aids (Nabelek, 2006). Hence it is not possible to establish a common benchmark and say that there is an absolute level of background noise beyond which speech perception cannot occur. This tolerance level is thought to be differentially distributed, however, with recent surveys indicating that approximately one third of high ANL users are able to hear satisfactorily in noisy places, another third only use the hearing aid on a part time basis and are functionally hearing impaired for the rest of the time. Another third try the device but reject it. Users who can tolerate 50db of background noise are classified as high ANL users (Ross, N.D.). Thus the noise standards proposed for the Kapiti Expressway indicate that approximately two thirds of those residents who are hearing impaired and living nearby are likely to have difficulty understanding speech and may be unable to use their hearing aid.

For elderly residents who are not hearing impaired, negative impacts may still occur. Nijland et al (2003) note that adverse health impacts can occur at levels as low as 40 db, particularly in children and elderly residents. In discussing the costs and benefits of reducing road noise, reducing traffic volumes or enlarging the distance between source and recipient are seen as two of the most effective noise abatement measures. During the planning stage of transportation, the authors note that enlarging the distance between source and recipient (that is, creating a greater separation between road and residential dwellings) is a feasible option.

Improving public transport services in the region is also an effective means of reducing traffic volumes.

Exposure to Loud Noise

Hearing loss can occur with exposure to loud noise. Noise over 100 db is considered dangerous if this exceeds 15 minutes in duration. Long term exposure to noise over 80 db can also cause hearing loss (American Tinnitus Association, 2012). It is not usually practical for most people to measure the decibel level of noises they regularly encounter, however a good rule of thumb is that if it is necessary to raise ones voice in order to be heard by another person within 1-3 meters, then that noise level should be regarded as hazardous. (Kim, 2011). Commonly experienced noises may vary in sound intensity. However, approximate levels of everyday sounds include the following:

	db
Normal conversation	20-50
Refrigerator humming	40
Loud conversation/shouting	50-65
Piano Practice	60-70
Vacuum Cleaner	75-80
Hair dryer	80
City Traffic	80
Rubbish removal truck	100

Source: http://www.hearingaidknow.com/2007/03/07/how-loud-is-too-loud-decibel-levels-of-common-sounds/http://www.hearingaidknow.com/2011/09/18/me-hearing-aids/ www.newton.dep.anl.gov/askasci/eng99

126. Paradoxically, people with hearing loss frequently perceive many loud sounds as louder than those with normal hearing. This is because remaining neurons can compensate for the damaged nerves by lower threshold levels, enabling, for example, some neurons to respond to higher frequencies than they would normally do (http://www.hearingaidknow.com/2011/06/21/hearing-loss-loud-comfort/) Tinnitus typically occurs following exposure to loud noise, with delayed onset. Sufferers are advised to take care of their hearing and avoid all exposure to further loud noise. Sound therapy is one method of treatment which involves masking, with low level sound, the ringing in the ears associated with the condition This can be a whirring fan, background music or other device. (American Tinnitus Association 2012)

Low Frequency Noise and Vibration

Long term exposure to low frequency noise below 500 Hz, usually experienced as vibration, can produce vibroacoustic disease. Clinical symptoms of this disease include behavioral changes such as irritability and depression, disturbance of vision, audition and balance, epileptic seizures and neurological damage or transient ischemic attacks (TIA). This disorder frequently shows increased blood pressure and arterial hypertension alongside cognitive effects (Kujala & Brattico 2009). The disease can also produce a range of symptoms that target the respiratory tract. Collagen disruption and degeneration of lung tissue is commonly seen and lung tumors are also thought to be associated with vibroacoustic

disease. Thickened blood and lymphatic vessels are also consistent with this condition (Ferreira et al 2006).

The WHO has recognized that general assessments of disturbance from low frequency noise experienced as vibration are deficient (Waye, 2011). Pereira, Nuno and Branco (2007) note that valid dose-response relationships have been difficult to identify and large scale epidemiological studies are still needed. Thus, this is an emerging area of research. It is thought however, that individuals who are sensitive to noise or electromagnetic radiation may have a low threshold for contracting the disease.

Vibrations from road traffic are mainly caused by road unevenness, irregularities that are intrinsic features of the road landscape (Lak, Degrande & Lombaert 2011). The unstable nature of the geology of the Wellington area will make it more likely that road unevenness will occur, resulting in traffic-induced vibrations that will cause discomfort to residents, are capable of disturbing sleep and causing physiological stress reactions.

Appendix B: Wellington Inner City Bypass

A Brief Case Study

This case study examines the assumptions used in support of the Wellington Inner City Bypass project and the arguments put forward, at the time planning permission was sought, to enable the project to commence. Follow up data will then be presented to establish whether the initial assumptions were correct and the overall outcome of the project will be examined along with its relevance to the proposed Kapiti expressway.

Background

Construction on the Wellington Inner city bypass commenced in January 2005. The new northbound route was open to traffic in February 2007, with the southbound route (Vivian Street) and Ghuznee St also reconfigured in 2007. The cost of the bypass was $39.9 million.

At the time of application for construction, the bypass was thought to offer significant benefits to traffic movement in the southern part of the city. As NZTA noted in its project information:

> *"The Bypass will relieve traffic congestion. Traffic heading for the airport, the hospital and to the southern and eastern suburbs will be separated from traffic heading into the Central Business District. Motorists will spend less time queuing, decrease their overall travel distance, and save time moving across the city. Because the current route is congested and traffic is stop/start, noise and exhaust emissions are very high. The Bypass will allow traffic to flow more smoothly, reducing congestion and exhaust emissions in the area. As a result, studies show that overall, pollution will reduce as a result of the Bypass being built."*

(source: NZTA: available online: http://www.nzta.govt.nz/projects/wicb/environment/wellington.html#Environmental)

The project however was not without its opposition. It was planned to be built through the middle of residential areas and areas with high cultural significance. Community groups were opposed to the project given the severance of the Te Aro community and the damage caused to historical buildings in the area. As one engineering source commented:

> *"The bypass has taken far longer to build than it probably should have. The initial vision seems to have been rather over-designed, and the opposition the project engendered was probably self-inflicted. If there are any lessons engineers can draw from the saga of the Inner City Bypass, they are that projections into the distant future are almost always unreliable, and that designing a project a community wants, the way it wants it, is ultimately the only way in the current era to get them built at all."*
> (source: *htttp://www.techlink.org.nz/Case-studies/Technological-practice/Materials/Print-PDFs/techlink-tp-wgtn-bypass.pdf).*

In terms of the current proposal for the Kapiti Expressway therefore, there are a number of similarities between the two projects. Both are contentious, with community groups opposed to the project and its proposed route, on the grounds of community impacts and destruction of housing. Both projects are described as allowing traffic to flow more freely, reducing congestion and producing an overall benefit in terms of air quality. The value of a comparison lies in the fact that the Kapiti expressway has not yet been built, whereas the inner city bypass has been operating for 5 years.

