

# Nelson Arterial Traffic Study 2010

## APPENDIX A: Noise

### **Interference with communication** (WHO 1999, HPA 2010)

- Masking effect of noise is a common adverse effect
- Masking can interfere with understanding speech, listening to TV or radio and social engagement
- Commonly occurs with road traffic noise

### **Sleep disturbance (and consequent effects on mood and performance)**

Sleep disturbance is considered an important adverse effect as it “reduces nightly recuperation, which affects our waking performance as well as our health and mood” (Hume 2008).

- Effects on sleep may vary widely for different people (WHO 1999)
- Effects may also vary for the same level of noise in individuals over time and with different stages of sleep and the time of night (WHO 1999)
- WHO reports there is a causal relationship between night time noise exposure and reported sleep disturbance and insomnia (WHO 2009)
- Sleep is known to be more affected if ambient noise is higher but, even if low ambient noise, maximum levels or low frequency noise can disturb sleep
- Disturbed sleep is associated with tiredness, poor performance and accidents, with greater risk of this occurring in people with certain occupations or day time activities that involve driving, operating machinery etc (HPA 2010, WHO 2009)
- WHO recently noted in the 2009 publication Night Noise Guidelines for Europe there is good quality evidence to support a causal relationship between disturbed sleep and fatigue, accidents, reduced performance (WHO 2009)
- It was reported by sleep researchers in 2009 that “traffic noise causally and relevantly disturbs sleep and, depending on noise levels, may impair behaviour and well being during the subsequent wake period” (HPA 2010).
- There is evidence that insomnia is related to increased mortality and physical morbidity but it is unclear whether long term health effects associated with environmental stressors, such as noise, are mediated through sleep disturbance or are direct effects alone (HPA 2010).
- Certain groups are more sensitive to such effects (often referred to as “vulnerable groups”) including:
  - the elderly, pregnant women, and children
  - people with certain, physical and mental health problems, people with pre-existing sleep difficulties and shift workers (WHO, 1999; WHO, 2009).

Defining an exposure response curve for noise and sleep disturbance is difficult due to the habituation that occurs to environmental noise and to specific psycho-acoustic issues (HPA, 2010).

Thresholds for sleep disturbance are given in the WHO Night Noise Guidelines for Europe (WHO, 2009) for levels recorded both inside bedrooms and outside. The inside thresholds use a maximum recorded reading whereas the outside thresholds use the recordings of an average 8 hour night time noise level ( $L_{\text{night}}$  – this is calculated for the time period 2300hrs to 0700hrs).

These thresholds and health effects are set out in Table 1. It should be noted that many people go to bed earlier than 2300hrs when noise levels are likely to be higher than the  $L_{\text{night}}$  time period with the consequence that these people will be exposed to an increased risk for adverse health effects.

**Table 1- Effects of different levels of night noise on the population's health (WHO 2009)**

<b>Threshold level (recorded inside bedroom)</b>	<b>Health effect observed in population</b>
32dB <sub>max,inside</sub>	motility (an early indication of disturbed sleep)
35 dB <sub>max,inside</sub>	changes in the duration of stages of sleep and in sleep structure
42 dB <sub>max,inside</sub>	waking up in the night or too early in the morning
<b>Average night noise level over a year</b> $L_{\text{night, outside}}$	<b>Health effect observed in population</b>
Up to 30 dB	Although individual sensitivities and circumstances may differ, it appears that up to this level no substantial biological effects are observed
30 to 40 dB	A number of effects on sleep observed in this noise level range: body movements, awakening, self-reported sleep disturbance, arousals. Intensity of effect depends on the nature of the noise source and number of events. Vulnerable groups more susceptible but even in worst cases effects seem modest
40 to 55 dB	Adverse health effects observed in exposed population in this range: most people have to adapt their lives to cope with the noise at night. Vulnerable groups are more severely affected.
Above 55 dB	This situation is considered dangerous for public health. Adverse health effects occur frequently, a sizeable proportion of the population is highly annoyed and sleep-disturbed. Evidence that cardiovascular risk increases.

It is important to note that although the Stage 3 study on noise by Malcolm Hunt Associates (Hunt, 2010) in relation to the road options uses  $dBLA_{\text{eq}(24\text{hr})}$ , the most relevant measures for sleep disturbance are night time measures such as  $L_{\text{night}}$  and single noise events (Basner, 2010; WHO, 2009).