Monitoring

The Regional Air Quality Management Plan (RAQMP) has been in operation since 2000 with the purpose of implementation of an ambient air quality monitoring plan sufficient to provide appropriate information on which to base future air quality management decisions. The program seeks to provide scientifically sound and relevant information to manage the region's air quality resource; protect human health; fulfill the statutory requirements of the

RMA 1991; and determine compliance with the national environmental standards for air quality (NES-AQ).

Greater Wellington council which administers the plan acknowledges that the adverse health effects associated with exposure to PM2.5 are widely recognised and thought to be more serious than those associated with PM10. Hence the WHO, US Environmental protection agency and the European Union have all adopted standards for PM2.5 which is now becoming the air quality indicator of choice due to its widespread use in epidemiological studies and the recommendations of the WHO. However monitoring of PM10 only is used in Wellington at this point. (Mitchell, 2012).

Traffic flows in the southern part of Wellington city show high levels of traffic pollutants generally. As Table 1 below illustrates, the area around the inner city bypass has recorded high levels of nitrogen dioxide. This traffic pollutant has been identified as having a role in the development of childhood asthma, as discussed in the literature review of the health impact assessment.

Table 1: The top five annual average concentrations of nitrogen dioxide (ug/m3) by passive monitoring in the Wellington region (2010)

Monitoring site	Site classification	Distance to road	Annual concentration
Riddiford St/Hall St, Newtown, Wellington	Local road	1 m	40.3 µg/m³
Rugby Street, Basin Reserve, Wellington	SH	1.5 m	35.5 µg/m³
Knights Road, Bloomfield Tce, Lower Hutt	Local road	1 m	30.5 µg/m³
Wellington central (Greater Wellington site)	SH	5 m	27.5 µg/m³
Western Hutt Rd / Manor Park Rd	SH	1 m	26.1 µg/m³

(Source: NZTA)

While some canyoning effects may be operating in the Riddiford Street area, the basin reserve is a more open landscape, with more opportunity for pollutants to disperse. Hence the build up of traffic pollutants in the inner city bypass area cannot be simply ascribed to canyoning effects.

Greater Wellington City council has taken measurements of the pollution levels prior to and following construction of the inner city bypass as part of the Regional Air Quality Management Plan. The pollution roses in figure 1 illustrate the levels of pollution present in the inner city bypass area prior to and following construction of the bypass.

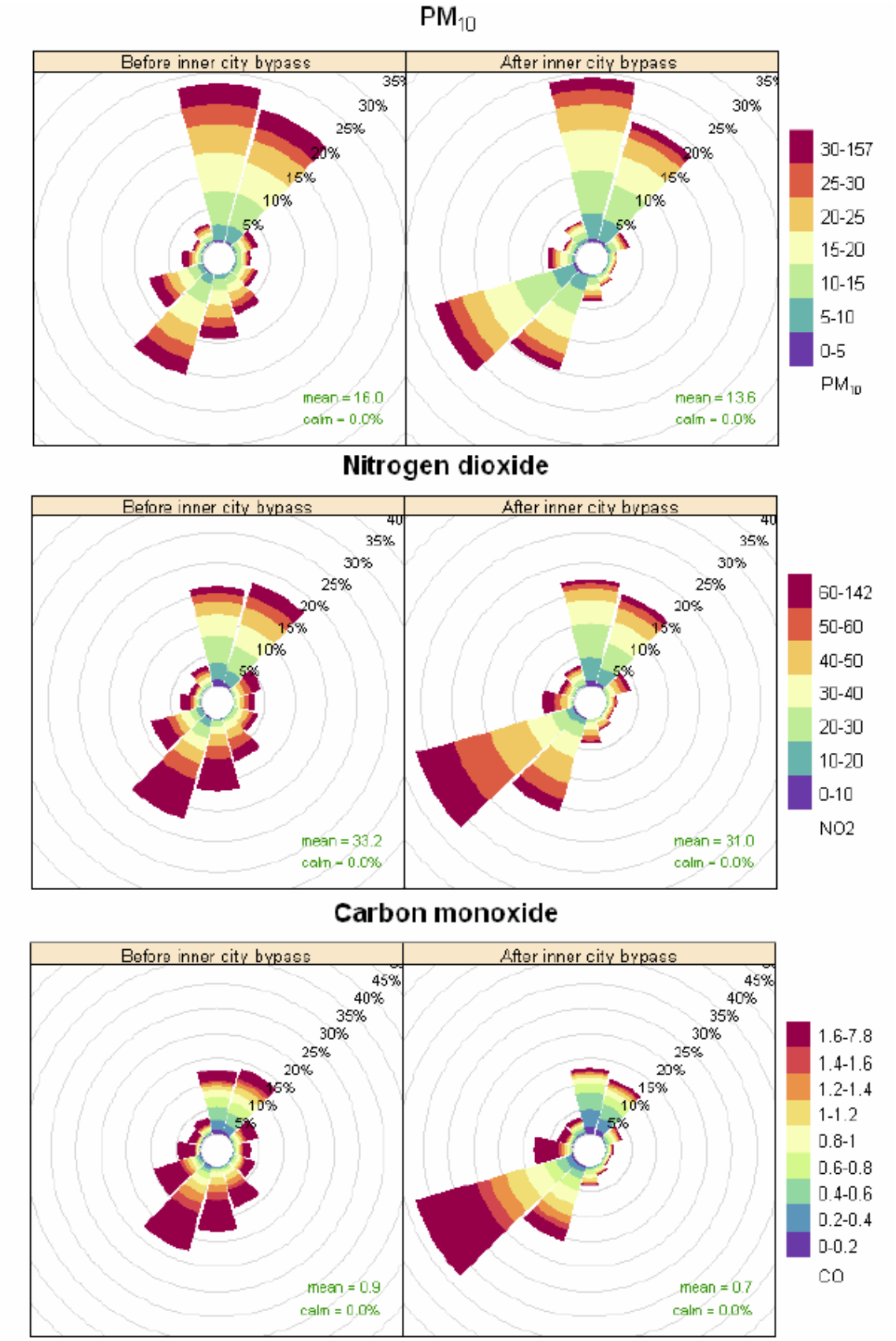


Figure 1. Pollution rose for one hour average of PM10 (ug/m3), nitrogen dioxide and carbon monoxide (all ug/m3) concentrations measured at Wellington central showing the

contribution to annual mean concentration by wind direction before (pre-2007) and after completion of the inner city bypass (post-2007). (Source: Mitchell, 2012).
While carbon monoxide and nitrogen dioxide levels have declined markedly at the Wellington central monitoring site between 2004 and 2007 this trend has not been observed with inner city bypass air quality levels. While there have been reduced traffic flows in Wellington city generally, attributed to increases in fuel prices, pollution concentrations in the inner city bypass area have increased. A greater concentration of levels has been recorded under light southwest conditions, indicating that the Vivian and Victoria street intersection approaching the bypass may now be more congested, with higher traffic flows recorded (Mitchell, 2012).

It appears, then, that the inner city bypass area, rather than reducing traffic congestion and improving air quality has become more congested with markedly poorer air quality in this area. As the bypass borders the residential area of the Aro valley, this is a cause of concern because of the potential health effects. The air quality data indicates that the assumptions of underlying benefits in construction of the inner city bypass have not been validated. The NZ Transport agency stated that this project would reduce congestion and improve air quality by improving traffic flow. They argued that " *studies show that overall, pollution will reduce as a result of the Bypass being built.".* This has not eventuated and the area has experienced significant increases in air pollution against improved air quality in most other parts of the central city.

Whether the increased pollution is the result of induced traffic or a redistribution of traffic flows is outside the scope of this brief report. However, it does establish that the project did not achieve its stated aim to improve traffic flow and air quality. This outcome has implications for the Kapiti expressway. The NZTA has once again argued that there will be a benefit from improved traffic flow on the proposed road, with less stop-start traffic and improved air quality, while at the same time acknowledging that there will be increased local congestion. (Traffic Modeling report, technical report 34, vol 3; aee-s-Air Quality report, p8) In the light of the results achieved with the inner city bypass, it is difficult to see how these benefits might occur. The NZTA's assumptions in this regard are not borne out by the evidence from the inner city bypass which has many parallels with the Kapiti project. The conclusions of techlink quoted on page 1, that such projections are frequently unreliable, is pertinent to this project as is their observation that community approval is necessary in the current climate.

In conclusion, the results of air quality monitoring of the inner city bypass indicate that the claimed benefits of better traffic flow and improved air quality for the Kapiti expressway are of dubious validity. Increased local congestion, greater emissions and more stop-start traffic is likely.

Marie O'Sullivan

6 November 2012

References and Bibliography:

Amato, F., Pandolfi, M., Viana, M., Querol, X., Alastuey, A & Moreno, T. (2009). Spatial and chemical patterns of PM10 in road dust deposited in urban environment. *Atmospheric Environment*, 43, 1650-1659.

American Tinnitus Association, (2012) http://www.ata.org/for-patients/how-loud-too-loud

Ameratunga, S., Hijar, M., & Norton, R. (2006). Road traffic injuries: confronting disparities to address a global health problem. A review. *The Lancet*, 367, 9521, 6-12, 1533-1540.

An Investigation of the Health Implications of the Wellington Northern Corridor Development: A Health Impact Assessment March 2011. Fourth year student project. Available online: http://www.otago.ac.nz/wellington/otago025414.pdf

Annesi-Maesano, I., Moreau, D, Caillaud, D., Lavaud, F., Le Moullec, Y., Taytard, A., Pauli, G., Charpin, D. (2007). Residential proximity fine particles related to allergic sensitization and asthma in primary school children. *Environmental Research*, 109, 8, 1004-1011.

Babish W. (2006). Transportation noise and cardiovascular risk review and synthesis of epidemiological studies, dose-effect curve and risk estimation. Berlin: *Umweltbundesamt;* Available from: http://www.umweltbundesamt.de

Bach, J., Anemüller, J., & Kollmeier B. (2011). Robust speech detection in real acoustic backgrounds with perceptually motivated features. *Speech Communication*, 53, 5, 690-706.

Baker, M., Keall, M., Lyn, Au E., Howden-Chapman, P., (2007). Letter to the editor: Home is where the heart is – most of the time. *New Zealand Medical Journal,* 120,1264.

Baldauf, R., Thoma, E., Khlystov, A., Isakov, V., Bowker, G., Long, T., & Snow, R., (2008). Impacts of noise barriers on near-road air quality. *Atmospheric Environment,* 42, (32), 7502-7507.

Bentayels, M., Helmer, C., Raherison, C., Dartigues, J.F., Tessier, J.F., & Annesi-Maesano I. (2010). Bronchitis-like symptoms and proximity to air pollution in French elderly. *Respiratory Medicine*, 104, 6, June 2010, 880-888

Bowker, G.E., Baldauf, R., Isakov, V., Khlystov, A., & Petersen, W., (2007). The effects of roadside structures on the transport and dispersion of ultrafine particles from highways. *Atmospheric Environment,* 41 (37), 8128e8139.

Brauer M, Avila-Casado C, Fortoul TI, Vedal S, Stevens B, Churg A.(2001). Air pollution and retained particles in the lung. *Environmental Health Perspectives* 109:1039–1043.

Briggs, D., de Hoogh, K., Morris, C., & Gulliver, J. (2008). Effects of travel mode on exposures to particulate air pollution. *Environment International*, 34, 1, 12-22.

British Medical Association, (BMA) July 2012. Press Release: Government urged to put health at heart of transport policies. Available online: http://bma.org.uk/news-views-analysis/news/2012/july/government-urged-to-put-health-at-heart-of-transport-policies

Brook, R., Rajagopalan, S., Pope C,Brook, J., Bhatnagar, A., Diez-Roux, A., Holguin, F., Hong, Y., Luepker, R., Mittleman, M., Peters, A., Siscovick, D., Smith, S., Whitsel L & Kaufman, J. (2010). Particulate matter air pollution and cardiovascular disease : An update to the scientific statement from the American Heart Association *Circulation,* 121:2331-2378.

Brown, C., & Bacon S. (2010). Fundamental frequency and speech intelligibility in background noise. *Hearing Research*, 266, 52-59.

Brown, S., & Cotton, A. (2003). Risk mitigating beliefs, risk estimates and self reported speeding in a sample of Australian drivers. *Journal of Safety Research*, 34, 183-188.

Brown, S. & Gould, R. (2012). A prospective study of relationships between propositions about risk and driver speeding. *Accident Analysis and Prevention*, 46, 1-7.

Brunekreef B, Janssen NA, de Hartog J, Harssema H, Knape M, van Vliet P (1997). Air pollution from truck traffic and lung function in children living near motorways. *Epidemiology* 1997, 8:298-303.

Boyle, P. (1997). Review. Cancer, cigarette smoking and premature death in Europe: a review including the Recommendations of European Cancer Experts Consensus Meeting, Helsinki, October 1996. *Lung Cancer*, 17, 1-60.

Bowling, A., & Stafford, M. (2007). How do objective and subjective assessments of neighbourhood influence social and physical functioning in older age? Findings from a British survey of ageing. *Social Science & Medicine*, 64, 12, 2533-2549.

Brugge D, Durant JL, Rioux C. (2006). Near-highway pollutants in motor vehicle exhaust: A review of epidemiologic evidence of cardiac and pulmonary health risks. *Environmental Health*. 6.

Calderón-Garcidueñas, L.,. D'Angiulli, A.,. Kulesza, R. Torres-Jardón, R. Osnaya, N., L. Romero L. (2011). Air pollution is associated with brainstem auditory nuclei pathology and delayed brainstem auditory evoked potentials. *International Journal of Developmental Neuroscience*, 29, 4, 365-375.

Calderón-Garcidueñas, L., A. Solt, A., & M. Franco-Lira M. (2008). Long-term air pollution exposure is associated with neuroinflammation, an altered innate immune response, disruption of the blood–brain-barrier, ultrafine particle deposition, and accumulation of amyloid beta 42 and alpha synuclein in children and young adults. *Toxicology Pathology*., 36, pp. 289–310

Chang, J., Pien, G., Duntley, S, & Macones, G. (2010) Sleep deprivation during pregnancy and maternal and fetal outcomes: Is there a relationship? *Sleep Med Rev*. 2010 April ; 14(2): 107–114. doi:10.1016/j.smrv.2009.05.00

Chen, Z., & Hong, L., (2004). An integrative methodology for environmental management in construction. *Automation in Construction*, 13, 621-628.