- High traffic densities are associated with continuous noise but at night time, typically with lower traffic densities, exposure of nearby residents to intermittent and single noise events is still very likely (Basner, 2010).
- It is the number and sound properties of these events that impact on sleep. "Clear exposure – response relationships have been demonstrated between single noise stimuli and arousals, awakenings or body movements. Several moderators (time of night, sleep depth etc) have been identified and quantified" (Basner, 2010).
- Measures of single noise events can supplement  $L_{\text{night}}$  measures in regard to the likelihood of sleep disturbance.

### **Physiological Effects of Traffic Noise (Heart Rate, Blood Pressure, Cardiovascular Disease)**

- Physiological effects on the body from noise at lower levels can occur and are believed to be linked to the release of stress hormones following noise exposure with these hormones influencing blood pressure and heart rate. In occupational settings long term exposure to noise has been linked to the risk of cardiovascular disease and a similar association shown in the community with regard to transport noise (HPA 2010, Basner 2010, WHO 2009).
- Recent epidemiological studies also suggest that nocturnal traffic noise exposure increases the risk for cardiovascular disease (HPA, 2010; Basner, 2010; WHO, 2009).

The WHO has also stated that there is:

- sufficient evidence to support a causal relationship between night time noise and increased use of medications as well as there being biological effects on heart rate, arousals, sleep stage changes and awakenings (WHO, 2009)
- limited evidence to support a causal relationship between night time noise and hormone level changes and conditions such as cardiovascular disease, depression and other mental illness (WHO, 2009).
- Research for the Swedish Road Administration (Kjellstrom 2008) on the health impacts and public health costs of road transport looked at studies and identified Risk Coefficients (RC's) for hypertension and ischaemic heart disease from long term exposure to road traffic noise. These Risk Coefficients (applied to all age groups older than 25 years) are:
  - RC - Hypertension 1.19 (19% increase) per 5 dB increase in  $L_{\text{eq}(24\text{hr})}$
  - RC - Ischaemic heart disease 1.045 (4.5% increase) per 5 dB increase in  $L_{\text{eq}(24\text{hr})}$
- The noise level considered as the threshold at which increased blood pressure effects started to appear was 55dB.

## Mental Health

- Link between noise and psychological symptoms has been clearly reported in studies from occupational settings - however these noise levels are much higher than in community settings.
- Evidence from studies in community settings is less clear:
  - People report symptoms such as being tense, headaches and restless nights
  - A link to more specific mental health symptoms has not been clearly shown
  - Community studies have generally been looking at noise from road traffic, aircraft or rail transport (HPA 2010)

## Performance

- Clear evidence of noise being associated with impaired cognitive performance both in children and adults with studies (Goines, 2007) showing:
  - Impaired task performance at school and at work
  - Increased errors, and decreased motivation.
  - Reading attention, problem solving, and memory strongly affected by noise.
- The authors concluded that noise produces negative after-effects on performance, particularly in children, and that schools and day-care centres should be located in areas that are as noise-free as possible (Goines, 2007)
- A large European study (Stansfeld, 2005) looked at aircraft and road traffic noise and children's performance at school and identified that:
  - Increasing exposure to both aircraft and road traffic noise was associated with increasing annoyance responses in children and impairment of quality of life.
  - Exposure to aircraft noise was associated with impaired reading comprehension and recognition memory.
- The WHO recommended Guideline Value for school classrooms and preschools indoors for community noise is 35dBLA(eq) during class (WHO, 1999).