Cox, E. (1995). *A truly civil society*. ABC: Boyer Lectures.

Currie J., Neidell,M., & Schmieder, J. (2009). Air pollution and infant health, lessons from New Jersey. *Journal of Health Economics*, 28, 688-703.

Ciccone G, Forastiere F, Agabiti N, Biggeri A, Bisanti L, Chellini E, (1998). Road traffic and adverse respiratory effects in children. SIDRIA Collaborative Group. *Occupational and Environmental Medicine*. 55(11):771-8.

Delhomme, P., Verlhiac J.F., Cécile Martha C. (2009). Are drivers' comparative risk judgments about speeding traffic realistic? *Safety Research*, 40, 333-339.

de Medeiros APP, Gouveia N, Machado RPP, de Souza MR, Alencar GP, & Novaes HMD, (2009). Traffic-related air pollution and perinatal mortality: a case-control study.(Children's Health)(Report). *Environmental Health Perspectives*. 2009;117(1):127(6).

Dravitzki V, Walton D, & Kvatch I, (eds) (2006). The Effects and Significance of New Zealand Road Surfaces on Traffic Noise. Acoustics, Christchurch, New Zealand. Available on line http://www.acoustics.asn.au/conference_proceedings/AASNZ2006/papers/p124.pdf

Dockery DW, Pope CA, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG, & Speizer FE (1993) An association between air pollution and mortality in six U.S. cities. *New England Journal of Medicine* 1993, 329:1753-9.

Dora, C., & Phillips, M. (eds)., (2000). *Transport, Environment & Health*. WHO European publications. European series no. 89. WHO, Austria.

Dora, C. & Racioppi F., (2003). Including health in transport policy agendas: the role of health impact assessment analyses and procedures in the European experience. *Bulletin of the World Health Organization*; 81:399-403.

Durie M.(2004). Understanding health and illness: research at the interface between science and indigenous knowledge. *International Journal of Epidemiology*. 33(5):1138-43.

Edwards J, Walters S, & Griffith R.K. (1994). Hospital admissions for asthma in preschool children: relationship to major roads in Birmingham, United Kingdom. Archives of *Environmental Health*, 49, 223-27.

Environmental Protection Agency Web site (2006). Air trends basic information. Available from: *www.epa.gov/air/airtrends/sixpoll.html*

Environmental Protection Agency Web site. Epidemiology of human health effects associated with ambient particulate matter.In: *Air quality criteria for particulate matter, vol. 2.* Available from: *http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid_87903*

Evans, G., Hygge S., & Bullinger, M. (1995). Chronic Noise and Psychological Stress. *Psychological Science* 6, 6, 333-338.

Evans G., Bullinger, M. & Hygge, S. (1998). Chronic Noise Exposure and Physiological Response: A Prospective Study of Children Living Under Environmental Stress. *Psychological Science*, 9, 1, 75-77.

Farhat, S., Silva, C., Orione, M., Campos, L., Sallum, A., & Braga, A. (2011) Air pollution in autoimmune rheumatic diseases: A review. *Autoimmunity Reviews*, 11, 1, 14-21.

Ferreira, J., Monteiro, M., Tavares, F., Serrano, I., Monteiro, E., Mendes, C., Alves-Pereira, M., Nuno A.A. & Branco C (2006). Involvement of central airways in vibroacoustic disease patients. *Revista Portuguesa de Pneumologia (English Edition*), 12, 2, 93-105.

Fowkes, F., Housley, E., Cawood, E., Macintyre, C., Ruckly, C & Prescott, R. (1991). Edinburgh Artery Study: Prevalence of asymptomatic and symptomatic peripheral arterial disease in the general population. *International Journal of Epidemiology*, 20, 2, 384-392.

Freedman, V., Grafova, I., Schoen, R., & Rogowski, J. (2008). Neighbourhoods and disability in later life. *Social Science & Medicine*, 66, 11, 2253-2267.

Fyhri, A. & Aasvang, G. (2010). Noise, sleep and poor health: Modeling the relationship between road traffic noise and cardiovascular problems. *Science of the Total Environment*, 408, 21, 4935-4942.

Gander, P., Graeber, R. & Belenky G. (2011). *Principles and Practice of Sleep Medicine* (Fifth Edition). Chapter 68 – Fatigue Risk Management. 760-768.

Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D, & Peters J (2005). The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age. *New England Journal of Medicine, 351,* 1057-1067.

Gaudeman, W., Avol, E., Lurmann, F., Kuenzli, N., Gilliland F., Peters, J., & McConnell R. (2005). Childhood asthma and exposure to traffic and nitrogen dioxide. *Epidemiology,* 16, 737-743.

Gauderman WJ, Vora H, McConnell R, Berhane K, Gilliland F, & Thomas D, (2007). Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study. *The Lancet*, 369, 571-7.

Gauderman WJ, McConnell , Gilliland F, London S, Thomas D, Avol E, Vora H, Berhane K, Rappaport EB, Lurmann F, Margolis HG, & Peters, J. (2000). Association between air pollution and lung function growth in Southern California Children. *American Journal of Respiratory*
and Critical Care Medicine, 162(4 Pt 1):1383-1390.

Gee, G.C., & Takeuchi, D., (2004). Traffic stress, vehicular burden and well-being: A multilevel analysis *Social Science & Medicine,* 59, 2, 405-414.

Ghosh, R., Joad, J., Benes, I, Dostal, M Sram, R., & Picciotto, I (2005). Ambient nitrogen oxides exposure and early childhood respiratory illnesses. *Journal of Allergy and Clinical Immunology,* 116, 2, 279-284.

González, V., &. Echaveguren T.(2012) Exploring the environmental modeling of road construction operations using discrete-event simulation. *Automation in Construction*, 24,

100–110.

Gruzieva, O., Tom Bellander, T., Eneroth, K., Kull, I., Mel_E., Nordling, E., van Hage, M., Wickman, M., Moskalenko, V. , Hulchiy, V, & Pershagen, G., (2012). Traffic-related air pollution and development of allergic sensitization in children during the first 8 years of life Traffic-related air pollution and development of allergic sensitization in children during the first 8 years of life. *Journal of Allergy & Clinical Immunology*, 129, 240-6.

Glymour, M., Mujahid, M., Wu, Q., White, K., & Tchetgen, E. (2010). Neighbourhood disadvantage, and self assessed health, disability and depressive symptoms. Longitudinal results from the health and retirement study. *Annals of Epidemiology*, 20, 11, 856-60.

Hackley B & Dixon J, (2007). Air pollution: Impact on maternal and perinatal health . *Journal of Midwifery & Womans Health.* Available online: www.jmwh.org 4351526-9523/07. doi:10.1016/j.jmwh.2007.03.020

Haglund, M., & Aberg, L (2000). Speed choice in relation to speed limit and influences from other drivers. *Transportation research*, part F, 3, 39-51.