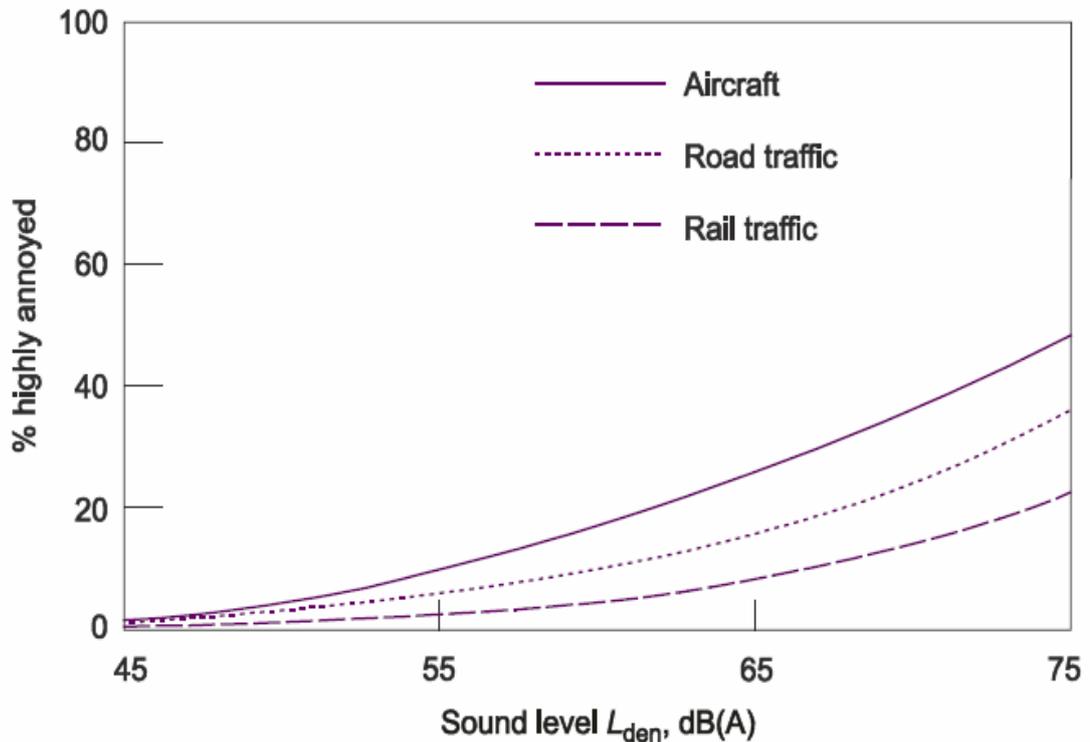
The issue of children's performance and noise is important particularly as in the NATS there are a number of schools close to roads in all the options under consideration. It is also noted that aircraft noise already impacts on parts of the Stoke Tahunanui community.

## Annoyance

- Annoyance is the most common adverse health effect of environmental noise. There are considerable differences in the response to noise by different people with annoyance being affected by a range of factors (Goines 2007) including:
  - the background noise level
  - the character of the noise and the ambient environment
  - the number of noise episodes

- the length of time the episodes last and the maximum sound level
  - the time the episodes occur
  - the ability to influence the noise
  - other social and personal factors
- The WHO has identified Guideline Values for serious (55 dBA) and moderate (50 dBA) annoyance for continuous noise (daytime and evenings – 16hrs) for the outside living areas. The Indoor Guideline Value for moderate annoyance is 35dBA (daytime and evenings – 16hrs) (WHO, 1999).
  - Research has been done on dose response curves for annoyance and transport noise and these have been refined over recent years to differentiate between the various sources of noise. The graph in Figure 1 shows the community response to increasing levels of noise from three different transportation sources including road traffic

**Figure 1: Dose-response relationships for the association between noise from different sources and annoyance**



**Source:** HPA 2010 (*Environmental Noise & Health in the UK, A Report of the Ad Hoc Expert Group on the Effects of Environmental Noise on Health*)

The following map (Figure 2) reflects the “do minimum” scenario in the year 2036. The most important areas affected by noise and vibration greater than 65dB $L_{Aeq(24hr)}$  are marked by the pink line which is located 15-18 metres from the near side edge of the closest traffic lane.

**Figure 2 - Predicted  $L_{Aeq(24hr)}$  traffic noise levels based on 24 hour traffic volumes and average vehicle as per ‘Do Minimum’ 2036**



**Source: Nelson Arterial Transport Study Stage 3: Evaluation of Options Noise Effects Study, August 2010. The permission from Malcolm Hunt Associates to reproduce this Figure is acknowledged.**

Note: This figure is not to Scale. The contours do not take account of site specific factors and are not suitable for assessing the traffic noise effects of any particular route option in isolation or at any specific geographical location.

**Figure 3 - Predicted  $LA_{eq} [24h]$  traffic noise levels based on Option B, 2036 24 hour traffic volumes and average vehicle**



**Source:** Nelson Arterial Transport Study Stage 3: Evaluation of Options Noise Effects Study, August 2010. The permission from Malcolm Hunt Associates to reproduce this Figure is acknowledged.

Note: This figure is not to Scale. The contours do not take account of site specific factors and are not suitable for assessing the traffic noise effects of any particular route option in isolation or at any specific geographical location.

### Glossary

<b>A-weighted sound level</b>	The level of sound in decibels, dB, after filtering with an A-weighting filter which mimics the frequency response of the human ear. Most environmental noise measurements use A-weighted sound levels
<b>A-weighting</b>	A way of filtering sounds to mimic the response of the human ear. It is used for almost all measurements of environmental noise. Its use is indicated by an uppercase 'A' in the measurement indicator, e.g. LA <sub>eq</sub>
<b>LA<sub>eq</sub></b>	Effectively the average sound level over a specified time. Formally it is the equivalent continuous A-weighted sound level, i.e. the level which if maintained constant for the stated time period contains the same sound energy as the real, varying sound over the same period
<b>LA<sub>max</sub></b>	The maximum A-weighted sound level occurring during a given period. Strictly speaking, the time constant of the instrument used for measurement should also be stated; normally this is 'F' (Fast), the other option (rarely used) being 'S' (Slow)
<b>L<sub>den</sub></b>	Day-evening-night level, a descriptor of noise level based on energy equivalent noise level (Leq) over a whole day with a penalty of 10 dBA for night-time noise (2300hr – 0700 hrs) and an additional penalty of 5 dBA for evening noise (i.e. 1900hr – 2300hr)
<b>L<sub>dn</sub></b>	Day-night level, the A-weighted equivalent sound level for a 24-hour period with an additional 10 dB imposed on the equivalent sound levels for night-time noise (2200hr –0700 hr)
<b>L<sub>night</sub></b>	The night-time noise indicator for self-reported sleep disturbance; the A-weighted sound level averaged over the night-time period (2300hr – 0700 hr), as defined in ISO 1996-2:1987. Note that although night-time noise attracts a penalty of 10 dBA in L <sub>den</sub> , the definition of L <sub>night</sub> does not include an addition of 10 dB

## APPENDIX B: Air Quality

Vehicle engines produce a number of air pollutants that may pose risks to health either as acute effects or through chronic exposure. These pollutants include Particulate Matter (PM<sub>10</sub> and PM<sub>2.5</sub>), nitrogen oxides (NOs) which include nitrogen dioxide (NO<sub>2</sub>), carbon monoxide (CO) sulphur dioxide (SO<sub>2</sub>) and ozone (O<sub>3</sub>). Specific health effects attributed to these pollutants are discussed in more detail below.

### Particulate Matter (PM<sub>10</sub> and PM<sub>2.5</sub>)

Particulate Matter is emitted in vehicle exhausts and is also formed in the atmosphere through chemical reactions between the various pollutants found in exhaust fumes.

PM<sub>10</sub> are 'thoracic' particles smaller than 10 microns in diameter which can penetrate into the lower respiratory system

PM<sub>2.5</sub> are 'respirable' or fine particles smaller than 2.5 microns that can penetrate into the gas-exchange region of the lung (Fisher, 2007).

- The National Environmental Standards (NES) for Air Quality recognise PM<sub>10</sub> as the primary measurement of Particulate Matter rather than PM<sub>2.5</sub>. The PM<sub>10</sub> Standard is 50µg/m<sup>3</sup> expressed as a 24-hour mean with only one exceedance permitted in a year. This exceedance limit is currently under review and may possibly increase from one to three.
- Studies (WHO 2005) in Europe have shown that near urban highways with more than 100,000 vehicles a day and greater than 5% of trucks, ultra-fine particles are significantly increased up to 250 metres away as compared to urban background concentrations.

Health effects of particulate matter:

- When inhaled may cause damage or aggravate symptoms in individuals who already suffer from respiratory or cardiovascular diseases
- Long term exposure to low dose concentrations of particulate matter, especially PM<sub>2.5</sub>, has been shown to be an important risk factor in cardiopulmonary and lung cancer mortality (Fisher 2007).

Recent studies show that:

- PM<sub>2.5</sub> is generally a better predictor of health effects than PM<sub>10</sub>
- It is probably the finer particles causing greater effects, owing to their ability to accumulate and reach the lower regions of the respiratory system (Fisher 2007).