Hanworth, N., & Rechnitzer, G. (1993). Description of fatal crashes involving various causal variables. Canberra, Australia: Federal Office of Road Safety.

Hawe, P., & Shiell, A., (2000). Social capital and health promotion: A review. *Social Science & Medicine*, 51, 6, 871-885.

Heist, D.K., Perry, S.G., & Brixey, L.A., (2009). A wind tunnel study of the effect of roadway configurations on the dispersion of traffic-related pollution. *Atmospheric Environment* 43 (32), 5101-5111.

Hitchins J., Mrawska, L., Wolff, R., Gilbert, D, Greenaway C., Rijnders E. (2000). A study of the horizontal and vertical profile of submicrometer particulates in relation to a busy road. *Atmospheric Environment* , 33, 1261-1274.

Hoek, G., Boogaard, H., Knol, A., Dehartoc, J., Slottje, P., Ayres, J., Borm, P., Brunekreef, B., Donaldson, K., Forastiere, F., Holgate, S., Kreyling, W., Emery, B., Kanen, J., Stone, V., Wichmann, E. & Vandersluijs, A. (2010). Concentration response functions for ultrafine particles and all cause mortality and hospital admissions. Results of a European expert panel elicitation. *Environmental Science & Technology*, 44, 476-482.

Hoek, G., Brunekreef, B., Goldbohm, S., Fischer, P., van den Brandt, P. (2002). Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. *The Lancet*, 360, 1203-09. available online: http://image.thelancet.com/extras/01art7366web.pdf

Hooda, P.S., Miller, A., Edwards, A.C., 2007. The distribution of automobile catalysts-cast platinum, palladium and rhodium in soils adjacent to roads and their uptake by grass. *Science of the Total Environment* 384, 384–392,

Hruba F, Fabianova E, Koppova K, Vandenberg JJ. (2001). Childhood respiratory symptoms, hospital admissions, and long-term exposure to airborne particulate matter. J Expo

Anal Environ Epidemiol. [Research Support, Non-U.S. Gov't Research Support, U.S. Gov't, Non-P.H.S.]. 11(1):33-40.

Hygge S. (2003). Classroom experiments on the effects of different noise sources and sound levels on long-term recall and recognition in children. *Applied Cognitive Psychology*. 17(8) 895-914.

Jacobsen, P. (2003). Safety in numbers: more walkers and bicyclists, safer walking and bicycling. *Injury Prevention*, 9(3): 205-9.

Jagger, C., Matthews, R., Matthews, F., Spiers, N., Nickson, J., & Paykel E., (2007). Cohort differences in disease and disability in the young-old: findings from the MRC Cognitive Function and Ageing Study (MRC-CFAS). *BMC Public Health*, 7 p. 156.

Janssen N.A.H, Brunekreef B, van Vliet P, Aarts F, Meliefste K, Harssema H, (2003). The relationship between air pollution from heavy traffic and allergic sensitization, bronchial hyperresponsiveness, and respiratory symptoms in Dutch schoolchildren. *Environmental Health Perspectives*. 111(12):1512-8.

Jarup L, Babisch W, Houthuijs D, (2008). Hypertension and exposure to noise near airports – the Hyena study. *Environmental Health Perspectives*. 2008; 116:329 –333.

Jephcote, C., & Chen, H. (2011). Environmental injustices of children's exposure to air pollution from road transport within the model British multicultural city of Leicester: 2000-09. *Science of the Total Environment*: in press, Sci Total Environ (2011), doi:10.1016/j.scitotenv.2011.11.040.

Jerrett, M., Finkelstein, M., Brook, J., Arain, M., Kanaroglou, P., Stieb, D., Gilbert, N., Verma, D., Finkelstein, N., Chapman, K., & Sears, M. (2009). A Cohort Study of Traffic-Related Air Pollution and Mortality in Toronto, Ontario, Canada. *Environmental Health Perspectives*, 117(5): 772–777.

Kaaresen, P., Ronning, J., Tunby, J., Nordhov, S., Ulvund, S., Dahl, L., (2008). A randomized controlled trial of an early intervention program in low birth weigh children: Outcome at 2 years. *Early Human Development*, 84, 3, 201-209.

Kalat, J (2008*). Biological Psychology*. Thompson UK.

Kalavrouziotis, I.K., Carter, J., Varnavas, S.P., Mehra, A., Drakatos, P.A.,(2006). Towards an understanding of metal contamination in food crops and soils related to road traffic. *Fresenius Environmental Bulletin* 15, 170–175.

Kapiti Observer, (27/02/2012). *Where's the Work*. Kapiti unemployment figures. Available online: http://www.stuff.co.nz/dominion-post/news/local-papers/kapiti-observer/6483161/Wheres-the-work

Kapiti Coast District Council *(KCDC) Planning Requirements (2005).* 4.9.14 KCDC Subdivisions and Development Principles and Requirements (2005). KCDC Streetscape Strategy and Guideline (2008)

Kaur, S., Nieuwenhuijsen, M., Colvile, R., (2005). Pedestrian exposure to air pollution along a major road in Central London, UK. Atmospheric Environment 39, 7307–7320.

KC News (2012) *Logic for road diminishing*. Available online: http://www.kcnews.co.nz/story.php?storyID=6271

Keuken, M., Roemer, M. & Elshout, S. (2009). Trend analysis of urban NO2 concentrations and the importance of direct NO2 emissions versus ozone/NOx equilibrium. *Atmospheric Environment,* 432, 31, 4780-4783.

Killgore, W., Kahn_Greene, E., Lipizzi, E., Newman, R., Kamimori, G., & Balkin, T., (2008) Sleep deprivation reduces perceived emotional intelligence and constructive thinking skills. *Sleep Medicine*, 9, 5, 517-526.

Kim, B., 2012. http://drbenkim.com/articles-decibel.html Updated on October 17,2011.

Kim JJ, Smorodinsky S, Lipsett M, Singer BC, Hodgson AT, & Ostro B (2004). Traffic-related air pollution near busy roads: The East Bay children's respiratory health study. *American Journal of Respiratory and Critical Care Medicine* 2004, 170:520-526.

Kingham,S., Pearce, J., Zawar-Reza, P. (2007). Driven to injustice? Environmental justice and vehicle pollution in Christchurch, New Zealand. *Transportation Research*, Part D, 12 254–263

Kjellstrom T, Hill S. (2002) *New Zealand Evidence for Health Impacts of Transport*. Public Health Advisory Committee, 2002, Wellington NZ.

Kobayashi, R., Okamoto, R.A., Maddalena, R.L., Kado, N.Y., 2008. Polycyclic aromatic hydrocarbons in edible grain: a pilot study of agricultural crops as a human exposure pathway for environmental contaminants using wheat as a model crop. *Environmental Research* 107, 145–151.

Kousoulidou, M., Ntziachristos, L., Mellios, G., & Samaras, Z., (2008). Road-transport emission projections to 2020 in European urban environments. *Atmospheric Environment* 43, 7465e7475.

Kozou, H., Kujala, T., Shtyrov, Y., Toppila, E., Starck, J., Alku, P., & Naatanen, R. (2005). The effect of different noise types on the speech and non-speech elicited mismatch negativity. *Hearing Research*, 199, 1-2, 31-39.

Kujala, I., & Brattico, E, (2000). Detrimental noise effects on brains speech functions. *Biological Psychology*, 61,3, 135, 143.

Kunzli N, Jerrett M, Mack WJ, Beckerman B, LaBree L, Gilliland F, Thomas D, Peters J, & Hodis HN (2005). Ambient air pollution and Atherosclerosis in Los Angeles. *Environmental Health Perspectives*, 113:201-206.

Lak, M., Degrande, G. & Lombaert, G. (2011). The effect of road unevenness on the dynamic vehicle response and ground-borne vibrations due to road traffic. *Soil Dynamics and Earthquake Engineering*, 31, 10, 1357-1377.

Lawson, S., Galbally, I, Powell, J., Keywood, M., Molloy, S., Cheng, M & Selleck, P. (2011). The effect of proximity to major roads on indoor air quality in typical Australian dwellings. *Atmospheric Environment*, 45, 2252-2259.

Leake, J., Adam-Bradford, A., Rigby, J.E., 2009. Health benefits of 'grow your own' food in urban areas: implications for contaminated land risk assessment and risk management. *Environmental Health* 8, S6. doi:10.1186/1476-069X-8-S1-S6.

Lee B, Ha E, Park H, Kim Y, Hong Y, & Kim H, (2003). Exposure to air pollution during different gestational phases contributes to risks of low birth weight. *Human Reproduction,*18:638–43.

Lercher, P., Evans, G. W., Meis, M., & Kofler, W. W. (2002). Ambient neighborhood noise and children's mental health. Occupational and *Environmental Medicine,* 59, 380–386.

Lin M, Stieb DM, & Chen Y. (2005). Coarse particulate matter and hospitalization for respiratory infections in children younger than 15 years in Toronto: a case-crossover analysis. *Pediatrics*. 116(2):e235-40.

Lipfert, F.W., Wyzqa, R., Baty, J., & Miller, J. (2003). Traffic density as a surrogate measure of environmental exposures in studies of air pollution health effects: Long-term mortality in a cohort of US veterans *Transport Policy*, 10, 2, 131-140.

Litman (2011).*Win-Win Emission Reduction Strategies* Victoria Transport Policy Institute. Available online: www.vtpi.org

Lyytimaki, J., Tapio, P., & Assmuth, T. (2012). Unawareness in environmental protection: The case of light pollution from traffic. *Land Use Policy*, 29, 3, 598-604.

Mariana Alves-Pereira, M., Nuno A.A. & Branco C. (2007). Vibroacoustic disease Biological effects of infrasound and low-frequency noise explained by mechanotransduction cellular signaling. Review. *Progress in Biophysics and Molecular Biology*, 93, 1–3, 256-279

Mata, M., Georgy, M & Ibrahim, M. (2008). Sustainable construction management : introduction of the operational context space(OCS). *Construction Management & Economics*, 26, 3, 261-275.

Maxwell, L., & Evans, G. (2000). The effects of noise on pre-school children's pre-reading skills. *Journal of Environmental Psychology* 20, 1, 91-97.

McAdam, K., Steer, P., & Perrotta, K. (2011). Using continuous sampling to examine the distribution of traffic related air pollution in proximity to a major road. *Atmospheric Environment*, 45, 12, 2080-2086.

McConnell R, Berhane K, Yao L, Jerrett M, Lurmann F, Gilliland F, Kunzli N, Gauderman J, Avol E, Thomas D, & Peters J (2006). Traffic susceptibility and childhood asthma. *Environmental Health Perspectives,* 114:766-772.

Meerlo, P., Sgoifi, A., & Suchecki, (2008) Physiological Review: Restricted and disrupted sleep: Effects on autonomic function, neuroendocrine stress systems and stress responsivity. *Sleep Medicine Reviews*, 12, 3, 197-210.

Mehrabian, S., Duron, E., Labouree, F., Rollot, F., Bune, A., Traykov, L., & Hanon O. (2010). Relationship between orthostatic hypotension and cognitive impairment in the elderly. *Journal of the Neurological Sciences*, 299, 1-2, 45-48.

Meiser-Stedman, R., Yule, W., Smith, P., Glucksman, E., & Dalgleish, T. (2005). Acute stress disorder and posttraumatic stress disorder in children and adolescents involved in assaults or motor vehicle accidents. *American Journal of Psychiatry,* 162:1381–1383.

Mercer BM, Merlino AA, Milluzzi CJ, (2008). Small fetal size before 20 weeks' gestation: associations with maternal tobacco use, early preterm birth, and low birth weight. *American Journal of Obstetrics and Gynecology* 198, :673.e1-673.e8.

Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, & Kaufman JD (2007). Long-term exposure to air pollution and incidence of cardiovascular events in women. *The New England Journal of Medicine* 2007, 356:447-458.

Ministry for the Environment. (2008) *Health and Air Pollution in New Zealand*. Available online http://www.mfe.govt.nz/issues/air/programme/hapinz.html.

Mitchell, T. (2012). Air quality in the Wellington region: State and trends. Greater Wellington Regional Council, Publication No. GW/EMI-T-12/137, Wellington. Available online: www.gw.govt.nz

Morrison, D., M. Petticrew, et al. (2003). What are the most effective ways of improving population health through transport interventions? Evidence from systematic reviews. *Journal of Epidemiology and Community Health* 57.

Ministry of Health, (2008). *A Portrait of Health. Key results of the 2006/07 New Zealand Health Survey*, Wellington, Ministry of Health. Available online: http://www.moph.govt.nz.

Ministry of Transport (MOT) (2006). *The Social Cost of Road Crashes and Injuries: June 2006* Update. Wellington, Ministry of Transport.

MSD (2005). *The Social Report*. Wellington, Ministry of Social Development.

Muzet, A. (2007). Environmental noise, sleep and health. Review. *Sleep Medicine Reviews*, 11, 2, 135-142.

Nabelek A, Freyaldenhoven M, Tampas J, (2006). Acceptable Noise Level as a predictor of hearing aid use. *Journal of the American Auditory Association,* 17 (9):626-639.

Nawrot, T., Perez, L., Kunzli, Munters & Nemery, (2011). Public health importance of triggers of myocardial infarction: A comparative risk assessment. *The Lancet*, 377, 732-40.

Nicolai T, Carr D, Weiland SK, Duhme H, von Ehrenstein O, Wagner C, & von Mutius E (2003). Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. *European Respiratory Journal,* 21:956-963.

Ning, Z., Hudda, N., Daher, N., Kam, W., Herner, J., Kozawa, K., Mara, S. & Sioutas C. (2010). Impact of roadside noise barriers on particle size distributions and pollutants concentrations near freeways. *Atmospheric Environment*, 44, 3118-3127.

Nijland, H.A., Van Kempen, E., Van Wee, G., & Jabben, J. (2003). Costs and benefits of noise abatement measures. *Transport Policy,* 10, 2, 131-140.