Mortality studies show:

- Association between daily average PM<sub>10</sub> readings and daily mortality was researched in Christchurch (Hales 2000), and showed that a 10µg/m<sup>3</sup> increase in PM<sub>10</sub> was associated with a 1% increase in all cause mortality and a 4% increase in respiratory mortality;
- For long term exposure to PM<sub>10</sub> and long term mortality impacts, the WHO identified a 10% increase in mortality for every 10µg/m<sup>3</sup> increase in PM<sub>10</sub> (Fisher 2007) with no threshold at which the effect on mortality begins;

- Other research has used a 4.3% increase in mortality for every 10  $\mu\text{g}/\text{m}^3$  increase in  $\text{PM}_{10}$  and used different thresholds to then calculate estimates of total deaths per year in urban settings in New Zealand. A greater effect is clearly seen if the threshold is set lower ( $5\mu\text{g}/\text{m}^3$  instead of 7.5 or 10  $\mu\text{g}/\text{m}^3$ ). From this research it was estimated (for adults greater than 30 yrs old) that the mortality rate due to traffic related air pollution was 196 per million; and that public health impacts from vehicle related pollution emissions are not insignificant (Fisher 2002);
- Other overseas research has been on mortality and long term exposure to fine particulate, ( $\text{PM}_{2.5}$ ) pollution (HPA 2009). This research estimated the following Risk Coefficients linking  $\text{PM}_{2.5}$  exposure and mortality:
  - Best estimate for all cause mortality 1.06 (95% CI 1.02-1.11)
  - Best estimate for cardiopulmonary mortality 1.09 (95% CI 1.03-1.16)
  - Best estimate for lung cancer mortality 1.08 (95%CI 1.01- 1.16)
  - These coefficients are expressed as relative risk per  $10\mu\text{g}/\text{m}^3$  rise in  $\text{PM}_{2.5}$  (annual average concentration); i.e., for all cause mortality, there is likely to be a 6% increase for every  $10\mu\text{g}/\text{m}^3$  rise in the annual average  $\text{PM}_{2.5}$  concentration.

It should be noted that  $\text{PM}_{2.5}$  is a better marker of health risk from vehicle pollution than  $\text{PM}_{10}$ , especially for diesel vehicles, and that measuring a range of pollutants may better reflect vehicle pollution, e.g.  $\text{PM}_{2.5}$ , benzene, carbon monoxide, nitrogen oxides.

### **Nitrogen Oxides (NO & NO<sub>2</sub>)**

- Engines burn nitrogen that is present in the air and in fuel producing Nitrogen oxides.
- The major source of nitrogen oxides in urban areas in NZ is motor vehicles (Fisher 2007).
- The NES for  $\text{NO}_2$  is  $200\mu\text{g}/\text{m}^3$  as a 1-hour average and the Ambient Air Quality Guideline Values for Nitrogen dioxide additionally recommend  $100\mu\text{g}/\text{m}^3$  as a 24-hour average.
- Studies (WHO 2005) in Europe have shown that near urban highways with more than 100,000 vehicles a day and greater than 5% of trucks, levels of nitrogen dioxide are significantly increased up to 250 metres away as compared to urban background concentrations.
- Nitrogen oxides can irritate airways, with nitrogen dioxide having an inflammatory reaction with the lung contributing to increased morbidity and mortality particularly for susceptible groups such as asthmatics, people with chronic bronchitis and young children.
- There is evidence that longer term exposure to  $\text{NO}_2$  of around  $80\mu\text{g}/\text{m}^3$  during childhood can give rise to the development of respiratory tract symptoms (Fisher 2007).

### **Carbon monoxide: (CO)**

- This pollutant is produced by incomplete combustion of fuel and all engine exhaust contains a certain amount of carbon monoxide. The amount will increase if engines are poorly maintained.
- Motor vehicles are the main source of CO in most urban areas, although domestic heating also contributes (Fisher 2007).

- The NES for CO is 10mg/m<sup>3</sup> expressed as an 8 hour running mean. The Standard allows for only one excess in a year. The Ambient Air Quality Guideline Values for Carbon monoxide are 30mg/m<sup>3</sup> as a 1-hour average as well as 10mg/m<sup>3</sup> as an 8-hour average.
- Carbon monoxide is readily absorbed into the blood combining with haemoglobin to form carboxy-haemoglobin. This decreases the ability of the blood to carry oxygen with consequent effects on body organs such as the heart and brain.
- The NES is based on keeping blood carboxy-haemoglobin concentration below 2.5% to protect people from an increased risk of heart attack. There is also some evidence of adverse health effects when carboxy-haemoglobin levels are less than 2.5 % (Fisher 2007).

### Volatile Organic Compounds (VOCs)

- These are a large family of carbon-containing compounds. Engine exhausts contain a number of different VOCs.
- An Ambient Air Quality Guideline Value has been set for a number of VOCs including benzene, a chemical constituent of petrol.
- The Guideline Value for benzene is 3.6µg/m<sup>3</sup> as an annual average (reduced in 2010 from 10µg/m<sup>3</sup>).
- Some VOCs, such as benzene, can cause cancer although the risk is small.
- The cancer risk from a lifetime exposure to benzene at 1.0µg/m<sup>3</sup> has been estimated at 4.4 to 7.5 per million population (Fisher 2007).

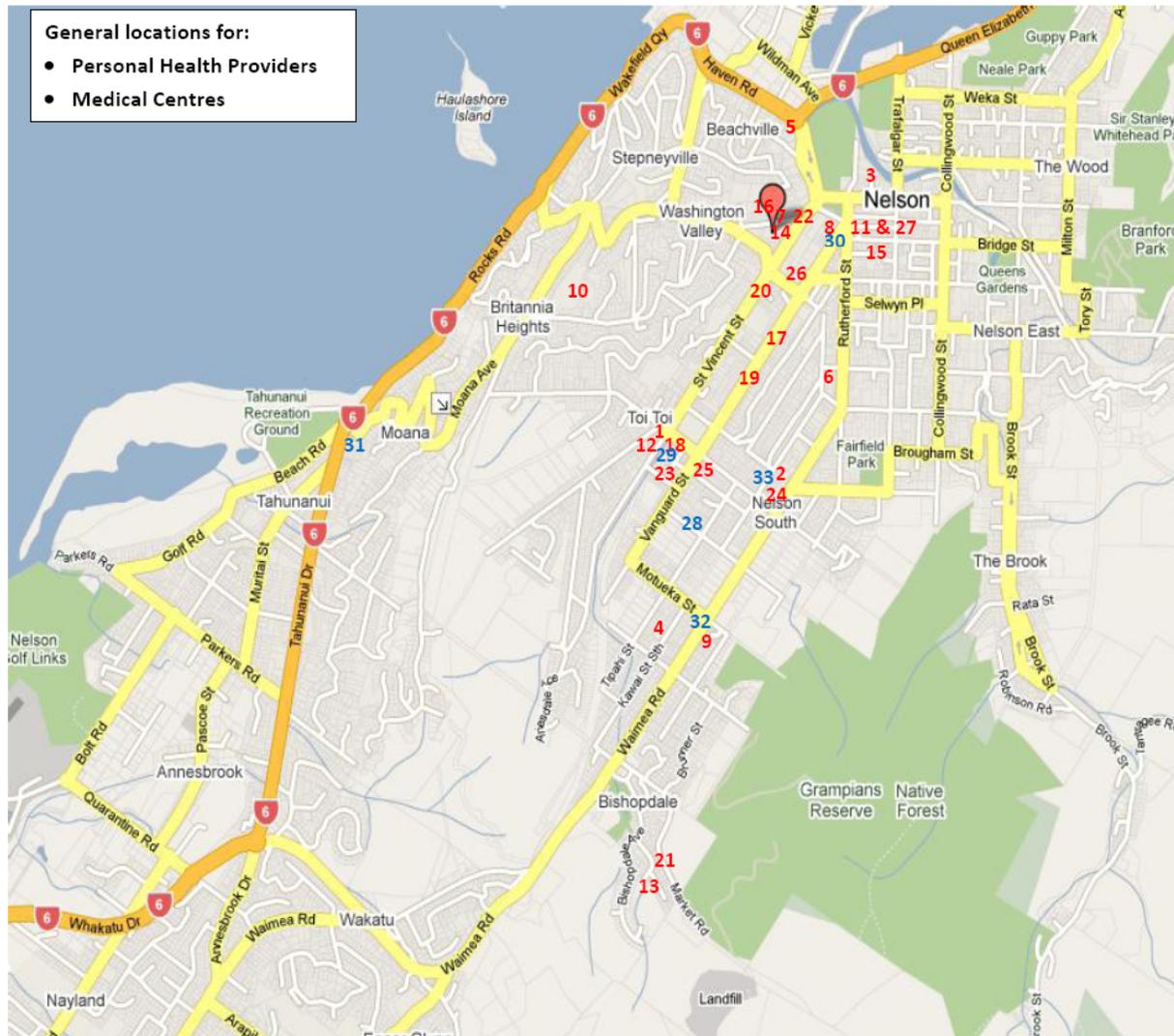
### Sulphur Dioxide (SO<sub>2</sub>)