Nyberg, F., Gustavsson, P, Järup, L, Bellander, T, Berglind, N, Jakobsson, R, & Pershagen, G (2000). Urban Air Pollution and Lung Cancer in Stockholm. *Epidemiology*: 11, 5 , 487-495

NZTA. (2008) Regional Summary-Wellington. In: NZTA, editor. Wellington: New Zealand Transport Agency; 2008.

Oberdoister, G., Sharp, Z., Atudorei, V., Elder, R., Gelein, R., Kreyling, W., & Cox C. (2004). Translocation of superfine particles to the brain. *Inhaled Toxicology*, 16, 437-445.

O'Connor GT, Neas L, Vaughn B, Kattan M, Mitchell H, & Crain E.F., (2008) Acute respiratory health effects of air pollution on children with asthma in US inner cities. *The Journal of Allergy and Clinical Immunology*. 121(5):1133-9.e1.

O'Dea D. and Wren J. (2012). *New Zealand Estimates of the Total Social and Economic Cost of Injuries. For All Injuries, and the Six Priority Areas. For Each of Years 2007 to 2010. At June 2010 prices.* Report to New Zealand Injury Prevention Strategy. Wellington, New Zealand.

Öhrström,' E & Skanberg, A. (2004). Sleep disturbances from road traffic and ventilation noise—laboratory and field experiments. *Journal of Sound and Vibration*, 271, 1-2, 279-296.

Öhrström E., Emina Hadzibajramovic, E., Maria Holmes, M., & Helena Svensson, H (2006). Effects of road traffic noise on sleep: Studies on children and adults Journal of Environmental Psychology, 26, 2, 116-126.

Olsson, A.C., et al., (2010). Exposure to diesel motor exhaust and lung cancer risk in a pooled analysis from case-control studies in Europe and Canada. Am. J. Respiritory Critical Care Medicine. 183, 7, 941-948.

Patelarou E., Chochlidaki M., Vivilaki V., & Brokalaki H. (2009). Is There a Link between Wheezing in Early Childhood and Adverse Birth Outcomes? A Systematic Review. *International Journal of Environmental Research and Public Health*. 6(11):2752-2761.

Petit, C., Aubry, C., & Rémy-Hall, E., (2011). Agriculture and proximity to roads: How should farmers and retailers adapt? Examples from the Ile-de-France region. *Land Use Policy*, 28, 867-876.

Pirrera, S., De Valck, E., & Cluydts, R, (2012). Nocturnal road traffic noise: A review on its assessment and consequences on sleep and health. Review. *Environment International*, 39, 1, 96-102.

Pénard-Morand C, Charpin D, Raherison C, Kopferschmitt C, Caillaud D, Lavaud F, et al. Long-term exposure to background air pollution related to respiratory and allergic health in schoolchildren. *Clinical & Experimental Allergy*. 2005;35(10):1279-87.

Plomp, R., (1986). A signal-to-noise ratio model for the speech-reception threshold of the hearing impaired. Journal of speech and hearing research 29, 146–154.

Pope CA, Thun MJ, Namboodiri MM, Dockery DW, Evans JS, Speizer FE, & Hath CW (1995). Particulate air pollution as a predictor of mortality in a prospective study of US adults. *American Journal of Respiratory and Critical Care Medicine* 1995, 151:669-674.

Pope CA, Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, & Thurston GD (2002). Lung Cancer, Cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *Journal of the American Medical Association* 2002, 287:1132-1141.

Peters A (2005). Particulate matter and heart disease: Evidence from epidemiological studies. *Toxicology and Applied Pharmacology,* 477-482.

Public Health Advisory Committee (2007) (PHAC). *An idea whose time has come: New Opportunities for Health Impact Assessment.* National Health Committee, Ministry of Health, Wellington, New Zealand.

Quigley R, Cunningham R, Ward M, Boer M.D., & Conland C. (2006). The Greater Wellington Regional Land Transport Strategy Health Impact Assessment. Health Impact Assessment.

Radio NZ, (2012) http://www.radionz.co.nz/news/national/110674/big-roading-projects-'drain-transport-coffers'

Ranft, U., Schikowski, T., Sugiri, D., Krutmann, J., Kramer, U. (2009) Long term exposure to traffic related particulate matter impairs cognitive function in the elderly. *Environmental Research* 109, 8, 1004-1011.

Rosenlund M, Berglind N, & Hallqvist J, (2006). Long-term exposure to urban air pollution and myocardial infarction. *Epidemiology*. 2006;17:383–390.

Ross, M. (ND) *Acceptable Noise Levels* (ANL). Rehabilitation Engineering Research Centre on Hearing Enhancements. Available online: http://www.hearingresearch.org/ross/hearing_aid_use/acceptable_noise_levels.php

Ryan P. , Masters, G, Biagini, J., Bernstein, D., Grinshpun, S, Shukla, R., Wilson, K., Villareal, M., Burkle, J.,& Lockey, J. (2007). Is it traffic type, volume or distance? Wheezing in infants living near truck and bus traffic. *Respiratory Medicine*, 101, 8, 1721-1729.

Ross, Z., Kerbed, I., Loughery, J., Ito, K., Matte, T., Markowitz, S., & Eisl, H. (2011). Santos, G., Behrendt, H., Maconi, L, Shirvani, T & Teytelb, A. (2010) Road transport externalities, economic policies, and other instruments for sustainable road transport. *Research in Transportation Economics*, 28, 1, 2-45.

Schilling, J., Miller, R., Sachs, M., & Young, E., (1998). Frequency shaped amplification changes the neural representation of speech with noise-induced hearing loss. *Hearing Research*, 117, 1–2, 1998, 57-70

Simkhovich, B., Kleinman, M., Kloner, R., (2008) Air Pollution and cardiovascular injury Epidemiology, toxicology, and mechanisms. *Journal of the American College of Cardiologists*. 2008; 52:719–26.

Shield B, Greenland E, Dockrell J, & Rigby K. (2008). Children′s perceptions of speech and hearing in open plan and enclosed classrooms. *Proceedings of the Institute of Acoustics*, 30: 10-19.

Simpson, S., Mahoney, M., Harris, E., Aldrich, R., Stewart-Willians, J. (2005). Equity focused health impact assessment: A tool to assist policy makers in addressing health inequalities. *Environmental Impact Assessment Review*, 25, 772-782.

Skene, K., Gent, J., McKay, L, Belanger B, & Leaderer, T (2010) Ambient nitrogen dioxide levels in Connecticut. Atmospheric Environment, 44, 5156-64

Slezakova, K., Castro, D., Begonha, A., Delerue-Matos, C., da Conceicao Alvin-Ferra, M, Morais, S., & do Carmo Pereira, M. (2011). Air pollution from traffic emissions in Oporto, Portugal: Health and environmental implications. *Microchemical Journal*, 99, 1, 51-59.

Stuart, A., & Zeager, M. (2011). An inequality study of ambient nitrogen dioxide and traffic levels near elementary schools in the Tampa area. *Journal of Environmental Management*, 92, 1923-30.

Suglia, S., Gryparis, A., Wright, R.O., Schwartz., J., Wright, R.J. (2008). Association of black carbon with cognition among children in a prospective birth cohort study. *American Journal of Epidemiology*, 167, 280-286.