- Motor vehicles are minor contributors to ambient SO<sub>2</sub> and the contribution has changed over the years with different concentrations of sulphur in fuel. New Zealand now has maximum limits on the amount of sulphur allowed in fuel.
- Sulphur dioxide is a respiratory irritant with some people such as asthmatics being particularly susceptible to this pollutant. It has been associated with increase in mortality and in increased hospital admissions for respiratory and cardio-vascular disease (Fisher 2007).

### Ground-level Ozone (O<sub>3</sub>)

- This is formed by chemical reactions between nitrogen oxides and VOCs. These reactions are stimulated by sunlight.
- The NZ National Environmental Standard for ozone is 150µg/m<sup>3</sup> expressed as a 1-hour mean and is not to be exceeded at any time.
- Ground-level ozone irritates airways and may trigger reactions in:
  - people who have asthma
  - people with chronic lung and cardiovascular disease
  - people taking active exercise outside over extended periods
 These effects are dependent on the ozone concentration being high enough. For example significant acute effects from exercising out doors require one hour exposures to ozone concentrations around 500µg/m<sup>3</sup> or higher (Fisher 2007).

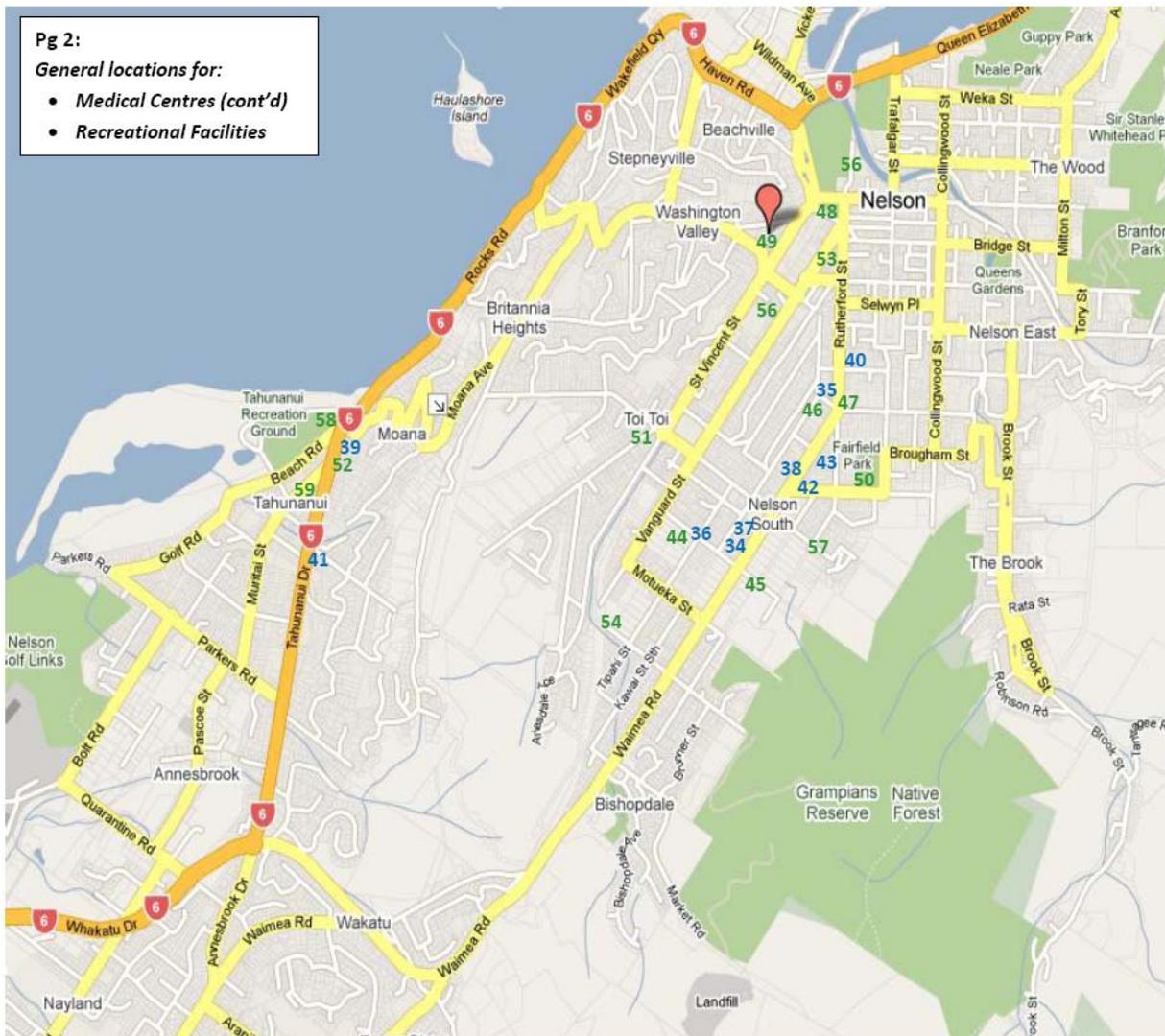
## APPENDIX C: Maps of Health Services



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Personal Health Providers	
1	"Journeys" Child & Family Counselling Service
2	Cancer Society - Nelson
3	Citizens Advice Bureau
4	Irlen Diagnostic Clinic
5	MoE - Special Education
6	Nelson Women's Refuge Inc
7	Nelson Neighbourhood Centre
8	Nelson Night Shelter
9	Sex Offenders Tx Programme
10	NZ Down Syndrome Ass
11	NZ Prisoners' Aid & Rehab
12	Parents As First Teachers
13	Plunket - Area Office
14	Real Dads Real Men
15	Salvation Army
16	SeniorNet Inc - Nelson
17	St Vincent de Paul
18	Stacey Bowker
19	Stopping Violence Services
20	Tautoko Services
21	The Gateway Housing Trust
22	The Male Room
23	Victory Homework Academy
24	WORKSTAR
25	YMCA Nelson OSCAR
26	Youth Nelson
27	English Language Partners
Medical Centres	
28	Public Health Nurses
29	Victory Comm. Health Centre
30	NZ Blood Service - Nelson
31	Tahuna Medical Centre
32	Dr Joseph Hassan
32	Natural Fertility
32	St Luke's Health Centre
33	Drs K D & J M F Jolly

File:Map of health providers May10



**Pg 2:**  
**General locations for:**

- **Medical Centres (cont'd)**
- **Recreational Facilities**

<b>Medical Centres (cont'd)</b>	
34	Afterhours & Duty Doctor
35	Health Centre
36	Dr K Hill
36	Dr David Leadbetter
37	Roundhay Medical Centre
38	Dr Elizabeth Narbey
38	Dr Susan Stubbs
38	Dr Liz Scott
39	Medlab South Ltd
40	Dental Care
41	Tahunanui Dental Centre
42	Grant McIntosh
43	Nelson Orthodontics
<b>Recreational Facilities</b>	
44	Nelson College sport fields
45	Nelson College gyms etc access from Franklyn Street
46	Nelson College for Girls sport field
47	Nelson College for Girls Gyms – access from Waimea Rd
48	ANZAC park
49	Pioneer Park (Athletics Nelson)
50	Fairfield Park
51	Victory Community Centre
52	Aikido Of Nelson and Motueka
53	All Saints Anglican Church
54	Gymnastics Nelson
55	Neighbourhood Connections
56	Nelson Lawn Tennis Club
57	Nelson South Swimming Club
58	Nightingale Library Memorial
59	Tahunanui Presbyterian Church Hall

## **APPENDIX D**

### **Nelson Marlborough District Health Board Staff who undertook this Health Impact Assessment**

Richard Butler: Nutrition Physical Activity Programme Manager

Geoff Cameron: Senior Health Protection Officer

Colleen Kem: Health Protection Officer ( Kaitiaki o te Tai Ao)

Dr Ed Kiddle: Medical Officer of Health

Dr Al Norrish: Public Health Analyst

Dr Jill Sherwood: Public Health Medical Specialist

Les Milligan: Health Promotion Co-ordinator