Sforza E, Chapotot F, Lavoie S, Roche F, Pigeau R, Buguet, A. Heart rate activation during spontaneous arousals from sleep: effect of sleep deprivation. *Clinical Neurophysiology* 2004; 115, 2442–51.

Tei, F., Benincasa, P., Farneselli, M., Caprai, M., (2010). Allotment Gardens for Senior Citizens in Italy: current status and technical proposals. Acta Horticulturae 881, 91e96.

Temple, J.B., (2008). Severe and moderate forms of food insecurity in Australia: are they distinguishable? *Australian Journal of Social Issues* 43, 649e668.

Trimmel, M., Atzlsdorfer, J., Tuopy N., Trimmel K. (2012). Effects of low intensity noise from aircraft or from neighbourhood on cognitive learning and electrophysiological stress responses *International Journal of Hygiene and Environmental Health* In press, Available online

Ugwi, O., Kumaraswamy, M., Wong, A & Ng, S. (2005). Sustainability appraisal in infrastructure projects (SUSAIP). Part 1. Development of indicators and computational methods. *Automation in Construction* 15, 239-251.

Van den Brandt PA, Goldbohm RA, van 't Veer P, Volovics A, & Hermus P. (1993). Arteriosclerosis, Thrombosis, and Vascular Biology. *Arteriosclerosis and Thrombosis* 13:661-668

Van Kempen, E., Fisher, P., Janssen, N., Houthuijs, D., Van camp, I., Stansfeld, S. & Cassee, F. (2012). Neurobehavioral effects of exposure to traffic-related air pollution and transportation noise in primary school children. *Environmental Research*, Article in press.

Venn AJ, Lewis SA, Cooper M, Hubbard R, & Britton J (2001). Living near a main road and the risk of wheezing illness in children. *American Journal of Respiratory and Critical Care Medicine* 2001, 164(12):2177-2180.

Verbrugge, L., & Jette, A., (1994). The disablement process. *Social Science & Medicine*, 38, 1, 1-14.

Villeneuve P., Parent, M.E., Sahni, V., & Johnson, K. (2011). Occupational exposure to diesel and gasoline emissions and lung cancer in Canadian men. *Environmental Research*, 111, 727-735.

Waitangi Tribunal (2012): Report No 7. available online: http://www.waitangi-tribunal.govt.nz/reports/viewchapter.asp?reportID=AF57CF1A-8E6A-4DE7-B7C6-0AA9F306E4B5&chapter=14

Wang, C., Qie, G., Yang, Y., Ren, Q., 2007a. Effects of highway forest belts on heavy metals pollution from tail gas of vehicles. Scientia Silvae Sinicae 43, 1–7.

Wang, C., Chen, Z.-L., Wang, J., Zhou, N.-S., Xu, S.-Y., 2007b. Heavy metal pollution of soils and vegetables on roadsides in Chongming Island. *Journal of Ecology and Rural Environment* 23, 89–93.

Wang, H., Colvile, R., Pain, C., Aristodemou, E., & ApSimon, H. (2011). Understanding peak pedestrian exposures due to traffic emissions within the urban environment. *Transportation Research*, D, 16, 392-401.

Walker, R., & Hiller, J. (2007). Places and health: A qualitative study to explore how older women living alone perceive the social and physical dimensions of their neighborhoods *Social Science & Medicine* 65, 6, 1154-1165.

Walsh, K., & Gannon, B. (2011). Perceived Neighbourhood context, disability onset and old age. Journal of Socio-Economics, doi: 10.1016/j.socec.2011.05.001. *Journal of Socio-Economics*,40, 631-636.

Waye, K. (2011) Effects of low frequency noise and vibrations: Environmental and occupational perspectives. *Encyclopedia of Environmental Health*, 240-253. available online: http://dx.doi.org/10.1016/B978-0-444-52272-6.00245-2

Weinberger, K., Lumpkin, T.A., 2007. Diversification into horticulture and poverty reduction: a research agenda. *World Development* 35, 1464–1480,

Weinmayr G, Romeo E, De Sario M, Weiland SK, & Forastiere F. (2010). Short-term effects of PM10 and NO2 on respiratory health among children with asthma or asthma-like

symptoms: a systematic review and meta-analysis. *Environmental Health Perspective* . [Meta-Analysis Review]. 118(4):449-57.

World Health Organization (WHO) (2004). *Social determinants of health: the solid facts*. 2nd Ed. Copenhagen, WHO.

World Health Organization (WHO), (2005). *Effects of Air Pollution on Children's Health and Development: a review of the evidence*. Geneva. Switzerland. WHO Monograph..

World Health Organization (WHO), (2009). *Air Quality Guidelines*. Air Quality Guidelines for Europe, 2nd ed., WHO Regional publications, European series no 91, Copenhagen.

WHO (1946). *Definition of Health*. Preamble to the Constitution of the World Health Organization as adopted by the International Health Conference, New York, 19-22 June, 1946 signed on 22 July 1946 by the representatives of 61 States (Official Records of the World Health Organization, no. 2, p. 100) and entered into force on 7 April 1948.

Wilhelm M, & Ritz B. (2005). Local variations in CO and particulate air pollution and adverse birth outcomes in Los Angeles County, California, USA. *Environmental Health Perspectives*, 113:1212–21.

Willeit, J., & Kiechl, S. (1993) Prevalence and risk factors of asymptomatic extracranial carotid artery atherosclerosis. A population-based study. *Arteriosclerosis, Thrombosis and Vascular Biology*, 13, 661-668.

Willich, S., et al.,(2005). Noise burden and the risk of myocardial infarction, *European Heart Journal* November 24, 2005, available online <http://eurheartj.oxfordjournals.org/cgi/content/abstract/ehi658v1>.

Willich, S., Wegscheider, K., Stallmann, M., & Keil, T. (2006). Noise burden and the risk of myocardial infarction. *European Heart Journal* (2006) 27, 276–282.

Woodward, A. and I. Kawachi (2000). Why reduce health inequalities? *Journal of Epidemiology and Community Health* 54: 923-29.

WRLTC (2006). *Wellington Regional Land Transport Strategy* (draft - 30/6/06).Wellington, Greater Wellington Regional Council: Wellington Regional Land Transport Committee.

Yen, I., Michael, Y. & Perdue, L., (2009). Neighbourhood environment in studies of health of older adults. A systematic review. *American Journal of Preventive Medicine*, 37, 5, 455-463.

Yorifuji, T., Naruse, H., Kashima, S., Murakoshi, T., Tsuda, T., Doi, H., & Kawachi, I. (2012). Residential proximity to major roads and placenta/birth weight ratio. *Science of The Total Environment*, 414, 98-102.

Zhu Y, Hinds WC, Kim S, & Sioutas C (2002)a. Concentration and size distribution of ultrafine particles near a major highway. *Journal of the Air and Waste Management Association*, 2(9): 1032-1042.

Zhu Y, Hinds WC, Kim S, Shen S, & Sioutas C (2002)b. Study of ultrafine particles near a major highway with heavy-duty diesel traffic. *Atmospheric Environment*, 36**:**4323-4335.